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Optimal control of an SIR model with effect of vaccination and a generic nonlinear infection rate

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Abstract

This paper introduces a SIR model with a nonlinear incidence rate and incorporates a vaccination scenario, offering a more realistic framework for disease dynamics. Assuming a vaccination coverage of $p\%$, we analyze its influence on epidemic outcomes. The model features a disease-free equilibrium E_0 from which we derive the basic reproduction number $\mathcal{R}_0$, serving as the threshold for disease eradication. When $\mathcal{R}_0 > 1$, an endemic equilibrium E_1 emerges; conversely, $\mathcal{R}_0 < 1$ guarantees the global stability of E_0, indicating disease elimination. A transcritical bifurcation at $\mathcal{R}_0 = 1$ captures the transition between disease extinction and persistence, with no evidence of Hopf bifurcations as shown by limit set analysis. Sensitivity analysis of $\mathcal{R}_0$ highlights key parameters influencing transmission, informing intervention strategies. We also develop an optimal control framework to determine the most effective vaccination coverage, providing actionable insights for public health policies. Numerical simulations validate the theoretical results, illustrating how variations in p impact outbreak trajectories and underscoring the importance of sustained vaccination efforts. By integrating nonlinear transmission with vaccination dynamics, this study advances epidemic modeling and offers practical tools for disease management.

Keywords. SIR model, Optimal control, Vaccination.

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1. Introduction

Epidemiology studies health patterns and factors affecting them in the population. While it has historically focused on infectious diseases, it has now prioritized non-communicable diseases such as stroke and heart disease, which dominate global mortality. However, infectious diseases such as pneumonia and HIV remain major health concerns. Infectious diseases are diseases caused by pathogens such as bacteria, viruses, fungi, parasites, or prions, examples of which include tuberculosis and HIV. Communicable diseases are a subset of infectious diseases that are transmitted between people, while some, such as tetanus, are non-communicable. Infectious diseases are spread by several routes: [**?**]:

- Person-to-person: direct contact (e.g., HIV) or indirect contact through objects or fluids (e.g., influenza);
- Airborne: inhalation of contaminated air (e.g., tuberculosis or measles);
- Food and water: consumption of contaminated sources (e.g., cholera);
- Vector-Borne: transmitted by vectors like mosquitoes (e.g., malaria);
- Vertical: transmitted from mother to child during pregnancy or birth (e.g., HIV).

Knowing the routes of disease transmission is actually essential for designing effective prevention strategies, controlling disease outbreaks, and making accurate mathematical models. In fact, by understanding and incorporating disease pathways into mathematical models, we increase our ability to predict outbreaks, implement targeted interventions, and reduce disease outbreaks.

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Mathematical epidemiology, which originated with Bernoulli's 1760 smallpox model [**?**], has made significant progress with the foundational theories developed in 1935. It has become a cornerstone of public health policy, helping to predict and control disease during outbreaks such as the 2001 foot-and-mouth disease crisis, the 2002–2003 SARS epidemic, and the COVID-19 pandemic. Indeed, mathematical epidemiology reached a turning point in 1927 with Kermack and McKendrick's influential model of the spread of infectious diseases, detailed in [**?**]. This deterministic model introduced the concepts of

- **Susceptible (S):** Healthy individuals who are at risk of contracting the disease,
- **Infected (I):** Individuals who have the disease and are assumed to be infectious,
- **Removed/Recovered (R):** Individuals who have recovered and are immune, and cannot be reinfected.

Each individual in the population is placed in one of these three groups, and the changes in each group are formulated as a differential equation. These categories form the basis of the SIR model used in mathematical epidemiology and modern epidemic modeling. With the rise of emerging diseases, there is a growing need for more flexible models. The study in [**?**] highlights the importance of developing integrated modeling frameworks to enable rapid responses to new threats. Addressing the shortcomings and refining epidemiological models has led to the introduction of numerous models for the study of infectious diseases. The rates of change used in the model, such as birth rates, disease incidence rates, and recovery rates, represent some of the most important differences between the introduced models and the work done in this field. For example, in the works of Kermack and McKendrick, their model initially focused on prevalence without considering natural birth and death rates. In subsequent works published in 1932 and 1933, they extended their framework to address diseases that persist within populations. Their original trilogy of papers, reprinted in 1991, remains a foundation for the field [**? ? ?**].

Recent advances in epidemic modeling emphasize the importance of capturing complex human behaviors and heterogeneous transmission patterns. Among various factors influencing disease dynamics, the incidence rate plays a particularly critical role and has attracted increasing scholarly attention. Traditional models, such as those in [**? ? ?**], often assume a linear incidence rate, typically expressed as βI, where β denotes the transmission rate.

However, more sophisticated approaches incorporate nonlinear incidence functions to better reflect behavioral factors impacting disease spread. For example, in [**?**], the incidence rate is modeled as $\beta I(1+\gamma I)$, enabling the analysis of effects arising from multiple contacts and behavioral responses that influence transmission dynamics.

In [**?**], the incidence rate is considered as $\beta I/(1+\alpha I)$, where α is referred to as the suppression effect, stemming from the behavioral changes of susceptible individuals in response to the increasing number of infected individuals. In [**?**], the incidence rate is defined as $\beta I^p/(1+\alpha I^q)$, and in [**?**], this model has been studied for $p=q$. In this paper, inspired by the previous work [**?**], we employ a nonlinear function for the disease incidence rate. This approach allows for a broader examination, accommodating various transmission rates relevant to the specified conditions.

Another important aspect of epidemic models is the implementation of disease control strategies. Among various methods, vaccination plays a pivotal role in limiting disease spread. Vaccination can be modeled in different ways; for instance, as noted in [**?**], vaccinated individuals are often assigned to a separate group. This approach increases the system's dimensionality, thereby elevating the complexity of calculations. Alternatively, vaccination can be represented by considering a specific percentage of the population as vaccinated, simplifying the model while still capturing its overall impact.

