



Research article

Nonlinear dynamics of reinfection with behavioral protection and saturated treatment: Bifurcation analysis and control insights

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Abstract: Mathematical modeling has been widely used by many researchers to understand the transmission mechanisms of infectious diseases and to evaluate how well different types of interventions work at the population level, including for COVID-19. In the context of COVID-19, many researchers have separately discussed the role of medical masks and the impact of reinfection on disease transmission. However, very few studies have effectively discussed these aspects in detail, particularly the impact of face masks on the reinfection process. Furthermore, many previous studies have discussed standard bifurcation phenomena, such as backward and forward bifurcations or Hopf bifurcations, but have neglected more complex dynamics, such as double limit cycles and cusp bifurcations. The main innovation of this study is the explicit incorporation of mask-induced reductions in both primary infection and reinfection pathways, combined with saturated treatment, and the systematic exploration of the resulting higher-codimension bifurcation structures. The next-generation matrix technique is used to compute the basic reproduction number $\mathcal{R}_0$. Our

analytical results reveal that the COVID-19-free equilibrium is locally asymptotically stable when $\mathcal{R}_0 < 1$ and unstable when $\mathcal{R}_0 > 1$. A center manifold approach is used to show that the system can exhibit either forward or backward bifurcation, depending on the parameter regime, indicating that disease persistence may occur even when $\mathcal{R}_0 < 1$. A numerical bifurcation study reveals several nonlinear dynamic phenomena, including Hopf bifurcations leading to oscillatory outbreaks, endemic bubbles, folds of limit cycles, and codimension-two bifurcations, including cusp and generalized Hopf points. These structures divide the parameter space into regions associated with different epidemiological outcomes. Our global sensitivity analysis, supported by optimal control simulations, reveals the importance of medical masks in reducing COVID-19 reinfection while also illustrating how reinfection may significantly alter the dynamic behavior of the model. These analytical and numerical results show that time-dependent adaptive interventions should be carefully considered to prevent recurrent outbreaks generated by the intrinsic periodic behavior of COVID-19's transmission dynamics and to ensure that the resources allocated to disease-control interventions are managed efficiently.

Keywords: COVID-19; reinfection; mask efficacy; recurrent outbreaks; global sensitivity; optimal control

1. Introduction

The COVID-19 pandemic was caused by the novel coronavirus Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2). More than 7 million deaths were reported from over 30 million reported cases worldwide [1, 2]. It is important to note that these figures are based on reported data, and actual numbers may be higher due to underreporting, limited testing, and differences in death certification practices across regions. COVID-19 is caused by the virus SARS-CoV-2, first identified in Wuhan, China, at the end of 2019. Although COVID-19 can be spread through airborne particles in poorly ventilated spaces and through contact with contaminated surfaces, the infection route is mainly respiratory droplets when an infected person coughs, sneezes, talks, or breathes. The symptoms for COVID-19 are very varied, from light symptoms such as fever or chills, cough, and diarrhea to more severe symptoms such as difficulty in breathing, persistent chest pain or pressure. It can even lead to death in more severe cases in some population groups (such as those with HIV/AIDS and/or tuberculosis, or people with weakened immunity) [3–5].

As of April 2025, the SARS-CoV-2 virus has evolved into numerous variants, with several currently circulating globally. According to the World Health Organization (WHO), notable variants under monitoring include LP.8.1, XEC, KP.3 and KP.3.1.1 [6]. With the emergence of various COVID-19 variants, reinfection has become increasingly common. Reinfection with SARS-CoV-2 occurs when an individual who has exited the infectious/exposed process becomes infected again. After the initial SARS-CoV-2 infection, individuals acquire temporary immunity against the same virus strain, primarily mediated by memory B- and T-cells. However, during this temporal immunity phase, individuals remain susceptible to infection by other respiratory viruses and, under certain conditions, by significantly mutated SARS-CoV-2 variants capable of evading immune memory [7, 8].

During the COVID-19 pandemic, a range of interventions were implemented globally to mitigate

the spread of the virus. These included nonpharmaceutical interventions (NPIs) such as lockdowns, social distancing, mask mandates, and travel restrictions, as well as pharmaceutical measures like vaccination campaigns and therapeutic treatments. The effectiveness of these interventions varied across regions, influenced by factors such as the healthcare infrastructure, public compliance, and timely implementation [9]. As of 2025, the focus has shifted towards sustainable management of COVID-19. The WHO's Global Health Strategy for 2025–2028 emphasizes the importance of leveraging scientific advancements, such as mRNA vaccine platforms, to enhance preparedness and the response to future health emergencies [10]. Additionally, the Centers for Disease Control and Prevention (CDC) has proposed updates to COVID-19 booster recommendations, prioritizing high-risk groups, reflecting a more targeted and sustainable approach to vaccination strategies.

Mathematical models have been used widely to model the spread of diseases since it was first introduced by Kermack and McKendrick in 1927 [11], such as for dengue [12], malaria [13, 14], measles [15], tuberculosis [16–18], HIV/AIDS [19, 20], pneumonia [21], and many other diseases, including COVID-19. A large number of mathematical models have been developed to study the spread of COVID-19, including the effectiveness of several types of intervention to control the spread of COVID-19. The authors of [22] proposed a comprehensive modeling approach in the early stages of the COVID-19 pandemic in 2020 to discuss the relevant question, exploring the potential of face masks during the COVID-19 pandemic. They analyzed the difference of the impact of implementing face masks only on symptomatic individuals versus the whole population. They reveals that with more asymptomatic individuals, in the population, then implementing mask measures to the general population becomes more necessary. In the same year, the authors of [23] also proposed another model to examine the potential of mask use on COVID-19 control efforts. Several incidence data from pre-lockdown and lockdown periods were used to estimate their model parameters. They found that enhanced diagnostic testing combined with universal face mask use significantly reduces COVID-19 transmission and can prevent post-lockdown resurgence and second waves.

A fractional order modeling approach was suggested by the authors of [24] to understand the implementation of face masks in Egypt. Froese and Angel [25] emphasized that even limited mask adoption notably decreased influenza infections, suggesting masks as a cost-effective complement or alternative to vaccination. Similarly, Ma et al. [26] reported that early adoption of masks by just 20% of the population could halve COVID-19 cases in New York City within weeks. Modeling by Ko et al. [27] indicated that while lifting mask mandates may be feasible under certain conditions, continued mask use remains crucial, especially when facing new variants. Darassi et al. [28] highlighted, through data fitting and sensitivity analysis, that mask use could contribute to infection elimination efforts, as evidenced in Saudi Arabia. Asempapa et al. [29] found that preventive measures by low-risk individuals, including mask wearing, significantly reduce infections across risk groups. Kolebaje et al. [30] suggested that maintaining precautionary behaviors, such as face mask use among over half of the susceptible population, could ensure the reproduction number stays below one. Ngonghala et al. [31] showed that widespread use of surgical masks offers greater population-level benefits compared with limited use of more protective N95 masks. In Tokyo, Nagata et al. [32] estimated that declining mask coverage could have led to a considerable increase in COVID-19-related deaths, highlighting the risk of prematurely relaxing mask mandates. Finally, Acheneje et al. [33] demonstrated that intensified mask usage, alongside other interventions, can substantially curb the spread of both COVID-19 and monkeypox. Collectively, these findings affirm

the critical importance of broad and sustained mask usage in pandemic control strategies.

Recently, the authors of [34] proposed a simple susceptible–exposed–infected–recovered (SEIR) model to study the impact of medical mask use together with saturated treatment on the spread of COVID-19. Their analysis highlighted the importance of mask usage and showed the possibility of recurrent outbreaks arising from periodic solutions generated by the model’s structure itself, rather than from sinusoidal or other time-dependent periodic parameters. Building upon the above mentioned references, here, in this research, we propose a refined formulation of the model that incorporates a dual protective role of masks, not only in the primary infection, but also in the incidence of reinfection. This is a simple yet essential modification to the model’s structure. This refinement leads to a more consistent analytical derivation and improves the accuracy of the resulting threshold quantities. Our analysis demonstrates that the interplay of these mechanisms may give rise to backward bifurcation and forward bifurcation with hysteresis at the epidemic threshold, which allows multiple endemic equilibria whether the basic reproduction number is below or above one. We also reveal the emergence of double periodic solutions due to the fold of limit cycles, a phenomenon not reported in previous literature. Furthermore, the identification of codimension-2 bifurcations, including cusp and generalized Hopf points, provides new insights into the mechanisms underlying periodic outbreaks and the transitions from stable equilibria to oscillatory behavior. By explicitly linking these bifurcation structures to epidemiological outcomes such as outbreak frequency, peak size, and interwave periods, our study offers novel policy-relevant guidance on the trade-offs introduced by mask usage and underscores the necessity of combining masks with other interventions to effectively reduce the disease burden.

This paper is organized as follows. In Section 2, we introduce the proposed model modifications together with the underlying assumptions and rationale. Section 3 presents the derivation of the COVID-19-free equilibrium and the basic reproduction number, while Section 4 provides a detailed analysis of the existence of endemic equilibria. In Section 5, we demonstrate the possible occurrence of backward bifurcation in the model. Section 6 is devoted to numerical experiments supporting our analytical results, ranging from numerical continuation to a global sensitivity analysis. The extension of the model using an optimal control approach is described in Section 7, where we characterize the optimal control problem and perform numerical simulations using the forward backward sweep method. Finally, Section 8 concludes the paper with a summary of the main findings and concluding remarks.

2. Mathematical model

Let the human population be classified into four different population groups according to their health status, namely the susceptible population ($S(t)$), which consists of individuals who are vulnerable to COVID-19; the exposed population ($E(t)$), which consists of individuals who have already been infected with COVID-19 but are not yet able to transmit the disease to others; the infected population ($I(t)$), which consists of infected individuals who have completed their incubation period and are able to transmit COVID-19 to others; and the removed and temporarily/partially protected class population ($R(t)$), which consists of individuals who have exited the infectious or exposed stage—either through abortive clearance of the infection or recovery following active infection—but who retain only partial and temporary immunity and therefore remain susceptible to reinfection. Hence, the total population

$N(t)$ is given by

$$N(t) = S(t) + E(t) + I(t) + R(t).$$

The model construction process is described below.

