THE EFFECT OF RISK TAKING BEHAVIOR INDUCED BY RECOVERY IN EPIDEMIC MODELS

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Abstract. We study several epidemic models that incorporate risk taking behavior as a response to an effective treatment or vaccine. We assume that knowledge about the number of recovered individuals has an effect in increasing the contact rate between susceptible and infectious individuals. We show that a relatively mild risk-taking behavior response changes the fate of an epidemic from disease clearance to disease persistence. Moreover, under certain conditions on the parameters, increasing the efficiency of treatment or vaccine has a counter-intuitive and unwanted effect of increasing the 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 a dramatic influence on how the epidemic evolves.

1. Introduction

Individuals facing an infectious disease, consciously or not, evaluate the contamination risk and engage or avoid risky situations or behaviors. The risk taking behavior of any individual is probably a very complex functional response that depends of many factors such as: morbidity of the disease, infectiousness, mode of transmission, existence and efficacy of treatment, etc. There are multiple studies that show individuals have dynamic responses to transmission risks that change with varying circumstances. For example, the advent of antibiotics makes all of us less concerned with exposure risks to common pathogens. Nobody is worried with getting tick bites in the woods since a course of penicillin would cure a possible exposure to Lyme disease. To the contrary, knowledge about the severity of disease (measured by number of infectious people, virrulence, lack of effective treatment) may cause susceptible individuals to be more cautious and reduce their exposure risk. From these remarks we can talk about two general ways in which behavioral attitudes influence the spread of a disease:

(i) Increasing risk-taking behavior as a response to "good news",
(ii) Decreasing risk-taking behavior as a response to "bad news"

The second case has been addressed by Del Valle et. al. in [2]. 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 shows that even small behavioral changes play a big role in counteracting the epidemic. Thus their model follows the framework in which the behavior is always positive from the perspective of individuals (i.e. reduction of risk behavior only). A general dependency of the contact rate of various types of individuals has been proposed by Chavez et. al. in [1, 2, 3]. This article is not focused on modeling risk-taking behavior but an example is proposed there in which the contact rate is assumed to depend on all types of people involved in the model (susceptible, infected, recovered) to illustrate how the population adjusts their behavior in response to knowledge about the epidemic status of the population. However, the purpose of the model was to illustrate 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

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risk-taking behavior in the evolution of the epidemics. Several other studies model the behavioral 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. [3]. Again, here the focus is on how sensitive the epidemic is with respect to behavioral changes.

In our paper we focus primarily on case (1) in which the contact rate increases as a response to knowledge about the size of the recovered class. A high percentage of recovered people signals that the treatment/vaccine was effective and either the worst of the epidemic has passed or that it is not that serious (easy recovery). Therefore, our contact rate will be modeled by an increasing function of the recovered individuals. Furthermore, we will assume that in the absence of risk taking behavior the fate is always positive, i.e. the disease will clear from the population and ask under what conditions this fate is reversed and the epidemic becomes endemic due to risk-taking behavior alone. Furthermore we will analyze the effect of increasing the treatment/vaccine efficiency in the presence of risk taking behavior and show that under certain conditions these measures, usually positive, have actually a detrimental effect in which the epidemic reproductive number is increased.

2. The basic SIR model

Let us first consider the basic SIR model:

$$\begin{cases} S' = \beta P - \lambda SI - \overline{\mu} S \\ I' = \lambda SI - \overline{\mu} I - rI \\ R' = rI - \overline{\mu} R \end{cases}$$

Here, S represents the number of susceptible (healthy) individuals, I represents the amount of people in the infected class, and R represents the number of people who have recovered from the infection. The rest are constants:

Variable	Meaning
β	birth rate
λ	interaction/infection rate
$\overline{\mu}$	logistic death rate
r	recovery rate

There are a couple of aspects of this model which need to be addressed. It is important to mention that $\overline{\mu} = \mu + bP, b \in \mathbb{R}^+$, where P is the total population. This means that the death rate of the population increases as the population itself increases. Also note, λSI denotes the number of new infectious individuals. This comes from the following:

$$\lambda P \left(\frac{S}{P} \right) I = \lambda SI$$

Here, λP denotes the amount of people that the average infected interacts with, $\frac{S}{P}$ represents the probability that an infected interacts with a susceptible in the population, and I represents the amount of infected in the model.

3. The SIR model with risk taking behavior dependent on treatment

Risk-taking behavior is characterized as the totality of measures an individual takes to avoid an infection. This depends on the virulence (severity of symptoms) of the disease and the treatments that are either

available or efficient. This characteristic of human behavior will be denoted using the variable ε. Considering the risk-taking behavior of humans into the equation results in an adjusted SIR model.

$$\begin{cases} S' = \beta P - \lambda(1+\varepsilon R)SI - \overline{\mu}S \\ I' = \lambda(1+\varepsilon R)SI - \overline{\mu}I - rI \\ R' = rI - \overline{\mu}R \end{cases}$$

The expression $1+\varepsilon R$ is added for a variety of reasons. It denotes the fact that humans are more likely to partake in risk-taking behavior whenever there are more recovered individuals in the population (in this case due to treatment), so the relationship εR should be present. However, if this is merely multiplied to λ (so that the new term is $\lambda\varepsilon R$), then as $R \to 0, \lambda\varepsilon R \to 0$. Biologically, this can be interpreted to mean that humans will quarantine themselves whenever there are no recovered individuals. There will be no new infections, and this is, in general, not realistic. The "*1 +*" is added in order to ensure that as $R \to 0$, there will still be new infections (as $R \to 0, \lambda(1+\varepsilon R) \to \lambda$). In addition, when risk-taking behavior is ignored ($\varepsilon = 0$), we should revert to the basic model. We use this linear equation because it meets all of the above criteria, and in addition, linear equations are also the easiest to work with.

