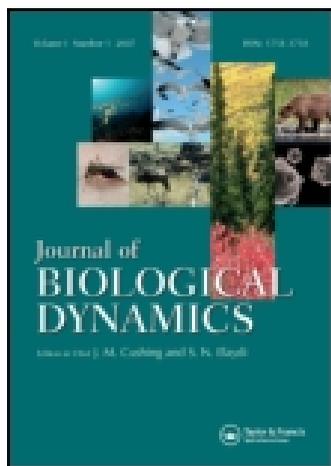


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### The effect of risk-taking behaviour in epidemic models

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## The effect of risk-taking behaviour in epidemic models

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We study an epidemic model that incorporates risk-taking behaviour as a response to a perceived low prevalence of infection that follows from the administration of an effective treatment or vaccine. We assume that knowledge about the number of infected, recovered and vaccinated individuals has an effect in the contact rate between susceptible and infectious individuals. We show that, whenever optimism prevails in the risk behaviour response, the fate of an epidemic may change from disease clearance to disease persistence. Moreover, under certain conditions on the parameters, increasing the efficiency of vaccine and/or treatment has the unwanted effect of increasing the epidemic reproductive number, suggesting a wider range of diseases may become endemic due to risk-taking alone. These results indicate that the manner in which treatment/vaccine effectiveness is advertised can have an important influence on how the epidemic unfolds.

**Keywords:** risk-taking behaviour; recovery; treatment; vaccination

*AMS Subject Classification:* 92D30

### 1. Introduction

Individuals facing an infectious disease may evaluate the contamination risk and engage or avoid risky situations or behaviours. The risk-taking behaviour of any individual is probably a very complex functional response that depends on many factors such as morbidity of the disease, infectiousness, mode of transmission or the existence and efficacy of a treatment. There are multiple studies that show individuals have dynamic responses to transmission risks (e.g. [13,16,20], and references therein) that change with varying circumstances. For example, the advent of antibiotics makes people less concerned with exposure risks to common pathogens that are known to be easily treated. To the contrary, knowledge about the severity of a disease (measured by the number of infectious people, virulence, lack of effective treatment) may cause

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susceptible individuals to be more cautious and reduce their exposure risk. Recent examples are given by SARS or swine flu epidemic when many individuals were clearly more pro-active in avoiding the contamination risk: avoiding crowds, wearing masks, etc. In general, we can talk about two primary ways in which behavioural attitudes influence the spread of a disease:

- Increasing risk-taking behaviour as a response to ‘good news’ (optimism),
- Decreasing risk-taking behaviour as a response to ‘bad news’ (pessimism).

It seems that the second case is more widely addressed in the literature (see Del Valle *et al.* [26] as an example). The model proposed there simulates a bioterrorist attack in which the contact rate is modified to decrease with the size of infectious individuals. Their results show that even small behavioural changes play a big role in counteracting the epidemic. Thus, their model follows the framework in which the behaviour is always positive from the perspective of individuals (i.e. reduction of risk behaviour only). A general dependency of the contact rate of various types of individuals has been proposed by Castillo-Chavez and Thieme [8]. Their article is not focused on modeling risk-taking behaviour but an example is proposed in which the contact rate is assumed to depend on all types of people involved in the model to illustrate how the population adjusts their behaviour in response to knowledge about the epidemic status of the population. However, the purpose of that model was to show the use of asymptotically autonomous differential systems in proving the global stability of an interior equilibrium and no detailed discussion was provided on the implications of risk-taking behaviour in the evolution of the epidemics. Several other studies model the behavioural change as constant rates (i.e. similar to weighted means that distinguish different contributions of various risk levels of individuals) such as Poletti *et al.* [22,23] or Brauer [6]. Again, here the focus is on how sensitive the epidemic is with respect to behavioural changes.

In this article we intend to model individual risk-taking behaviour in response to a *perceived* lower burden of an infectious disease given by the knowledge of the size of relevant categories of individuals present during an epidemic: infected, recovered and/or vaccinated. Specifically, we assume that optimism (or risk-taking) correlates in varying degrees with lower number of infected people and higher numbers of recovered/treated or vaccinated. Several studies show that behavioural responses of this nature are possible, though more sociological research is certainly needed to establish how likely they are.

To illustrate the aforementioned premises, take, for example, the changing landscape of human immunodeficiency virus (HIV) prevention and treatment efforts. It is known that as advances have been made to slow the spread of the HIV epidemic, beliefs – and subsequently behaviours – have shifted in response. Specifically, a handful of studies have documented associations between improvements in HIV treatment outcomes and greater sexual risk taking; that is, more events of condomless sex and/or increased number of sex partners in response to information that HIV treatments have improved [9,11,21,25,27]. Furthermore, there is evidence that these shifts in beliefs and behaviours are also related to new sexually transmitted infections, a proxy for predicting HIV transmission [17]. Not only is it known that advances in treatment options are associated with greater risk-taking for the disease, there is also evidence that people who perceive relatively lower overall burden of HIV engage in greater sexual risk taking as well [12]. This is a phenomenon coined as *intuitive epidemiology* [18]. Intuitive epidemiology works on the premise that people hold a working knowledge of how much disease there is in a given population (perceived prevalence) and the relative share of the disease across different geographical locations (perceived disease burden). Although an individual’s capacity to accurately estimate perceived prevalence and disease burden widely varies, there exist numerous examples of these perceptions as being related to risk-taking. For example, Kalichman *et al.* [19] found that perceived HIV disease burden is positively related to condom use, and Chao *et al.* [7] found that perceived

HIV prevalence is positively related to condom use. Moreover, engaging in diagnostic testing for HIV, a critical step in slowing the HIV epidemic [28], is also related to perceiving greater disease prevalence [29]. Finally, in addition to perceptions of the disease burden, there is also evidence that the advent of prevention tools themselves, such as the availability of a HIV vaccine, can alter people's risk-taking behaviours and has the potential to negatively impact vaccine effectiveness [3,10].

A specific question that we will address is whether risk-taking alone can cause an endemic situation to be more likely or, in extreme cases, to change the prediction from disease clearance to persistence. By analysing the effect of increasing the treatment/vaccine efficiency in the presence of risk-taking behaviour, we will show that, under certain conditions, these measures, usually positive, may actually have a detrimental effect. These conditions depend on the disease-specific parameters and the behavioural response of individuals.

Although many studies on the risk-taking behaviour were motivated by the HIV epidemic, we want to point out, from the onset, that our model does not address a specific disease (although the examples given in Appendices 1 and 2 do use some parameters related to Tuberculosis and HIV). Rather we want to show that the risk-taking component in a generic model may play an important role in many situations in which the optimism prevails in response to a perceived low disease burden.

This article is structured as follows: in the following section we introduce our general epidemic model and compute its reproductive number. We show that this number could increase with higher vaccination rates. In Section 3 we provide a more detailed analysis of the model with vaccination only. Section 4 provides a similar analysis for the model with treatment only. We show that risk-taking behaviour dependent on the treated class may cause bistability between the disease free equilibrium and an endemic state. Using a linear form for the risk-taking function we show that the bistability range of the infection rate may extend if the treatment rate increases within a certain critical interval. We conclude the paper with our thoughts on the implications and limitations of these results and ideas for further research.