Demographic process

First of all, we assume that the total population grows only through the natural recruitment of newborns at a rate Λ , and that all newborns enter the susceptible population. Furthermore, we assume that migration is neglected in this model. We have that each compartment has the same natural death rate μ , while individuals in compartment I has an additional death rate due to COVID-19, denoted δ .

Primary infection process

The infection process occurs through close contact between susceptible individuals S and infectious individuals I , with a transmission rate denoted by β . In this study, the mask-related reduction is interpreted from the source-control perspective. Hence, we assume that a constant proportion c_m of infectious individuals wear face masks. The parameter ϵ_{m_1} represents the efficacy of masks worn by infectious individuals in reducing transmission to susceptible individuals. In particular, $\epsilon_{m_1} = 1$ means that masks are fully effective in preventing primary transmission from masked infectious individuals. Therefore, the effective primary infection rate is reduced, and the infection term becomes $(1 - \epsilon_{m_1} c_m) \beta S I$. Newly infected individuals are not immediately infectious; instead, they undergo an incubation period. As a result, new infections enter the exposed compartment E and subsequently progress to the infected compartment I at a rate ν .

Recovery process

We assume that both exposed and infected individuals can recover, but at different recovery rates. For exposed individuals, we assume a constant natural recovery rate due to good early immunity denoted θ . Biologically, this pathway can be interpreted as abortive infection, in which early immune responses clear the virus before the individual becomes infectious or develops a detectable infection. Such a mechanism is supported by studies reporting SARS-CoV-2-exposed seronegative individuals with virus-specific T-cell responses, suggesting that some exposed individuals may clear the infection before seroconversion or detectable infection [35]. However, such abortive infections are not necessarily associated with the development of strong or long-lasting immunity, as the durability and protective capacity of T-cell responses generated during abortive infection remain uncertain and appear more limited than those following overt infection [35,36]. For this reason, we do not interpret individuals entering compartment R through this pathway as fully immune; instead, as mentioned before, R is defined more broadly as a *removed and temporarily/partially protected* class, encompassing both individuals who clear the infection abortively and those who recover from active infection through treatment. In contrast, infected individuals recover at a rate given by $\gamma + \frac{\rho}{1 + \kappa I}$, where γ is the baseline recovery rate, and ρ represents the maximum additional recovery rate due to treatment. This recovery rate is modeled as a monotonically decreasing function to reflect the saturation effect in hospitals as the number of infected individuals, I , increases. As I grows, the term $\frac{\rho}{1 + \kappa I}$ decreases, indicating that the additional benefit from treatment diminishes with higher

infection levels.

Reinfection process

We assume that individuals in the removed compartment R possess only partial and temporary immunity, regardless of whether they entered R through abortive infection or through recovery from active infection. Because this protection is incomplete, individuals in R may undergo two distinct processes. First, immunity may wane entirely over time, in which case individuals return to the fully susceptible compartment S at a waning rate η . Second, individuals may become reinfected while still retaining some degree of partial immunity; this corresponds to a direct transition from R to E . Importantly, this reinfection process does not require the assumption of an antigenically distinct or immune-escaping variant; rather, the term ζRI captures the reduced but nonzero susceptibility of removed individuals to reinfection by SARS-CoV-2, reflecting the imperfect nature of the immunity acquired through abortive infection or recovery [37]. Since primary infections and reinfections are assumed to progress through the same latent period, disease progression, and recovery dynamics, reinfected removed individuals enter the same exposed compartment E as primary infections. Consistent with the source-control interpretation used for primary infection, c_m denotes the proportion of infectious individuals wearing face masks, while ϵ_{m_2} represents the efficacy of those masks in reducing transmission to removed individuals. Consequently, the reinfection term is modeled as $(1 - \epsilon_{m_2} c_m) \zeta RI$.

Based on the above model description, the mathematical model for COVID-19 transmission considering reinfection, face mask use, and saturated recovery rate is given by

$$\frac{dS}{dt} = \Lambda - k_1 \beta S I + \eta R - \mu S, \quad (2.1a)$$

$$\frac{dE}{dt} = k_1 \beta S I - \nu E - \theta E - \mu E + k_2 \zeta RI, \quad (2.1b)$$

$$\frac{dI}{dt} = \nu E - \delta I - \mu I - \left(\gamma + \frac{\rho}{1 + \kappa I} \right) I, \quad (2.1c)$$

$$\frac{dR}{dt} = \theta E + \left(\gamma + \frac{\rho}{1 + \kappa I} \right) I - \eta R - \mu R - k_2 \zeta RI, \quad (2.1d)$$

where $k_1 = 1 - c_m \epsilon_{m_1}$ and $k_2 = 1 - c_m \epsilon_{m_2}$. A description of the model's parameters is given in Table 1.

Table 1. Description of the parameter values in System (2.1).

Parameter	Description	Value	Unit
Λ	Recruitment of human	$\Lambda > 0$	$\frac{\text{human}}{\text{day}}$
β	Transmission rate	$\beta > 0$	$\frac{1}{\text{day} \times \text{human}}$
ϵ_{m_1}	Masks' efficacy to prevent COVID-19 infection	$\epsilon_{m_1} \in [0, 1]$	Nondimensional
c_m	Proportion of infected individuals who use face masks	$c_m \in [0, 1]$	Nondimensional
η	Dropout rate due to the loss of temporary immunity	$\eta > 0$	$\frac{1}{\text{day}}$
θ	Removed rate of exposed individuals due to abortive infection	$\theta > 0$	$\frac{1}{\text{day}}$
μ	Natural death rate	$\mu > 0$	$\frac{1}{\text{day}}$
δ	Death rate due to disease	$\delta > 0$	$\frac{1}{\text{day}}$
ν	Transition from E to I due to incubation of the virus	$\nu > 0$	$\frac{1}{\text{day}}$
ζ	Reinfection rate	$\zeta > 0$	$\frac{1}{\text{day} \times \text{human}}$
γ	Natural recovery rate of infected I individuals	$\gamma > 0$	$\frac{1}{\text{day}}$
ρ	Additional recovery rate	$\rho > 0$	$\frac{1}{\text{day}}$
κ	Saturation parameter of additional recovery rate	$\kappa > 0$	$\frac{1}{\text{human}}$
ϵ_{m_2}	Mask efficacy to prevent reinfection with COVID-19.	$\epsilon_{m_2} \in [0, 1]$	Nondimensional

2.1. Preliminary analysis of the basic properties of the model

To ensure that the proposed epidemic model (2.1) is biologically meaningful and mathematically well-posed, it is essential to analyze the properties of the solutions. In this section, we establish the existence, uniqueness, non-negativity, and boundedness of the solutions.

Existence and uniqueness of solutions

Theorem 1. For any given initial condition $X(0) = (S(0), E(0), I(0), R(0))^T \in \mathbb{R}_+^4$, there is a unique local solution $X(t) = (S(t), E(t), I(t), R(t))^T$ to the System (2.1) for all $t \geq 0$.

Proof. Let the System (2.1) be rewritten in the vector form $\frac{dX}{dt} = f(X)$, where $X = (S, E, I, R)^T$ and $f(X) = (f_1, f_2, f_3, f_4)^T$ represents the right-hand side functions of the system

$$\begin{aligned}
 f_1(X) &= \Lambda - k_1\beta SI + \eta R - \mu S, \\
 f_2(X) &= k_1\beta SI - \nu E - \theta E - \mu E + k_2\zeta RI, \\
 f_3(X) &= \nu E - \delta I - \mu I - \left(\gamma + \frac{\rho}{1 + \kappa I}\right)I, \\
 f_4(X) &= \theta E + \left(\gamma + \frac{\rho}{1 + \kappa I}\right)I - \eta R - \mu R - k_2\zeta RI.
 \end{aligned}$$

Note that the denominator in the treatment function is $1 + \kappa I$. Since we consider the non-negative region where $I \geq 0$, the denominator never vanishes ($1 + \kappa I \neq 0$). All the component functions f_i are

continuously differentiable with respect to the state variables S, E, I , and R . Consequently, all partial derivatives $\frac{\partial f_i}{\partial x_j}$ (where $x_j \in \{S, E, I, R\}$) exist and are continuous on $\mathbb{R}_+^4$. Since the vector field $f(X)$ is $C^1(\mathbb{R}_+^4)$, it is locally Lipschitz. By the Picard–Lindelöf Theorem (see [38]), the System (2.1) admits a unique maximal solution for every non-negative initial condition. Since the solution remains bounded on every finite interval, the maximal solution is global. $\square$

Positivity of solutions

Theorem 2. *Given the initial conditions satisfy $S(0) \geq 0, E(0) \geq 0, I(0) \geq 0, R(0) \geq 0$, then the solutions $S(t), E(t), I(t)$, and $R(t)$ of System (2.1) will remain non-negative for all $t > 0$.*

Proof. Evaluating the vector field on each coordinate hyperplane gives:

- From Eq (2.1a), when $S = 0$, we have $\frac{dS}{dt}\big|_{S=0} = \Lambda + \eta R > 0$ since $\Lambda > 0$ and $R \geq 0$.
- From Eq (2.1b), when $E = 0$, we have $\frac{dE}{dt}\big|_{E=0} = k_1 \beta S I + k_2 \zeta R I \geq 0$ since $S, I, R \geq 0$.
- From Eq (2.1c), when $I = 0$, we have $\frac{dI}{dt}\big|_{I=0} = \nu E \geq 0$ since $E \geq 0$.
- From Eq (2.1d), when $R = 0$, we have $\frac{dR}{dt}\big|_{R=0} = \theta E + \left(\gamma + \frac{\rho}{1+\kappa I}\right) I \geq 0$ since $E, I \geq 0$.