3.1. **Equilibrium Points.** The total population P is just the summation of $S+I+R$. The population also changes over time, and can be modeled by the following differential equation:

$$P' = S' + I' + R' = (\beta - \overline{\mu})P = (\beta - \mu - bP)P$$

So P follows a logistic model with carrying capacity K as $t \to \infty$. In the context of this model, $K = \frac{\beta-\mu}{b}$. As $t \to \infty$, $P = K = \frac{\beta-\mu}{b}$. This relationship can be used to show that $\overline{\mu} = \mu + bP \to \mu + b\left(\frac{\beta-\mu}{b}\right) = \beta$. So as $t \to \infty$, $\overline{\mu} \to \beta$. Looking at the population as $t \to \infty$, $S+I+R = K$. In other words, $S = K - I - R$. We can use this latter equality in order to reduce the system from three variables to two. Now we have the following system:

$$\begin{cases} I' = \lambda(1+\varepsilon R)(K-I-R)I - \beta I - rI \\ R' = rI - \beta R \end{cases}$$

The Jacobian matrix for this system is as follows:

$$J = \begin{bmatrix} -\lambda(1+\varepsilon R)I + \lambda(1+\varepsilon R)(K-I-R) - \beta - r & ; & I[\lambda\varepsilon(K-I-R) - \lambda(1+\varepsilon R)] \\ r & ; & -\beta \end{bmatrix}$$

Next, we will set both I' and R' equal to 0. I^* and R^* will be used to denote the infected and recovered population, respectively, at the time of equilibrium. Looking at $R' = rI - \beta R$:

$$\begin{aligned} I^* &= \frac{\beta R^*}{r} \\ R^* &= \frac{rI^*}{\beta} \end{aligned}$$

We will later use the above relationships with respect to the Jacobian matrix. Now looking at $I' = \lambda(1+\varepsilon R^*)(K-I^*-R^*)I^* - \beta I^* - rI^*$:

$$0 = I^*[\lambda(1+\varepsilon R^*)(K-I^*-R^*) - \beta - r]$$

Thus, either $I^* = 0$ or $\lambda(1+\varepsilon R^*)(K-I^*-R^*) - \beta - r = 0$. We can see that our first equilibrium point is $(I^*, R^*) = (0,0)$. This comes from the fact that if $I^* = 0$ (as in our first case), then $R^* = \frac{rI^*}{\beta} = \frac{r*0}{\beta} = 0$. Now let us consider the case where $\lambda(1+\varepsilon R^*)(K-I^*-R^*) - \beta - r = 0$. Using the substitution for I^*

and expanding out the expression, we get:

$$(\lambda\varepsilon\beta + \lambda\varepsilon r)R^2 + (\lambda\beta + \lambda r - \lambda\varepsilon K r)R - \lambda K r + \beta r + r^2 = 0$$

Using the quadratic equation, we get that:

$$R = \frac{\lambda(-1 + \varepsilon K)r - \beta)) \pm \sqrt{[(1 + \varepsilon K)^2\lambda - 4\varepsilon r(\beta + r)^2]\lambda}}{2\lambda\varepsilon(\beta + r)}$$

3.2. **Analyzing the Equilibrium Points.**

3.2.1. *Disease-Free Equilibrium Point.* Substituting in the point (0, 0) for (I, R), we get that the Jacobian matrix now looks like:

$$J(0,0) = \begin{bmatrix} \lambda K - \beta - r & ; & 0 \\ r & ; & -\beta \end{bmatrix}$$

In order for this equilibrium point to be stable, both of the eigenvalues have to be negative and real. Since the Jacobian is a lower triangular matrix in this case, the two eigenvalues are along the diagonal. Thus, $\lambda K - \beta - r < 0$ and $-\beta < 0$. The second condition is always true by the definition of β. Therefore, the only condition that must be satisfied in order for the system to be stable is that $\lambda K - \beta - r < 0$, or $\frac{\lambda K}{\beta + r} < 1$. So $\mathcal{R}_0 = \frac{\lambda K}{\beta + r} < 1$.