## 2. The general model

We consider an epidemic model that includes treatment (including also the possible natural recovery) and vaccination. We assume that individuals adjust their risk-taking behaviour based on the knowledge of how the epidemic affects the community. Specifically, we assume risk avoidance with respect to the size of the infectious class combined with risk taking (or optimism) with higher numbers of those who are recovered or become immune (treatment, natural recovery and/or vaccination). For greater generality we will assume also several other features that may or may not be present in a particular real situation: vaccination available both at birth and later in life, waning of vaccine effectiveness in time or treatment failure due to a multitude of possible factors such as lack of adherence, drug resistance. The general model is as follows:

$$\begin{aligned}
 S' &= (1 - \xi)\beta P - f(I, T, V)\frac{SI}{P} - \bar{\mu}S - \nu S + \eta V, \\
 V' &= \xi\beta P + \nu S - \bar{\mu}V - \eta V, \\
 I' &= f(I, T, V)\frac{SI}{P} - \bar{\mu}I - rI - \alpha I + \delta T, \\
 T' &= rI - \bar{\mu}T - \delta T.
 \end{aligned}
 \tag{1}$$

$S$ ,  $I$ ,  $T$  and  $V$  denote the susceptible, infected, treated and vaccinated classes, respectively.  $\beta$  is the per-capita birth rate,  $\bar{\mu} := \mu + bP$  is the logistic natural mortality rate where  $P = S +$

$I + T + V$  is the total population size. We assume that a fraction  $0 < \xi < 1$  of the newborn are vaccinated at birth while  $v$  is the rate at which susceptible are vaccinated as adults later on.  $\eta$  and  $\delta$  denote the rate at which vaccinated and treated individuals re-enter the susceptible and infected classes, respectively, due to a possible waning of the effectiveness of the vaccine or treatment failure.  $r$  denotes the treatment rate and  $\alpha$  is the additional mortality due to the disease.

The incidence rate that includes the risk-taking behaviour is given by  $f(I, T, V)$  which is assumed to be a continuously differentiable function with the following monotonicity assumptions:

$$\frac{\partial f}{\partial I} < 0, \quad \frac{\partial f}{\partial T} > 0 \quad \text{and} \quad \frac{\partial f}{\partial V} > 0.$$

The model (1) always admits the following disease-free equilibrium (DFE):

$$\bar{S} = \frac{K[\beta(1 - \xi) + \eta]}{\beta + \eta + v}, \quad \bar{I} = 0, \quad \bar{T} = 0, \quad \bar{V} = \frac{K(\beta\xi + v)}{\beta + \eta + v}.$$

The Jacobian of Equation (1) evaluated at the DFE is

$$\begin{bmatrix} -\xi\beta - b\bar{S} - v & (1 - \xi)\beta - b\bar{S} + \eta & (1 - \xi)\beta - \frac{\bar{S}}{K}f(0, 0, \bar{V}) - b\bar{S} & (1 - \xi)\beta - b\bar{S} \\ \xi\beta + v - b\bar{V} & \xi\beta - b\bar{V} - \beta - \eta & \xi\beta - b\bar{V} & \xi\beta - b\bar{V} \\ 0 & 0 & \frac{\bar{S}}{K}f(0, 0, \bar{V}) - r - \alpha - \beta & \delta \\ 0 & 0 & r & -\beta - \delta \end{bmatrix}.$$

This is a block matrix and the eigenvalues corresponding to the  $(S, V)$  block are both negative:

$$-bK \quad \text{and} \quad -(v + \beta + \eta).$$

The eigenvalues corresponding to the  $(I, T)$  block have negative real parts provided that the following conditions are met:

$$\begin{aligned} \frac{\bar{S}}{K}f(0, 0, \bar{V}) &< \delta + 2\beta + \alpha + r \quad \text{and} \\ \frac{\bar{S}}{K}f(0, 0, \bar{V}) &< \frac{\beta^2 + \beta(\delta + \alpha + r) + \delta\alpha}{\beta + \delta}. \end{aligned}$$

A straightforward computation shows that the second condition implies the first. Hence, the DFE is locally stable when the following epidemic reproductive number is less than 1:

$$\mathcal{R}_0 := \frac{\beta + \delta}{\beta^2 + (\alpha + r + \delta)\beta + \delta\alpha} \frac{\beta(1 - \xi) + \eta}{\beta + \eta + v} f\left(0, 0, \frac{K(\beta\xi + v)}{\beta + \eta + v}\right).$$

This quantity has the usual epidemiological meaning of representing the secondary number of infections caused by a single infected individual introduced in a healthy population: the first term represents the mean duration of the infectious period with an indefinite number of treatment failures, the second term is the fraction of the total population that is susceptible and the last term is the rate of infection.

Notice that, if we do not have a risk-taking behaviour response (i.e. if  $f$  were a constant) then increasing either the fraction of vaccination at birth ( $\xi$ ) or the rate of adult vaccination ( $v$ ) will decrease the epidemic reproductive number as expected, thus lowering the chance of an epidemic. However, if the risk-taking behaviour is present, there are conditions on the parameters that will cause the epidemic reproductive number to increase with vaccination terms up to a

certain critical value. To see this, we consider first  $\mathcal{R}_0$  as a function of  $\xi$ . Then  $d\mathcal{R}_0/d\xi > 0$  provided that

$$\frac{\partial f}{\partial V} \left( 0, 0, \frac{K(\beta\xi + v)}{\beta + \eta + v} \right) > \frac{\beta + \eta + v}{K[\beta(1 - \xi) + \eta]} f \left( 0, 0, \frac{K(\beta\xi + v)}{\beta + \eta + v} \right).$$

Suppose that this condition is satisfied in the absence of vaccination at birth (i.e.  $\xi = 0$ ):

$$\frac{\partial f}{\partial V} \left( 0, 0, \frac{vK}{\beta + \eta + v} \right) > \frac{\beta + \eta + v}{K(\beta + \eta)} f \left( 0, 0, \frac{vK}{\beta + \eta + v} \right). \tag{2}$$

Then, due to continuity, there exists a critical value  $\xi^*$  such that  $\mathcal{R}_0(\xi)$  is increasing on the interval  $0 < \xi < \xi^*$ . The inequality (2) provides a lower bound on the risk-taking behaviour that occurs in a population where there is no vaccination at birth. The existence of the critical value  $\xi^*$  suggests that by introducing a rate of vaccination at birth that is too low, the epidemic reproductive number will increase.

A similar situation occurs if we analyse the monotonicity of  $\mathcal{R}_0$  with respect to the rate of adult vaccination  $v$ . Its derivative with respect to  $v$  is positive provided that

$$\frac{\partial f}{\partial V} \left( 0, 0, \frac{K(\beta\xi + v)}{\beta + \eta + v} \right) > \frac{\beta + \eta + v}{K[\beta(1 - \xi) + \eta]} f \left( 0, 0, \frac{K(\beta\xi + v)}{\beta + \eta + v} \right),$$

and, if this condition is met without adult vaccination ( $v = 0$ ), it becomes

$$\frac{\partial f}{\partial V} \left( 0, 0, \frac{\xi\beta K}{\beta + \eta} \right) > \frac{\beta + \eta}{K[\beta(1 - \xi) + \eta]} f \left( 0, 0, \frac{\xi\beta K}{\beta + \eta} \right). \tag{3}$$

It follows that there exists a critical vaccination rate  $v^*$  such that on the interval  $0 < v < v^*$  the epidemic reproductive number increases. Condition (3) has a similar interpretation: it provides a lower bound on the initial growth rate of risk-taking behaviour present in a population without adult vaccination.