Since the vector field points inward (or is tangent) on every boundary hyperplane, any trajectory originating within the non-negative orthant $\mathbb{R}_+^4$ cannot cross the coordinate axes into the negative regions. Thus, the solutions remain non-negative for all $t \geq 0$. $\square$

Boundedness of the solutions

Theorem 3. *All solutions of System (2.1) that initiate in $\mathbb{R}_+^4$ are uniformly bounded within a biologically feasible region Ω .*

Proof. Let $N(t) = S(t) + E(t) + I(t) + R(t)$ denote the total population at time t . Summing all the equations in System (2.1), the interaction terms cancel out, yielding

$$\begin{aligned} \frac{dN}{dt} &= \frac{dS}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dR}{dt} \\ &= \Lambda - \mu S - \mu E - \mu I - \mu R - \delta I \\ &= \Lambda - \mu N - \delta I. \end{aligned}$$

Since the death rate due to disease is $\delta \geq 0$ and the infected population $I(t) \geq 0$ for all $t \geq 0$, we can establish the following differential inequality:

$$\frac{dN}{dt} \leq \Lambda - \mu N.$$

and we obtain

$$N(t) \leq N(0)e^{-\mu t} + \frac{\Lambda}{\mu} (1 - e^{-\mu t}).$$

Taking the limit superior as $t \rightarrow \infty$, we get

$$\limsup_{t \rightarrow \infty} N(t) \leq \frac{\Lambda}{\mu}.$$

This indicates that the total population $N(t)$ is uniformly bounded. Furthermore, if $N(0) \leq \frac{\Lambda}{\mu}$, then $N(t) \leq \frac{\Lambda}{\mu}$ for all $t \geq 0$. Therefore, the biologically feasible region for System (2.1) is given by the compact, positively invariant set

$$\Omega = \left\{ (S, E, I, R) \in \mathbb{R}_+^4 : S + E + I + R \leq \frac{\Lambda}{\mu} \right\}.$$

This completes the proof. $\square$

3. The COVID-19-free equilibrium and the basic reproduction number

The COVID-19 model represented in System (2.1) has two types of equilibrium. The first equilibrium is the COVID-19-free equilibrium point, denoted $\mathcal{E}_0$, and the COVID-19 endemic equilibrium point, denoted $\mathcal{E}_*$, which will be discussed Section 4. Setting the right-hand side of System (2.1) equal to zero, $\mathcal{E}_0$ is given by

$$\mathcal{E}_0 = (S_0, E_0, I_0, R_0) = \left(\frac{\Lambda}{\mu}, 0, 0, 0 \right). \quad (3.1)$$

For the calculation of the basic reproduction number (denoted $\mathcal{R}_0$) of Model (2.1), we use the next-generation matrix approach formula given in [39]. The transition (Σ) and transmission (T) matrix from the infected subcompartment are given by

$$\Sigma = \begin{bmatrix} -\nu - \mu - \theta & 0 \\ \nu & -\mu - \delta - \gamma - \rho \end{bmatrix}, \quad \mathbf{T} = \begin{bmatrix} 0 & \frac{\beta k_1 \Lambda}{\mu} \\ 0 & 0 \end{bmatrix}.$$

Since the second row of T is 0, then we define $M = \begin{bmatrix} 1 \\ 0 \end{bmatrix}$ which spans every column of $\mathbf{T}$, and the next-generation matrix is given by

$$\begin{aligned} \mathbf{K} &= -\mathbf{M}^T \mathbf{T} \Sigma^{-1} \mathbf{M}, \\ &= \left[\frac{\beta k_1 \Lambda \nu}{\mu (\nu + \mu + \theta) (\mu + \delta + \gamma + \rho)} \right]. \end{aligned}$$

Hence, the basic reproduction number of System (2.1), which is taken from the spectral radius of $\mathbf{K}$, is given by

$$\mathcal{R}_0 = \frac{\beta k_1 \Lambda \nu}{\mu (\nu + \mu + \theta) (\mu + \delta + \gamma + \rho)}. \quad (3.2)$$

Local stability of $\mathcal{E}_0$

To analyze the local stability of $\mathcal{E}_0$, we calculate the linearization of System (2.1) at $\mathcal{E}_0$ which yields

$$\mathcal{J}(\mathcal{E}_0) = \begin{bmatrix} -\mu & 0 & -\frac{\Lambda \beta k_1}{\mu} & \eta \\ 0 & -\nu - \mu - \theta & \frac{\Lambda \beta k_1}{\mu} & 0 \\ 0 & \nu & -\mu - \delta - \gamma - \rho & 0 \\ 0 & \theta & \gamma + \rho & -\eta - \mu \end{bmatrix}.$$

The eigenvalues of $\mathcal{J}(\mathcal{E}_0)$ are $-\mu, -(\mu+\eta)$, while the other two are taken from the roots of the following characteristic polynomial

$$\lambda^2 + (\gamma + \delta + 2\mu + \nu + \rho + \theta)\lambda + (1 - \mathcal{R}_0)(\nu + \mu + \theta)(\mu + \delta + \gamma + \rho) = 0.$$

It can be seen that all coefficient of the polynomial above will be positive if $\mathcal{R}_0 > 1$. Hence, $\mathcal{E}_0$ will be locally asymptotically stable if $\mathcal{R}_0 > 1$. This result is represented in the following theorem.

Theorem 4. *The COVID-19 free equilibrium $\mathcal{E}_0$ of System (2.1) is always locally asymptotically stable if $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$.*

4. The COVID-19 endemic equilibrium point

The next equilibrium of System (2.1) is the COVID-19 endemic equilibrium, denoted $\mathcal{E}_*$. This equilibrium is given by

$$\mathcal{E}_* = (S, E, I, R) = (S_*, E_*, I_*, R_*), \quad (4.1)$$

where

$$\begin{aligned} S_* &= \frac{1}{\beta k_1 I_*} ((\nu + \mu + \theta)E_* - \zeta R_* I_*), \\ E_* &= \frac{1}{\nu} \left((\mu + \delta + \gamma)I_* + \frac{\rho I_*}{1 + \kappa I_*} \right), \\ R_* &= \frac{1}{\mu + \eta + \zeta I_*} \left(\theta E_* + \gamma I_* + \frac{\rho I_*}{1 + \kappa I_*} \right), \end{aligned}$$

where I_* is the positive solution of the following polynomial:

$$\mathcal{V}(I) = A_3 I^3 + A_2 I^2 + A_1 I + A_0 = 0, \quad (4.2)$$

where

$$\begin{aligned} A_3 &= \beta k_1 \kappa \zeta (\mu^2 + (\nu + \delta + \gamma)\mu + \nu\delta) k_2, \\ A_2 &= \kappa (k_1 \beta + k_2 \zeta) \mu^3 + (\beta ((\theta + \nu + \eta + \delta + \gamma) \kappa + k_2 \zeta) k_1 + k_2 \zeta \kappa (\nu + \delta + \gamma)) \mu^2 \\ &\quad + (((\eta + \delta + \gamma) \nu + (\delta + \gamma) (\theta + \eta)) \kappa + k_2 \zeta (\nu + \rho + \delta + \gamma)) \beta k_1 + k_2 \nu \zeta \delta \kappa \mu \\ &\quad - k_1 \nu ((\Lambda \zeta k_2 - \delta \eta) \kappa - k_2 \zeta \delta) \beta, \\ A_1 &= \kappa \mu^4 + (k_1 \beta + (\theta + \nu + \eta + \delta + \gamma) \kappa + k_2 \zeta) \mu^3 + (\beta (\theta + \nu + \eta + \rho + \delta + \gamma) k_1) \\ &\quad + ((\eta + \delta + \gamma) \nu + (\theta + \delta + \gamma) \eta + \theta (\delta + \gamma)) \kappa + k_2 \zeta (\nu + \rho + \delta + \gamma) \mu^2 \\ &\quad + ((-\Lambda \nu \kappa + (\eta + \rho + \delta + \gamma) \nu + (\rho + \delta + \gamma) (\theta + \eta)) \beta k_1 + \eta (\delta + \gamma) (\nu + \theta) \kappa + \delta \nu \zeta k_2) \mu \\ &\quad - k_1 \nu \beta (\Lambda \eta \kappa + \Lambda \zeta k_2 - \delta \eta), \\ A_0 &= (\eta + \mu) \mu (1 - \mathcal{R}_0) (\nu + \mu + \theta) (\mu + \delta + \gamma + \rho). \end{aligned}$$

Since $A_3 > 0$, when $A_0 < 0 \Leftrightarrow \mathcal{R}_0 > 1$, we will always have at least one positive root that satisfies $\mathcal{V}(I) = 0$; hence, we have the following theorem.

Theorem 5. *System (2.1) always has at least one COVID-19 endemic equilibrium if $\mathcal{R}_0 > 1$.*

Since the polynomial $\mathcal{V}(I)$ is cubic, it is necessary to analyze the possible existence of multiple endemic equilibria. To calculate the possible number of positive root of $\mathcal{V}(I)$, we use Descartes' rule of sign table as shown in Table 2. It can be seen clearly that whenever $\mathcal{R}_0 > 1$, the model always has at least one COVID-19 endemic equilibrium, and it may have three endemic equilibria if $A_3, A_1 > 0$, and $A_2, A_0 < 0$. On the other hand, in the case of $\mathcal{R}_0 < 1$, it is not always the case that we will have no COVID-19 endemic equilibrium. Once it appears when $\mathcal{R}_0 < 1$, then we will always have two $\mathcal{E}_*$ at the same time for some specific value of $\mathcal{R}_0$. Hence, it is necessary to analyze further on the number of endemic equilibrium around $\mathcal{R}_0 = 1$. For this purpose, we conduct a gradient analysis at the branching point (BP) $\mathcal{R}_0 = 1, I = 0$. If the gradient is negative, then we will have $\mathcal{E}_*$ for some specific value of $\mathcal{R}_0$.