In [**?**], the authors incorporated vaccination, assuming 100% efficacy, and conducted a comprehensive analysis of its effects. In this paper, however, we will explore the effects of vaccination and the percentage of vaccinated individuals within the community in our model. Our focus in this paper is on modeling diseases whose transmission mechanisms are not sensitive to population density, such as Hepatitis B and HIV. Therefore, we will utilize the standard transmission rate.

2. Model formulation

Let $N(t)$ represent the total population size. Therefore, we have: $N(t)=S(t)+I(t)+R(t)$, where $S(t)$ is the number of susceptible individuals, $I(t)$ is the number of infected individuals, and $R(t)$ is the number of recovered individuals. Since the spread of an infectious disease is directly related to the interaction between class class I (infected) and S (susceptible), it makes sense for the infection rate we propose to accurately reflect the impact of their interaction.

TABLE 1. Description of the parameters used in the system (2.1).

Parameter	Description
λ	Birth rate
β	Disease transmission rate
p	Percentage of vaccinated population
v	Vaccine effectiveness rate
μ	Natural death rate
α	The rate of recovery from the disease

What is important for us to consider is that if the number of infected individuals exceeds a certain threshold, then the rate of infection resulting from the interaction will decline. To relate these aspects, we consider the function $g(x)$ under the following conditions:

(1) $g(0) = 1$.
(2) For $x > 0$, $g(x) > 0$.
(3) There exists a small positive constant $\sigma > 0$ such that if $0 < x < \sigma$, then $\left(\frac{x}{g(x)}\right)' > 0$, and if $x > \sigma$, then $\left(\frac{x}{g(x)}\right)' < 0$.

Now we consider the infection rate in the form: $\frac{\beta IS}{Ng(I/N)}$. In this case, we see that condition (3) fulfills our requirement regarding the trend of patient infections. Furthermore, we assume that a portion of the population, denoted by p, is vaccinated, with an efficacy rate of v. With these clarifications, we propose the following SIR model:

$$\begin{cases} S' = \lambda N - \dfrac{\beta IS}{Ng\,(I/N)} - pvS - \mu S, \\ I' = \dfrac{\beta IS}{Ng\,(I/N)} - (\alpha + \mu)I, \\ R' = pvS + \alpha I - \mu R. \end{cases} \tag{2.1}$$

The description of all parameters is given in Table 1. The diagram of this model is shown in Figure 1. Using the

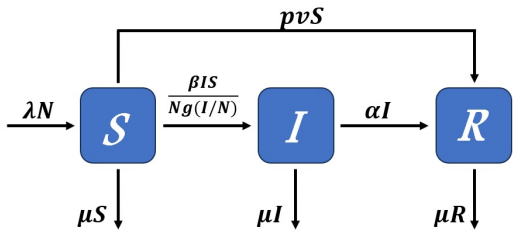


FIGURE 1. The diagram of SIR model (2.1).

change of variables $x = S/N$, $y = I/N$, and $z = R/N$, we rewrite system (2.1) as the following:

$$\begin{cases} x' = \lambda - \dfrac{\beta xy}{g(y)} - (\lambda + pv)x, \\ y' = \dfrac{\beta xy}{g(y)} - (\lambda + \alpha)y, \\ z' = pvx + \alpha y - \lambda z. \end{cases} \tag{2.2}$$

3. Equilibria and basic reproduction number

Since the variable z does not appear in the first and second equations of system (2.2), we can consider the following two-dimensional system by using the equality $z = 1 - x - y$:

$$\begin{cases} x' = \lambda - \dfrac{\beta xy}{g(y)} - (\lambda + pv)x, \\ y' = \dfrac{\beta xy}{g(y)} - (\lambda + \alpha)y. \end{cases} \tag{3.1}$$

This system has a disease-free equilibrium point given by $E_0 = (\lambda/(\lambda + pv), 0)$. Corresponding to this equilibrium point, the basic reproduction number can be obtained using the next-generation technique [**?**]. We define the functions $f(x, y)$ and $v(x, y)$ as follows:

$$f(x, y) = \frac{\beta xy}{g(y)}, \ w(x, y) = (\lambda + \alpha)y.$$

Then, we can write y' as $y' = f(x, y) - w(x, y)$. Denote

$$F = \left[\frac{\partial f}{\partial y}(E_0)\right] = \frac{\beta\lambda}{\lambda + pv}$$

and

$$W = \left[\frac{\partial w}{\partial y}(E_0)\right] = \lambda + \alpha.$$

Then, the basic reproduction number, which is the spectral radius of the matrix FW^{-1}, is equal to

$$\mathscr{R}_0 = \frac{\beta\lambda}{(\lambda + \alpha)(\lambda + pv)}.$$

System (3.1) has another equilibrium point, which we call the endemic equilibrium point. To find this equilibrium point, we solve the following system:

$$\begin{cases} \lambda - \dfrac{\beta xy}{g(y)} - (\lambda + pv)x = 0, \\ \dfrac{\beta xy}{g(y)} - (\lambda + \alpha)y = 0. \end{cases}$$

Since, in this case, $y \neq 0$, we obtain from the second equation that $x = (\lambda + \alpha)\, g(y)/\beta$ and by substituting it into the first equation, we get that

$$\lambda - (\lambda + \alpha)y - \frac{(\lambda + pv)\,(\lambda + \alpha)}{\beta} g(y) = 0.$$

We set

$$G(y) = \lambda - (\lambda + \alpha)y - \frac{(\lambda + pv)\,(\lambda + \alpha)}{\beta} g(y). \tag{3.2}$$

In this case:

$$\lim_{y \to \infty} G(y) = -\infty$$

and

$$G(0) = \lambda - \frac{(\lambda + pv)(\lambda + \alpha)}{\beta} = \frac{(\lambda + pv)(\lambda + \alpha)}{\beta}(\mathscr{R}_0 - 1).$$

As for $y > 0$ we have $g'(y) > 0$, thus

$$G'(y) = -(\lambda + \alpha) - \frac{(\lambda + pv)(\lambda + \alpha)}{\beta} g'(y) < 0.$$

Therefore, if $\mathscr{R}_0 > 1$, then $G(0) > 0$, which implies that $G(y)$ has a unique positive root y^*, leading to the endemic equilibrium point $E_1 = (x^*, y^*)$. It is worth noting that if $\mathscr{R}_0 \leq 0$, then $G(0) < 0$. Based on the strict monotonicity of $G(y)$ and by Bolzano's theorem, the function $G(y)$ will not have any positive roots.

