The effect of information transmission on epidemic outbreaks

Istvan Z. Kiss [1,*], Jackie Cassell [2], Mario Recker [3] & Péter L. Simon [4]

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[1] Department of Mathematics, University of Sussex, Falmer, Brighton BN1 9RF, UK
[2] Brighton and Sussex Medical School, University of Sussex, Falmer, Brighton BN1 9PH, UK
[3] University of Oxford, Department of Zoology, South Parks Road, Oxford OX1 3PS, UK
[4] Institute of Mathematics, Eötvös Loránd University Budapest, Budapest, Hungary

∗ corresponding author
email: istvan.kiss@sussex.ac.uk

Abstract

For many diseases (e.g., sexually transmitted infections, STIs), most individuals are aware of the potential risks of becoming infected, but choose not to take action ('respond') despite the information that aims to raise awareness and to increases the responsiveness or alertness of the population. We propose a simple mathematical model that accounts for the diffusion of health information disseminated as a result of the presence of a disease and an 'active' host population that can respond to it by taking measures to avoid infection or if infected by seeking treatment early. In this model, we assume that the whole population is potentially aware of the risk but only a certain proportion chooses to respond appropriately by trying to limit their probability of becoming infectious or seeking treatment early. The model also incorporates a level of responsiveness that decays over time. We show that if the dissemination of information is fast enough, infection can be eradicated. When this is not possible, information transmission has an important effect in reducing the prevalence of the infection. We derive the full characterisation of the global behaviour of the model, and we show that the parameter space can be divided into three parts according to the global attractor of the system which is one of the two disease-free steady states or the endemic equilibrium.

Keywords: differential equations, compartmental models, disease transmission, epidemic, information spread.

1 Introduction

Many compartmental models of disease transmission assume a 'passive' population that will not 'respond' (change its behaviour) following an infectious disease outbreak or an ongoing endemic infection [2, 7]. For many diseases (e.g., sexually transmitted infections (STIs), SARS, Pandemic Influenza, Childood diseases) this is rarely the case since through targeted campaigns or simple diffusion of news through various media (e.g., TV, newspaper, social networking sites) and individual to individual contact, the population can be alerted to the presence of a disease that is spreading through the population. This will usually result in individuals taking a range of measures to lower their probability of becoming infected. These measures, depending on the disease, can range from the use of face masks, vaccination, taking antiviral drugs [8], condoms to individuals choosing to limit their number of contact with others, or in particular avoid contact with persons known to be infectious [10, 17]. In the case when already infected, as a results of the information, some individuals will seek early treatment.

Here, in the context of STIs, we propose a simple compartmental model that describes such an 'active' population. For many infectious diseases, prevention is desirable, but when individuals become infectious it is vital they seek treatment early. By the time that most people become sexually active it is likely that many will be aware of the potential risk of becoming infected and of the measures that can be taken to avoid becoming infected. Hence, the most important factor is the willingness or responsiveness of individuals to act upon the information that is made available. For example, there is evidence that mass media campaigns resulted in less than 1% of young adults taking a Chlamydia test[1, 14, 16]; although there may be an increase in testing during and shortly after campaigns. However, these data reflect only testing behaviour following a media campaign and cannot assess any possible change in sexual beaviour and consequently in risk of acquiring infection. To reflect this in the model, we differentiate between individuals based on the willingness to respond to the information generated by the presence of the disease. Individuals that are not yet infected and are willing to respond can take basic measures to reduce their probability of becoming infected. If infected, responsive individuals are likely to seek treatment early and thus have a shorter infectious period compared to infected individuals that remain passive. The willingness to respond is likely to degrade with time either due to susceptible individuals becoming less cautious with time or as a result of limited diffusion of information when prevalence is low [1, 16]. These are important factors that are incorporated in the model.

Simple compartmental models have been previously used to describe the influence of the information and information-related delays on vaccination campaigns [3]. Specifically, for STIs, Chen [4] developed a simple model to capture the interplay between the quality of information, the prevalence of the infection and disease dynamics. In a recent paper, Funk et al. [9] discussed the spread of awareness about the disease and its effect on epidemic outbreaks. They investigated an *SIR* type compartmental model and compared results to findings based on individual-based simulations. In the compartmental model that they

propose, the spread of awareness has no effect on the epidemic outbreak threshold R_0, but decreases the proportion of infected individuals. When considering disease and awareness spread on theoretical network models, they show that if the disease transmission is not too fast the transmission of awareness can halt the outbreak. The model that we propose is motivated by and analysed in the context of STIs and uses assumptions that are relevant in this context. In the case of STIs, most individuals are aware of the risk, but only few respond accordingly. Most campaigns are aimed at raising the responsiveness of the population to a level where a significant number of individuals will take measures to avoid infection or seek treatment early [1, 14, 15, 16]. The present model apart from capturing individual to individual transmission of information also accounts for a population wide transmission and we discuss the overall implications of the dissemination of information for disease outbreak threshold, disease dynamics and long-term behaviour of the system.

2 Model

We extend the simple *SIS* model to account for the treatment class that is a common feature for many STIs. To account for non-responsive and responsive individuals, the population is divided into five compartments as follows: susceptible non-responsive (S_{nr}), susceptible responsive (S_r), infectious non-responsive (I_{nr}), infectious responsive (I_r) and treatment class (T). This model captures basic features of STIs (e.g., Chlamydia and Gonorrhea) without considering heterogeneity in the number of contacts. The equations corresponding to the transitions between the various classes are (see Fig. 1 for a diagram of possible transitions)

$$\begin{aligned}
\frac{dS_{nr}}{dt} &= -\beta_{nr}(I_{nr}+I_r)\frac{S_{nr}}{N} - \alpha_s f_s(S_{nr};S_r,I_r,T) - \delta_s g_s(I_{nr},I_r)S_{nr} \\
&+ h_s(I_{nr},I_r)S_r + prT, && (1)\\
\frac{dS_r}{dt} &= -\beta_r(I_{nr}+I_r)\frac{S_r}{N} + \alpha_s f_s(S_{nr};S_r,I_r,T) + \delta_s g_s(I_{nr},I_r)S_{nr} \\
&- h_s(I_{nr},I_r)S_r + (1-p)rT, && (2)\\
\frac{dI_{nr}}{dt} &= \beta_{nr}(I_{nr}+I_r)\frac{S_{nr}}{N} - \alpha_i f_i(I_{nr};S_r,I_r,T) - \delta_i g_i(I_{nr},I_r)I_{nr} \\
&- \gamma_{nr}I_{nr} + h_i(I_{nr},I_r)I_r, && (3)\\
\frac{dI_r}{dt} &= \beta_r(I_{nr}+I_r)\frac{S_r}{N} + \alpha_i f_i(I_{nr};S_r,I_r,T) + \delta_i g_i(I_{nr},I_r)I_{nr} \\
&- \gamma_r I_r - h_i(I_{nr},I_r)I_r, && (4)\\
\frac{dT}{dt} &= \gamma_{nr}I_{nr} + \gamma_r I_r - rT, && (5)
\end{aligned}$$

where $(S_{nr}+S_r+I_{nr}+I_r+T)(t) = N$ for all $t \geq 0$ and N is the population size. The model given above, considers two different means of information dissemination: (i) information

dissemination via direct contact between individuals given by f_s and f_i (e.g. mass-action or some form of nonlinear incidence), and (ii) population wide dissemination of disease related information given by g_s and g_i. As a result of either of these, non-responsive susceptible and infectious individuals move to the responsive class. In general, g_s and g_i depend on the level of infection prevalence with high prevalence of infection enhancing information transmission (e.g., TV, newspaper, social networking sites) which in turn results in a higher rate of transition from the non-responsive to the responsive class. However, information that covers the same topic repeatedly will loose its value over time. This can be captured by including a saturation effect in g_s and g_i for increasing levels of infection prevalence. The value of the information degrades in time and many individuals that are aware of the disease and are prepared to respond are going to become less willing to do so. This is captured by h_s and h_i which represent the rates at which responsive individuals move to the non-responsive class. These rates can depend on time and can increase when the level of infection prevalence is low (i.e., low levels of prevalence can make individuals even less responsive) and decrease when prevalence increases.

Responsive individuals are less likely to be infected ($\beta_{nr} > \beta_r$) and seek treatment faster ($\gamma_r > \gamma_{nr}$) compared to non-responsive individuals. Disease and information is likely to be transmitted through contacts that are non-overlapping and hence information transmission is possible from any responsive individual (i.e., S_r, I_r and T).

2.1 Choice of model

2.1.1 Contact-based transmission of information

For STIs contact between individuals is best characterised by frequency dependent contact (i.e., mass action). Thus, upon assuming that the disease and information spread on different routes, the natural choice for f_s and f_i is given by

$$f_s(S_{nr}; S_r, I_r, T) = f_i(I_{nr}; S_r, I_r, T) = f(X; S_r, I_r, T) = \frac{X(S_r + I_r + T)}{N}. \tag{6}$$

This accounts for the spread of information triggered by the disease from individuals that are aware and responsive (i.e., S_r, I_r and T) to those that are non-responsive (i.e., S_{nr} and I_{nr}).

2.1.2 Population wide transmission of information

The rate of population wide transmission of information is assumed to depend on the disease prevalence. This is based on the assumption that more cases will generate an increased volume of information and more efficient diffusion of information. However, this will be limited and can saturate for higher prevalence when information is likely to have a smaller

impact with time. This is similar to the case where the bilinear incidence is replaced by a non-linear function to capture the saturation effect as the number of infectious people in the population increases [6, 11, 13]. There are different ways to account for these aspects and we propose a relatively simple form where g_s and g_i are given by

$$g_s(I_{nr}, I_r) = g_i(I_{nr}, I_r) = g(I_{nr}, I_r) = \frac{(I_{nr} + I_r)^n}{K + (I_{nr} + I_r)^n}, \tag{7}$$

where $n = 1, 2, \ldots$. For small n, these functions have a quick initial growth compared to the case of large n. However, for large n, the saturation level is reached quicker. In the limit of large n, these function become step like with sudden transition from small values to close to saturation. By choosing $n = 1$ and $n = 2$, the function above will be equivalent to Michaelis-Menten and Holling-type II function, respectively.