Table 2. Possible number of positive roots of $\mathcal{V}(I) = 0$.

Case of $\mathcal{R}_0$	A_3	A_2	A_1	A_0	Number of sign changes	Possible number of positive roots
$\mathcal{R}_0 < 1$	+	+	-	+	2	0 or 2
$\mathcal{R}_0 < 1$	+	-	+	+	2	0 or 2
$\mathcal{R}_0 < 1$	+	-	-	+	2	0 or 2
$\mathcal{R}_0 < 1$	+	+	+	+	0	0
$\mathcal{R}_0 > 1$	+	+	-	-	1	1
$\mathcal{R}_0 > 1$	+	-	+	-	3	1 or 3
$\mathcal{R}_0 > 1$	+	-	-	-	1	1
$\mathcal{R}_0 > 1$	+	+	+	-	1	1

Gradient analysis at the BP $\mathcal{R}_0 = 1, I = 0$

The first step to conduct this gradient analysis, we will rewrite $\mathcal{V}(I)$ explicitly as a function of $\mathcal{R}_0$ and I by substituting $\beta(\mathcal{R}_0)$ into $\mathcal{V}(I)$, where $\beta(\mathcal{R}_0)$ is taken from the equation of $\mathcal{R}_0$ in (4.1). From direct calculation, we have

$$\beta(\mathcal{R}_0) = \frac{\mu(\mu + \delta + \gamma + \rho)(\nu + \mu + \theta)}{\Lambda \nu k_1}.$$

Substituting this $\beta(\mathcal{R}_0)$ into $\mathcal{V}(I)$ gives

$$A_3(\mathcal{R}_0)I^3 + A_2(\mathcal{R}_0)I^2 + A_1(\mathcal{R}_0)I + A_0(\mathcal{R}_0) = 0. \quad (4.3)$$

Taking the implicit derivative of I respect to $\mathcal{R}_0$ of the equation above gives

$$\begin{aligned} & \frac{\partial A_3(\mathcal{R}_0)}{\partial \mathcal{R}_0} I^3 + 3A_3(\mathcal{R}_0)I^2 \frac{\partial I}{\partial \mathcal{R}_0} + \frac{\partial A_2(\mathcal{R}_0)}{\partial \mathcal{R}_0} I^2 \\ & + 2A_2(\mathcal{R}_0)I \frac{\partial I}{\partial \mathcal{R}_0} + \frac{\partial A_1(\mathcal{R}_0)}{\partial \mathcal{R}_0} I + A_1(\mathcal{R}_0) \frac{\partial I}{\partial \mathcal{R}_0} \\ & + \frac{\partial A_0(\mathcal{R}_0)}{\partial \mathcal{R}_0} = 0. \end{aligned}$$

Substituting $I = 0, \mathcal{R}_0 = 1$ into the equation above and solving it with respect to $\frac{\partial I}{\partial \mathcal{R}_0}$ yields

$$\left. \frac{\partial I}{\partial \mathcal{R}_0} \right|_{I=0, \mathcal{R}_0=1} = (\eta + \mu) \mu (\nu + \mu + \theta) (\mu + \delta + \gamma + \rho) \frac{1}{A_1(1)}, \quad (4.4)$$

where

$$A_1(1) = \nu\Lambda(c_m\epsilon_{m2} - 1)(\delta\theta + \gamma\nu + \theta\gamma + \mu\theta + \nu\rho + \rho\theta)\mu\zeta - (\nu + \mu + \theta)\mu \dots, \\ \left(-\mu(\mu + \rho + \delta + \gamma)^2(\eta + \mu + \theta) - \nu(\mu^3 + (\eta + 2\rho + 2\delta + 2\gamma)\mu^2, \right. \\ \left. (\delta^2 + (2\eta + 2\rho + 2\gamma)\delta + (\rho + \gamma)\eta + \rho^2 + (-\Lambda\kappa + 2\gamma)\rho + \gamma^2)\mu, \right. \\ \left. \eta(\delta^2 + (\rho + \gamma)\delta - \rho\kappa\Lambda)\right)).$$

It can be seen clearly that $A_1(1)$ can be positive or negative. Hence, it is possible to have $\frac{\partial I}{\partial \mathcal{R}_0}\Big|_{I=0, \mathcal{R}_0=1}$ to be positive or negative, depending on the sign of $A_1(1)$.

On the basis of this calculation and results from Descartes' rules of sign in Table 2, we have the following theorem.

Theorem 6. *If $A_1(\mathcal{R}_0 = 1) < 0$, then System (2.1) will have two endemic equilibrium for some values at $\mathcal{R}_0 < 1$.*

Applying the Cardano formula [40], we get the discriminant of the cubic polynomial $\mathcal{V}(I)$ is given by

$$\Delta = 18A_0A_1A_2A_3 - 4A_1^3A_3 - 4A_0A_2^3 + A_1A_2^2 - 27A_0^2A_3^2. \quad (4.5)$$

If the equation $\Delta = 0$ has solutions within a non-negative parameter domain, then the curves formed in the two-parameter plane represent a fold bifurcation curve. If two of these curves intersect tangentially, their intersection points can be considered as potential candidates for cusp bifurcation points. We will demonstrate the detection of these cusp bifurcations numerically in Section 6.3.

5. Existence of backward bifurcation

From Theorem 6, we have shown the possibility of the existence of endemic equilibrium $\mathcal{E}_*$ for some values of $\mathcal{R}_0 < 1$. However, the theorem only discusses about the existence of the endemic equilibrium, not the stability. Hence, here, in this section, we analyze the stability of $\mathcal{E}_*$ around $\mathcal{R}_0 = 1$ using the Castillo–Song bifurcation theorem [41].

To apply the results from [41], we rewrite System (2.1) by taking $S = x_1, E = x_2, I = x_3$, and $R = x_4$. Hence, we have

$$g_1 = \frac{dx_1}{dt} = \Lambda - k_1\beta x_1x_3 + \eta x_4 - \mu x_1, \quad (5.1a)$$

$$g_2 = \frac{dx_2}{dt} = k_1\beta x_1x_3 - \nu x_2 - \theta x_2 - \mu x_2 + k_2\zeta x_4x_3, \quad (5.1b)$$

$$g_3 = \frac{dx_3}{dt} = \nu x_2 - \gamma x_3 - \delta x_3 - \mu x_3 - \frac{\rho x_3}{1 + \kappa x_3}, \quad (5.1c)$$

$$g_4 = \frac{dx_4}{dt} = \theta x_2 + \gamma x_3 + \frac{\rho x_3}{1 + \kappa x_3} - \eta x_4 - \mu x_4 - k_2\zeta x_4x_3. \quad (5.1d)$$

From previous theorems, we can argue that $\mathcal{R}_0 = 1$ as the BP of our system. Hence, we choose β as the bifurcation parameter. Solving the expression of $\mathcal{R}_0 = 1$ respect to β , we have

$$\beta^* = \frac{\mu(\mu + \delta + \gamma + \rho)(\nu + \mu + \theta)}{\Lambda\nu k_1}, \quad (5.2)$$

By substituting β^* and $\mathcal{E}_0$ on the Jacobian matrix of System (5.1), we have

$$\mathcal{A} = \begin{bmatrix} -d & 0 & -\frac{(d + \delta + \gamma + \rho)(v + d + \mu)}{\nu} & \eta \\ 0 & -(v + d + \mu) & \frac{(d + \delta + \gamma + \rho)(v + d + \mu)}{\nu} & 0 \\ 0 & \nu & -(d + \delta + \gamma + \rho) & 0 \\ 0 & \mu & \gamma + \rho & -(d + \eta) \end{bmatrix}. \quad (5.3)$$

Calculating the eigenvalues of $\mathcal{A}$, we have $\lambda = 0$ appears as simple zero eigenvalue, while the other three are negative, i.e., $-\mu$, $-(\eta + \mu)$, and $-(\gamma + \delta + \nu + \rho + 2\mu)$. Hence, A center manifold approach was used to analyze the local stability of our System (5.1) around $\mathcal{R}_0 = 1$. To do this, next, we calculate the left and right eigenvector of $\mathcal{A}$ respect to eigenvalue 0. By direct calculation, we have the left eigenvectors are

$$\mathbf{v} = [v_1 \ v_2 \ v_3 \ v_4] = \begin{bmatrix} 0 & v_2 & \frac{(\nu + \mu + \theta)v_2}{\nu} & 0 \end{bmatrix}.$$

Similarly, the right eigenvectors are

$$\mathbf{w} = [w_1 \ w_2 \ w_3 \ w_4]^T,$$

where

$$w_1 = -\frac{w_3}{\mu\nu(\eta + \mu)} \left(\mu^3 + (\nu + \eta + \rho + \delta + \gamma + \theta)\mu^2 + [(\nu + \rho + \delta + \gamma)\eta + (\rho + \delta + \gamma)(\nu + \theta)]\mu + \delta\eta\nu \right),$$

$$w_2 = \frac{(\mu + \delta + \gamma + \rho)w_3}{\nu}, \quad w_3 = w_3, \quad w_4 = \frac{((\mu + \delta + \gamma + \rho)\theta + \nu(\gamma + \rho))w_3}{(\eta + \mu)\nu}.$$

Having the left and right eigenvectors of $\mathcal{A}$ with respect to the eigenvalue 0 in hand, we are ready to calculate the bifurcation coefficient $\mathcal{A}$ and $\mathcal{B}$ from [41] which are given by the following formulas

$$\mathcal{A} = \sum_{k,i,j=1}^4 v_k w_i w_j \frac{\partial^2 g_k}{\partial x_i \partial x_j}(\mathbf{0}, 0),$$

$$\mathcal{B} = \sum_{k,i=1}^4 v_k w_i \frac{\partial^2 g_k}{\partial x_i \partial \beta}(\mathbf{0}, 0).$$

From direct calculation, we have

$$\mathcal{A} = 2\beta v_2 w_1 w_3 k_1 + 2\kappa \rho v_3 w_3^2 + 2v_2 w_3 w_4 \zeta k_2.$$

Furthermore

$$\mathcal{B} = k_1 \Lambda \mu \nu^2 (\mu + \eta) > 0.$$

Since $\mathcal{B}$ is always positive, while $\mathcal{A}$ could be positive or negative, then our model (2.1) could undergo forward or backward bifurcation at $\mathcal{R}_0 = 1$ depends on the value of $\mathcal{A}$. This results stated in the following theorem.