What has been discussed so far about system (3.1) is summarized in the following theorem:

Theorem 3.1. *Consider system* (3.1).

a) *If $\mathscr{R}_0 \leq 1$, then the system has a unique disease-free equilibrium point.*
b) *If $\mathscr{R}_0 > 1$, then the system, in addition to the disease-free equilibrium point, possesses an endemic equilibrium point.*

Epidemiological interpretation of $\mathscr{R}_0$. Two points regarding $\mathscr{R}_0$ deserve attention and explanation:

1. **Formula**: It is essential to clarify the formula used to calculate $\mathscr{R}_0$ in the context of this model.
2. **Behavior Near 1**: The second point concerns how changes in $\mathscr{R}_0$, particularly around the value of 1, affect the dynamics of system (3.1).

The variations of $\mathscr{R}_0$ around 1 indicate that if an infected individual cannot infect at least one other person, the disease will disappear from the population. Conversely, if $\mathscr{R}_0$ is greater than 1, the disease will persist within the community.

The epidemiological interpretation of the formula for $\mathscr{R}_0$ will be discussed below. It is shown in [**?**] that if ρ represents the exit rate from a group, then $\frac{1}{\rho}$ corresponds to the average time spent in that group. Since $\lambda + \alpha$ is the rate of exit from the infectious class y, the average time spent in this class for each individual will be $\frac{1}{\lambda+\alpha}$. On the other hand, the exit rate for each individual from the susceptible class is $\lambda + pv$. Therefore, the average time of susceptibility for each individual will be $\frac{1}{\lambda+pv}$. The disease is transmitted to a susceptible individual at a rate β. Consequently, an infectious person can infect a susceptible person at a rate of $\frac{\beta}{(\lambda+pv)(\lambda+\alpha)}$ during the time they are infected. Since individuals are born and enter the susceptible class at a rate λ, an infected individual can infect during the time $\frac{\beta\lambda}{(\lambda+pv)(\lambda+\alpha)}$, which is equal to $\mathscr{R}_0$. It is important to note that as more individuals are vaccinated, $\mathscr{R}_0$ will decrease, indicating a reduced spread of the disease within the community.

4. Local stability of the equilibrium points

4.1. **Local stability of the disease-free equilibrium point E_0.** To analyze the stability of the disease-free equilibrium point in system (3.1), we employ the linearization method and the Jacobian matrix. The Jacobian matrix of the system at the equilibrium point $E_0 = (\lambda/(\lambda + pv), 0)$ is given by:

$$J_{E_0} = \begin{bmatrix} -(\lambda+pv) & \frac{-\beta\lambda}{\lambda+pv} \\ 0 & \frac{\beta\lambda}{\lambda+pv} - (\lambda+\alpha) \end{bmatrix}.$$

This matrix has two eigenvalues: $-(\lambda+pv)$ and $\frac{\beta\lambda}{\lambda+pv} - (\lambda+\alpha)$. The first eigenvalue is negative, indicating stability in that direction. For the second eigenvalue, we need to analyze it further. We have that

$$\frac{\beta\lambda}{\lambda+pv} - (\lambda+\alpha) = (\lambda+\alpha)\left(\frac{\beta\lambda}{(\lambda+pv)(\lambda+\alpha)} - 1\right) = (\lambda+\alpha)(\mathscr{R}_0 - 1).$$

Therefore, if $\mathscr{R}_0 < 1$, the disease-free equilibrium point E_0 is locally stable. Conversely, if $\mathscr{R}_0 > 1$, it becomes unstable. At $\mathscr{R}_0 = 1$, this equilibrium point is non-hyperbolic, and considering the change in stability, one can expect a bifurcation to occur. We will examine this case in section 6.

4.2. **Local stability of the endemic equilibrium point E_1.** The Jacobian matrix of the system (3.1) at the equilibrium point $E_1 = (x^*, y^*)$ is given by:

$$J_{E_1} = \begin{bmatrix} -\dfrac{\beta y^*}{g(y^*)} - (\lambda + pv) & -\beta x^* \left(\dfrac{g(y^*) - y^* g'(y^*)}{g^2(y^*)} \right) \\ \dfrac{\beta y^*}{g(y^*)} & \beta x^* \left(\dfrac{g(y^*) - y^* g'(y^*)}{g^2(y^*)} \right) - (\lambda + \alpha) \end{bmatrix}.$$

The characteristic polynomial of this matrix in the variable t is given by $p(t) = t^2 + a_1 t + a_2$, where

$$a_1 = -\mathrm{tr}(J_{E_1}) = (\lambda + pv) + \frac{\beta y^*}{g(y^*)} - \beta x^* \left(\frac{g(y^*) - y^* g'(y^*)}{g^2(y^*)} \right) + (\lambda + \alpha),$$

$$a_2 = \det(J_{E_1}) = \frac{\beta y^* (\lambda + \alpha)}{g(y^*)} - (\lambda + pv)\beta x^* \left(\frac{g(y^*) - y^* g'(y^*)}{g^2(y^*)} \right) + (\lambda + pv)(\lambda + \alpha).$$

According to the Routh-Hurwitz test, two roots of $p(t)$ have negative real parts if and only if both a_1 and a_2 are positive. Since E_1 is the equilibrium point of system (3.1), from the second equation of this system, we obtain that

$$\frac{\beta x^* y^*}{g(y^*)} - (\lambda + \alpha) y^* = 0,$$

which yields $\frac{\beta x^*}{g(y^*)} = \lambda + \alpha$. Consequently,

$$a_1 = (\lambda + pv) + \frac{\beta y^*}{g(y^*)} - \frac{\beta x^*}{g(y^*)} + \frac{\beta x^* y^* g'(y^*)}{g^2(y^*)} + (\lambda + \alpha) = (\lambda + pv) + \frac{\beta y^*}{g(y^*)} + \frac{\beta x^* y^* g'(y^*)}{g^2(y^*)} > 0,$$

$$\begin{aligned} a_2 &= \frac{\beta(\lambda + \alpha) y^*}{g(y^*)} - \frac{(\lambda + pv)\beta x^*}{g(y^*)} + \frac{(\lambda + pv)\beta x^* y^* g'(y^*)}{g^2(y^*)} + (\lambda + pv)(\lambda + \alpha) \\ &= \frac{\beta(\lambda + \alpha) y^*}{g(y^*)} + \frac{(\lambda + pv)\beta x^* y^* g'(y^*)}{g^2(y^*)} > 0. \end{aligned}$$

Hence, the equilibrium point E_1 is locally asymptotically stable because the real part of both eigenvalues at this point is negative.