2.1.3 Decaying value of information

The value of information is likely to decay with time. For example, individuals that responsive and remain susceptible for a certain amount of time are likely to become less cautious with time. Moreover, low levels of disease prevalence in the population might increase the rate at which individuals that are responsive become less cautious. For high levels of prevalence this is likely to decrease and individuals will remain more cautious for longer. Below we give two different possible choices for h_s and h_i,

$$h_b^1(I_{nr}, I_r) = d_b, \;\; h_b^2(I_{nr}, I_r) = \frac{D_b}{M_b + (I_{nr} + I_r)} \;\; \text{for} \;\; b \in \{s, i\}. \tag{8}$$

In the first case, h is independent of the prevalence level and in the second case, h depends on the proportion of infectious individual in the population.

3 Baseline model and its analysis

All dependent variables are non-dimensionalised by N. For $n = 1$, $h = h^1$ and upon using $s_{nr} = S_{nr}/N$, $s_r = S_r/N$, $i_{nr} = I_{nr}/N$, $i_r = I_r/N$, $\tau = T/N$, $k = K/N$ we obtain

$$\frac{ds_{nr}}{dt} = -\beta_{nr}(i_{nr} + i_r)s_{nr} - \alpha_s(s_r + i_r + \tau)s_{nr} - \frac{\delta_s(i_{nr} + i_r)}{k + (i_{nr} + i_r)}s_{nr} + d_s s_r + pr\tau, \tag{9}$$

$$\frac{ds_r}{dt} = -\beta_r(i_{nr} + i_r)s_r + \alpha_s(s_r + i_r + \tau)s_{nr} + \frac{\delta_s(i_{nr} + i_r)}{k + (i_{nr} + i_r)}s_{nr} - d_s s_r + (1 - p)r\tau, \tag{10}$$

$$\frac{di_{nr}}{dt} = \beta_{nr}(i_{nr} + i_r)s_{nr} - \alpha_i(s_r + i_r + \tau)i_{nr} - \frac{\delta_i(i_{nr} + i_r)}{k + (i_{nr} + i_r)}i_{nr} - \gamma_{nr}i_{nr} + d_i i_r, \tag{11}$$

$$\frac{di_r}{dt} = \beta_r(i_{nr}+i_r)s_r + \alpha_i(s_r+i_r+\tau)i_{nr} + \frac{\delta_i(i_{nr}+i_r)}{k+(i_{nr}+i_r)}i_{nr} - \gamma_r i_r - d_i i_r, \tag{12}$$

$$\frac{d\tau}{dt} = \gamma_{nr} i_{nr} + \gamma_r i_r - r\tau. \tag{13}$$

The goal of our investigation is to reveal the dynamic behaviour of the system. In the first Subsection we determine the two disease-free steady states and their stability. In the next Subsection we will show how the endemic steady state can be determined numerically. We will give numerical evidence that the endemic steady state (if it exists) is unique and it is globally asymptotically stable in the positive orthant, that is all trajectories starting from a positive initial condition tend to this point as time tends to infinity. To illustrate the behaviour of the model, based on previous studies of the spread and control of Chlamydia [1, 16], some model parameters are fixed. In the case of Chlamydia, the average infectious period for individuals that are unaware and do not seek treatment early is found to be in the region of 6 months (i.e., $\gamma_{nr} = 1/(26 \text{ weeks})$). For individuals that seek treatment early, mainly due to being aware, the average infectious period is around 3 months (i.e., $\gamma_r = 1/(13 \text{ weeks})$). The time spent in treatment on average is around one week giving an estimate of $r = 1/(1 \text{ week})$. To show the qualitative behaviour of the model, we numerically integrated Eqs. (9)-(13) and varied the rate at which infection (β_{nr}) and information (α_s and α_i) is transmitted. This preliminary investigation reveals three different model outcomes: trivial and non-trivial disease-free steady states and an endemic equilibrium (see Fig. 2). In the sections below, the stability of these steady state is analysed in detail.

3.1 Disease-free steady states, stability analysis and R_0

There are two disease-free steady states (DFSSs). The trivial DFSS is $ss_{triv} = (s_{nr}, s_r, i_{nr}, i_r, t) = (1, 0, 0, 0, 0)$. The second DFSS can be obtained by setting i_{nr}, i_r and τ to zero and determining the values of s_{nr} and s_r such that Eqs. (9-10) are at equilibrium (i.e., $ds_{nr}/dt = ds_r/dt = 0$). Provided that, $\alpha_s > d_s$, the second DFSS is given by

$$dfss_{non-triv} = (s_{nr}, s_i, i_{nr}, i_i, t) = \Big(1 - s_0 = \frac{d_s}{\alpha_s}, s_0 = \frac{\alpha_s - d_s}{\alpha_s}, 0, 0, 0\Big). \tag{14}$$

There is also an endemic steady state (i.e., non-zero prevalence of infection) with its existence and uniqueness discussed in the following subsection.

3.1.1 Linear stability analysis

The linear stability analysis can be carried out easier when Eqs. (9)-(13) are considered as a four variable system (Eqs. (9)-(12)) with $\tau = 1 - s_{nr} - s_i - i_{nr} - i_i$. Then the two disease-free steady states can be written in the form $(1 - s_0, s_0, 0, 0)$, where $s_0 = 0$ for the trivial DFSS

and $s_0 = \frac{\alpha_s - d_s}{\alpha_s}$ for the non-trivial DFSS. Then the 4×4 Jacobian at the two disease-free steady states $(1 - s_0, s_0, 0, 0)$ takes the form

$$J = \begin{pmatrix} L & N \\ 0 & M \end{pmatrix}$$

with

$$L = \begin{pmatrix} \alpha_s(1 - 2s_0) - pr & d_s - pr \\ \alpha_s(2s_0 - 1) + pr - r & -d_s + pr - r \end{pmatrix}$$

$$M = \begin{pmatrix} \beta_{nr}(1 - s_0) - \alpha_i s_0 - \gamma_{nr} & \beta_{nr}(1 - s_0) + d_i \\ \beta_r s_0 + \alpha_i s_0 & \beta_r s_0 - \gamma_r - d_i \end{pmatrix}$$

The block form of the Jacobian J yields that its eigenvalues are the eigenvalues of L and M (the matrix N does not affect the eigenvalues). The eigenvalues of L are denoted by λ_1, λ_2 and those of M are denoted by λ_3, λ_4. For the trivial DFSS (when $s_0 = 0$) the eigenvalues are

$$\lambda_1 = -r,\ \lambda_2 = \alpha_s - d_s,\ \lambda_3 = \beta_{nr} - \gamma_{nr},\ \lambda_4 = -\gamma_r - d_i. \tag{15}$$

Therefore the trivial DFSS is stable if and only if $\alpha_s < d_s$ and $\beta_{nr} < \gamma_{nr}$. For the non-trivial DFSS (when $s_0 = \frac{\alpha_s - d_s}{\alpha_s}$) the eigenvalues are

$$\lambda_1 = -r,\ \lambda_2 = d_s - \alpha_s,\ \lambda_{3,4} = \frac{1}{2}(\mathrm{Tr}M \pm \sqrt{(\mathrm{Tr}M)^2 - 4\det M}). \tag{16}$$

It is easy to show that $\mathrm{Re}\lambda_{3,4} < 0$ if and only if $\mathrm{Tr}M < 0$ and $\det M > 0$. Using that

$$\det M = -\beta_{nr}(1 - s_0)(\gamma_r + d_i + \alpha_i s_0) - \beta_r s_0(\gamma_{nr} + d_i + \alpha_i s_0) + \alpha_i s_0 \gamma_r + \gamma_{nr}\gamma_r + \gamma_{nr} d_i,$$

one can easily prove that $\det M > 0$ implies $\mathrm{Tr}M < 0$. Therefore the non-trivial DFSS is stable if and only if $d_s < \alpha_s$ and $\det M > 0$. Summarising the above results, we have proved the following about the local stability of the DFSSs.

Proposition 1 *The system given by Eqs. (9)-(13) can have two disease-free steady states: a trivial DFSS* $(1, 0, 0, 0, 0)$ *and in the case* $\alpha_s > d_s$ *a non-trivial DFSS* $(1 - s_0, s_0, 0, 0, 0)$, *where* $s_0 = \frac{\alpha_s - d_s}{\alpha_s}$.

1. *The trivial DFSS is locally stable if and only if* $\alpha_s < d_s$ *and* $\beta_{nr} < \gamma_{nr}$.