Theorem 7. *System (2.1) will*

- (1) *Undergo a forward bifurcation at $\mathcal{R}_0 = 1$ if $\mathcal{A} < 0$,*
- (2) *Undergo a backward bifurcation at $\mathcal{R}_0 = 1$ if $\mathcal{A} > 0$.*

Epidemiological consequences of Theorem 7

The emergence of backward bifurcation in our COVID-19 model, as described in System (2.1), leads to several important consequences. First, reducing $\mathcal{R}_0$ below one is no longer sufficient to eliminate COVID-19 from the population. Although the COVID-19-free equilibrium is locally stable when $\mathcal{R}_0 < 1$, a stable endemic equilibrium may also exist simultaneously. Second, unlike traditional models with forward bifurcation—where the disease-free equilibrium is globally stable when $\mathcal{R}_0 < 1$ —the presence of backward bifurcation makes the outcome dependent on the initial conditions. This results in a hysteresis effect, making it more difficult to eliminate COVID-19 once it becomes established, even if interventions are introduced later. Lastly, in terms of intervention strategies, a stronger or combined set of interventions is required to drive the disease toward elimination. Therefore, policymakers must carefully design control strategies to steer the system toward the desired equilibrium.

6. Numerical investigation of bifurcation and global sensitivity analysis

6.1. Parameter estimation

Table 3. Parameter values and references.

Par.	Interval values	References	Baseline
Λ	(0, 5)	Assumption	3.5
μ	$\left[\frac{1}{77.18 \times 365}, 0.01 \right]$	Assumption	0.009
β	$[1 \times 10^{-9}, 0.4251]$	[42, 43]	0.003601
c_m	[0, 1]	[34, 42]	0.1
ϵ_{m_1}	[0, 1]	[34, 42]	0.5
ϵ_{m_2}	[0, 1]	Assumption	0.5
η	(0, 1]	[34, 44, 45]	0.0001
ν	$\left[\frac{1}{14}, 1 \right]$	[22, 46, 47]	0.8
θ	[0, 0.2]	[48]	$\frac{1}{7}$
ζ	$[6.3 \times 10^{-5}, 0.333]$	[43, 49]	0.00013
δ	[0, 0.5]	[50–52]	0.014
γ	$\left[\frac{1}{30}, \frac{1}{3} \right]$	[22, 46, 47]	$\frac{1}{14}$
κ	[0, 1)	[34]	0.05
ρ	$\left[\frac{1}{30}, \frac{1}{3} \right]$	Assumption	0.2

In this study, the parameter values are drawn from previously published and peer-reviewed works where rigorous data calibration has been performed in the context of COVID-19 transmission. This approach allows the model to operate within biologically validated ranges that have been systematically estimated in the literature. This methodological choice is intentional. Since the

emergence of COVID-19 in late 2019, extensive modeling efforts have been devoted to parameter estimation using diverse dataset across countries, time periods, and different strains of viruses. As a result, key epidemiological parameters, such as the transmission rate, incubation period, recovery rates, and so on, have been repeatedly estimated and validated in the literature. To assess robustness, sensitivity analysis are performed in the following subsection using partial rank correlation coefficient (PRCC) method combined with Latin hypercube sampling (LHS).

6.2. Global sensitivity analysis

In this paper, we cite several references for the possible interval values of our model parameters. Therefore, it is important to understand the effect of the redundancy of model parameter values not only on the outcome of the basic reproduction number, but also on the model's behavior. Hence, in this section, we conduct a global sensitivity analysis using the PRCC combined with LHS on our model in System (2.1) and on the expression of our basic reproduction number $\mathcal{R}_0$. This approach has been widely used in many epidemic models to measure the relationship between model inputs and outputs, while taking into account parameter uncertainty with an efficient computational effort [53]. The PRCC values provide insight into how variations in the model parameters influence the trajectories of the state variables $S(t)$, $E(t)$, $I(t)$, and $R(t)$, as well as the threshold quantity $\mathcal{R}_0$, which determines the potential for disease invasion or persistence. In total, 30,000 parameter sets were generated using LHS within $\pm 25\%$ of their respective baseline values. We use the baseline parameter values as shown in Table 3. Numerical simulations were performed for each sample to compute time-dependent PRCCs for all compartments, and a one-time PRCC analysis was carried out to identify the most influential parameters affecting $\mathcal{R}_0$.

Global sensitivity analysis of $\mathcal{R}_0$

Figure 1 shows our first numerical experiment results on the global sensitivity analysis using the PRCC–LHS approach, where we illustrate how our model's parameters impact the behavior of the basic reproduction number. By definition, the basic reproduction number only calculates the expected number of new infections generated by one primary infected individual. Hence, it is reasonable that the expression of the basic reproduction number of our model in (3.2) does not depend on the reinfection parameters, such as ζ , κ , and ϵ_{m_2} . We will analyze the impact of these reinfection parameters on the global sensitivity analysis for the model's variables in the next numerical experiment.

Figure 1(a) reveals the PRCC results of $\mathcal{R}_0$, and Figure 1(b) shows the frequency distribution of the magnitude of $\mathcal{R}_0$ from 30,000 samples taken using the LHS approach. The positive PRCC values mean that there is a positive relationship between the specific parameter and $\mathcal{R}_0$. For example, since β has a positive PRCC value, increasing β , which represents the primary infection rate, will increase the magnitude of $\mathcal{R}_0$. Hence, it is reasonable that one way to control the size of $\mathcal{R}_0$ is by controlling the value of β , for example, by implementing medical mask use. On the other hand, negative PRCC values represent a negative relationship between the parameter and $\mathcal{R}_0$. For instance, since ρ , which represents the additional recovery rate of infected individuals, has a negative PRCC value, increasing ρ will reduce $\mathcal{R}_0$. Among all model parameters in the expression of $\mathcal{R}_0$ in (3.2), not all of them are easy to control, such as Λ and μ , which represent the natural birth and natural death rates, respectively. In contrast, parameters like β , c_m , ϵ_{m_1} , and ρ are easier to control, where these

parameters represent the infection rate, the proportion of medical mask use, the efficacy of medical masks, and the additional recovery rate, respectively. The distribution of $\mathcal{R}_0$ obtained from 30,000 samples using the LHS approach is shown in Figure 1(b). It is clearly observed that the most frequently occurring values of $\mathcal{R}_0$ lie between 3 and 4, suggesting that the majority of the sampled parameter combinations are associated with an endemic transmission scenario.

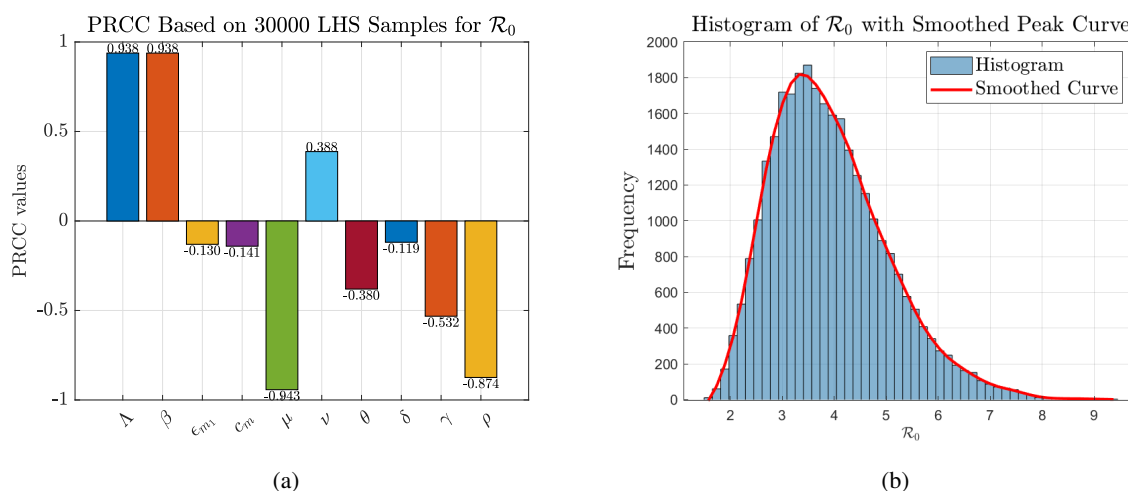


Figure 1. Global sensitivity analysis result of $\mathcal{R}_0$ with respect to each parameter value that appears on $\mathcal{R}_0$. Panel (a) shows the PRCC values using 30,000 sample points from LHS approach, and Panel (b) shows the distribution of $\mathcal{R}_0$ from the same sample point.

Time-dependent PRCC analysis of model outputs

The next global sensitivity analysis presented in our numerical experiment is the time-dependent PRCC analysis of the model output from System (2.1). This analysis is crucial because, as previously discussed, not all parameters in System (2.1) appear in the expression for $\mathcal{R}_0$. Consequently, understanding the behavior of $\mathcal{R}_0$ alone is insufficient to capture the full dynamical complexity of the model. This assertion is further supported by the analytical results in Theorem 7, which show that reducing $\mathcal{R}_0$ below one does not necessarily guarantee disease eradication from the population.