*Remark 1* These results suggest that it is possible to have conditions for disease clearance before the introduction of a vaccine (i.e.  $\mathcal{R}_0 < 1$ ) while vaccination levels that are too low will change the conditions to make an epidemic likely ( $\mathcal{R}_0 > 1$ ).

We show that this is possible using the following risk-taking function:

$$f(I, T, V) := \lambda(1 + \epsilon_1 T + \epsilon_2 V - aI). \tag{4}$$

The advantage of this form lies in its linearity making the computation of all the thresholds as simple as possible. One can also think of this example as a linear approximation of a more complicated form. However, this choice requires an artificial condition on  $\partial f/\partial I$  from the outset in order to prevent the contact rate  $f$  from taking negative values which would not be biologically correct. Since the maximum population size is  $K$ , then condition  $a < 1/K$ , assumed throughout, ensures that  $f(I, T, V)$  is positive for all values in the biologically relevant interval  $(0, K)$ .

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Notice that  $\epsilon_1 = \epsilon_2 = a = 0$  correspond to no risk-taking behaviour. Condition (2) now becomes

$$\epsilon_2 > \frac{\beta + \eta + v}{K(\beta + \eta - v)} \quad \text{and} \quad \beta + \eta > v.$$

The corresponding critical value  $\xi^*$  up to which vaccination at birth increases  $\mathcal{R}_0$  is

$$\xi^* = \frac{\epsilon_2 K(\beta + \eta - v) - (\beta + \eta + v)}{2\epsilon_2 \beta K}.$$

As an additional consequence, we can see that the vaccination at birth will have this critical value only if the adult vaccination is low enough, ( $v < \beta + \eta$ ).

With respect to adult vaccination, condition (3) becomes

$$\epsilon_2 > \frac{\beta + \eta}{K(\beta + \eta - 2\beta\xi)} \quad \text{and} \quad \xi < \frac{\beta + \eta}{2\beta}.$$

The critical value of adult vaccination  $v^*$  up to which  $\mathcal{R}_0$  increases is

$$v^* = \frac{\epsilon_2 K(\beta + \eta - 2\beta\xi) - (\beta + \eta)}{1 + \epsilon_2 K}.$$

Similarly, we see from the upper bound on  $\xi$  that increasing adult vaccination from 0 to  $v^*$  has a detrimental effect only if the fraction of vaccination at birth is low enough.

In Appendix 1 we present several examples to illustrate the possible detrimental effect of increasing  $\xi$  and  $v$  past their critical values described above.

The analysis of any possible endemic state is too complex for our model in its most general form. However, we show in Section 4, for a simplified model, that the component of the risk-taking behaviour associated with the treated class  $T$  may cause the existence of multiple endemic equilibria. This, in turn, creates the possibility of a bistability regimen between the DFE and an endemic one. Using the explicit form Equation (4), we show that the range of the infection rate  $\lambda$  that leads to bistability, may actually increase if the treatment rate  $r$  increases between two critical values. We conjecture that this phenomenon is directly related to the component of  $f$  that depends on the treated class  $T$  at least for diseases with low disease-induced mortality. We prove this in the case of no disease mortality ( $\alpha = 0$ ) in the following theorem:

**THEOREM 2.1** *If no disease mortality is present and the risk-taking behaviour depends only on the infected and the vaccinated class, a locally stable disease free equilibrium cannot co-exist with an endemic state for the model (1).*

*Proof* Assuming no disease mortality (i.e.  $\alpha = 0$ ), the total population approaches an equilibrium denoted by  $K := \beta - \mu/b$ . Any possible endemic state denoted by  $(S^*, I^*, T^*, V^*)$  satisfies the following:

$$S^* = K - I^* - T^* - V^*, \quad I^* = \frac{\beta + \delta}{r} T^*, \quad V^* = \frac{rK(\beta\xi + v) - v(\beta + \delta + r)T^*}{r(\beta + v + \eta)},$$

while  $T^*$  is any possible positive root for

$$\begin{aligned} & f\left(\frac{\beta + \delta}{r} T, T, \frac{rK(\beta\xi + v) - v(\beta + \delta + r)T}{r(\beta + v + \eta)}\right) \\ &= \frac{Kr\beta(\beta + r + \delta)(\beta + v + \eta)}{(\beta + \delta)\{rK[\beta(1 - \xi) + \eta] - (\beta + \eta)(\beta + r + \delta)T\}}. \end{aligned}$$

Denoting by  $A(T)$  and  $g(T)$  the left- and right-hand side, respectively of the above equality, it is easy to see that  $g(T)$  is a concave upward increasing function in  $T$  with a vertical asymptote at

$T = rK[\beta(1 - \xi) + \eta]/(\beta + \eta)(\beta + r + \delta)$ . The stability of the DFE (i.e.  $\mathcal{R}_0 < 1$ ) is equivalent to

$$f(0, 0, \bar{V}) < g(0).$$

For an endemic equilibrium to exist under this condition,  $A(T)$  must be increasing on some interval taking values sufficiently large to intersect the graph of  $g(T)$ . Notice also that if that happens it is likely that there will be a second intersection (thus, a second endemic state) due to the vertical asymptote of  $g(T)$ .

However, if the risk-taking behaviour would depend *only* on the infected class and/or the vaccinated class,  $A(T)$  would be a decreasing function in  $T$  which means there will not be any endemic states if the disease free state is locally stable. To see this notice that

$$A'(T) = \frac{\partial f}{\partial I} \frac{\beta + \delta}{r} + \frac{\partial f}{\partial T} - \frac{\partial f}{\partial V} \frac{v(\beta + \delta + r)}{r(\beta + v + \eta)}.$$

Since  $\partial f/\partial I < 0$  and  $\partial f/\partial V > 0$  then  $\partial f/\partial T = 0$  implies  $A'(T) < 0$ . ■

From the above theorem, we conclude that, for the bistability to occur, not only the risk-taking behaviour should depend on the treated class  $T$  but that its influence should be stronger than the risk-taking behaviour dependent on the infected and vaccinated classes.

In all of the special cases treated below, we will assume no-disease induced mortality ( $\alpha = 0$ ). This allows us to assume that the total population is at its demographic equilibrium,  $P(t) \rightarrow K$ , and effectively reduce the number of equations in the original model. Furthermore, the local stability for the DFE is only stated without proof since this is already shown for the general model (1) and we only concentrate on the existence and stability of the endemic states, whether bistability is present and its impact on increasing the recovery rate  $r$ .