2. *The non-trivial DFSS is locally stable if and only if* $d_s < \alpha_s$ *and*

$$-\beta_{nr}(1 - s_0)(\gamma_r + d_i + \alpha_i s_0) - \beta_r s_0(\gamma_{nr} + d_i + \alpha_i s_0) + \alpha_i s_0 \gamma_r + \gamma_{nr}\gamma_r + \gamma_{nr} d_i > 0. \tag{17}$$

3.1.2 Basic reproduction number

The local stability of the DFSSs can be expressed in terms of the basic reproduction number R_0. First consider the trivial DFSS and its stability. To determine R_0 the next generation matrix approach proposed by van den Driessche and Watmough [18] is used. The rate of appearance of new infections F and the rate of transfer of individuals out of the three compartments V are given by

$$F = \begin{array}{c} \\ s_r \\ i_{nr} \\ i_r \end{array} \begin{array}{c} \begin{array}{ccc} s_r & i_{nr} & i_r \end{array} \\ \begin{pmatrix} \alpha_s & \frac{\delta_s}{k} & \alpha_s + \frac{\delta_s}{k} \\ 0 & \beta_{nr} & \beta_{nr} \\ 0 & 0 & 0 \end{pmatrix} \end{array}, \; V = \begin{array}{c} \\ s_r \\ i_{nr} \\ i_r \end{array} \begin{array}{c} \begin{array}{ccc} s_r & i_{nr} & i_r \end{array} \\ \begin{pmatrix} d_s & 0 & 0 \\ 0 & \gamma_{nr} & -d_i \\ 0 & 0 & \gamma_r + d_i \end{pmatrix} \end{array}. \tag{18}$$

The basic reproduction number R_0 is then defined as the leading eigenvalue of the next generation matrix FV^{-1}. Solving the resulting equation, two eigenvalues are obtained and hence the basic reproduction number is given by

$$R_0 = \max\left(R_0^r = \frac{\alpha_s}{d_s}, R_0^i = \frac{\beta_{nr}}{\gamma_{nr}}\right). \tag{19}$$

The non-trivial disease-free steady state can be perturbed through the s_r and/or i_{nr} class. If the initial seeding is in the s_r class alone, the disease cannot spread independently of the value of R_0^i. If $R_0^r < 1$, the trivial disease-free steady state is stable. However, if $R_0^r > 1$ more individuals become responsive and the system will converge to the non-trivial DFSS. R_0^r is equivalent to the basic reproduction number and represents the number of new responsive individuals to whom information about the disease has been transmitted from a responsive individual. Thus the trivial DFSS is locally stable if and only if $R_0^r < 1$ and $R_0^i < 1$.

Let us consider now the non-trivial DFSS that represents the case when the responsive individuals have reached an 'endemic' level and the spread of infection is not possible. The conditions of local stability in Proposition 1 can be conveniently rearranged to give

$$R^r = \frac{\alpha_s}{d_s} \geq 1 \tag{20}$$

$$R^i = \frac{\beta_{nr}(1-s_0)(\gamma_r + d_i + \alpha_i s_0) + \beta_r s_0(\gamma_{nr} + d_i + \alpha_i s_0)}{\alpha_i s_0 \gamma_r + \gamma_{nr}(\gamma_r + d_i)} \leq 1. \tag{21}$$

where $s_0 = \frac{\alpha_s - d_s}{\alpha_s}$. The first condition is the obvious requirement that $R_0^r = \frac{\alpha_s}{d_s} > 1$. This means that the spread of responsiveness has to be fast enough in order to reach the non-trivial DFSS. An initial small proportion of infectious individuals can kick start the spread of responsiveness. If neither of the two conditions above (Eqs. (20) & (21)) are fulfilled the non-trivial DFSS is not stable and the system will tend to the trivial DFSS $(1, 0, 0, 0, 0)$.

The present system can be considered as a multi-strain system where individuals can become infected by the information (i.e., becoming responsive) and/or by the disease. This

is equivalent to considering the spread of information (i.e., first strain) and the spread of the disease (second strain) as two competing strains [5]. The second strain can infect individuals already infected with the first strain. Hence, 'superinfection' is possible since disease can be transmitted to individuals that are already aware. Considering the stability of the non-trivial DFSS is equivalent to establishing whether i_{nr} and i_r can invade the population when s_r is already at equilibrium. We show that upon perturbing the trivial DFSS the system will tend to either the non-trivial DFSS or an endemic equilibrium. The condition in Eq. (21) can be rewritten in terms of R_0^r and R_0^i to give

$$R_0^i - 1 \leq A(R_0^r - 1), \text{ with } A = \frac{(\gamma_r - \beta_r)(\alpha_i + \gamma_{nr}) + B(\gamma_{nr} - \beta_r)}{\gamma_{nr}(\alpha_i + \gamma_r + B)}, \; B = d_i - \frac{\alpha_i}{R_0^r}. \tag{22}$$

Therefore we can reformulate Proposition 1 in terms of R_0^r and R_0^i.

Proposition 2 *1. The trivial DFSS is locally stable if and only if $R_0^r < 1$ and $R_0^i < 1$.*

2. The non-trivial DFSS is locally stable if and only if $R_0^r > 1$ and $R_0^i - 1 < A(R_0^r - 1)$ (A given in Eq. (22)).

In Fig. 3a the local stability regions of the disease-free and endemic steady states are illustrated for a particular set of parameters. In the case $\alpha_i = \alpha_s$, $d_i = d_s$ (a reasonable assumption from the biological point of view) we have $B = 0$, hence A does not contain R_0^r and $R_0^i - 1 \leq A(R_0^r - 1)$ gives a linear relation between R_0^i and R_0^r. In Fig. 3b the local stability regions are shown for the case $\alpha_i = \alpha_s$, $d_i = d_s$. Numerical studies show that in the case when both DFSSs are unstable there exists a unique endemic equilibrium and this is discussed in the next Subsection.

3.2 Existence and uniqueness of the endemic steady state

In this Subsection our aim is to show numerical evidence that in the case $R_0^i > 1$ and $R_0^i - 1 > A(R_0^r - 1)$ there exists a unique endemic steady state. Numerical investigations and partial analytical results, that will be presented later, show that when conditions in Proposition 2 hold, the two DFSSs are also globally stable. The analysis starts by reducing the five variable system given by Eqs. (9)-(13) (with zeros in the l.h.s.) to two equations with two unknowns. Then the endemic equilibrium can be obtained as the intersection point of two curves (in the plane of the two unknowns). We will show numerically that the two curves have a unique intersection point for different values of the parameters. In this Subsection we assume that $\delta_s = \delta_i = 0$ to make the calculations easier.

The existence and uniqueness of the endemic equilibrium can be investigated via introducing the following new variables

$$i = i_{nr} + i_r \quad \text{and} \quad n = s_{nr} + i_{nr},$$

which denote total infection prevalence and the proportion of non-responsive individuals, respectively. The system based on Eqs. (9)-(13) has four independent variables since $\dot{s}_{nr} + \dot{i}_{nr} + \dot{s}_a + \dot{i}_a + \dot{\tau} = 0$ implies that the sum of these variables is constant and equal to 1. Instead of the original variables the following new variables τ, i, n and i_{nr} are used. The original variables can be expressed easily using the new ones

$$s_{nr} = n - i_{nr}, \quad s_r = 1 - \tau - i - n + i_{nr}, \quad i_r = i - i_{nr}. \tag{23}$$

Hence, in terms of the new variables the system can rewritten to give

$$\begin{aligned}
\dot{\tau} &= \gamma_r i - \gamma i_{nr} - r\tau, && (24)\\
\dot{i} &= i\left(\beta(n - i_{nr}) + \beta_a(1 - \tau - i)\right) - \gamma_r i + \gamma i_{nr}, && (25)\\
\dot{n} &= (d - \alpha n)(1 - n) - \gamma_{nr} i_{nr} + (pr - d)\tau, && (26)\\
\dot{i}_{nr} &= \beta_{nr} i(n - i_{nr}) + \alpha n i_{nr} - (\alpha + \gamma_{nr} + d) i_{nr} + di, && (27)
\end{aligned}$$

where $\beta = \beta_{nr} - \beta_r$ and $\gamma = \gamma_r - \gamma_{nr}$. For simplicity we assume that $\alpha_s = \alpha_i := \alpha$ and $d_s = d_i := d$. We note that this is not a restriction, and all our calculations can be carried out without this assumption.

In order to determine the positive steady state, the system given by Eqs. (24)-(27) is reduced to two equations with two unknowns. Upon expressing one of the unknowns from these equations, a graphical illustration of the two curves determined by the two equations is possible. A systematic numerical study for different parameter values allows to show that these two curves have a unique intersection point that determines the unique endemic equilibrium. In order to carry out this analysis an additional new variable is introduced

$$x = \frac{i_{nr}}{i} \tag{28}$$

and the left hand side of Eqs. (24)-(27) is set to zero to consider all possible steady states. From Eq. (24) we obtain

$$\tau = i\left(\frac{\gamma_r}{r} - x\frac{\gamma}{r}\right). \tag{29}$$

Substituting this expression for τ in Eq. (25) we obtain

$$i = r\frac{\beta_r - \gamma_r + \beta n + \gamma x}{\beta_r(\gamma_r + r) + x(\beta r - \beta_r \gamma)}. \tag{30}$$

Similarly, upon substituting τ in Eq. (26) with the expression given in Eq. (29), we get

$$(d - \alpha n)(1 - n) + i\left((p - \frac{d}{r})(\gamma_r - x\gamma) - \gamma_{nr} x\right) = 0.$$

Finally, by substituting i in the above equation, with the expression given in Eq. (30), the following equation is obtained

$$(d-\alpha n)(1-n)\left(\beta_r(\gamma_r + r) + x(\beta r - \beta_r\gamma)\right) + (\beta_r - \gamma_r + \beta n + \gamma x)\left((pr - d)(\gamma_r - x\gamma) - r\gamma_{nr} x\right) = 0.$$

This gives a quadratic equation in x

$$A_0 + A_1 x + A_2 x^2 = 0 \tag{31}$$

where

$$\begin{aligned}
A_0 &= (d-\alpha n)(1-n)\beta_r(\gamma_r + r) + (\beta_r - \gamma_r + \beta n)(pr - d)\gamma_r, \\
A_1 &= (d-\alpha n)(1-n)(\beta r - \beta_r \gamma) - (\beta_r - \gamma_r + \beta n)\left((pr-d)\gamma + r\gamma_{nr}\right) + \gamma(pr-d)\gamma_r, \\
A_2 &= -\gamma\left((pr-d)\gamma + r\gamma_{nr}\right).
\end{aligned}$$