The time-dependent PRCC analysis is performed by taking the model compartments $S(t)$, $E(t)$, $I(t)$, and $R(t)$ as output variables. For each parameter set generated by LHS, the model is solved numerically over a 500-day simulation period. At each time point t_j , the PRCC between each input parameter and the corresponding output value $S(t_j)$, $E(t_j)$, $I(t_j)$, or $R(t_j)$ is computed. Therefore, the resulting PRCC curves describe how the sensitivity of each compartment to the model parameters changes over time. Furthermore, we also calculate the PRCC value for each variables at $t = 500$ days to summarize the final-time PRCC values and identify the dominant parameters affecting each compartment at the end of the simulation period.

The results of the time-dependent PRCC analysis for all state variables over a 500-day simulation period are presented in Figure 2(a). It can be seen clearly for all model variables that the time-dependent PRCC values fluctuate significantly during the first 300 days of the simulation period. This behavior reflects the transient dynamics of each variable due to the epidemic growth of

COVID-19, the infection outbreak, or even the early recovery of infected individuals. After 300 days of simulation, the dynamics of the time-dependent PRCC for each variable become more stabilized, reflecting that the system has reached its quasi-steady-state condition, where the model parameters' sensitivities become more consistent.

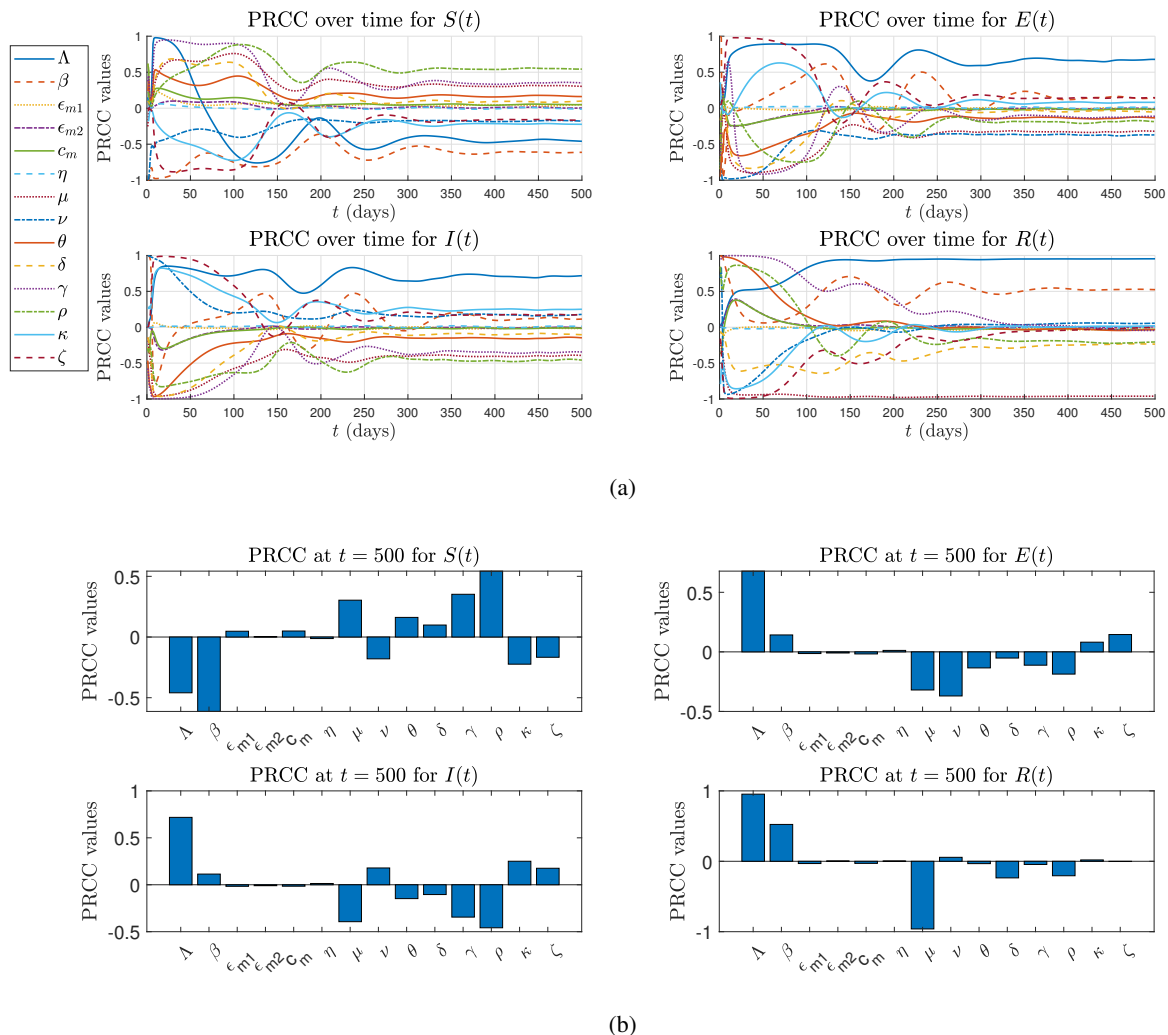


Figure 2. (a) PRCC dynamics over time for the model compartments $S(t)$, $E(t)$, $I(t)$, and $R(t)$ of System (2.1). PRCC values are computed using LHS (30,000 samples) across $\pm 25\%$ variation of each parameter. This figure illustrates the time-dependent sensitivity of model outputs to each parameter over a 500-day simulation period. (b) Histogram of PRCC values at the final time $t = 500$ for the compartments of System (2.1). This figure highlights the most influential parameters affecting each compartment at the end of the simulation period.

From Figure 2(a), we observe that the infection rate β and the reinfection parameter ζ consistently have negative PRCC values with respect to the susceptible compartment but positive values for the remaining compartments. This implies that even though the reinfection term directly involves only the R and E compartments, it indirectly affects all compartments—particularly the I compartment, which

increases as reinfection becomes more prevalent in the population. Another noteworthy result is the role of the recovery-related parameter ρ , which does not appear in the expression for $\mathcal{R}_0$, yet has a significant impact on the system's dynamics. In our model, ρ is the additional recovery rate due to treatment. Therefore, increasing this parameter will reduce I but increase S . From the PRCC results, it can be seen that increasing ρ will also reduce E and R for a long-term implementation. It is interesting to mention that the impact of ρ on R is not trivial. It has a positive PRCC value at the beginning, indicating that increasing the treatment rate will increase the recovery compartment. However, in the long run, increasing ρ will reduce R , which indicates that implementing an additional recovery rate from treatment for infected individuals may ultimately reduce the number of removed individuals, even though it initially reduces the number of infected individuals.

In Figure 2(b), we present the PRCC values of each parameter with respect to each compartment at the final simulation time ($t = 500$). It is evident that β and ρ are the most influential parameters affecting the susceptible population at the end of the simulation. Specifically, increasing β or decreasing ρ leads to a decline in the susceptible population. The progression rate ν has the biggest negative PRCC values in the E compartment, which suggests that increasing this parameter will reduce the E compartment. This is reasonable since ν moves people from the E to the I compartment due to the incubation period of infection. In the I compartment, it can be seen that this compartment is highly affected by ρ and γ , where these parameters represent the additional recovery rate and the natural recovery rate, respectively. Last but not least, the R compartment is highly affected by the primary infection parameter β but, interestingly, is not highly affected by the reinfection parameter ζ . The reason that ζ has a small impact on the R compartment is perhaps due to the number of susceptible and infected populations moving closer to their equilibrium condition.

This numerical experiment on the time-dependent PRCC values of each variable suggests an important insight: The impact of the parameters on variable dynamics may change over time. It may be significant in the early phase of the disease introduced in the population, but become less significant when it already approaches a quasi-steady-state condition. Hence, analyzing the time-dependent PRCC can help improve the possible chosen strategies to control COVID-19, whether in the early stage of disease invasion or when it is already approaching its stable condition.

6.3. Bifurcation diagram

To produce the bifurcation diagram of the model, we began by assigning baseline values to all model parameters and calculating the corresponding basic reproduction number $\mathcal{R}_0$, along with identifying the equilibrium point(s) that arise under these conditions. Once the initial equilibrium was determined, we utilized the numerical continuation software packages AUTO and MATCONT to systematically track the changes in the equilibrium as one of the key parameters was varied. Through this continuation process, we were able to detect the emergence of additional equilibria and observe qualitative changes in the system's behavior, which were then summarized in the bifurcation diagram.

Forward bifurcation with endemic bubble

To perform this simulation, we use the baseline parameter values in Table 3. With these parameter values, we have two equilibrium points that appear from our model, i.e., the COVID-19 endemic

equilibrium which is stable and is given by

$$\mathcal{E}_* = (S_*, E_*, I_*, R_*) = (64.05, 3.6, 13.47, 286.8).$$

The second equilibrium that appears is the COVID-19 free equilibrium point which is unstable and is given by

$$\mathcal{E}_0 = (S_0, E_0, I_0, R_0) = (388.8, 0, 0, 0).$$

Using this endemic equilibrium as the starting point, we conduct our continuation to seek the other equilibrium branches using β as the bifurcation parameter. The result is depicted in Figure 3. Figure 3(a) shows the bifurcation diagram in the plane of β – I , while Figure 3(b) represents the same bifurcation diagram, but in a three-dimensional space of β – S – I . It can be seen that with the mentioned parameter values, we have $\mathcal{R}_0 = 1$ at $\beta = 0.000942276$, which also serves as the transcritical point or BP. Before the BP, for any value of β , the solution always tends to the COVID-19 free equilibrium (see Figure 4(a)).