### 3. The case of vaccination only

In this section we consider a disease without recovery and treatment ( $r = \delta = 0$ ) for the sole purpose of analysing the endemic state in the presence of vaccination only. While the goal here is to study the risk-taking behaviour effect tied up with vaccination only, several diseases do have vaccination available but no treatment such as *Poliomyelitis* or diseases for which the cost of treatment is very high and thus unavailable in certain communities (such as the cost of interferon therapy for *Hepatitis B*). We assume therefore that risk-taking behaviour is a reaction to a successful implementation of a vaccine and the model becomes

$$\begin{aligned} S' &= (1 - \xi)\beta P - f(I, V) \frac{SI}{P} - \bar{\mu}S - vS + \eta V, \\ I' &= f(I, V) \frac{SI}{P} - \bar{\mu}I, \\ V' &= \xi\beta P + vS - \bar{\mu}V - \eta V. \end{aligned} \tag{5}$$

Assuming  $P(t)$  reached its limit  $K$ , Equation (5) can be reduced to the following limiting planar system:

$$\begin{aligned} I' &= \frac{1}{K} f(I, V)(K - I - V)I - \beta I, \\ V' &= \xi\beta K + v(K - I - V) - \beta V - \eta V. \end{aligned} \tag{6}$$

The main result concerning (6) is proved in the following:

**THEOREM 3.1** *The behaviour of the solutions of Equation (6) is completely determined by the epidemic reproductive number  $\mathcal{R}_0 = \beta(1 - \xi) + \eta/\beta(\beta + v + \eta)f(0, K(\beta\xi + v)/(\beta + v + \eta))$ . The model always admits a Disease Free Equilibrium which is locally stable if  $\mathcal{R}_0 < 1$  and unstable otherwise. If  $\mathcal{R}_0 > 1$  there exists a unique endemic state that is locally asymptotically stable.*

*Proof* The Jacobian of this system is

$$\begin{bmatrix} \frac{1}{K} \frac{\partial f}{\partial I}(I, V)(K - I - V)I + \frac{1}{K} f(I, V)(K - 2I - V) \\ -\beta \frac{1}{K} \frac{\partial f}{\partial V}(I, V)(K - I - V)I - \frac{1}{K} f(I, V)I \\ -v - (\beta + v + \eta) \end{bmatrix}.$$

As shown in Section 2, the DFE  $(\bar{I}, \bar{V}) = (0, K(\beta\xi + v)/(\beta + v + \eta))$  is locally stable whenever  $\mathcal{R}_0 < 1$ . Any possible endemic equilibrium  $(I^*, V^*)$  must satisfy

$$h(V^*) := f\left(\frac{(\beta\xi + v)K - (\beta + v + \eta)V^*}{v}, V^*\right) [(\beta + \eta)V^* - \xi\beta K] - K\beta v = 0 \quad \text{and}$$

$$I^* = \frac{(\beta\xi + v)K - (\beta + v + \eta)V^*}{v}.$$

Furthermore,  $V^*$  must be in the biologically feasible interval  $(\beta\xi K/\beta + \eta, (\beta\xi + v)K/(\beta + v + \eta))$  (due to positivity conditions on  $I$  and  $K - I - V$ ). Notice that  $h(V)$  is an increasing function in  $V$  on this interval and  $h(\beta\xi K/(\beta + \eta)) < 0$ . Hence, the equation  $h(V) = 0$  has a unique root in the feasible interval if and only if

$$h\left(\frac{\beta\xi + v}{\beta + v + \eta}K\right) > 0.$$

A straightforward computation shows that this is equivalent to  $\mathcal{R}_0 > 1$ . Furthermore, the endemic equilibrium is stable whenever it exists since the trace and the determinant of the Jacobian evaluated at this point are

$$\text{trace} = \frac{\beta I^*}{f(I^*, V^*)} \frac{\partial f}{\partial I}(I^*, V^*) - \frac{1}{K} f(I^*, V^*) I^* - (\beta + v + \eta) < 0 \quad \text{and}$$

$$\det = \frac{\beta I^*}{f(I^*, V^*)} \left[ v \frac{\partial f}{\partial V}(I^*, V^*) - \frac{\partial f}{\partial I}(I^*, V^*) (\beta + v + \eta) \right] + \frac{\beta + \eta}{K} f(I^*, V^*) I^* > 0,$$

after we replaced  $K - I^* - V^*$  with  $\beta K/f(I^*, V^*)$ . ■

*Remark 2* As expected, from the remarks made on the previous section, in the absence of the treated/recovered class, the epidemic reproductive number  $\mathcal{R}_0$  is the only threshold that separates disease persistence from extinction and there is no bistability possible between the DFE and an endemic state.

#### 4. The case of treatment only

In this section we assume that there is no vaccination available ( $v = \xi = \eta = 0$ ). The model becomes

$$\begin{aligned} S' &= \beta P - f(I, T) \frac{SI}{P} - \bar{\mu} S, \\ I' &= f(I, T) \frac{SI}{P} - \bar{\mu} I - rI + \delta T, \\ T' &= rI - \bar{\mu} T - \delta T. \end{aligned} \tag{7}$$

which can be reduced to the following planar one:

$$\begin{aligned} I' &= \frac{1}{K} f(I, T)(K - I - T)I - \beta I - rI + \delta T, \\ T' &= rI - \beta T - \delta T. \end{aligned} \tag{8}$$

The following theorem provides the main result concerning (8):

**THEOREM 4.1** *The model (8) always admits a disease-free equilibrium which is locally asymptotically stable if  $\mathcal{R}_0 = (\beta + \delta/\beta(\beta + r + \delta))f(0, 0) < 1$  and unstable otherwise. The endemic equilibria alternate their stability whenever they exist in the following way:*

- (a) *from locally stable to unstable if the DFE is unstable,*
- (b) *from unstable to locally stable if the DFE is locally stable.*

Furthermore, a necessary condition for bistability is that the function  $f((\beta + \delta)/rT, T)$  is initially increasing in the domain of  $T$ .

*Proof* The first part of the theorem concerning the stability of the DFE is shown in Section 2. The Jacobian of Equation (8) is

$$\begin{bmatrix} \frac{1}{K} \frac{\partial f}{\partial I} f(I, T)(K - I - T)I + \frac{1}{K} f(I, T)(K - 2I - T) \\ -\beta - r - \frac{1}{K} \frac{\partial f}{\partial T} (K - I - T)I - \frac{1}{K} f(I, T)I + \delta \\ r - (\beta + \delta) \end{bmatrix}.$$

Concerning any possible endemic equilibrium we first establish its stability condition. We denote by  $(I^*, T^*)$  a typical endemic steady state where

$$\frac{1}{K} f(I^*, T^*)(K - I^* - T^*) = \frac{\beta(\beta + r + \delta)}{\beta + \delta} \quad \text{and} \quad I^* = \frac{(\beta + \delta)T^*}{r}.$$

Using the first equality, we can write the Jacobian evaluated at  $(I^*, T^*)$  as

$$\begin{bmatrix} \frac{\beta(\beta + r + \delta)}{\beta + \delta} \frac{I^*}{f(I^*, T^*)} \frac{\partial f}{\partial I}(I^*, T^*) - \frac{1}{K} f(I^*, T^*)I^* \\ -\frac{\delta r}{\beta + \delta} \frac{\beta(\beta + r + \delta)}{\beta + \delta} \frac{I^*}{f(I^*, T^*)} \frac{\partial f}{\partial T}(I^*, T^*) - \frac{1}{K} f(I^*, T^*)I^* + \delta \\ r - (\beta + \delta) \end{bmatrix}.$$