Using that $i_{nr} = ix$ and upon dividing Eq. (27) by i, we obtain

$$\beta_{nr}(n - ix) + \alpha n x - (\alpha + \gamma_{nr} + d)x + d = 0.$$

Now substituting i using the expression given in Eq. (30), the equation above yields

$$\beta_{nr} n + \alpha n x - (\alpha + \gamma_{nr} + d)x + d - \beta_{nr} x r \frac{\beta_r - \gamma_r + \beta n + \gamma x}{\beta_r(\gamma_r + r) + x(\beta r - \beta_r \gamma)} = 0.$$

The equation above is a linear equation in terms of n

$$B_0 + B_1 n = 0, \tag{32}$$

where

$$\begin{aligned}
B_0 &= (d - (\alpha + \gamma_{nr} + d)x)(\beta_r(\gamma_r + r) + x(\beta r - \beta_r \gamma)) - \beta_{nr} x r(\beta_r - \gamma_r + \gamma x), \\
B_1 &= (\beta_{nr} + \alpha x)(\beta_r(\gamma_r + r) + x(\beta r - \beta_r \gamma)) - \beta_{nr} x r \beta.
\end{aligned}$$

Thus the endemic equilibrium can be obtained as follows. First, the curves given by Eqs. (31) & (32) have to be plotted in terms of the two independent variables (x, n). Their point of intersection defines the x and n coordinate of the endemic equilibrium. Then Eqs. (28), (29) & (30) will yield the other coordinates of the equilibrium of the system given by Eqs. (24)-(27). Finally, Eq. (23) gives the coordinates of the endemic equilibrium in terms of the original variables. Since all coordinates of the endemic equilibrium have to be positive (i.e., $s_{nr}, s_r, i_{nr}, i_r, \tau > 0$), inequalities for the new variables can be obtained. Using the transformation formulas given in Eq. (23), the following conditions for the new variables are obtained. From $i_{nr} > 0$ follows that $i > 0$ and $x > 0$. Similarly, $s_{nr} > 0$ yields $n > ix$. From $i_r > 0$ immediately follows that $i > 0$ and $x < 1$. These conditions, through Eq. (29), automatically imply that $\tau > 0$ since $\gamma_r > \gamma$. Finally, $s_r > 0$ yields $1 - \tau - n - i(1-x) > 0$ and upon using Eq. (29) it follows that

$$i < \frac{r(1-n)}{\gamma_r + r - x(\gamma + r)}.$$

Thus the following conditions for the new variables ensure the positivity of the endemic equilibrium

$$0 < x < 1, \quad 0 < i < \frac{n}{x}, \quad i < \frac{r(1-n)}{\gamma_r + r - x(\gamma + r)}. \tag{33}$$

Using the expression for i given in Eq. (30), conditions in Eq. (33) can be expressed in terms of x and n as follows

$$0 < \quad x \quad < 1 \ \textbf{(A)}, \quad \beta_r - \gamma_r + \beta n + \gamma x > 0 \ \textbf{(B)}, \quad \frac{rx(\beta_r - \gamma_r + \gamma x)}{\beta_r(\gamma_r + r) - x\gamma\beta_r} < n \ \textbf{(C)}, \tag{34}$$

$$n \quad < \quad \frac{\gamma_r(\gamma_r + r) + x((\beta_{nr} - \gamma_{nr})r - 2\gamma(\gamma_r + r)) + x^2\gamma(\gamma + r)}{\beta_{nr}(\gamma_r + r) - x(\beta r + \beta_r\gamma)}. \tag{35}$$

In Fig. 4, for a particular set of parameters, the uniqueness of the endemic equilibrium is illustrated numerically. The endemic steady state can be computed based on the intersection point of the continuous red and blue lines, provided that this point lies in the appropriate area defined by positivity constraints given in Eqs. (34) & (35). Fig. 4 illustrates, that when close to the boundaries delimiting the stability of the different steady states (see Fig. 3), the endemic equilibrium approaches the trivial DFSS or the non-trivial DFSS. When $R_0^r = 0.5$, the endemic equilibrium approaches $(1, 0, 0, 0, 0)$ as R_0^i approaches one from above. In the case of $R_0^r = 2$, the endemic equilibrium approaches $(d/\alpha, (\alpha - d)/\alpha), 0, 0, 0) = (0.5, 0.5, 0, 0, 0)$ as R_0^i decreases to satisfy the condition given by Eq. (22).

3.3 Global dynamical behaviour of the system

In Proposition 2, we determined the local stability of the disease-free steady states. Numerical investigations show that under the assumptions given in the Proposition, the steady states are not only locally but also globally stable, that is all trajectories starting from positive initial condition tend to the given equilibrium. We can prove global stability analytically only in the case $R_0^i < 1$.

Proposition 3 *1. If $R_0^i < 1$ and $R_0^r < 1$, then the trivial disease free steady state $(1, 0, 0, 0, 0)$ is globally asymptotically stable in the positive orthant.*

2. If $R_0^i < 1$ and $R_0^r > 1$, then the non-trivial disease free steady state $(d_s/\alpha_s, 1 - d_s/\alpha_s, 0, 0, 0)$ is globally asymptotically stable in the positive orthant.

PROOF. Let us add Eqs. (11) & (12) and introduce $i = i_{nr} + i_i$. In the case of $R_0^i < 1$, it follows that $\dot{i} < 0$, hence, i tends to zero as $t \to \infty$. Using Eq. (13), implies that also τ tends to zero. Hence, the differential equation for s_{nr}, in the limit $t \to \infty$, will give

$$\dot{s}_{nr} = (d_s - \alpha_s s_{nr})(1 - s_{nr}).$$

This equation can have two equilibria 1 and d_s/α_s. If $R_0^r < 1$, then the only biologically relevant equilibrium is $s_{nr} = 1$ and it is globally stable. If $R_0^r > 1$, then there are two equilibria, with $s_{nr} = 1$ being unstable, while $s_{na} = d_s/\alpha_s$ is globally stable. □

The above proof does not work in the case $R_0^i > 1$; however, we have numerical evidence for the following full characterisation of the global behaviour of the system, see Fig. 3.

1. If $R_0^i < 1$ and $R_0^r < 1$, then the trivial disease free steady state $(1, 0, 0, 0, 0)$ is globally asymptotically stable in the positive orthant.

2. If $R_0^r > 1$ and $R_0^i - 1 < A(R_0^r - 1)$ (A is given in Eq. (22)), then the non-trivial disease free steady state $(d_s/\alpha_s, 1 - d_s/\alpha_s, 0, 0, 0)$ is globally asymptotically stable in the positive orthant.

3. If $R_0^i > 1$ and $R_0^i - 1 > A(R_0^r - 1)$ then there exists a unique endemic steady state and it is globally asymptotically stable in the positive orthant.

In the special case of $\beta_{nr} = \beta_r$ and $\gamma_{nr} = \gamma_r$, all the above statements can be proved analytically. It is important to note that this case corresponds to considering the limit of $\frac{\beta_r}{\beta_{nr}} \to 1$ and $\frac{\gamma_{nr}}{\gamma_r} \to 1$ in the full system. Hence, when close to this regime, the full system can be viewed as a perturbed version of the special case with results from the special case expected to hold for the full system. When $\beta_{nr} = \beta_r$ and $\gamma_{nr} = \gamma_r$, $R_0^i - 1 < A(R_0^r - 1)$ is equivalent to $R_0^i < 1$. Hence we have the following Theorem,

Theorem 1 *If $\beta_{nr} = \beta_r$ and $\gamma_{nr} = \gamma_r$, three different cases follow:*

1. *If $R_0^i \leq 1$ and $R_0^r < 1$, then the trivial disease free steady state $(1, 0, 0, 0, 0)$ is globally asymptotically stable in the positive orthant.*

2. *If $R_0^i \leq 1$ and $R_0^r > 1$, then there exists a non-trivial disease-free steady state $(d_s/\alpha_s, 1 - d_s/\alpha_s, 0, 0, 0)$ that is is globally asymptotically stable in the positive orthant.*

3. *If $R_0^i > 1$, then there exists a unique endemic steady state that is globally asymptotically stable in the positive orthant.*

The proof of Theorem 1 is given in **Appendix 1**.

4 Discussion

The spread and persistence of STIs is a result of the complex interaction between the behaviour of the individuals, the characteristics of the disease and various control programmes that are aimed at limiting disease transmission or bringing prevalence of infection to as low

levels as possible. While more and more data describing the attitudes and lifestyle of individuals is becoming available, it is challenging to capture the interaction of these with the transmission dynamics. In this simple model, we relaxed the assumption of a 'passive' population that will not react to the presence of the disease and we also accounted for the spread of the information about the diseases. The assumption of the model is that individuals who choose to respond to information triggered by the presence of the disease will lower their probability of becoming infected through behavioural change or seek treatment early. The spread of responsiveness competes with the spread of infection and contributes to reducing the number of individuals becoming infected.

We derived the characterisation of the global behaviour of the system using a mixture of analytical and numerical methods and investigated to what extent can the spread of information stop the spread of the infection. For the most general case, the existence and uniqueness of the endemic state is difficult to derive and so is the proof of the global stability results of the disease-free steady states. However, numerical investigations and complete analytical results, for particular choice of parameters, provide a good description of the system. Given the negative feedback between the rate of information transmission at the population level and infection prevalence, the existence of a Hopf bifurcation is possible and in future work this will be investigated further.