5. Global stability

To investigate the global stability of the equilibrium points, we will use the Poincaré-Bendixson theorem. For this purpose, we will first demonstrate the boundedness of the solutions of the system (3.1).

Theorem 5.1. *The set $\mathbb{R}^2_+ = \{(x, y) \in \mathbb{R}^2 \mid x > 0,\ y > 0\}$, which represents the positive area of the plane, is positively invariant under the flow of the system (3.1), and the solutions of the system (3.1) are bounded in this region.*

Proof. To prove the first part of the theorem, we show that the flow is directed inward or tangent to the boundary of the region $\mathbb{R}^2_+$. For this purpose, we observe that on the boundary of $x = 0$, we have $x' = \lambda$, which indicates that the flow is directed inward into the region $\mathbb{R}^2_+$. Along the boundary $y = 0$, we have $y' = 0$, which shows that the flow is directed neither to the left nor to the right but is tangent to the boundary $y = 0$. This implies that the solutions cannot cross the boundary of $y = 0$, confirming that the region $\mathbb{R}^2_+$ is positively invariant and the flow remains within this bounded area.
To prove the boundedness of the solutions, it is enough to show that the function $n(t) := x(t) + y(t)$ is bounded. We have:

$$n'(t) = x'(t) + y'(t) = \lambda - \lambda n(t) - pvx(t) - \alpha y(t) \leq \lambda(1 - n(t)).$$

Therefore,

$$n(t) \leq (n_0 - 1)e^{-\lambda t} + 1 \leq 1.$$

□

Theorem 5.2. *The system (3.1) does not have periodic orbits in the first quadrant.*

Proof. We will prove this theorem using the Dulac criterion. We consider the function $B(x, y) = \frac{g(y)}{xy}$ as the Dulac function. Let $\begin{bmatrix} f_1 \\ f_2 \end{bmatrix}$ be the vector field associated with the system (3.1). Then:

$$Bf_1 = \frac{\lambda g(y)}{xy} - \frac{(\lambda + pv)g(y)}{y} - \beta, \; Bf_2 = \beta - \frac{(\lambda + \alpha)g(y)}{x}.$$

Therefore,

$$\operatorname{div}(Bf) = \frac{\partial(Bf_1)}{\partial x} + \frac{\partial(Bf_2)}{\partial y} = -\frac{\lambda y g(y)}{x^2 y^2} - \frac{(\lambda + \alpha)g'(y)}{x} < 0.$$

Now, since the divergence of the vector field Bf in the first quadrant of the xy plane does not change in sign and has a constant sign, by the Dulac criterion, this system will not have periodic orbits in the first quadrant. □

Theorem 5.3. *If $X(t) = (x(t), y(t))$ is a solution of the system (3.1) in the region $\mathbb{R}^2_+$, then $\lim_{t\to\infty} X(t)$ exists and its value is equal to $E_1 = (x^*, y^*)$.*

Proof. Assume that $N > 1$. Consider the set $M = \{(x, y) \in \mathbb{R}^2 \mid 0 \leq x \leq N, \; 0 \leq y \leq N - x\}$. Then M is clearly a compact set and is positively invariant under the flow of the system (3.1) because along the line $x + y = N$, we have:

$$x' + y' = \lambda - \lambda(x + y) - \alpha y = \lambda - \lambda N - \alpha y \leq \lambda(1 - N) < 0.$$

Now, if $(x_0, y_0) \in M$ such that $y_0 > 0$, then it follows from the Poincaré-Bendixson theorem combined with theorem 5.2 that the omega limit set $\omega(x_0, y_0)$ of the point (x_0, y_0) is an equilibrium point. Specifically, in the case that $y_0 = 0$, we have $\omega(x_0, 0) = \{E_0\}$. As we have seen, system (3.1) can have at most two equilibrium points E_0 and E_1. Therefore, there are two cases, as described below:

- If $\mathscr{R}_0 \leq 1$, then there is no equilibrium point E_1. Thus, in this case, we have:

  $$\lim_{t\to\infty} (x(t), y(t)) = E_0,$$

  which means that the equilibrium point E_0 is globally stable.
- If $\mathscr{R}_0 > 1$, then it is proved that E_0 is unstable. Therefore,

  $$\lim_{t\to\infty} (x(t), y(t)) = E_1,$$

  which means that the equilibrium point E_1 is globally stable.

□

6. Analysis of the Transcritical Bifurcation

As we saw in section 4, the stability status of the equilibrium point E_0 changes as $\mathscr{R}_0$ passes through 1, and another equilibrium point is added to the system (3.1) for $\mathscr{R}_0 = 1$. Since E_0 is a non-hyperbolic equilibrium point for $\mathscr{R}_0 = 1$, it is predicted that the system (3.1) experiences a bifurcation at $\mathscr{R}_0 = 1$. We will demonstrate by the Sotomayor theorem that a transcritical (or a stability exchange) bifurcation occurs.