3.2.2. *Endemic Equilibrium Point.* Now that we have looked at the disease free equilibrium point, let us consider what happened in the second instance, the one with the quadratic. We want R to be real, so this can only happen if the following condition is met:

$$\lambda > \frac{4\varepsilon r(\beta + r)^2}{[(1 + \varepsilon K)r + \beta]^2}$$

Thus, we can make the assumption that if λ is less than the above value, R is not a real value, so there only exists one equilibrium point, which is the DFE point. Using the Vieta relations for a quadratic, we notice that:

$$\begin{aligned} -\frac{b}{a} &= \frac{-\beta - r + \varepsilon K r}{\varepsilon(\beta + r)} \\ \frac{c}{a} &= \frac{-\lambda K r + \beta r + r^2}{\lambda\varepsilon\beta + \lambda\varepsilon r} \end{aligned}$$

So if we have two solutions, denoted R_1 and R_2 (such that $R_2 > R_1$), then $R_1 + R_2 = \frac{-\beta - r + \varepsilon K r}{\varepsilon(\beta + r)}$ and $R_1 R_2 = \frac{-\lambda K r + \beta r + r^2}{\lambda\varepsilon\beta + \lambda\varepsilon r}$. It is important to note that we are only concerned with the sign of these values. In both the above equations, the denominator is always positive, so now we can concern ourselves with the numerator. Looking at the numerator, we can propose the following conditions:

If	Then
$\lambda < \frac{r+\beta}{K}$	$R_1 R_2 > 0$
$\lambda > \frac{r+\beta}{K}$	$R_1 R_2 < 0$
$r\varepsilon > \frac{\beta + r}{K}$	$R_1 + R_2 > 0$
$r\varepsilon < \frac{\beta + r}{K}$	$R_1 + R_2 < 0$

Let us first consider the case where $\lambda > \frac{r+\beta}{K}$. If we manipulate this inequality, we get that:

$$1 < \frac{\lambda K}{\beta + r}$$

However, this inequality is in direct contradiction to the condition needed for the stable DFE point. So if $\lambda > \frac{r+\beta}{K}$, then the DFE point is unstable. Since $R_1, R_2 \in \mathbb{R}$, and $R_2 > R_1$, then $R_1 \in \mathbb{R}^-$. Thus, if $R_1 R_2 < 0$, then $\exists$ an equilibrium point at R_2 and the DFE point is unstable.

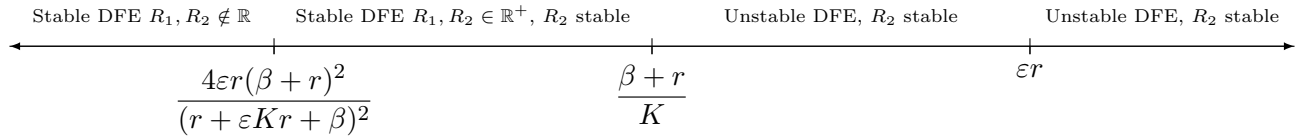
Now let us consider the case where $\lambda < \frac{r+\beta}{K}$. So $R_1 R_2 > 0$. Since, $R_1, R_2 \in \mathbb{R}$, either $R_1, R_2 \in \mathbb{R}^+$ or $R_1, R_2 \in \mathbb{R}^-$. The sign of R_1 and R_2 depends on $r\varepsilon$ and $\frac{\beta+r}{K}$. If $r\varepsilon < \frac{\beta+r}{K}$, then $R_1 + R_2 < 0$, so $R_1, R_2 \in \mathbb{R}^-$. But if $r\varepsilon > \frac{\beta+r}{K}$, then $R_1 + R_2 > 0$, so $R_1, R_2 \in \mathbb{R}^+$. Thus, if $r\varepsilon < \frac{\beta+r}{K}$, then there is only one equilibrium point which makes sense biologically, which is the DFE point. But if $r\varepsilon > \frac{\beta+r}{K}$, then there are three equilibrium points to examine: the DFE, R_1, and R_2.

3.3. **Conditions.** So if we combine the above, we can see that there are four scenarios:

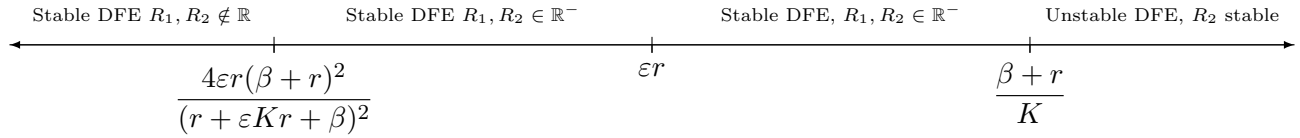
Condition	DFE	Other Equilibria
$\lambda > \frac{\beta + r}{K}$	unstable	$R_1 \in \mathbb{R}^-, R_2 \in \mathbb{R}^+$, R_2 - stable
$\frac{4\varepsilon r(\beta + r)^2}{(r + \varepsilon K r + \beta)^2} < \lambda < \frac{\beta + r}{K}, r\varepsilon > \frac{\beta + r}{K}$	stable	$R_1, R_2 \in \mathbb{R}^+$, R_1 - unstable, R_2 - stable
$\frac{4\varepsilon r(\beta + r)^2}{(r + \varepsilon K r + \beta)^2} < \lambda < \frac{\beta + r}{K}, r\varepsilon < \frac{\beta + r}{K}$	stable	$R_1, R_2 \in \mathbb{R}^-$, no other equilibria other than DFE
$\lambda < \frac{4\varepsilon r(\beta + r)^2}{(r + \varepsilon K r + \beta)^2}$	stable	$R_1, R_2 \notin \mathbb{R}$, no other equilibria other than DFE

The following are another visual representation of the above data in the form of number lines:

Case 1: $\varepsilon r > \frac{\beta + r}{K}$

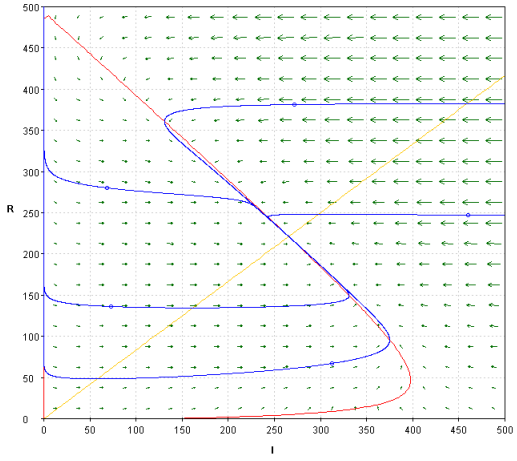


Case 2: $\varepsilon r < \frac{\beta + r}{K}$



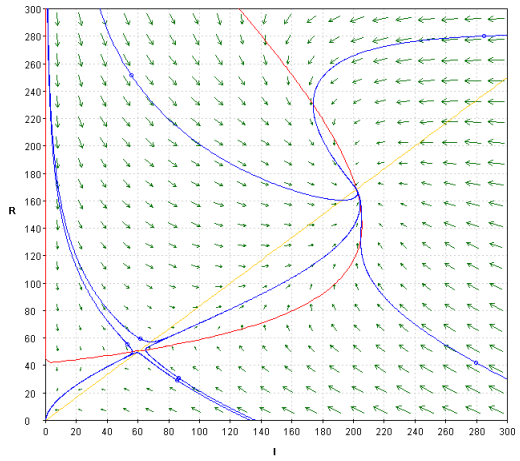
3.4. **Examples.** We provide several examples to illustrate the cases established above.

3.4.1. *Condition 1.* $\lambda > \dfrac{\beta + r}{K}$
$\lambda = 0.0015$, $\varepsilon = 0.12$, $r = 0.25$, $K = 500$, $\beta = 0.3$ (implies $\mu = 0.1$, $b = 0.0004$)

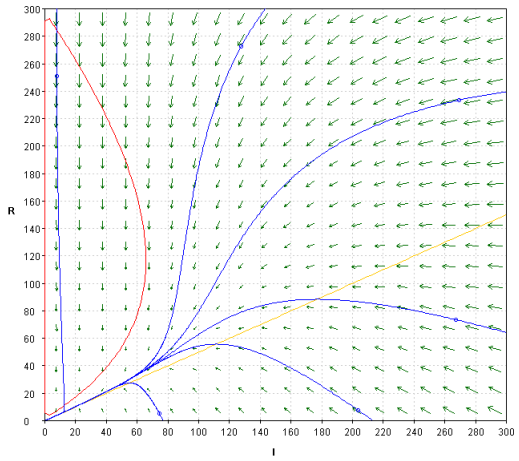


Here, we can see that there is a single stable equilibrium point located in the middle of the graph. Our disease free equilibrium point is unstable, and any initial condition leads to an epidemic. All initial conditions eventually meet at the intersection of the nullclines of I' and R' in the first quadrant, also denoted as R_2.

3.4.2. *Condition 2.* $\dfrac{4\varepsilon r(\beta + r)^2}{(r + \varepsilon K r + \beta)^2} < \lambda < \dfrac{\beta + r}{K}, r\varepsilon > \dfrac{\beta + r}{K}$
$\lambda = 0.0002$, $\varepsilon = 0.12$, $r = 0.25$, $K = 500$, $\beta = 0.3$ (implies $\mu = 0.1$, $b = 0.0004$)

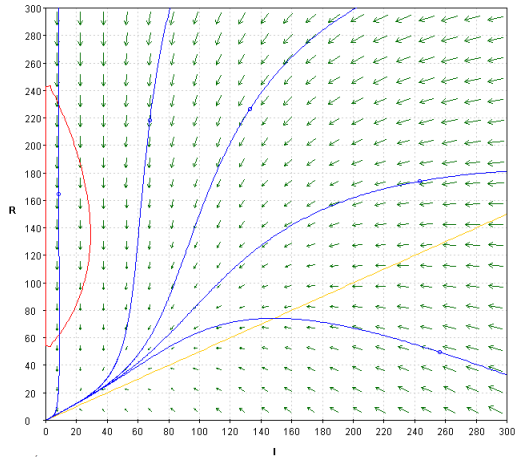


This case is the most interesting one, and it is the one that we shall examine further. There are several important features to notice. First, there are two stable equilibrium points: one at the disease free state, and one at R_2. There appears to be a certain arc along the graph which determines the stable equilibrium point that an initial condition will approach. Interestingly enough, if we look closely enough, we can see that any point close to this arc will approach the unstable equilibrium point, R_1, before approaching its end behavior. We will make the conjecture that the radius of the stable endemic point passes through the unstable equilibrium point, but we will discuss this later if necessary.