The proposed model incorporates two ways in which information or responsiveness can spread and it is important to separately consider the effect of these. The transmission of information due to direct contact between individuals, under appropriate conditions, changes the endemic threshold and can prevent the spread of infection. However, the population wide transmission does not affect the endemic threshold (see Proposition 1, 2 and 3 and Theorem 1), but leads to smaller levels of infection prevalence at the endemic equilibrium (Fig. 5). While the effect of the information transmission, due to direct contact between individuals, is clear from the analysis, the precise implications of the population wide transmission (δ_s, δ_i and k) and the proportion of treated individuals that return to the non-responsive group (p) are less obvious. These parameters do not play a role in determining the stability of the steady states, but have an impact on the level prevalence. Fig. 5 shows that the prevalence decreases considerably faster when $\delta = \delta_s = \delta_i$ increases compared to the case when p decreases. Therefore, even though the population wide transmission cannot completely eradicate infection, it has a significant effect in reducing infection prevalence to low levels.

The discrepancy between the potential impact of population wide and of individual to individual transmission of information on endemic thresholds has important public health implications. These are exemplified in the United Kingdom's early AIDS epidemic, which was concentrated largely among men who have sex with men (MSM). Informal information campaigns within the male homosexual community can be dated to early 1983, prior to dissemination in the gay press (1983-4) and long before the wider government sponsored campaigns of 1986-7. It is estimated that HIV transmission peaked around 1983 among MSM [15], followed by a rapid decrease which was paralleled by a marked fall in male syphilis incident cases, a disease which also concentrates among MSM in the UK. The associated

reduction in the force of infection is thought to have been a major factor in limiting the size of the HIV epidemic in the UK, both through reducing spread among MSM, and limiting bridging to the heterosexual population. The wider population information campaigns of 1986-7 were however associated with much less dramatic changes both in rates of STI diagnosis among women and heterosexual males.

This is a simple model that captures some important features, and although contact heterogeneity is not accounted for, it illustrates how an active host population and the transmission of information triggered by the disease can eradicate or minimise infection levels. We have suggested different model choices, but focused our analysis on the most basic one. Further analysis can help to better understand how disease dynamics is affected by the population wide transmission of information and by how fast the value of information decays over time.

5 Appendix

Here we prove Theorem 1 (the case of $\beta_{nr} = \beta_r$, $\gamma_{nr} = \gamma_r$).

5.1 Existence an uniqueness of the endemic equilibrium

In this special, case the original system (Eqs. (9)-(13)) can be rewrite in terms of τ, i, s_{nr}, i_{nr}. The original variables can be easily expressed in terms of the new ones as follows

$$i_r = i - i_{nr}, \qquad s_r = 1 - \tau - i - s_{nr}.$$

The differential equations take the following form in terms of the new variables,

$$\begin{aligned}
\dot{\tau} &= \gamma_r i - r\tau, && (36)\\
\dot{i} &= i(\beta_r - \gamma_r - \beta_r\tau - \beta_r i), && (37)\\
\dot{s}_{nr} &= -\beta_r i s_{nr} - \alpha_s(1 - s_{nr} - i_{nr})s_{nr} - \frac{\delta_s i}{k+i}s_{nr} + d_s(1 - \tau - i - s_{nr}) + pr\tau, && (38)\\
\dot{i}_{nr} &= \beta_r i s_{nr} - \alpha_i(1 - s_{nr} - i_{nr})i_{nr} - \frac{\delta_i i}{k+i}i_{nr} - \gamma_r i_{nr} + d_i(i - i_{nr}). && (39)
\end{aligned}$$

The main advantage of this special case and the choice of the variables is that the first two equations form an independent subsystem within the whole system. It is easy to see that the steady states of the subsystem given by Eqs. (36)-(37) are

$$(0,0) \quad \text{and} \quad \frac{\beta_r - \gamma_r}{\beta_r(\gamma_r + r)}(\gamma_r, r).$$

Thus the endemic equilibrium may exist if $\beta_r > \gamma_r$ and its τ and i coordinates are given by

$$\tau = \frac{\gamma_r(\beta_r - \gamma_r)}{\beta_r(\gamma_r + r)}, \qquad i = \frac{r(\beta_r - \gamma_r)}{\beta_r(\gamma_r + r)}. \tag{40}$$

The s_{nr} and i_{nr} coordinates of the endemic equilibrium are determined by

$$0 = -\beta_r i s_{nr} - \alpha_s(1 - s_{nr} - i_{nr})s_{nr} - \frac{\delta_s i}{k+i} s_{nr} + d_s(1 - \tau - i - s_{nr}) + pr\tau, \quad (41)$$

$$0 = \beta_r i s_{nr} - \alpha_i(1 - s_{nr} - i_{nr})i_{nr} - \frac{\delta_i i}{k+i} i_{nr} - \gamma_r i_{nr} + d_i(i - i_{nr}), \quad (42)$$

where τ and i are given by Eq. (40). First we prove the uniqueness of the endemic equilibrium.

Proposition 4 *If $\beta_r > \gamma_r$, then the system given by Eqs. (36)-(39) has a unique equilibrium with positive coordinates.*

PROOF. We have seen that the subsystem given by Eqs. (36)-(37) has a unique positive solution. Hence we have to prove that Eqs. (41)-(42) have a unique positive solution such that $s_r > 0$ and $i_r > 0$ is fulfilled, that is

$$s_{nr} < 1 - \tau - i = \frac{\gamma_r}{\beta_r}, \quad \text{and} \quad i_{nr} < i. \quad (43)$$

Solving Eq. (41) for i_{nr} we obtain

$$i_{nr} = 1 + a_1 - \frac{a_0}{s_{nr}} - s_{nr} := h_1(s_{nr}) \quad (44)$$

where

$$a_0 = \frac{d_s \gamma_r}{\alpha_s \beta_r} + \frac{p\gamma_r i}{\alpha_s}, \qquad a_1 = \frac{\beta_r i + d_s}{\alpha_s} + \frac{\delta_s i}{\alpha_s(k+i)},$$

and i is given by Eq. (40). Solving Eq. (42) for s_{nr} we obtain

$$s_{nr} = \frac{i_{nr}(b_1 + 1) - b_0 - i_{nr}^2}{b_2 + i_{nr}} := h_2(i_{nr}) \quad (45)$$

where

$$b_0 = \frac{d_i i}{\alpha_i}, \qquad b_1 = \frac{\gamma_r + d_i}{\alpha_i} + \frac{\delta_i i}{\alpha_i(k+i)}, \qquad b_2 = \frac{\beta_r i}{\alpha_i}.$$

Thus we have to prove that the curves $i_{nr} = h_1(s_{nr})$ in the domain $0 < s_{nr} < \gamma_r/\beta_r$ and $s_{nr} = h_2(i_{nr})$ in the domain $0 < i_{nr} < i$ have a unique intersection point. In order to prove the uniqueness it is enough to show the following.

(i) The functions h_1 and h_2 are concave,

(ii) $h_1(\gamma_r/\beta_r) > i$,

(iii) $h_2(i) > \gamma_r/\beta_r$.

Namely, assume that there would be two intersection points. Then the straight line determined by these two points separates the endpoints of the two curves $(\gamma_r/\beta_r, h_1(\gamma_r/\beta_r))$ and $(h_2(i), i)$ because of the concavity of the curves. One can see from Fig. 6 that this contradicts to (ii) and (iii). Hence we have to prove (i)-(iii).

The concavity of h_1 is obvious. For the concavity of h_2 the following equality can be used

$$i_{nr}(b_1+1) - b_0 - i_{nr}^2 = (b_2 + i_{nr})(b_2 + b_1 + 1 - i_{nr}) - b_2(b_2 + b_1 + 1) - b_0,$$

hence

$$h_2(i_{nr}) = b_2 + b_1 + 1 - i_{nr} - \frac{b_2(b_2 + b_1 + 1) + b_0}{b_2 + i_{nr}}$$

from which $h_2''(i_{nr}) < 0$ easily follows. For (ii)

$$h_1(\frac{\gamma_r}{\beta_r}) = 1 - \frac{\gamma_r}{\beta_r} + \frac{(1-p)\beta_r i}{\alpha_s} + \frac{\delta_s i}{\alpha_s(k+i)}.$$

From Eq. (40) it follows that $1 - \frac{\gamma_r}{\beta_r} > i$, implying $h_1(\gamma_r/\beta_r) > i$. In order to verify (iii) we have

$$h_2(i) = \frac{i(b_1+1) - b_0 - i^2}{b_2 + i} = \frac{D + \gamma_r + \alpha_i(1-i)}{\beta_r + \alpha_i}$$

where $D = \delta_i i/(k+i)$. It is easy to prove that

$$\frac{\gamma_r + \alpha_i(1-i)}{\beta_r + \alpha_i} > \frac{\gamma_r}{\beta_r}.$$

However, this implies $h_2(i) > \gamma_r/\beta_r$.

□

5.2 Dynamical behaviour of the system

Let us first consider the case $R_0^i \leq 1$, that is $\beta_r \leq \gamma_r$. In this case the only nonnegative steady state of the system given by Eqs. (36)-(37) is the origin. From Eq. (37) one can see that $\dot{i} < 0$ when i and τ are positive, thus $i(t) \to 0$ as $t \to +\infty$. Hence Eq. (36) implies that $\tau(t)$ also tends to zero. Thus the origin is a globally stable equilibrium of (36)-(37), and $i_{nr}(t) \to 0$, $i_r(t) \to 0$ as $t \to +\infty$. The dynamical behaviour of s_{nr} can then be determined from Eq. (38) by substituting $i = 0$ and $\tau = 0$. In this case, $s_r = 1 - s_{nr}$ and Eq. (38) takes the following form

$$\dot{s}_{nr} = (d_s - \alpha_s s_{nr})(1 - s_{nr}).$$

This equation can have two equilibria 1 and d_s/α_s. If $d_s/\alpha_s > 1$, then the only biologically relevant equilibrium is $s_{nr} = 1$ and it is globally stable. If $d_s/\alpha_s < 1$, then there are two equilibria, and $s_{nr} = 1$ is unstable, while $s_{nr} = d_s/\alpha_s$ is globally stable. Thus, we have proved the first two statements of Theorem 1.