Theorem 6.1. ***[Sotomayor [?]]]*** *Let $X \in \mathbb{R}^n$ and $\mu \in \mathbb{R}$. Assume for the system $X' = f(X, \mu)$ that $f(X_0, \mu_0) = 0$ and the Jacobian matrix $A = Df(X_0, \mu_0)$ has a simple zero eigenvalue. If V_L and V_R are the left and right eigenvectors corresponding to the zero eigenvalue of the matrix A, then the system $X' = f(X, \mu)$ admits a transcritical bifurcation at the point (X_0, μ_0) whenever:*

(1) $V_L f_\mu(X_0, \mu_0) = 0$;
(2) $V_L D f_\mu(X_0, \mu_0) V_R \neq 0$;
(3) $V_L D^2 f(X_0, \mu_0)(V_R, V_R) \neq 0$.

Since $\mathscr{R}_0 = 1$ is equivalent to $\beta_0 = \dfrac{(\lambda+\alpha)(\lambda+pv)}{\lambda}$, we consider β as the bifurcation parameter of the system (3.1). The Jacobian matrix of the system (3.1) at the point E_0 and at the parameter value β_0 is given by

$$J_0 = J_{(E_0,\beta_0)} = \begin{bmatrix} -(\lambda+pv) & -(\lambda+\alpha) \\ [15pt]0 & 0 \end{bmatrix}.$$

The left and right eigenvectors corresponding to the zero eigenvalue of the matrix J_0 are, respectively, denoted by

$$V_L = [0\ 1]\,, \qquad V_R = \begin{bmatrix} 1 \\ \\ \dfrac{-(\lambda+pv)}{\lambda+\alpha} \end{bmatrix}.$$

Considering system (3.1) as $(x', y') = f(x, y, \beta)$, we will examine the points mentioned in Theorem 6.1. Since

$$\frac{\partial f}{\partial \beta}(x, y, \beta) = f_\beta(x, y, \beta) = \begin{bmatrix} -\dfrac{xy}{g(y)} \\ \\ \dfrac{xy}{g(y)} \end{bmatrix}, \qquad f_\beta(E_0, \beta_0) = \begin{bmatrix} 0 \\ 0 \end{bmatrix},$$

we see that the first condition

$$V_L f_\beta(E_0, \beta_0) = 0$$

of Theorem 6.1 holds. To examine the second condition, we note that

$$Df_\beta(x, y, \beta) = \begin{bmatrix} -\dfrac{y}{g(y)} & -x\dfrac{g(y) - yg'(y)}{(g(y))^2} \\ \\ \dfrac{y}{g(y)} & x\dfrac{g(y) - yg'(y)}{(g(y))^2} \end{bmatrix}.$$

Hence,

$$Df_\beta(E_0, \beta_0) = \begin{bmatrix} 0 & \dfrac{-\lambda}{\lambda+pv} \\ \\ 0 & \dfrac{\lambda}{\lambda+pv} \end{bmatrix},$$

which yields that

$$V_L D f_\beta(E_0, \beta_0) V_R = \frac{-\lambda}{\lambda+\alpha} \neq 0.$$

Thus, the second condition is also satisfied. We will now examine the third condition of Theorem 6.1. We have that

$$Df = \begin{bmatrix} -\dfrac{\beta y}{g(y)} - (\lambda+pv) & -\beta x\dfrac{g(y) - yg'(y)}{(g(y))^2} \\ \\ \dfrac{\beta y}{g(y)} & \beta x\dfrac{g(y) - yg'(y)}{(g(y))^2} - (\lambda+\alpha) \end{bmatrix} = \begin{bmatrix} Df_1 \\ \\ Df_2 \end{bmatrix},$$

$$D(Df_1)^T = \begin{bmatrix} 0 & -\beta\dfrac{g(y)-yg'(y)}{(g(y))^2} \\ -\beta\dfrac{g(y)-yg'(y)}{(g(y))^2} & \beta x\dfrac{yg(y)g''(y)+2g(y)g'(y)-2y(g'(y))^2}{(g(y))^3} \end{bmatrix},$$

$$D(Df_2)^T = \begin{bmatrix} 0 & \beta\dfrac{g(y)-yg'(y)}{(g(y))^2} \\ \beta\dfrac{g(y)-yg'(y)}{(g(y))^2} & -\beta x\dfrac{yg(y)g''(y)+2g(y)g'(y)-2y(g'(y))^2}{(g(y))^3} \end{bmatrix}.$$

Therefore

$$D^2f_1(E_0,\beta_0) := D(Df_1)^T(E_0,\beta_0) = \begin{bmatrix} 0 & -\dfrac{(\lambda+\alpha)(\lambda+pv)}{\lambda} \\ -\dfrac{(\lambda+\alpha)(\lambda+pv)}{\lambda} & 2(\lambda+\alpha)g'(0) \end{bmatrix},$$

$$D^2f_2(E_0,\beta_0) := D(Df_2)^T(E_0,\beta_0) = \begin{bmatrix} 0 & \dfrac{(\lambda+\alpha)(\lambda+pv)}{\lambda} \\ \dfrac{(\lambda+\alpha)(\lambda+pv)}{\lambda} & -2(\lambda+\alpha)g'(0) \end{bmatrix}.$$

Assume that $\{e_1, e_2\}$ is the standard basis for $\mathbb{R}^2$. Then

$$D^2f(E_0,\beta_0)(V_R,V_R) = \alpha_1 e_1 + \alpha_2 e_2,$$

where

$$\alpha_1 = V_R^T D^2f_1(E_0,\beta_0)V_R = \begin{bmatrix} 1 & \dfrac{-(\lambda+pv)}{\lambda+\alpha} \end{bmatrix} \begin{bmatrix} 0 & -\dfrac{(\lambda+\alpha)(\lambda+pv)}{\lambda} \\ -\dfrac{(\lambda+\alpha)(\lambda+pv)}{\lambda} & 2(\lambda+\alpha)g'(0) \end{bmatrix} \begin{bmatrix} 1 \\ \dfrac{-(\lambda+pv)}{\lambda+\alpha} \end{bmatrix}$$

$$= \frac{2(\lambda+pv)^2}{\lambda} + \frac{2(\lambda+pv)^2}{\lambda+\alpha}g'(0) > 0,$$

$$\alpha_2 = V_R^T D^2f_2(E_0,\beta_0)V_R = \begin{bmatrix} 1 & \dfrac{-(\lambda+pv)}{\lambda+\alpha} \end{bmatrix} \begin{bmatrix} 0 & \dfrac{(\lambda+\alpha)(\lambda+pv)}{\lambda} \\ \dfrac{(\lambda+\alpha)(\lambda+pv)}{\lambda} & -2(\lambda+\alpha)g'(0) \end{bmatrix} \begin{bmatrix} 1 \\ \dfrac{-(\lambda+pv)}{\lambda+\alpha} \end{bmatrix}$$

$$= -\frac{2(\lambda+pv)^2}{\lambda} - \frac{2(\lambda+pv)^2}{\lambda+\alpha}g'(0) < 0.$$

Hence,

$$V_L D^2f(E_0,\beta_0)(V_R,V_R) = \alpha_2 \neq 0,$$

which means that the third condition of Theorem 6.1 also holds. Consequently, system (3.1) at $\beta = \beta_0$ has a transcritical bifurcation.