This matrix has a negative trace and its determinant is equal to

$$I^*(\beta + r + \delta) \left[ \frac{1}{K} f(I^*, T^*) - \frac{\beta}{f(I^*, T^*)} \left( \frac{r}{\beta + \delta} \frac{\partial f}{\partial T}(I^*, T^*) + \frac{\partial f}{\partial I}(I^*, T^*) \right) \right].$$

Hence any possible endemic state is stable provided that

$$f^2 \left( \frac{\beta + \delta}{r} T^*, T^* \right) > \beta K \left[ \frac{r}{\beta + \delta} \frac{\partial f}{\partial T} \left( \frac{\beta + \delta}{r} T^*, T^* \right) + \frac{\partial f}{\partial I} \left( \frac{\beta + \delta}{r} T^*, T^* \right) \right]. \quad (9)$$

Concerning the existence of endemic equilibria,  $T^*$  should satisfy the following equation:

$$h(T) := f \left( \frac{\beta + \delta}{r} T, T \right) [rK - (\beta + r + \delta)T] - \frac{r\beta K(\beta + r + \delta)}{\beta + \delta} = 0,$$

and belong to the biologically feasible interval  $(0, rK/(\beta + r + \delta))$ . This means that, for any possible solution  $T^*$ , we have

$$f \left( \frac{\beta + \delta}{r} T^*, T^* \right) = \frac{r\beta K(\beta + r + \delta)}{(\beta + \delta)[rK - (\beta + r + \delta)T^*]}. \quad (10)$$

In what follows, we assume that  $h(T)$  is never tangent to the horizontal axis, i.e.  $h(T^*) = 0$  and  $h'(T^*) = 0$ . This is because these possible fixed points are not hyperbolic (the Jacobian evaluated at them has one zero eigenvalue) and, while their stability can still be determined, from a practical standpoint they are unlikely since they require additional artificial conditions on the parameters that are not likely to occur in reality.

Notice that

$$h(0) = rKf(0, 0) - \frac{r\beta K(\beta + r + \delta)}{\beta + \delta} = rKf(0, 0) \left( 1 - \frac{1}{\mathcal{R}_0} \right) \quad \text{and} \quad h \left( \frac{rK}{\beta + r + \delta} \right) < 0.$$

Suppose now that the DFE is locally stable. This implies  $h(0) < 0$  and together with  $h(rK/(\beta + r + \delta)) < 0$  it means that there are either none, or at least two endemic steady states. On the other hand, if the DFE is unstable, i.e.  $h(0) > 0$ , then there is at least one endemic steady state. Given an equilibrium  $(I^*, T^*)$ , suppose that  $h'(T^*) > 0$ . This implies

$$\frac{\partial f}{\partial I} \left( \frac{\beta + \delta}{r} T^*, T^* \right) \frac{\beta + \delta}{r} + \frac{\partial f}{\partial T} \left( \frac{\beta + \delta}{r} T^*, T^* \right) > \frac{\beta + r + \delta}{rK - (\beta + r + \delta)T^*} f \left( \frac{\beta + \delta}{r} T^*, T^* \right).$$

Together with Equation (10) this implies

$$\frac{\partial f}{\partial I} \left( \frac{\beta + \delta}{r} T^*, T^* \right) \frac{\beta + \delta}{r} + \frac{\partial f}{\partial T} \left( \frac{\beta + \delta}{r} T^*, T^* \right) > \frac{\beta + \delta}{r\beta K} f^2 \left( \frac{\beta + \delta}{r} T^*, T^* \right),$$

and from Equation (9) we see that, in this case, the equilibrium is unstable. Similarly,  $h'(T^*) < 0$  implies the equilibrium is stable.

Hence, the stability of the multiple endemic equilibria will alternate from unstable to stable if the DFE is stable. If the DFE is unstable then the first crossing of  $h(T)$  with the  $T$ -axis will be at a point  $T^*$  with  $h'(T^*) < 0$  so, in that case, the stability of the multiple endemic states will alternate from stable to unstable. ■

*Remark 3* As mentioned in Section 2, an important feature of this model is that, in the presence of risk-taking behaviour, the epidemic reproductive number is no longer the only indicator of whether the disease persists or not. Because of the possibility of multiple equilibria and the bistability between DFE and an endemic state, the fate of the epidemic depends on the initial

value of the population in each class. Notice also that the stability condition of the endemic state, under the assumption that the DFE is stable, may never hold if the dependence on the infected class of the risk behaviour function is strong enough (i.e.  $\partial f/\partial I$  negative enough to compensate).

We now use the explicit form Equation (4) for the risk-taking function  $f$  and show that the bistability condition translates into a lower and an upper bound on the baseline infection rate (without the risk behaviour effect). Then we show that this range may actually extend in both directions by increasing  $r$ .

The risk-taking function  $f$  introduced in Equation (4) and adjusted for the treatment only model (8) is

$$f(I, T) = \lambda(1 - aI + \epsilon T).$$

Equation (10) becomes

$$\lambda \left[ 1 + T^* \left( \epsilon - \frac{a(\beta + \delta)}{r} \right) \right] = \frac{r\beta K(\beta + r + \delta)}{(\beta + \delta)[rK - (\beta + r + \delta)T^*]}.$$

Since the left term is linear in  $T^*$  then a necessary condition for the existence of two endemic equilibria is

$$\epsilon > \frac{a(\beta + \delta)}{r}, \tag{11}$$

and

$$\lambda < L_2 := \frac{\beta(\beta + r + \delta)}{\beta + \delta}.$$

The equation that solves for  $T$  can be written in the following quadratic form:

$$\begin{aligned} &(\beta + \delta + r) \left[ \epsilon - \frac{a(\beta + \delta)}{r} \right] T^2 + \left[ (\beta + r + \delta) - rK \left( \epsilon - \frac{a(\beta + \delta)}{r} \right) \right] T \\ &+ rK \left[ \frac{\beta(\beta + \delta + r)}{\lambda(\beta + \delta)} - 1 \right] = 0. \end{aligned} \tag{12}$$

The roots of this quadratic have positive real part provided that

$$\epsilon > E := \frac{a(\beta + \delta)}{r} + \frac{\beta + r + \delta}{rK}. \tag{13}$$

Notice that Equation (13) implies Equation (11) as well. Finally, the solutions are real provided that

$$\lambda > L_1 := \frac{4\beta K(\beta + \delta + r)^2[\epsilon r - a(\beta + \delta)]}{(\beta + \delta)[K(\epsilon r - a(\beta + \delta)) + (\beta + \delta + r)]^2}.$$

Notice also that the bounds on  $\lambda$  are consistent since  $L_2 > L_1$  is equivalent to

$$\frac{\beta(\beta + r + \delta)[K(\epsilon r - a(\beta + \delta)) - (\beta + \delta + r)]^2}{(\beta + \delta)[K(\epsilon r - a(\beta + \delta)) + (\beta + \delta + r)]^2} > 0.$$