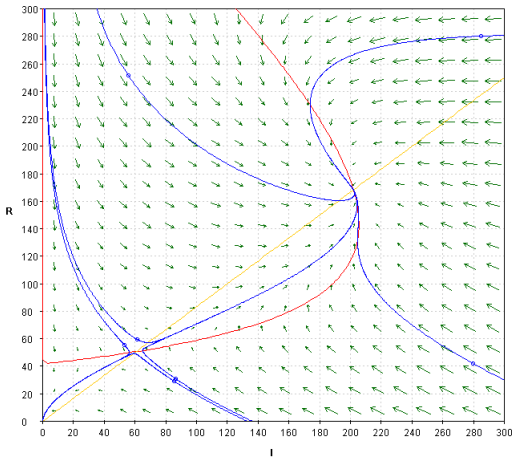
3.4.3. *Condition 3.* $\dfrac{4\varepsilon r(\beta+r)^2}{(r+\varepsilon Kr+\beta)^2} < \lambda < \dfrac{\beta+r}{K}, r\varepsilon < \dfrac{\beta+r}{K}$
$\lambda = 0.000895$, $\varepsilon = 0.005$, $r = 0.15$, $K = 500$, $\beta = 0.3$ (implies $\mu = 0.1$, $b = 0.0004$)
This is the case where there are two negative real intersections of the nullclines. Thus, the only equilibrium point that matters biologically is the disease free equilibrium point, which is stable.

3.4.4. *Condition 4.* $\lambda < \dfrac{4\varepsilon r(\beta+r)^2}{(r+\varepsilon Kr+\beta)^2}$
$\lambda = 0.0008$, $\varepsilon = 0.12$, $r = 0.25$, $K = 500$, $\beta = 0.3$ (implies $\mu = 0.1$, $b = 0.0004$)

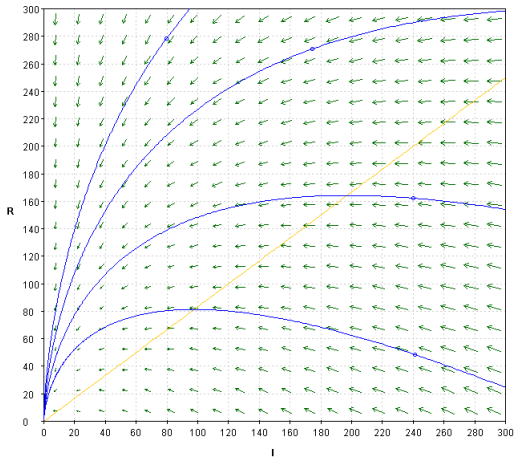


This case is very similar to Condition 3. The biological interpretation is the same; there is only one equilibrium point we can examine, which is the DFE. This occurs because there is no real intersection of the nullclines; it only occurs on the complex plane.

3.5. **Condition 2 Revisited.** The case that we are most interested in examining is the second one, which can also be referred to as the bistability condition. For the other three conditions, there is a set equilibrium point that will be the end result and it is independent of the initial condition. However, with the second condition, we can see that there are two possible equilibria. Another important factor of this model is that the risk-taking factor is present ($\varepsilon \neq 0$). But let us examine what takes place whenever the risk factor is removed.
In this figure, we see an important difference. The only parameter that was changed from the second condition is that $\varepsilon = 0$. In the figure on the left, the disease can become an epidemic based on the initial condition, but in the figure on the right, all initial conditions lead to a disease free equilibrium. Thus, a relatively low lever of risk-taking behavior leads to a big difference in the outcome of the disease.



(A) $\varepsilon \neq 0$



(B) $\varepsilon = 0$

3.6. **The range of the infection rate as a function of ε.** If we look back at the table in Section 2.3, we can see that $\frac{4\varepsilon r(\beta+r)^2}{(r+\varepsilon Kr+\beta)^2}$ is the lower bound for λ in order to have two real equilibrium points. By taking the derivative with respect to ε, setting it equal to zero, and solving for ε, we can look at the span of λ for which the bistability regimen is maintained. Denoting the lower bound as a function of ε,

$$f(\varepsilon) = \frac{4\varepsilon r(\beta + r)^2}{(r + \varepsilon Kr + \beta)^2}$$

we get that $\varepsilon = \frac{\beta+r}{Kr}$, or equivalently, $\varepsilon r = \frac{\beta+r}{K}$ is a critical value. We can use a sign chart to establish which direction the lower bound for λ will change based off of ε. If we do this, we can find that the slope of the derivative is negative $\forall\ \varepsilon > \frac{\beta+r}{Kr}$. This is the only part of the chart that matters, as we start with the assumption that $\varepsilon r > \frac{\beta+r}{K}$ (as in Condition 2). So the range of λ increases as ε increases, which is an expected result. Biologically, this can be interpreted to mean that the infection rate has a larger variance which can result in an epidemic as humans engage in more risk-taking behavior.

3.7. **The range of the infection rate as a function of r.** We are now going to treat the lower bound of λ as a function of r:

$$f(r) = \frac{4\varepsilon r(\beta + r)^2}{(r + \varepsilon Kr + \beta)^2}$$

If we differentiate this equation with respect to r, we get the following:

$$f'(r) = \frac{4\left((1 + \varepsilon K)r^2 + \beta(2 - \varepsilon K)r + \beta^2\right)(\beta + r)\varepsilon}{(r + r\varepsilon K + \beta)^3}$$

Notice that the sign of the derivative matters only on the value of the first parenthesis: $(1 + \varepsilon K)r^2 + \beta(2 - \varepsilon K)r + \beta^2$which is a quadratic in r. The roots of this quadractic are:

$$r_{12} = \frac{1}{2}\ \frac{\left(-2 + \varepsilon K \pm \sqrt{-8\varepsilon K + \varepsilon^2 K^2}\right)\beta}{1 + \varepsilon K}$$