7. Sensitivity index of $\mathcal{R}_0$

The sensitivity index is a tool used to evaluate how changes in a model's input parameters affect its outputs. It helps identify which parameters have the most significant influence, enhancing our understanding of the model and guiding improvements. In epidemiological models, the sensitivity index is particularly valuable for analyzing how different parameters impact disease prevalence. By using this measure, we can pinpoint the factors that most strongly influence the spread of the disease. These parameters can serve as suitable targets for health interventions. In addition, the sensitivity index helps decision-makers in allocating resources in a way that maximizes the impact of reducing disease outbreaks. By changing the parameters and observing their effects on the model results, more effective control strategies can be identified or designed. It is worth noting that the basic reproduction number can be a criterion for the severity and weakness of an epidemic. In this section, to demonstrate the impact of each parameter on controlling or spreading the disease, we obtain the sensitivity index of the basic reproduction number for each parameter, which is used to identify the parameter that has the most significant effect on the disease prevalence.

Definition 7.1. The normalized sensitivity index of $\mathcal{R}_0$ with respect to the parameter δ is equal to

$$C_\delta^{\mathscr{R}_0} = \frac{\partial \mathscr{R}_0}{\partial \delta} \times \frac{\delta}{\mathscr{R}_0}.$$

From the recent formula, it is clear that the sensitivity index regarding each parameter indicates a direct relationship between the parameter changes and disease prevalence. For example, the sensitivity index of $\mathcal{R}_0$ with respect to the vaccination parameter p is represented as:

$$C_p^{\mathscr{R}_0} = \frac{\partial \mathscr{R}_0}{\partial p} \times \frac{p}{\mathscr{R}_0} = \frac{-\beta\lambda v}{(\lambda+\alpha)^2(\lambda+pv)^2} \times \frac{p(\lambda+\alpha)(\lambda+pv)}{\beta\lambda} = \frac{-pv}{\lambda+pv} < 0,$$

which is negative regardless of any initial data. Therefore, as we expect, with the increase in vaccination, we observe a decrease in the basic reproduction number, which ultimately leads to the end of the epidemic. Additionally, the sensitivity index of $\mathcal{R}_0$ with respect to the parameter β, the disease transmission rate, is denoted as:

$$C_\beta^{\mathscr{R}_0} = \frac{\partial \mathscr{R}_0}{\partial \beta} \times \frac{\beta}{\mathscr{R}_0} = \frac{\lambda}{(\lambda+\alpha)(\lambda+pv)} \times \frac{\beta(\lambda+\alpha)(\lambda+pv)}{\beta\lambda} = 1 > 0.$$

In this case, we also see that regardless of any data, the transmission rate has a direct relationship with epidemic prevalence. Comparing the magnitudes of the indices clarifies the impact of each parameter on controlling the epidemic relative to the other parameters.

8. Optimal Control System

Optimal control methods can be used to obtain the minimum number of vaccinated individuals in the community to control the epidemic. Here, we will examine the general steps to form an optimal control system using the control variable p as a function of time, $p : [0,\infty) \to [0,1]$. Therefore, system (3.1) becomes:

$$\begin{cases} x' = \lambda - \dfrac{\beta xy}{g(y)} - (\lambda + vp(t))x, \ x(0) = x_0 \geq 0, \\ y' = \dfrac{\beta xy}{g(y)} - (\lambda+\alpha)y, \ y(0) = y_0 \geq 0, \\ x(T) \text{ and } y(T) \text{ are free}, \end{cases} \tag{8.1}$$

where the terminal time T is the final vaccination time. We introduce the following set as acceptable optimal solutions:

$$\mathcal{A} := \{p \in L^1(0,T) \mid p(t) \in A = [0,1]\}.$$

We apply the optimal control theory to determine the best number of vaccinated individuals to minimize the disease spread and vaccination costs. In particular, we seek a control p^* that minimizes the payoff functional

$$J[p^*] = \min_{p\in\mathcal{A}} J[p] := \min_{p\in\mathcal{A}} \int_0^T \left(\frac{w_1}{2}y^2(t) + \frac{w_2}{2}p^2(t)\right) dt, \tag{8.2}$$

where w_1 and w_2 are some fixed weight coefficients, and that $(x(t), y(t))$ solves (8.1) for the specified control $p(t)$. We first prove the existence of an optimal pair $(X^*(t), p^*(t))$. We use the Filippov-Cesari Existence Theorem given by Theorem 9.1 of [**?**].