We established that Equation (8) exhibits bistability between the DFE and an endemic state if and only if

$$\epsilon > E \quad \text{and} \quad L_1 < \lambda < L_2.$$

It is easy to see that the lower bound on  $\epsilon$  is not affected by an increase in  $r$  since  $dE/dr < 0$ . Furthermore,  $L_2$  is increasing in  $r$  which is expected since this bound is related to the local

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stability condition on the DFE, i.e.  $\mathcal{R}_0 < 1$ . We now proceed to establish the monotonicity of  $L_1$  with respect to  $r$ . A straightforward computation shows that  $dL_1/dr$  has the sign of the following quadratic in  $r$ :

$$B(r) := \epsilon(1 + \epsilon K)r^2 - \epsilon(\beta + \delta)[K(\epsilon + 3a) - 2]r + (\beta + \delta)^2[\epsilon(1 + aK) + 2a^2K].$$

The roots of  $B(r)$  are

$$r_{1,2} = \frac{\beta + \delta}{2\epsilon(1 + \epsilon K)} [\epsilon K(\epsilon + 3a) - 2\epsilon \pm (\epsilon + a)\sqrt{\epsilon K(\epsilon K - 8)}].$$

$B(r)$  is negative (and therefore  $L_1$  decreases) provided that  $r_1, r_2$  are real, positive and  $r_1 < r < r_2$ . This is equivalent to

$$\epsilon > \frac{8}{K}.$$

We still need to verify whether the treatment interval  $(r_1, r_2)$  is within the constraint imposed by Equation (13). Notice that  $\epsilon > E$  can be written as

$$r > r^* := \frac{(\beta + \delta)(1 + aK)}{\epsilon K - 1}.$$

Furthermore,

$$B(r^*) = \frac{2K(\beta + \delta)^2(\epsilon + a)^2}{(\epsilon K - 1)^2} > 0 \quad \text{and} \quad B'(r^*) = -\frac{\epsilon K(\beta + \delta)(\epsilon + a)(\epsilon K - 5)}{\epsilon K - 1} < 0.$$

This shows that

$$r^* < r_1 < r_2.$$

To summarize, the bistability range of the infection rate  $\lambda$  increases with the treatment rate  $r$  provided that

$$\epsilon > \max \left\{ E, \frac{8}{K} \right\} \quad \text{and} \quad r_1 < r < r_2.$$

In the examples provided in Appendix 2, we will consider the situation when  $\lambda$  is just outside to the left of this range. This corresponds to a stable DFE and no endemic state. Then, by increasing  $r$  within the corresponding critical interval  $(r_1, r_2)$  we will observe how  $L_1$  decreases and  $\lambda$  falls within the bistability range,  $L_1 < \lambda < L_2$ , and the disease may become endemic depending on the initial population size.

## 5. Conclusions

We analysed a model of a typical infectious disease where individuals increase their infection risk as a response to a perceived lower disease burden measured by the knowledge about the size of the infected, recovered and vaccinated classes. Our main result shows that under several conditions on the parameters, an increase in the effectiveness of a vaccine or treatment may be detrimental with respect to the chance of the disease to become endemic. A possible interpretation of our results can be summarized as follows: if enough risk-taking response with respect to the recovered or vaccinated class is present then

- it may be better to not vaccinate at all if we cannot vaccinate above a certain threshold;
- increases of the treatment rate from 0 to low positive values and from a higher threshold and above are beneficial, but in between two thresholds  $r_1 < r_2$  may be detrimental.

Another important consideration is that, for large populations, it takes a relatively small risk-taking behaviour for the detrimental effects to take place. For example, the lower bounds imposed on the risk-taking components are small if  $K$ , the carrying capacity, is large.

There are several important limitations of these results. First, there is no precisely defined function  $f(I, T, V)$  that models risk-taking behaviour and the explicit form we used should be interpreted as a linear approximation of a (possible) more complex function. Furthermore, there is no easy way of collecting data on how individuals respond when having a certain information about the disease prevalence. Even more important, from the perspective of our results, it is also difficult to differentiate the risk-taking response with respect to a specific class of individuals and we intend to pursue more research on addressing these questions. At the very least, to measure the willingness of a community to engage in risky behaviour, one can design a sociological experiment with a carefully designed survey. It is encouraging that such experiments are possible and were done already. For example, in [4], Andersson *et al.* measure the anticipated decrease in condom use (i.e. risk-taking) as a response to a hypothetical low-efficiency HIV vaccine. In Adler *et al.* [2] an Internet survey is used to assess the incidence and risk-factors associated with Influenza-like-illnesses. In this study a slightly higher incidence risk was measured for the female cohort which suggests that it may be worthwhile to study epidemic models that involve both risk behaviour and gender structure.

Another limitation (which is evident from the example in Appendix 2) is that, for certain diseases, the critical interval of the treatment rate where the bistability range of  $\lambda$  increases with  $r$  can be rather narrow and over values which may never be taken by the relevant parameters.

We conclude that, in addition to a continued focus on risk taking in response to changes in disease epidemiology, it is important to examine and model how prevention tools affect and are used by individuals in the general public. This approach would add to the current knowledge as much of the available literature is derived from participants in clinical trials where conditions likely do not match real-world settings. As such, other avenues of future research would be to analyse the problems of partial protection and inadequate adherence to medical regimens when examining biomedical forms of HIV prevention (see also [1,5,15,24]).

Finally, another point of interest for us, especially in light of the emergence of drug resistant infections, is to study mathematically whether the evolution towards resistant strains is significantly influenced by behavioural changes in the exposed population. We expect to pursue these avenues of research in the near future.

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## Appendix 1. Example of the negative effect of increasing vaccination rates

To illustrate the possible detrimental effect of increasing vaccination rates, we use some parameter values related to Tuberculosis (TB), a disease for which both treatment and vaccination exist. However, the vaccine (*Bacillus Calmette-Gurin* or BCG), varies widely in its application and effectiveness across the world. For a comprehensive study on TB prevention and treatment practices in various countries, see Gerberry *et al.* [14]. Using demographic data from USA in 2013 from the Center of Disease Control and Prevention (CDC, <http://www.cdc.gov/>) we approximate the birth and the death rates:  $\beta = 0.01244$  and  $\mu = 0.00822$ . We adjusted the logistic term in the natural mortality such that the carrying capacity is more or less close to the US population in 2013 which is 316 million. This is an overestimation of the logistic effect but we preferred it because most of the proofs in our paper assume the population is at its demographic equilibrium. In fact, a higher carrying capacity  $K$  will impose even lower bounds on the risk-taking components of  $f$  and, as one can see from the conditions imposed on the partial derivatives of  $f$  and the values chosen in this example, the higher the total population is, the lower is the risk-taking necessary to cause the detrimental effect.

From [14] we use the following TB specific parameters: contact rate  $\lambda = 12$ , disease mortality  $\alpha = 0.12$ , waning effectiveness of the vaccine  $\eta = 0.02$ . While the average duration of treatment is between 6 and 12 months the patient is non-infectious after about 4 weeks which can be approximated in our model by a high recovery rate  $r = 13.004$ .