Let us now consider the case of $R_0^i > 1$, that is $\beta_r > \gamma_r$. In this case, the system given by Eqs. (36)-(37) has two nonnegative equilibria, the origin and the equilibrium given in Eq. (40). Linearisation shows that the origin is a saddle point and the endemic equilibrium is stable. Upon adding Eqs. (36) & (37), immediately follows that $\dot{\tau} + \dot{i} < 0$ when $\tau + i \geq 1$. This implies that all the trajectories (in the nonnegative part of the phase plane) are bounded. Thus the Poincaré-Bendixsson theory implies that all trajectories tend to the endemic equilibrium or to a periodic orbit. The existence of the periodic orbit can be excluded by the Bendixsson-Dulac criterion using the Bendixsson function $1/i$, since dividing the coordinates of the vector field by i the divergence is $-r/i - \beta_r < 0$. Thus we proved that the equilibrium given by Eq. (40) is a globally stable equilibrium of the system given by Eqs. (36)-(37). The dynamical behaviour of s_{nr} and i_{nr} can then be determined from Eqs. (38)-(39) by substituting i and τ with the expressions given in Eq. (40). Drawing the nullclines, given by Eqs. (41)-(42), and the direction field of this two dimensional system, one can see that the unique equilibrium is globally stable, since all trajectories are bounded and the existence of a periodic orbit is excluded by the position of the nullclines that are shown in Fig. 6.

References

[1] B. Andersen, L. Ostergaard,J.K. Moller & F. Olesen 2001. Effectiveness of a mass media campaign to recruit young adults for testing of Chlamydia trachomatis by use of home obtained and mailed samples. Sex. Transm. Infect. 77, 416-418.

[2] R.M. Anderson & R.M. May 1991. *Infectious diseases of humans: dynamics and control*, Oxford University Press.

[3] B. Buonomo, A. D'Onofrio & D. Lacitignola 2008. Global stability of ans SIR epidemic model with information dependent vaccination, Math. Biosci. 216, 9-16.

[4] F.H. Chen 2009. Modeling the effect of information quality on risk behaviour change and the transmission of infectious diseases. Math. Biosci. 217, 125-133.

[5] C. Castillo-Chavez & Z. Feng 1997. To treat or not to treat: the case of tuberculosis J. Math. Biol. 35, 629-656.

[6] W.R. Derrick and P. van den Driessche 1993. A disease transmission model in a non-constant population, J. Math. Biol. 31, 495-512.

[7] O. Diekmann, & J.A.P. Heesterbeek 2000. Mathematical epidemiology of infectious diseases: model building, analysis and interpretation. John Wiley & Sons Ltd, Chichester, UK.

[8] N.M. Ferguson, D.A.T. Cummings, C. Fraser, J.C. Cajka, P.C. Cooley & D.S. Burke 2006. Strategies for mitigating an influenza pandemic, Nature 442, 228-452.

[9] S. Funk, E. Gilad, C. Watkins & V. A. A. Jansen 2009. The spread of awareness and its impact on epidemic outbreaks, PNAS, early edition, www.pnas.org/cgi/doi/10.1073/pnas.0810762106.

[10] T. Gross, C.J. Dommar DĹima & B. Blasius 2006. Epidemic Dynamics on an Adaptive Network, Phys. Rev. Lett. 96, 208701.

[11] H.W. Hethcote and P. van den Driessche 1991. Some epidemiological models with non-linear incidence, J. Math. Biol. 29, 271-287.

[12] H.W. Hethcote & J.A. Yorke 1984. Gonorrhea Transmission Dynamics and Control, Lecture Notes in Biomathematics 56, Springer-Verlag, New York, 1984.

[13] A. Korobeinikov and P.K. Maini 2005. Non-linear incidence and stability of infectious disease models, Math. Med. Biol. 22, 113-128.

[14] N. Low, N. Bender, L. Nartey, S. Redmond, A. Shang & J. Stephenson 2006. Revised rapid review of evidence for the effectiveness of screening for genital chlamydia infection in sexually active young women and men. Review 2, National Institute of Clinical Excellence. Ref Type: Report.

[15] A. Nicoll, G. Hughes, M. Donnelly, S. Livingstone, D. De Angelis, K. Fenton, B. Evans, O. Nöel Gill & M. Catchpole 2001. Assessing the impact of national anti-HIV health campaigns: trends in the transmission of HIV and other sexually transmitted infections in England. Sex. Transm. Inf. 77, 242-247.

[16] M.K. Oh, D.M. Grimley, J.S. Merchant, P.R. Brown, H. Cecil & E.W. Hook 3rd 2002. Mass media as a population-level intervention tool for Chlamydia trachomatis screening: report of a pilot study. Journal of Adolescent Health 31, 40-47.

[17] S. Risau-Gusman & D.H. Zanette 2009. Contact switching as a control startegy for epidemic outbreaks published online in J. Theor. Biol. 257, 52-60.

[18] P. van den Driessche & J. Watmough 2002. Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission, Math. Biosci. 180, 29-48.

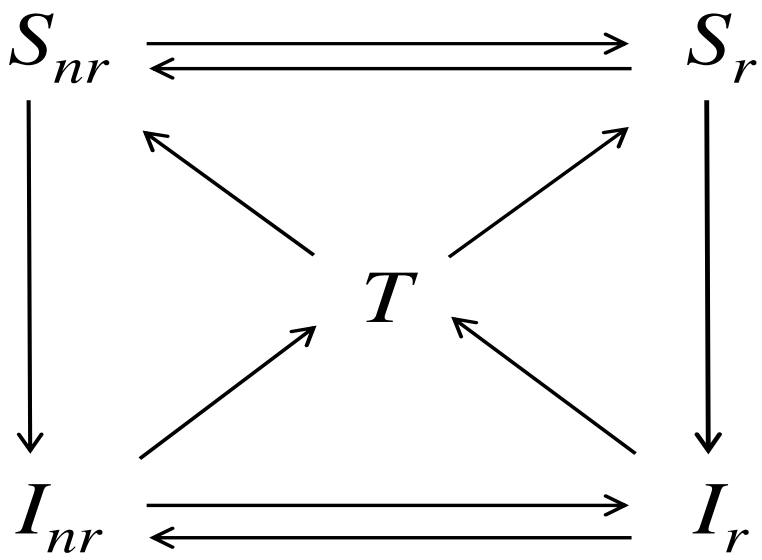

Figure 1: Illustration of all possible transitions.