Proposition 8.1. *The optimal control problem* (8.1)-(8.2) *has a solution.*

Proof. We consider the set $N(t, X)$ as follows:

$$N(t,X) = \left\{ \left(\frac{w_1}{2} y^2 + \frac{w_2}{2} p^2 + \xi, f(X,p) \right) \ : \ \xi \le 0, \ p \in A = [0,1] \right\},$$

where f is the vector field of the system (8.1) and $X = (x, y) \in \mathbb{R}^2$. We show that $N(t, X)$ is convex for every $(t, X) \in \mathbb{R} \times \mathbb{R}^2$. To do this, we consider $Y_1, Y_2 \in N(t, X)$ and show that for any $a \in [0, 1]$ we have that

$$aY_1 + (1-a)Y_2 \in N(t,X).$$

The fact that $Y_i \in N(t, X)$ for $i = 1, 2$, implies that there exist $\xi_i \le 0$ and control variables $p_i \in [0, 1]$, $i = 1, 2$, such that

$$Y_i = \left(\frac{w_1}{2} y^2 + \frac{w_2}{2} p_i^2 + \xi_i, f(X,p_i) \right), \ i = 1, 2.$$

Then, we have

$$a\left(\frac{w_1}{2} y^2 + \frac{w_2}{2} p_1^2 + \xi_1 \right) + (1-a)\left(\frac{w_1}{2} y^2 + \frac{w_2}{2} p_2^2 + \xi_2 \right) = \frac{w_1}{2} y^2 + \frac{w_2}{2}(ap_1^2 + (1-a)p_2^2) + (a\xi_1 + (1-a)\xi_2).$$

Letting $p_3 = \sqrt{ap_1^2 + (1-a)p_2^2}$, we see that $p_3 \in A = [0, 1]$. Furthermore, letting $\xi_3 = a\xi_1 + (1-a)\xi_2$, we observe that $\xi_3 \le 0$. Thus, the first component satisfies the convexity condition. Next, we check the second component. By a simple computation, we see that $af(X, p_1) + (1-a)f(X, p_2) = f(X, ap_1 + (1-a)p_2)$. Letting $p_4 = ap_1 + (1-a)p_2$, we have that $p_4 \in A = [0, 1]$. This completes the proof of the convexity of $N(t, X)$. Clearly, $A = [0, 1]$ is compact. We also proved the boundedness of the solutions of system (8.1) in Theorem 5.1. Then, by Theorem 9.2 of [**?**], there exists an optimal pair $(X^*(t), p^*(t))$, where $p^* \in \mathcal{A}$. □

After verifying the existence of an optimal solution, it can be found with Pontryagin's principle of minimum (Theorem 9.2 of [**?**]). Considering the time-varying vector $\eta(t) = (\eta_1(t), \eta_2(t)) \in \mathbb{R}^2$ of the Lagrange multipliers, whose elements are called the adjoint variables of the system, we define the Hamiltonian H for all $t \in [0, T]$ as follows:

$$\begin{aligned} H\left(X(t), p(t), \eta(t)\right) &= \frac{w_1}{2} y^2(t) + \frac{w_2}{2} p^2(t) + \sum_{i=1}^{2} \eta_i(t) f_i\left(X(t), p(t)\right) \\ &= \frac{w_1}{2} y^2 + \frac{w_2}{2} p^2 + \eta_1\Big(\lambda - \frac{\beta xy}{g(y)} - (\lambda + vp(t))x\Big) + \eta_2\Big(\frac{\beta xy}{g(y)} - (\lambda + \alpha)y\Big). \end{aligned}$$

The optimal control p^* must be a critical point of the Hamiltonian function. Thus, we must have $\frac{\partial H}{\partial p}(p^*) = 0$. This leads to the following condition on the optimal control:

$$p^*(t) = \frac{v}{w_2} \eta_1(t)\, x(t).$$

On the other hand, we have $\frac{\partial^2 H}{\partial p^2} = w_2 > 0$, which indicates that the critical point p^* is a minimum of the Hamiltonian H. Since p^* must belong to $\mathcal{A}$, we must have

$$p^*(t) = \min\left\{1, \max\left\{0, \frac{v}{w_2} \eta_1(t)\, x(t)\right\}\right\}. \tag{8.3}$$

The adjoint system is given by

$$\begin{cases} \eta_1' = -\frac{\partial H}{\partial x} = (\eta_1 - \eta_2)\frac{\beta y}{g(y)} + \eta_1\left(\lambda + vp^*(t)\right), \\ \eta_2' = -\frac{\partial H}{\partial y} = -w_1 y + \beta x(\eta_1 - \eta_2)\frac{g(y) - yg'(y)}{g^2(y)} + (\lambda + \alpha)\eta_2, \\ \eta_1(T) = \eta_2(T) = 0. \end{cases}$$

TABLE 2. Parameters used in the system (3.1).

parameter	p	v	λ	β	α
value	0.8	0.6	0.3	0.8	0.2

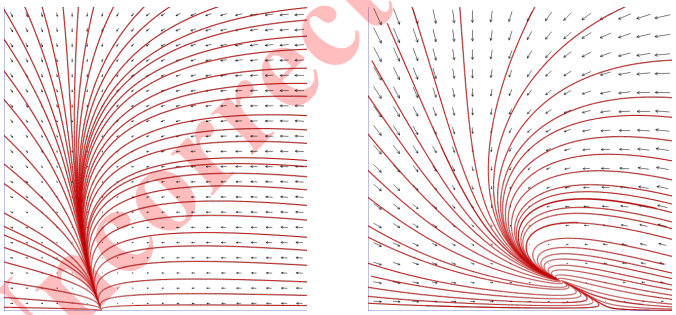
To find the optimal solution, we must solve the following system:

$$\begin{cases} x' = \lambda - \dfrac{\beta xy}{g(y)} - (\lambda + vp^*(t))x, \ x(0) = x_0 \geq 0, \\ y' = \dfrac{\beta xy}{g(y)} - (\lambda + \alpha)y, \ y(0) = y_0 \geq 0, \\ \eta_1' = (\eta_1 - \eta_2)\dfrac{\beta y}{g(y)} + \eta_1\big(\lambda + vp^*(t)\big), \ \eta_1(T) = 0, \\ \eta_2' = -w_1 y + \beta x(\eta_1 - \eta_2)\dfrac{g(y) - yg'(y)}{g^2(y)} + (\lambda + \alpha)\eta_2, \ \eta_2(T) = 0, \end{cases} \tag{8.4}$$

where $p^*(t)$ is given by (8.3). System (8.4) is an optimal control system that cannot be solved manually, and numerical methods must be used. In the next section, this system will be solved numerically using the discretization method implemented in Maple. A concise explanation of this method is provided in Section 9.1.