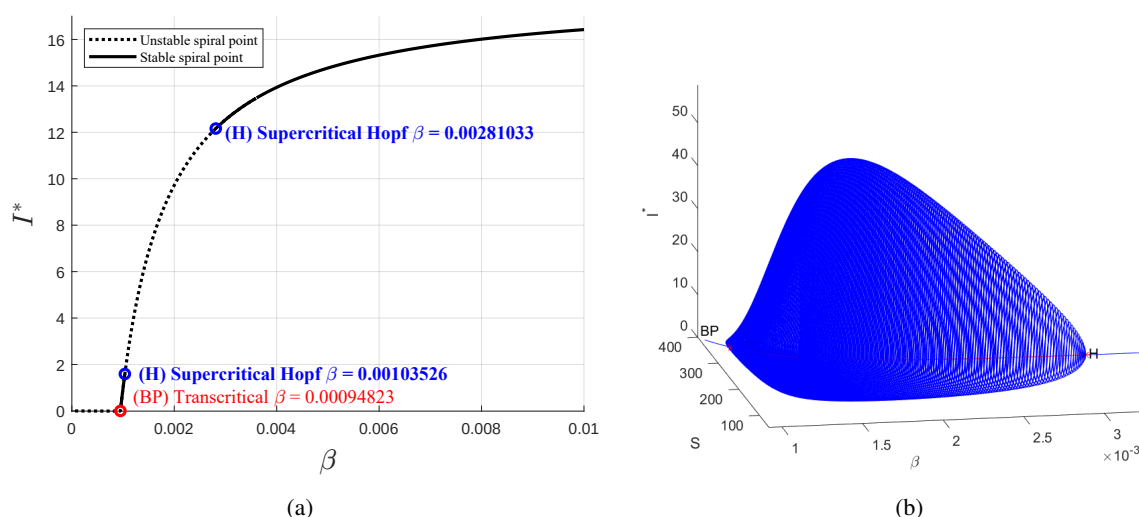


Figure 3. Forward bifurcation occurs at $\mathcal{R}_0 = 1$, with the branching point (BP) located at $\beta = 0.00094823$. Two Hopf points are identified at $\beta = 0.00103526$ and $\beta = 0.00281033$. Panel (a) shows the two-dimensional bifurcation diagram, while Panel (b) presents the three-dimensional bifurcation structure, including a family of periodic solutions.

After the BP, for values of β such that $0.00094823 < \beta < 0.00103526$, the COVID-19 free equilibrium becomes a saddle point, and a stable endemic equilibrium emerges (see Figure 4(b)). Following the first Hopf bifurcation at $\beta = 0.00103526$, a stable limit cycle appears, leading to the instability of the endemic equilibrium. This stable limit cycle exists for $0.00103526 < \beta < 0.00281033$, with various periods depending on the parameter β (see Figure 5(a)). All trajectories will converge to this limit cycle, indicating that they approach a periodic solution. Each periodic solution has a maximum range of variables; in Figure 5(b), we show how the maximum range of I^* depends on the parameter β . Among these periodic solutions, the one with the longest period occurs at $\beta = 0.00107$ (indicated by the red circle in Figure 5), while the greatest maximum range of I^* is observed at $\beta = 0.0015$ (marked by the blue circle in Figure 5). The dynamics of the

system at these β values are illustrated in Figure 6(a),(b). After the second Hopf bifurcation at $\beta = 0.00281033$, the limit cycle disappears, and all trajectories converge back to the endemic equilibrium (see Figure 6(c)).

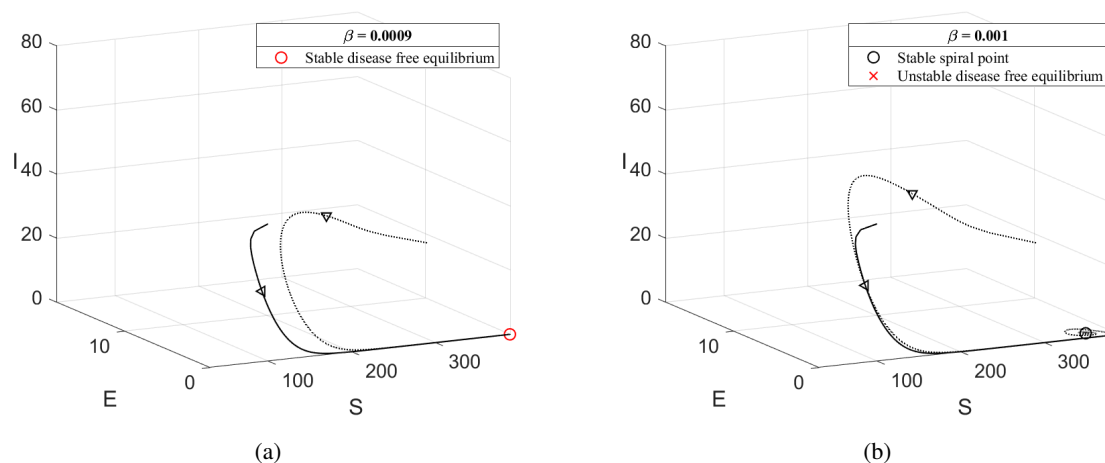


Figure 4. Simulation I: (a) There is one stable COVID-19-free equilibrium, denoted $\mathcal{E}_0$. (b) After a transcritical bifurcation, $\mathcal{E}_0$ becomes a saddle point, leading to the emergence of the endemic equilibrium, denoted $\mathcal{E}_*$.

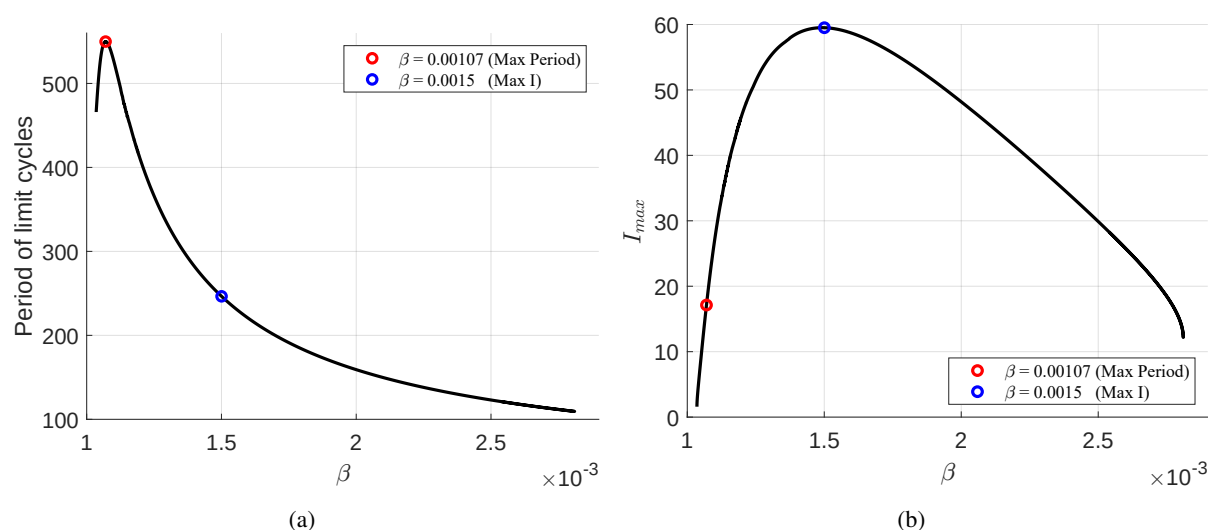


Figure 5. Relationship between the infection parameter β and epidemic characteristics. Panel (a) shows the period of successive outbreaks. The longest period occurs near $\beta \approx 1.07 \times 10^{-3}$, indicating very slow and infrequent waves, whereas increasing β shortens the interval between outbreaks, leading to more frequent epidemic recurrences. Panel (b) shows the outbreak peak $I_{\max}$, which does not increase monotonically with β . The peak is largest at $\beta \approx 1.5 \times 10^{-3}$ and becomes smaller for larger β .

Figure 5 conveys crucial information for guiding strategies on COVID-19 eradication and outbreak anticipation. The numerical results reveal that the transmission rate β strongly shapes the temporal patterns of COVID-19 outbreaks. When β lies in the oscillatory regime, the system generates recurrent epidemic waves. As β increases, the period between waves becomes shorter, leading to more frequent outbreaks. This, of course, makes the work of public health authorities more difficult, since outbreaks occur more frequently and, for large values of β , there may even be more than two outbreaks in a single year. However, the peak size of the infected population is not a simple increasing function of β : The largest epidemic peak is observed at an intermediate value of β , while both lower and higher values produce smaller peaks. This finding has direct epidemiological implications. Partial reductions in transmission that still leave β inside the oscillatory window may not always mitigate the epidemic burden; in fact, they may shift the system toward the range that produces the most severe outbreak peaks. Therefore, control measures should aim either to push β below the oscillatory threshold to eliminate sustained cycles, or to reduce transmission sufficiently far from the parameter region that maximizes the outbreak's size. This suggests that stronger transmission does not always correspond to more severe outbreaks but rather determines a trade-off between outbreaks' size and frequency. Such insights are crucial for designing interventions that not only delay outbreaks but also prevent excessively high epidemic peaks.