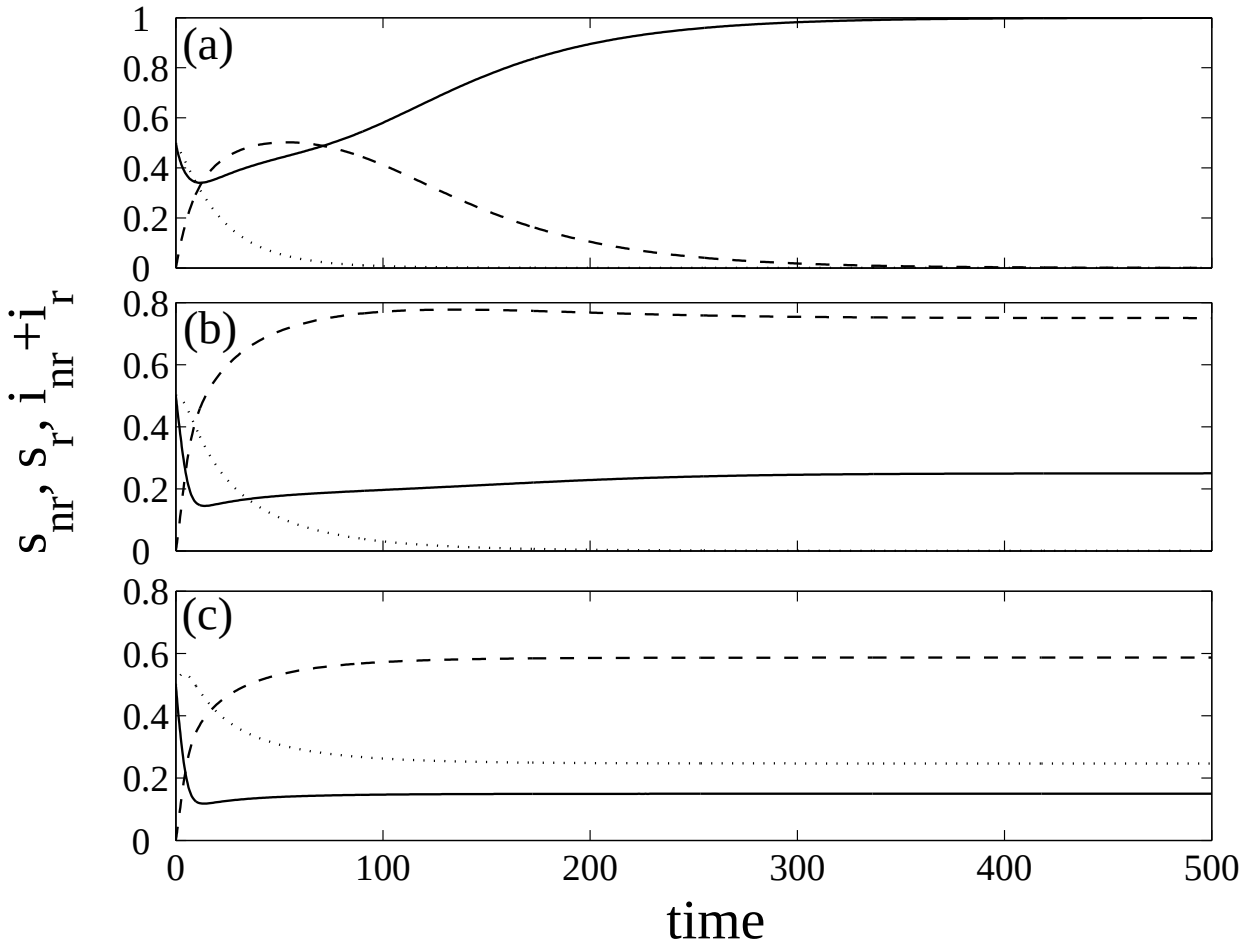

Figure 2: Three different model outcomes, (a) trivial disease-free steady state (b) non-trivial disease-free steady state and (c) endemic steady state, illustrated in terms of s_{nr} (continuous), s_r (dashed) and $i_{nr} + i_r$ (dotted). Parameter values are $\gamma_{nr} = 1/(26 \text{ weeks})$, $\gamma_r = 1/(13 \text{ weeks})$, $r = 1/(1 \text{ week})$, $d = d_s = d_i = 1/(12 \text{ weeks})$, $\delta_s = d_s$, $\delta_i = d_i$, $p = 0.5$ and $k = 0.01$. The other parameter values for the different panels are: (a) $\beta_{nr} = 0.5\gamma_{nr}$, $\beta_r = 0.5\beta_{nr}$ and $\alpha_s = \alpha_i = 0.5d_s$, (b) $\beta_{nr} = 2\gamma_{nr}$, $\beta_r = 0.5\beta_{nr}$ and $\alpha_s = \alpha_i = 4d_s$, and (c) $\beta_{nr} = 4\gamma_{nr}$, $\beta_r = 0.5\beta_{nr}$ and $\alpha_s = \alpha_i = 4d$. The initial condition for all cases is $(s_{nr}, s_r, i_{nr}, i_r, \tau) = (0.5, 0, 0.5, 0, 0)$.

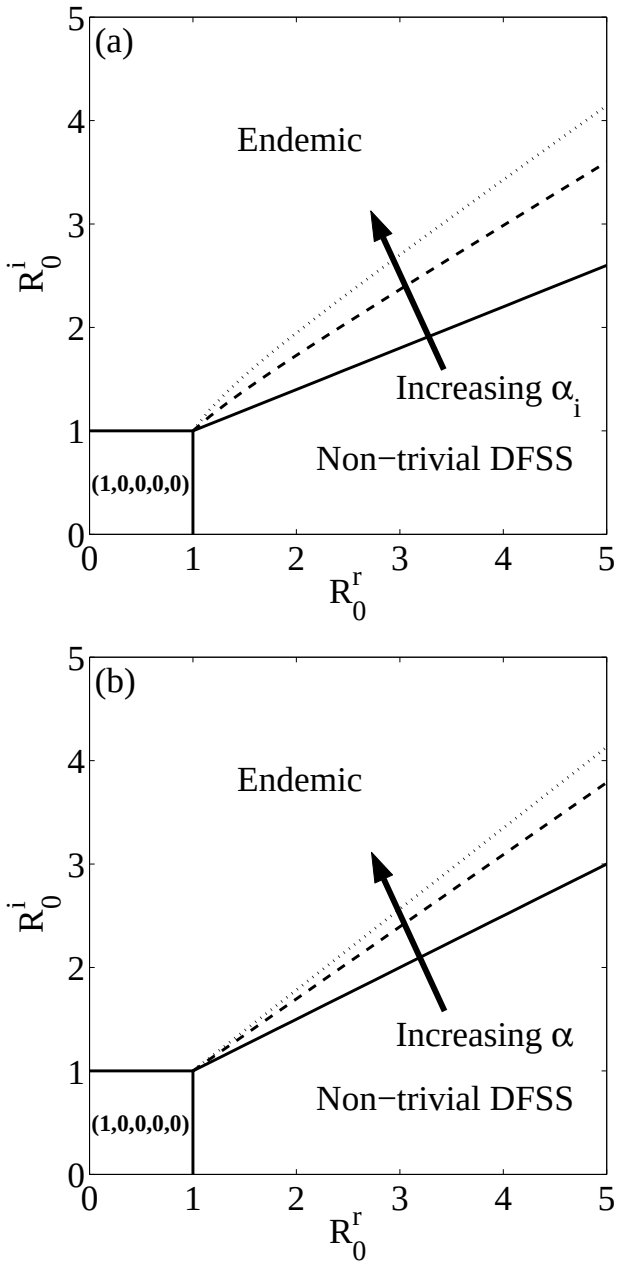

Figure 3: Illustration of the long term behaviour of the system as a function of R_0^r and R_0^i for increasing values of $\alpha_i = 0.05\,j\,(j = 0, 1, 2)$ and $d_i = 1/(52 \text{ weeks})$ (a), and $\alpha = \alpha_s = \alpha_i = 0.05\,j\,(j = 0, 1, 2)$ (b). For both cases, $\gamma_{nr} = 1/(26 \text{ weeks})$, $\gamma_r = 1/(13 \text{ weeks})$ and $\beta_r = \gamma_{nr}$.

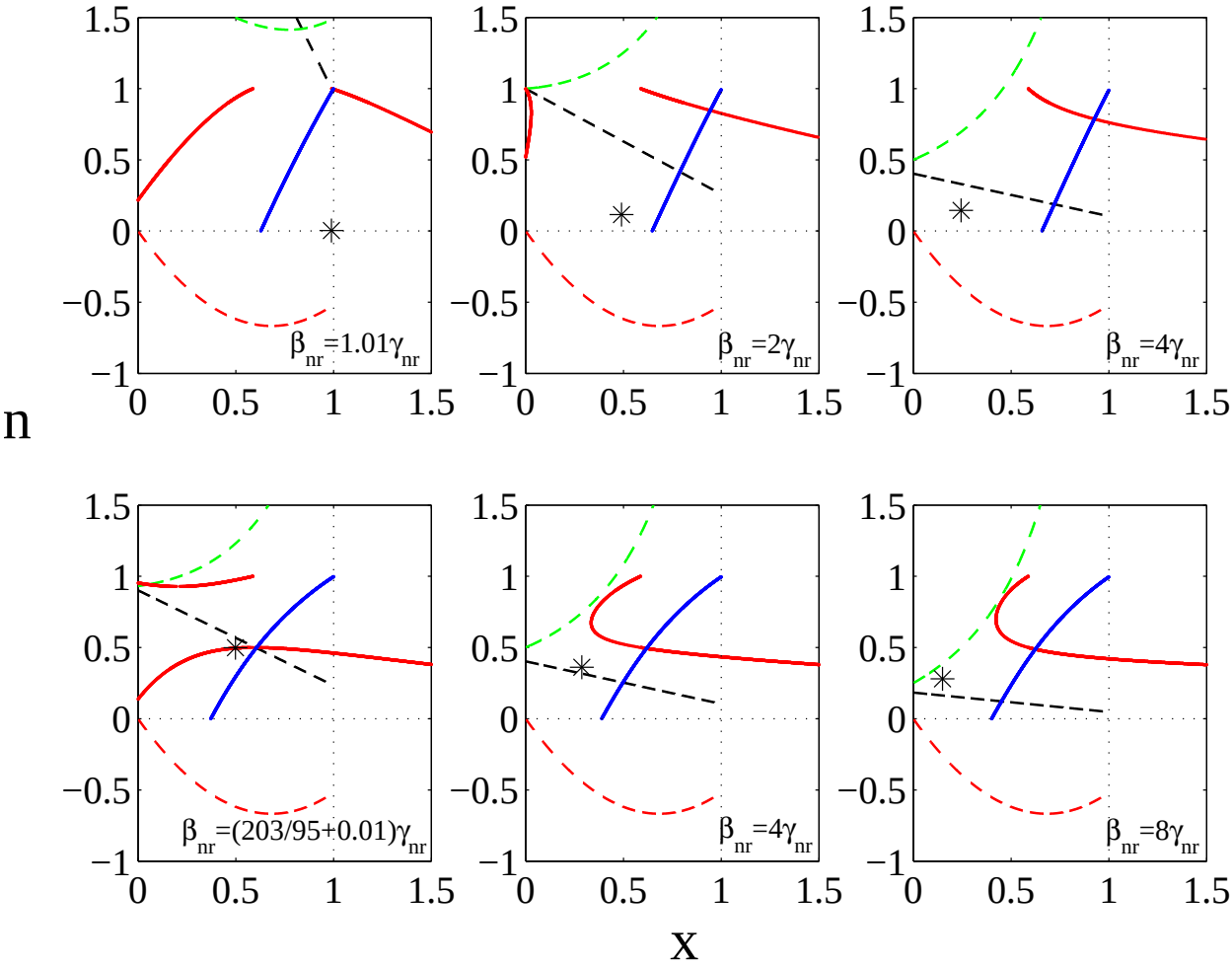

Figure 4: Illustration in terms of the (x, n) coordinates of the uniqueness of the endemic equilibrium. Dashed lines represent the positivity conditions given in Eqs. (34) and (35) (Eq. (34) **B**, black; Eq. (34) **C**, red; Eq. (35), green). Continuous lines correspond to the curves given by Eq. (31) (red) and Eq. (32) (blue). The intersection of these two curves determine the (x_0, n_0) pair that is used to compute the endemic equilibrium in terms of the original variables. The asterisks, in all panels, denote the (s_{nr}, s_r) pair. The top row corresponds to $\alpha = 0.5d$ (i.e., $R_0^r = 0.5$), while the bottom row illustrates the case of $\alpha = 2.0d$ (i.e., $R_0^r = 2$). The other parameter values are $\gamma_{nr} = 1/(26 \text{ weeks})$, $\gamma_r = 1/(13 \text{ weeks})$, $r = 1/(1 \text{ week})$, $d = d_s = d_i = 1/(12 \text{ weeks})$, $\delta_s = \delta_i = 0$, $p = 0.5$ and $\beta_r = 0.025$. For completeness, positivity bounds outside biologically plausible regions ($0 < x < 1$ and $0 < n < 1$) are also shown.