9. NUMERICAL SIMULATION

To examine the results obtained in the previous sections, we consider the function g in system (3.1) as $g(y) = 1 + y^2$ and the data from Table 2. In this case, $\mathscr{R}_0 = 0.6153846154 < 1$ implies that system (3.1) has only one disease-free equilibrium point $E_0 = (0.3846153846, 0)$, which is a globally stable node, as shown in Figure 2(a).



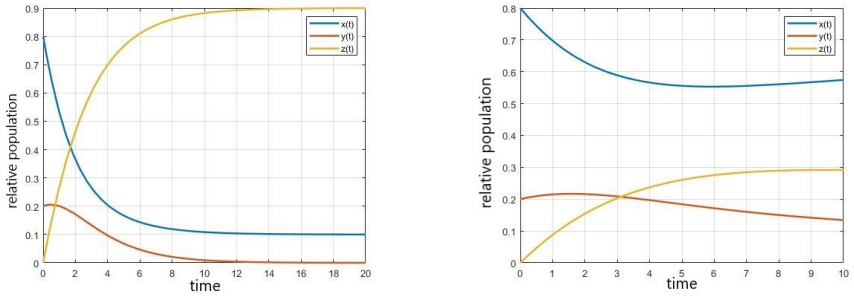
(a) $p = 0.9$. (b) $p = 0.2$.

FIGURE 2. The phase portrait of system (3.1).

Now, we examine system (3.1) with the same data from Table 2, but with only a change, $p = 0.2$. In this case, we have $\mathscr{R}_0 = 1.142857143 > 1$, which, as expected, indicates that system (3.1) has two equilibrium points, $E_0 = (0.7142857143, 0)$ and $E_1 = (0.6282633338, 0.07225879959)$, where E_0 is the disease-free equilibrium point and E_1 is the endemic equilibrium point. E_0 is unstable, and E_1 is a globally stable node, as shown in Figure 2(b).

Figure 3 shows the changes in each of the groups $x(t)$, $y(t)$, and $z(t)$, which represent the relative population of susceptible, infected, and recovered individuals under the initial condition $(x_0, y_0, z_0) = (0.8, 0.2, 0)$.

9.1. **Optimal Control.** To find the optimal trajectories, we simulate the optimal control system (8.4). For this, we utilize the discretization method. First, we consider the data from Table 2 and use the function $g(y) = 1 + y^2$. We set the initial population to $x_0 = 0.8$ and $y_0 = 0.2$, with $w_1 = w_2 = 1$ and the final time $T = 1$.



(a) $p = 0.9$. (b) $p = 0.2$.

FIGURE 3. The graph shows the changes in the solutions of system (3.1).

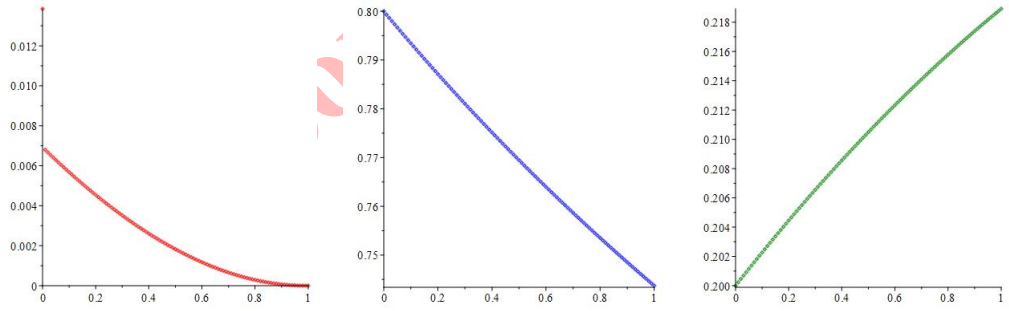
In this method, the integral appearing in (8.2) is approximated using the Trapezoidal-Romberg formula:

$$\int_a^b f(x)\,dx \approx \frac{h}{2}\left[f(x_0) + 2\sum_{i=1}^{n-1} f(x_i) + f(x_n)\right],$$

where h is the step size, which we set to 0.01 for this study. The first and second equations in the system (8.4) are discretized using finite differences to formulate the optimization constraints:

$$\frac{x_i - x_{i-1}}{h} = f_1(x_{i-1}, y_{i-1}, p_{i-1}), \quad \frac{y_i - y_{i-1}}{h} = f_2(x_{i-1}, y_{i-1}), \quad 0 \le x_i, y_i, p_i \le 1,$$

where f_1 and f_2 are the components of the vector field in the system (8.4). The optimal control p is then computed using the command Optimization[NLPSolve](J,s) in Maple, where J is the cost function and s denotes the set of constraints. The optimal control trajectory is illustrated in Figure 4(a). Additionally, the corresponding optimal state trajectories are shown in Figures 4(b) and (c).



(a) Graph of the optimal control $p^*(t)$. (b) Graph of the state variable $x^*(t)$. (c) Graph of the state variable $y^*(t)$.

FIGURE 4. Optimal path diagrams.

10. Conclusion

In this paper, we introduced an SIR model with a nonlinear infection rate assuming 100% vaccine efficacy. We observed that the corresponding system has a disease-free equilibrium point E_0 and an endemic equilibrium point E_1. Corresponding to E_0, we derived the basic reproduction number $\mathcal{R}_0$. The equilibrium point E_0 is globally stable for

$\mathscr{R}_0 < 1$ and unstable for $\mathscr{R}_0 > 1$, and the equilibrium point E_1, if it exists, will be stable. The system possesses a stability exchange bifurcation at $\mathscr{R}_0 = 1$. In addition, by calculating the sensitivity index of $\mathscr{R}_0$, the effects of vaccine-related parameters and disease transmission rate were examined. In the final section, the results were discussed using numerical examples. In that section, the function $g(y)$ was chosen as $1 + y^2$. As expected, the system exhibited only one equilibrium point E_0 when $\mathcal{R}_0 < 1$, while a second equilibrium point E_1 appeared when $\mathcal{R}_0 > 1$. The phase portrait of the system confirms that the stability of these equilibrium points aligns with the theoretical predictions in each case.

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