First notice that $r_2 > r_1$. We want $r_1, r_2 \in \mathbb{R}^+$, so let us now look at the conditions which make it so. The radicand cannot be a negative real number, so let us look at this condition. Thus, $-8\varepsilon K + \varepsilon^2 K^2 > 0$, or equivalently, $\varepsilon K > 8$. Now that we have this inequality which makes our values real, we can use Vieta's formulas to determine the sign of r_1 and r_2. We can see that $r_1 + r_2 = \frac{\beta(\varepsilon K-2)}{1+\varepsilon K}$, which is positive as long as $\varepsilon K > 2$, which we believed to be true by our assumption. Thus, $r_1 + r_2 > 0$. Now, we can observe that $r_1 r_2 = \frac{\beta^2}{1+\varepsilon K}$, which is always positive, so $r_1 r_2 > 0$. Thus, we can conclude that whenever $f'(r) = 0$, both solutions r_1 and r_2 are positive real numbers. Due to the fact that the sign of $f'(r)$ can be reduced to a quadratic with a positive coefficient in front of the squared variable, we can conclude that $f'(r)$ is decreasing between r_1 and r_2. Thus, if r increases from r_1 to r_2, the span of λ actually increases.

Biologically speaking, a wider infection rate is tolerable to create an epidemic if the recovery rate increases while it is between r_1 and r_2. However, before we progress any further, we must first check to make sure that we are still working within the bistability condition, which is $\varepsilon r > \frac{\beta+r}{K}$, or equivalently, $r > \frac{\beta}{\varepsilon K-1}$. In order for r_1 and r_2 to be values that are acceptable in the bistability environment, the previous inequality most hold. So we must check to make sure that:

$$\frac{\beta}{\varepsilon K-1} < \frac{\beta\left(-2+\varepsilon K+\sqrt{K^2\varepsilon^2-8K\varepsilon}\right)}{2(1+\varepsilon K)}$$

If we simplify the previous inequality, we get:

$$\varepsilon K(5-\varepsilon K) < (\varepsilon K-1)\sqrt{K^2\varepsilon^2-8K\varepsilon}$$

This inequality is always true based on our assumption that $\varepsilon K > 8$. Thus, r_1 and r_2 are always within the bistability condition. Thus, we can conclude that as long as the bistability condition holds, the range of λ will decrease as r increases, except for when r increases between r_1 and r_2. This is counter-intuitive, as it can mean that a disease is more likely to become an epidemic if treatment for the disease becomes more successful within a certain range.

4. Vaccination vs. Treatment

In this section, we will explore the differences between the advertisement of an effective treatment (as in Section 3) versus the effects of advertising a successful vaccine. This model does not take into account vertical transmission or mortality rate of the disease. As we used r previously to denote the recovery rate, we will now use v to represent the vaccination rate. The model is as follows:

$$\begin{cases} S' = \beta P - \lambda(1+\varepsilon R)SI - \overline{\mu}S - vS \\ I' = \lambda(1+\varepsilon R)SI - \overline{\mu}I \\ R' = vS - \overline{\mu}R \end{cases}$$

4.1. **Equilibrium Points.** Similar to the previous models, the population will approach a carrying capacity K as time goes to infinity. Also, we have a disease-free equilibrium point, but it occurs at $I = 0$, $R = \frac{vK}{\beta+v}$. There also exist two endemic equilibrium points, denoted R_1 and R_2 (such that $R_1 < R_2$). In the vaccination case, both roots are always real, but only R_2 is positive; R_1 is always negative. This stems for the fact that we get the following expression of R when solving the system of I' and R':

$$\lambda\varepsilon R^2 + \lambda R - v$$

The above is a quadratic of R, and there will always exist exactly one positive real root, R_2.

4.2. **Analyzing the Equilibrium Points.**

4.2.1. *Disease-Free Equilibrium Point.* When we substitute the DFE point into the Jacobian, we get the following:

$$J\left(0, \tfrac{vK}{\beta+v}\right) = \begin{bmatrix} \frac{\lambda(v+\beta+\varepsilon vK)K\beta}{(v+\beta)^2} - \beta & ; & 0 \\ -r & ; & -r-\beta \end{bmatrix}$$

From the Jacobian, we get the condition that the disease-free equilibrium point is stable if $\lambda < \frac{(v+\beta)^2}{(v+\beta+\varepsilon vK)K}$. Equivalently, we get that $\mathcal{R}_0 = \frac{\lambda K(v+\beta+\varepsilon vK)}{(v+\beta)^2} < 1$.

4.2.2. *Endemic Equilibrium Point.* As stated before, although there exist two equilibrium points, only one is biologically feasible. This endemic equilibrium is always positive, real, and stable. We get this from the fact that the determinant of the Jacobian is always positive and the trace is always negative. However, for certain values of R, a negative I value can result, so we have to find the condition that I is positive. It turns out that I is positive whenever $\lambda > \frac{(v+\beta)^2}{(v+\beta+\varepsilon vK)K}$.

4.3. **Conditions.** If we combine the above information, we can see that we get a simple chart: So we can see that there only exist two possible cases: either the disease-free equilibrium exists, or the endemic equilibrium exists. Both cannot simultaneously occur.