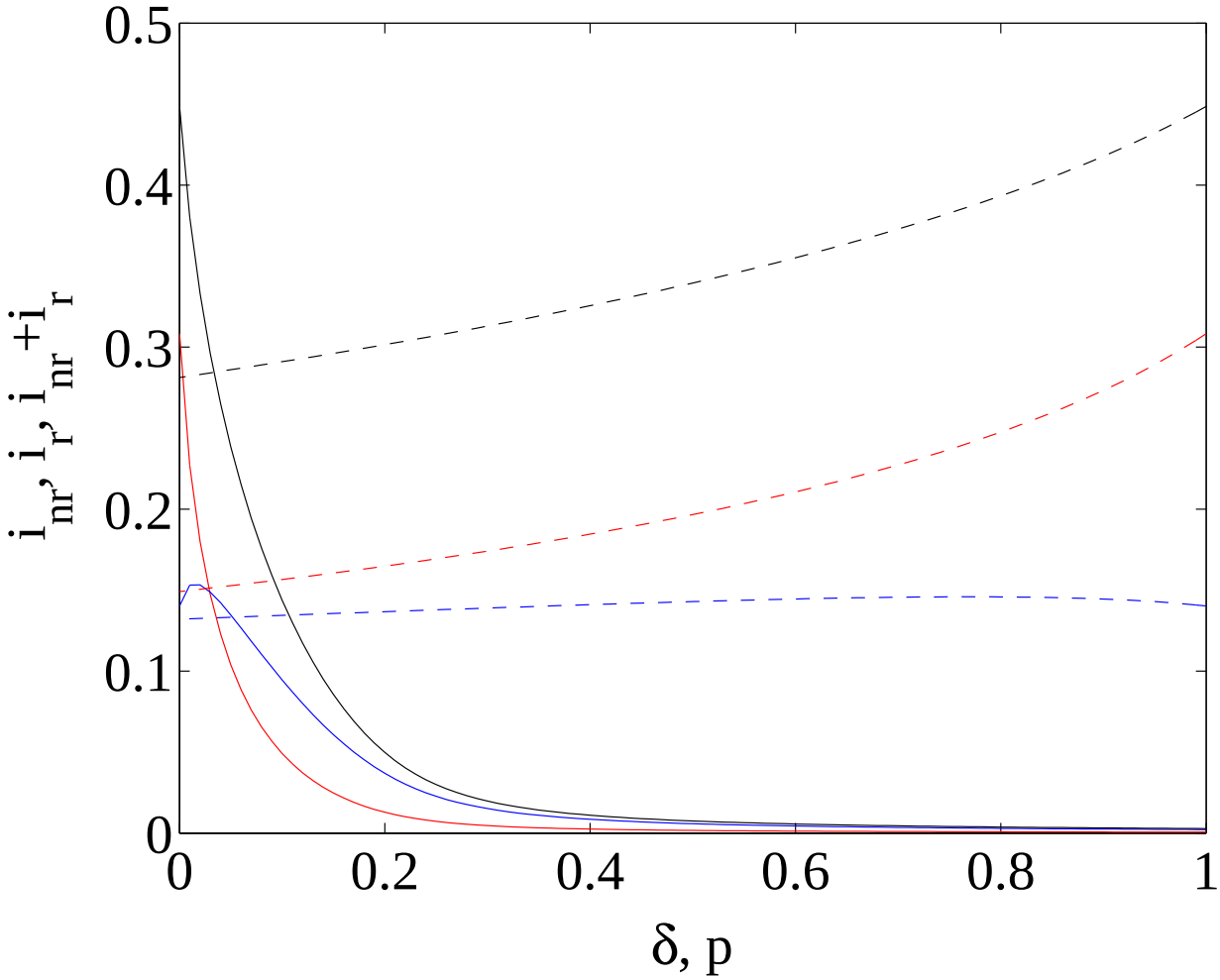

Figure 5: Illustration of the level of prevalence once the endemic equilibrium is reached (i_{nr}-red, i_r-blue, $i_{nr}+i_r$-black). The starting point is the worst case scenario when $\delta = \delta_s = \delta_i = 0$ and $p = 1.0$. The case of constant $p = 1$ (continuous lines, increasing δ) and constant $\delta = \delta_s = \delta_i = 0$ (dashed lines, decreasing p) are shown. The other parameter values are $\gamma_{nr} = 1/(26 \text{ weeks})$, $\gamma_r = 1/(13 \text{ weeks})$, $r = 1/(1 \text{ week})$, $d = d_s = d_i = 1/(12 \text{ weeks})$, $\beta_{nr} = 3\gamma_{nr}$, $\beta_r = 0.5\beta_{nr}$, $\alpha = \alpha_s = \alpha_i = 2d$ and $k = 0.01$.

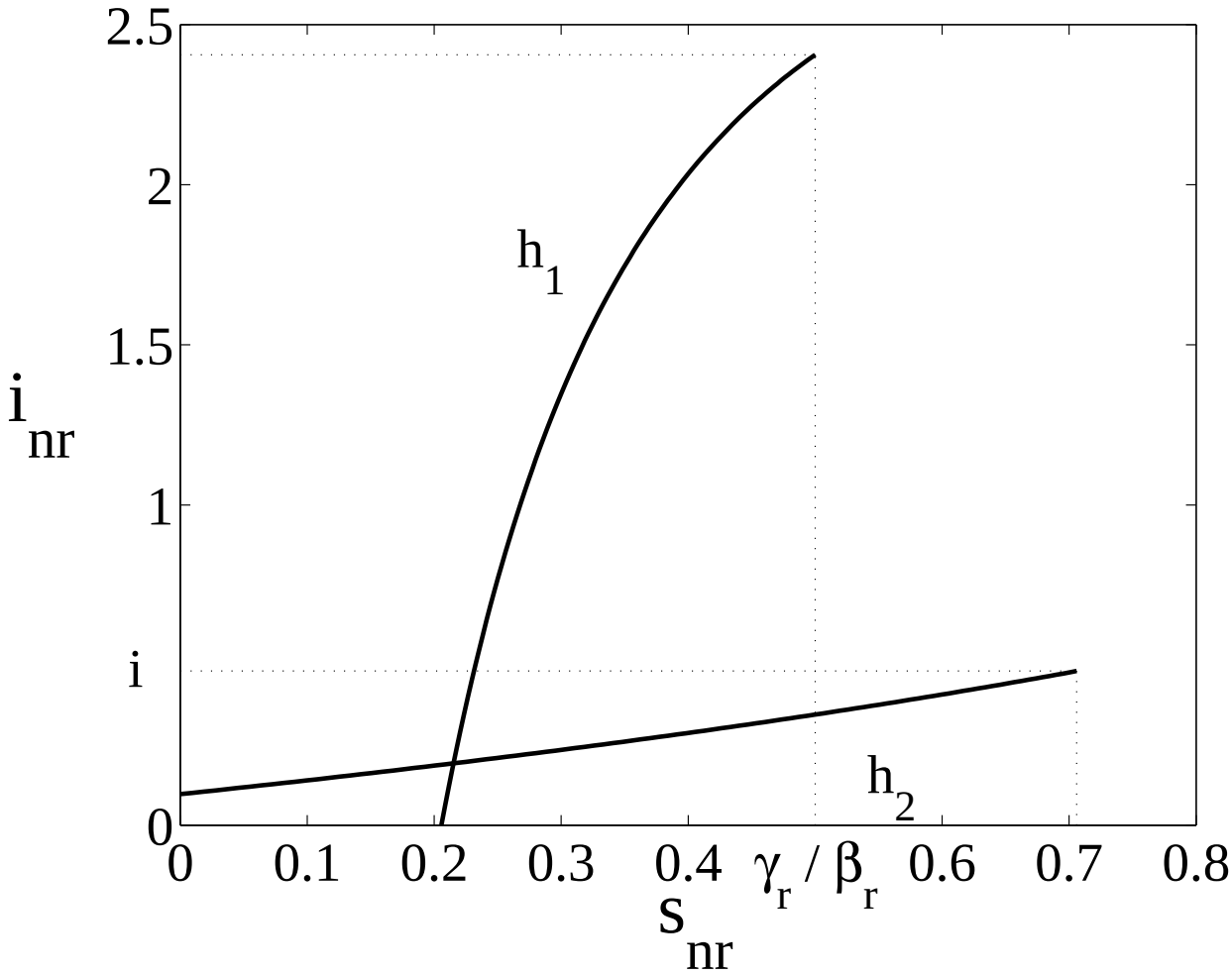

Figure 6: Illustration of the functions $i_{nr} = h_1(s_{nr})$ and $s_{nr} = h_2(i_{nr})$. As indicated by Eq. (43), s_{nr} and i_{nr} are restricted to $s_{nr} < \frac{\gamma_r}{\beta_r}$ and $i_{nr} < i$, respectively. The values of the parameters are $\beta_{nr} = \beta_r = 1/13$, $\gamma_{nr} = \gamma_r = 1/26$, $\alpha_s = \alpha_i = 0.02$, $d_s = d_i = 1/52$, $\delta_s = \alpha_s$, $\delta_i = \alpha_i$, $r = 1$, $p = 0.5$ and $k = 0.01$.