In the case of either vaccination at birth only ( $v = 0$ ) or adult vaccination only ( $\xi = 0$ ), conditions (2) and (3) become

$$\epsilon_2 > \frac{1}{K} \approx 3.16 \times 10^{-9}.$$

Recall that we need  $a < 1/K$  as well, in order to prevent  $f$  from ever becoming negative. With these restrictions, we consider the following values for the risk-behaviour coefficients:  $a = 3 \times 10^{-9}$ ,  $\epsilon_1 = 2 \times 10^{-9}$  and  $\epsilon_2 = 9 \times 10^{-9}$ .

In Figure A1 we show an example in which  $\mathcal{R}_0 < 1$  in the absence of vaccination. Then, we show the effect of increasing the fraction of vaccinated individuals at birth. Assuming no adult vaccination  $v = 0$ , the critical value is  $\xi^* = 0.86$  up to which  $\mathcal{R}_0$  increases. We then increase  $\xi$  it to  $\xi = 0.5$  and show the disease becomes endemic in Figure A2. In this particular case  $\mathcal{R}_0$  never reaches a value less than 1 even with 100% vaccination at birth ( $\xi = 1$ ) as seen in Figure A3.

A similar example is provided concerning the effect of increasing the adult vaccination rate  $v$ . Assuming no vaccination at birth ( $\xi = 0$ ), and all other parameters the same as before, the critical vaccination rate is  $v^* = 0.016$  up to which  $\mathcal{R}_0$  increases. Increasing vaccination to  $v = 0.015$  we see in Figure A4 how the disease become endemic. Finally, in Figure A5, we increase vaccination beyond the critical value to  $v = 0.06$  and the DFE becomes stable again.

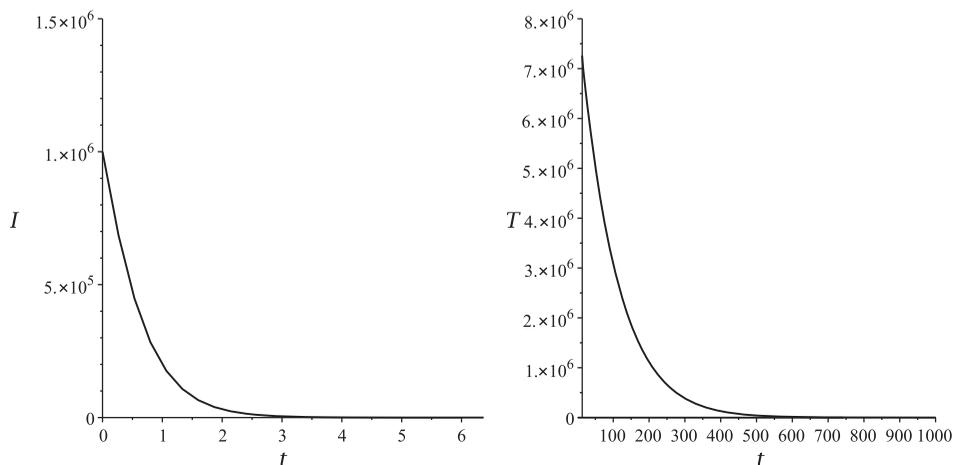


Figure A1. Disease clearance in the absence of vaccination ( $v = 0$ ,  $\xi = 0$ ,  $\mathcal{R}_0 = 0.913$ ).

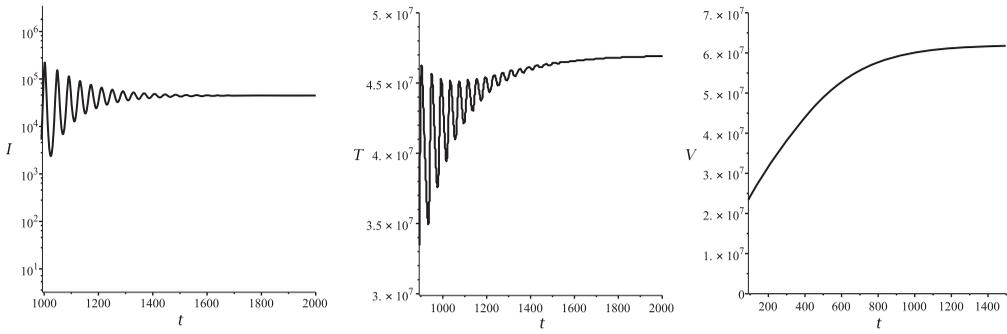


Figure A2.  $\xi = 0.5, \mathcal{R}_0 = 1.152$ .

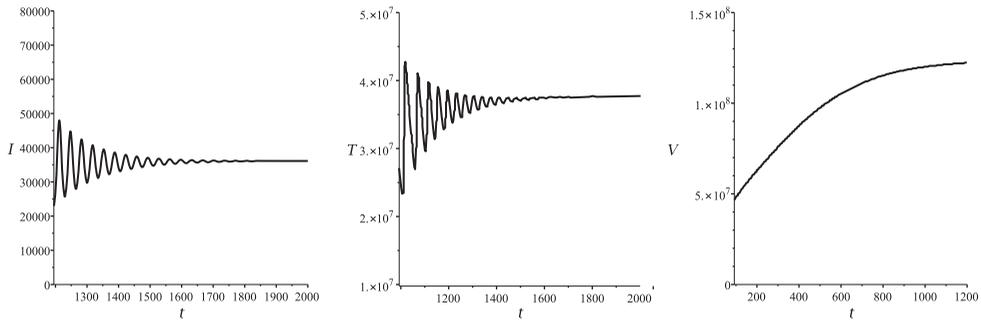


Figure A3.  $\xi = 1.0, \mathcal{R}_0 = 1.19$  (less than the maximum 1.2 attained at  $\xi = \xi^*$  but still greater than 1).

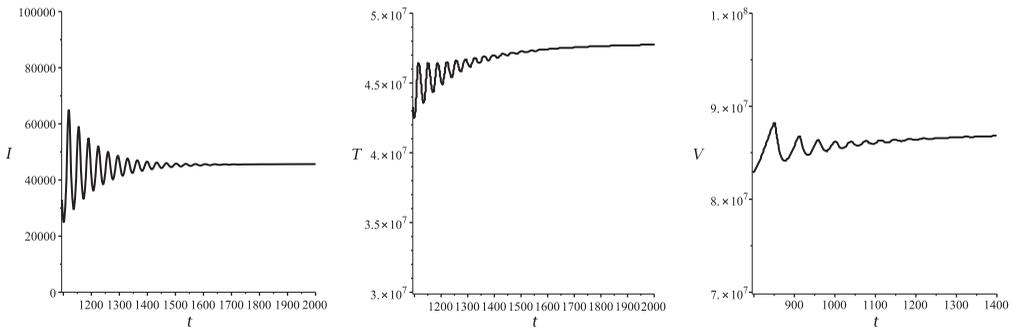


Figure A4.  $\nu = 0.015, \mathcal{R}_0 = 1.202$ .