Condition	DFE	Other Equilibria
$\lambda < \dfrac{(v+\beta)^2}{(v+\beta+\varepsilon vK)K}$	stable	$R_1 \in \mathbb{R}^-, R_2 \in \mathbb{R}^+$, but $I \in \mathbb{R}^-$
$\lambda > \dfrac{(v+\beta)^2}{(v+\beta+\varepsilon vK)K}$	unstable	$R_1 \in \mathbb{R}^-, R_2 \in \mathbb{R}^+$, R_2 - stable

4.4. **Monotonicity of v.** Similar to before, we want to treat the value for the infection rate as a function with respect to a variable and see how the function changes for different values. This time, there is only one bound, so there will not be a bistability case as there was in the previous model. So let:

$$f(v) = \frac{(v+\beta)^2}{(v+\beta+\varepsilon vK)K}$$

If we differentiate f with respect to v, we obtain one critical value $v^* = \frac{\beta(\varepsilon K-1)}{\varepsilon K+1}$. Now we make the assumption that $\varepsilon K > 1$ in order to have a biologically feasible v^* (otherwise, $v^* < 0$). This is a safe assumption to make, especially in a population with a larger carrying capacity. A minimal change in risk behavior $\left(\varepsilon > \frac{1}{K}\right)$ is required, so we can continue safely under this assumption. For any values of v from 0 up until v^*, we see that the derivative is negative, so f decreases in this range, and any values after v^* yield a positive derivative, so f increases in this range. We find a similar counter-intuitive result: as the vaccination rate increases within a certain range, it actually worsens the situation and allows the disease a wider range to be able to spread.

5. Vaccination and Treatment

Now, we will combine both of the above models in order to create a model where individuals can join the recovered group through either vaccination or treatment. We will consider the following model:

$$\begin{cases} S' = \beta P - \lambda(1+\varepsilon R)SI - \overline{\mu}S - vS \\ I' = \lambda(1+\varepsilon R)SI - \overline{\mu}I - rI \\ R' = rI + vS - \overline{\mu}R \end{cases}$$

It is important to mention that if the vaccination rate is set equal to 0, then the treatment model will be the result. Likewise, whenever the recovery rate is set equal to 0, the vaccination model will be the result.

5.1. **Equilibrium Points.** As time approaches infinity, our population will once again approach a carrying capacity, K. We will once again have a disease-free equilibrium point when $I^* = 0$ and $R^* = \frac{vK}{\beta+v}$. As with before, there are also two other equilibrium points, R_1 and R_2, which depend on the the values of r and v.

5.2. **Analyzing the Equilibrium Points.**

5.2.1. *Disease-Free Equilibrium Point.* When we substitute the DFE point into the Jacobian, we get the following:

$$J\left(0, \tfrac{vK}{\beta+v}\right) = \begin{bmatrix} \frac{\lambda(v+\beta+\varepsilon vK)K\beta}{(v+\beta)^2} - \beta - r & ; & 0 \\ r - v & ; & -v-\beta \end{bmatrix}$$

From the Jacobian, we get the condition that the disease-free equilibrium point is stable if $\lambda < \frac{(v+\beta)^2(\beta+r)}{(v+\beta+\varepsilon vK)K\beta}$. Equivalently, we get that $\mathcal{R}_0 = \frac{\lambda(v+\beta+\varepsilon vK)K\beta}{(v+\beta)^2(\beta+r)} < 1$.

5.2.2. *Endemic Equilibrium Points.* As mentioned before, we are going to have two equilibrium points which are not the disease-free case, R_1 and R_2. However, in order for these to be real, the following condition for the infection rate must hold true:

$$\lambda > \frac{4\varepsilon(r+\beta)^2(r-v)}{((1+\varepsilon K)r+\beta)^2}$$

We are also able to obtain the following table for the sign of R_1 and R_2: We also have to take notice of I.

If	Then
$\lambda < \frac{(r+\beta)(r-v)}{rK}$	$R_1R_2 > 0$
$\lambda > \frac{(r+\beta)(r-v)}{rK}$	$R_1R_2 < 0$
$r\varepsilon > \frac{\beta+r}{K}$	$R_1 + R_2 > 0$
$r\varepsilon < \frac{\beta+r}{K}$	$R_1 + R_2 < 0$

5.3. **The Condition v < r.** First, we will focus on the case where the vaccination rate is greater than the recovery rate. This will lead to a model which is actually very similar to the vaccination model of Section 4. As mentioned before, there will be two endemic equilibria, which will be denoted similarly to before. However, it is also important to note that I must not be negative, so we must establish a separate condition for which this is true. In order for I to be non-negative, the following condition must hold:

$$\lambda > \frac{(\beta + r)(v + \beta)^2}{K\beta(v + \beta + \varepsilon vK)}$$

It is important to note that this inequality is the exact opposite of the condition for disease clearance. Thus, in the case where the vaccination rate is greater than the recovery rate, then there will only be two states: disease clearance and disease persistence. Further investigating the endemic equilibrium points, we get that (under the condition that $v > r$) both R_1 and R_2 will always be stable. This stems from the fact that the condition for the infection rate will consist of the infection rate being greater than a negative number, which is always the case. However, using the Vieta relations, we can see that $R_1 \in \mathbb{R}^-$ and $R_2 \in \mathbb{R}^+$. So there will always be only one biologically feasible endemic equilibria under this condition. There will be no case of bistability as there was in the treatment model. So, similarly to the vaccination model, we get the following table, with two cases:

Condition	DFE	Other Equilibria
$\lambda < \frac{(v+\beta)^2(\beta+r)}{(v+\beta+\varepsilon vK)K\beta}$	stable	$R_1 \in \mathbb{R}^-, R_2 \in \mathbb{R}^+$, but $I \in \mathbb{R}^-$
$\lambda > \frac{(v+\beta)^2(\beta+r)}{(v+\beta+\varepsilon vK)K\beta}$	unstable	$R_1 \in \mathbb{R}^-, R_2 \in \mathbb{R}^+$, R_2 - stable

5.3.1. *Monotonicity of v.* With regards to how the thresholds respond when the vaccination rate is changed we obtained the same result as in the vaccination model: as the vaccination rate increases from zero up until a critical value, the range for the infection rate increases, meaning that a higher vaccination rate actually can make the epidemic worse just due to overcompensation with respect to risk behavior. We also have the same result as before from the critical value to infinity. This is the intuitive result, where increasing the vaccination rate results in a smaller range for the infection rate.

5.3.2. *Monotonicity of r.* Now that we have a model considering both the vaccination and the treatment rates, we want to see how the recovery rate affects the threshold for the model. It turns out that the derivative of the threshold with respect to the recovery rate is a positive constant. So as the recovery rate increases, as long as $v > r$, the range for the infection rate will get smaller.

5.4. **The Condition r > v.** Similar to how the model reflected the vaccination model whenever the vaccination rate was greater than the recovery rate, we expect the model to reflect the treatment model under the condition that the recovery rate is greater. There is the general condition for disease clearance which was stated in Section 5.2.1, but there is also a possibility that the disease may persist whenever

the condition for the infection rate in Section 5.2.2 is satisfied. It is important to note that these two thresholds are different, so this is more likely to be similar to the treatment model than the vaccination one. There are three separate thresholds for the infection rate in this case, but only two are necessary for comparing the different end behavior of the population, so will denote the thresholds as below:

$$T_1 = \frac{4\varepsilon(\beta+r)^2(r-v)}{(\beta+r+\varepsilon rK)^2},\ T_2 = \frac{(\beta+r)(\beta+v)^2}{\beta K(\beta+v+\varepsilon vK)}$$

There will also be two separate thresholds with respect to the risk-taking term. We will denote these thresholds as follows:

$$E_1 = \frac{\beta+r}{r},\ E_2 = \frac{\beta^2+\beta(r+v)+rv}{\beta(r-v)}, V_1 := \frac{\beta r}{2\beta+r}$$

Stability results:

(i) $0 < v < V_1$
- $\varepsilon K < E_2$ then we have DFE for $0 < \lambda < T_2$ and EE for $\lambda > T_2$
- $\varepsilon K > E_2$ then we have DFE for $0 < \lambda < T_1$, bistability between DFE and EE for $T_1 < \lambda < T_2$ and EE for $\lambda > T_2$

(ii) $V_1 < r$ then we have DFE for $0 < \lambda < T_2$ and EE for $\lambda > T_2$

We can show (similar to the treatment only case) that there is a range of the treatment that causes the bistability range of λ to increase. First notice that $\frac{dT_2}{dr} > 0$ and $\frac{dE_2}{dr} < 0$. The last condition actually shows that as r increases, it takes less risk-taking behavior for the bistability regimen to be maintained. Consider now the monotonicity of T_1. This depends on the sign of

$$(1+\varepsilon K)r^2 + \beta(2-\varepsilon K)r + 2v\beta\varepsilon K + \beta^2.$$

There are two positive critical values r_1 and r_2 such that T_1 decreases if $r_1 < r < r_2$ provided that

$$\varepsilon K > \max\left\{E_2, \frac{8(\beta+v)}{\beta-8v}\right\} \text{ and } v < \min\left\{\frac{\beta}{8}, \frac{\beta r}{2\beta+r}\right\}$$

6. Conclusions

We introduced and analyzed a simple SIR model where the infection rate is made increasingly dependent on the recovered class to account for a possible risk-taking behavior effect as a response to a successful intervention (treatment or vaccine). In order to simplify the mathematical analysis we assumed a closed population which always approaches its carrying capacity and a disease without re-infection or loss of immunity.

We found that risk taking behavior not only increases the epidemic reproductive number (as expected) but, for certain mild conditions on the parameters, it reverses the expected benefit of intervention. Mathematically, this is illustrated by an increasing reproductive number with higher levels of treatment or vaccination. While, in the treatment case, the dangerous interval is in between two critical treatment rates, in the vaccination case, that interval is always $(0, v^*)$ where v^* is a critical vaccination rate. This would suggest that low vaccination efficacy is worse than not vaccinating at all if even a small risk taking behavior is present.

This research can be continued and improved in several ways. First one must consider a disease with induced mortality and re-infection in order to account for a wider ranges of diseases that fit our modeling framework. Secondly, a generalization of the risk-taking term would be necessary since there is no universal mathematical way to describe it. In fact, one major drawback is the difficulty to measure ε and an investigation to collect data for it must involve carefully designed social experiments. Finally, an extension to two-sex models would be necessary to account not only for Sexually Transmitted Diseases but also for the possible skewed risk-taking behavior response between genders. We plan to address some or all of these questions in the near future.

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