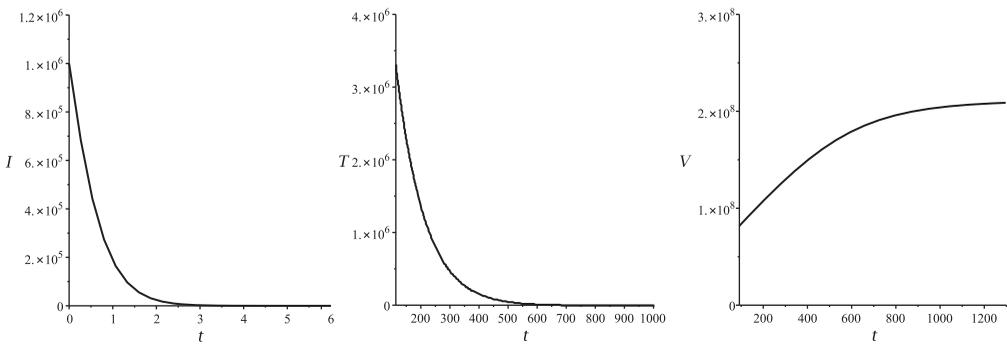


Figure A5.  $\nu = 0.06, \mathcal{R}_0 = 0.928$ .

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## Appendix 2. Example of the negative effect of increasing the recovery rate

We provide an example related to the simplified model (8). We will use the same demographic parameters as in the previous appendix:  $\beta = 0.01244$ ,  $\mu = 0.00822$ . We will adjust some parameters in order to illustrate, as in the vaccination case, the possibility of an endemic state caused by increasing the treatment rate:  $a = 2 \times 10^{-9}$ ,  $\epsilon_1 = \epsilon_2 = \epsilon = 3 \times 10^{-8}$ . With these values, the bistability range for  $\lambda$  is  $(0.0153, 0.016)$  and the critical recovery values between which this range increases with  $r$  are  $r_1 = 0.0043$  and  $r_2 = 0.0092$ . In Figure A6 we show how the disease is eliminated with an initial recovery rate  $r = 0.0044$  inside the critical interval and with a value of  $\lambda$  just outside the bistability range. In Figure A7 we increase the recovery to  $r = 0.008$  keeping the same initial conditions. This extends the bistability interval and  $\lambda$  falls into it which causes the disease to become endemic for the same initial conditions. However, since now we are in the bistability regimen, low enough initial values for  $I$  will cause the disease to be eliminated as seen in Figure A8.

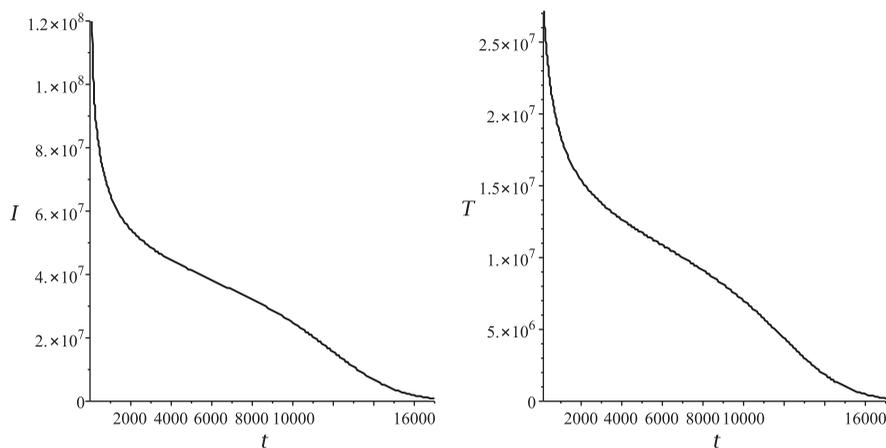


Figure A6. Simplified model (8).  $r = 0.0044$ ,  $\lambda = 0.0152$ ,  $\lambda$ -bistability range  $(0.0153, 0.016)$ ,  $\mathcal{R}_0 = 0.94996$ .

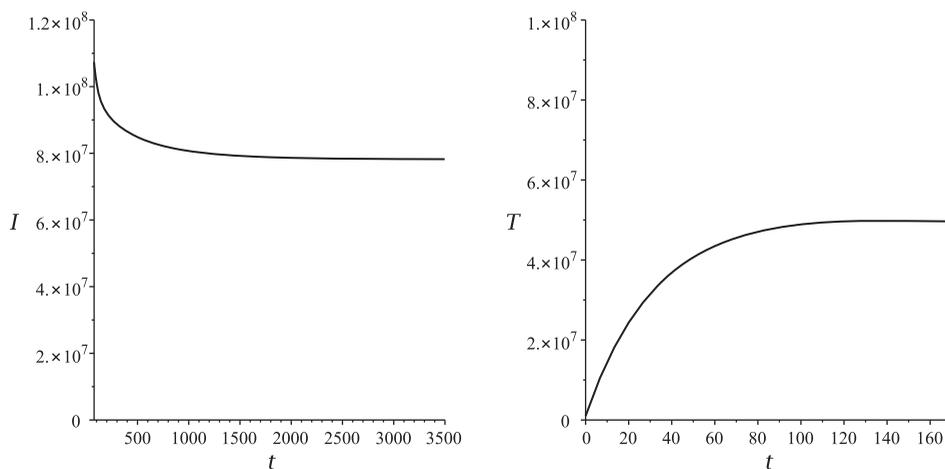


Figure A7. Simplified model (8).  $r = 0.008$ ,  $\lambda = 0.0152$ ,  $\lambda$ -bistability range  $(0.01497, 0.0189)$ ,  $\mathcal{R}_0 = 0.804$ .

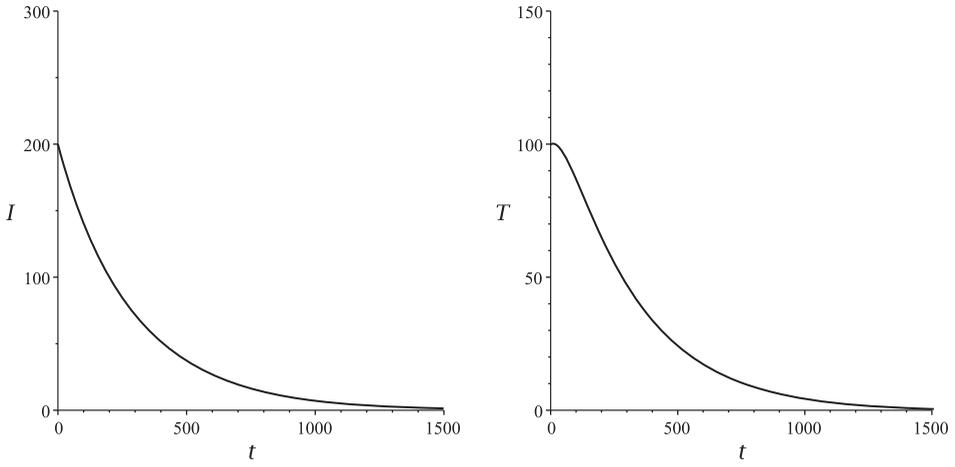


Figure A8. Simplified model (8).  $r = 0.008$ ,  $\lambda = 0.0152$ ,  $\lambda$ -bistability range  $(0.01497, 0.0189)$ ,  $\mathcal{R}_0 = 0.804$ . Remark: initial population size within the basin of attraction of the DFE.