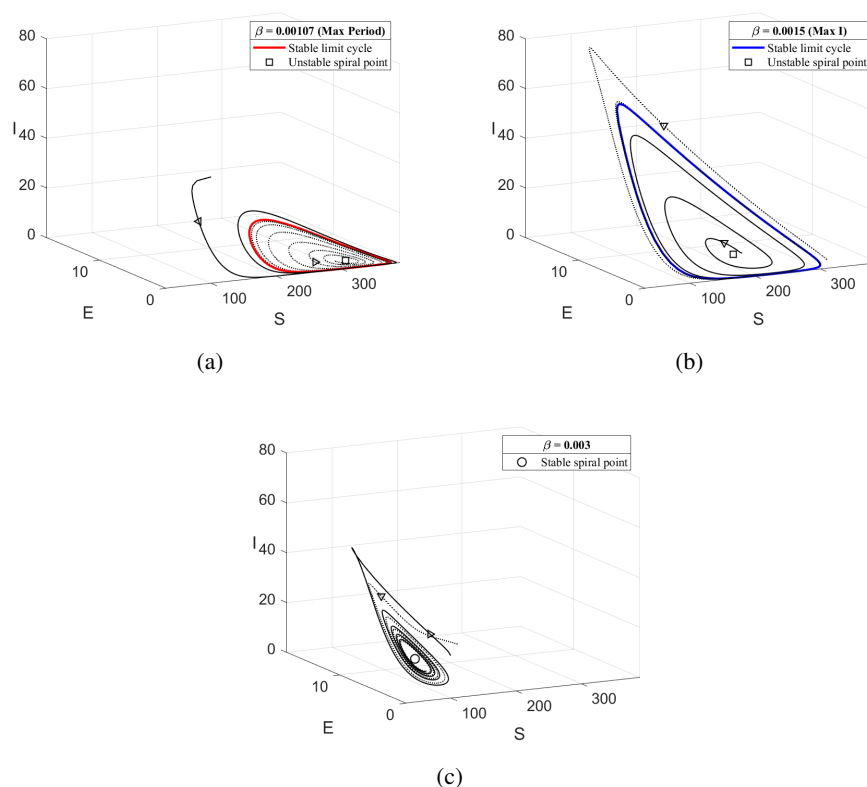


Figure 6. (a) Following a Hopf bifurcation, a limit cycle appears, and when $\beta = 0.00107$, its period reaches a maximum. (b) At $\beta = 0.0015$, the limit cycle reaches its maximum amplitude. (c) After the second Hopf bifurcation, the limit cycle disappears and all trajectories converge to the endemic equilibrium.

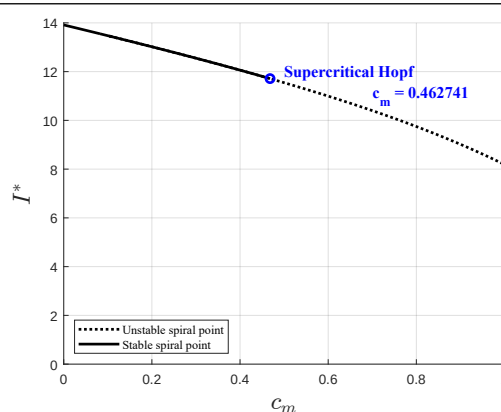


Figure 7. Forward bifurcation as the parameter c_m varies. A Hopf bifurcation point is identified at $c_m = 0.462741$.

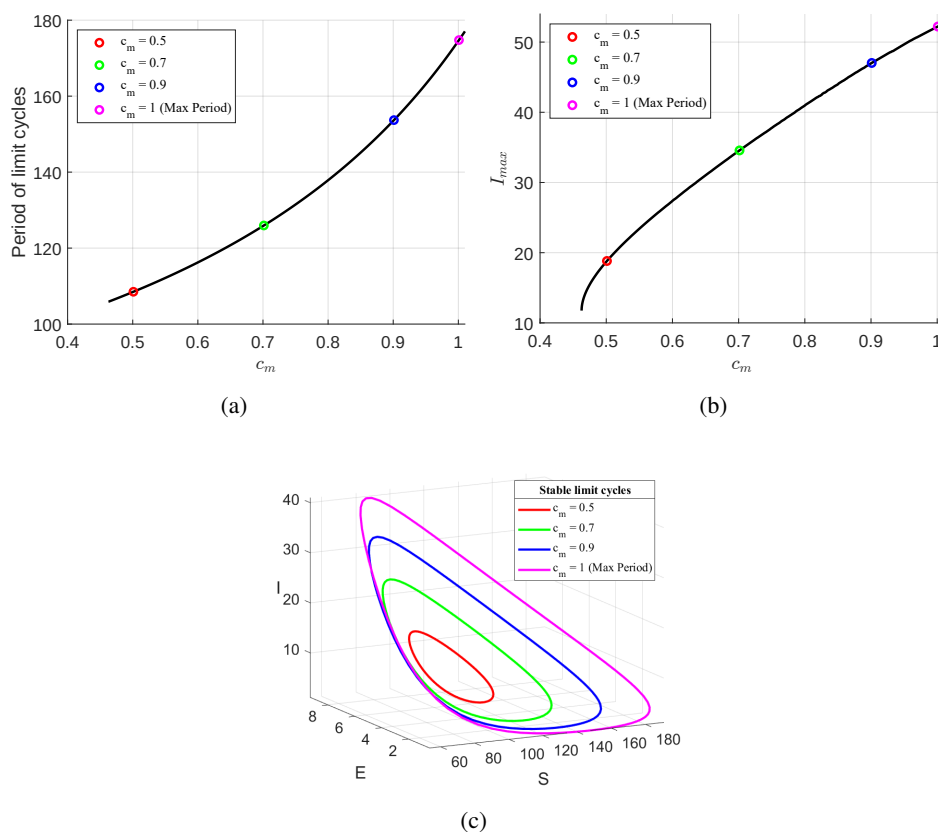


Figure 8. Relationship between the proportion of mask use (c_m) and epidemic characteristics. Panel (a) shows the period of successive outbreaks. The longest period occurs as c_m increases. Smaller values of c_m lead to more frequent epidemic recurrences. Panel (b) shows the outbreak peak I_{max} , which is monotonically increasing respect to c_m . Panel (c) depicts some periodic solutions with respect to four different values of c_m .

Using the same endemic equilibrium as the starting point, we conduct a continuation analysis to explore other equilibrium branches, utilizing c_m as the bifurcation parameter. The bifurcation diagram is presented in Figure 7. For $0 \leq c_m < 0.462741$, the trajectories converge to the endemic equilibrium. However, for $c_m \geq 0.462741$, the trajectories converge to some limit cycles. As shown in Figure 8(a),(b), both the period of the limit cycle and the maximum range of I^* are proportional to the parameter c_m . As anticipated, having more infected individuals who wear face masks lengthens the period of periodic solutions but, at the same time, leads to larger outbreak peaks. This outcome can be explained by the fact that a longer period allows more susceptible individuals to accumulate before the next wave, so that when an outbreak eventually occurs, it affects a larger fraction of the population. This highlights a trade-off between outbreak frequency and outbreak size, and suggests that mask usage alone may not be sufficient to mitigate the epidemic burden without complementary interventions such as vaccination or early detection. We provide examples of stable limit cycles that occur for $c_m = \{0.5, 0.7, 0.9, 1\}$ as shown in Figure 8(c).

Figure 9 presents a codimension-2 bifurcation diagram of model (2.1), using β and c_m as the bifurcation parameters. The parameter space is divided into four regions (R-1 to R-4), where each region corresponds to a distinct combination of β and c_m , resulting in qualitatively different dynamics of System (2.1). In Region R-1, where $\mathcal{R}_0 < 1$, there is a unique equilibrium point—the COVID-19 free equilibrium—which is locally asymptotically stable. In Region R-2, where $\mathcal{R}_0 \in (1, 1.0965)$, the COVID-19 free equilibrium becomes unstable, while the COVID-19 endemic equilibrium is locally asymptotically stable. In Region R-3, where $\mathcal{R}_0 \in (1.0965, 3.083688)$, the COVID-19 free equilibrium remains unstable and the COVID-19 endemic equilibrium loses stability, giving rise to periodic solutions. Finally, in Region R-4, where $\mathcal{R}_0 > 3.083688$, the COVID-19 free equilibrium remains unstable, but the endemic equilibrium regains its local asymptotic stability. This diagram illustrates how varying transmission rate β and medical mask use c_m parameters can lead to critical transitions in disease dynamics. Understanding these regions provides insight into the thresholds that govern the emergence of complex behaviors such as periodic outbreaks.

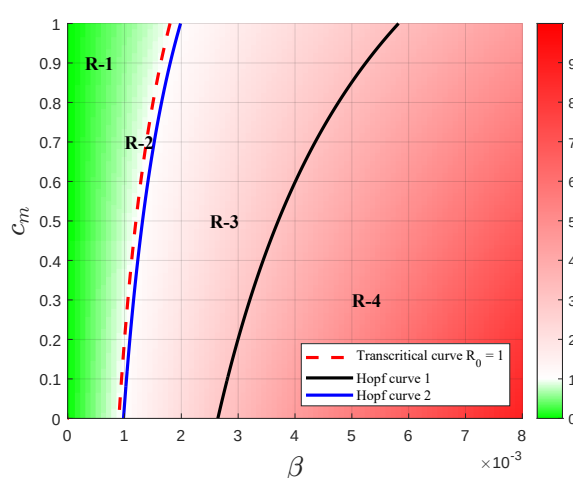


Figure 9. Codimension-2 bifurcation diagram in the plane of $\beta - c_m$.

Backward bifurcation

The next simulation conducted to show the appearance of backward bifurcation from our model. From Theorem 6, we see that backward bifurcation may appears at $\mathcal{R}_0 = 1$ if $\mathcal{A} > 0$. To run this simulation, we use the following parameter values:

$$\begin{aligned} \Lambda = 0.5, \quad \beta = 0.08995, \quad \epsilon_{m_1} = 0.5, \quad \epsilon_{m_2} = 0.5, \quad c_m = 0.5, \quad \eta = 0.001, \\ \mu = 0.097, \quad \nu = 0.6, \quad \theta = \frac{1}{7}, \quad \delta = 0.05, \quad \gamma = 0.1, \quad \rho = 0.001, \quad \kappa = 0.0005. \end{aligned} \quad (6.1)$$

We substitute this parameter values into $\mathcal{A}$ and solve it with respect to ζ to get the threshold of ζ such that we have forward or backward bifurcation from our model. By this substitution, we see that threshold ζ is $\zeta^* = 0.195843611$. By choosing $\zeta = 0.2154272972 > \zeta^*$, we will have backward bifurcation phenomena at $\mathcal{R}_0 = 1$. Similar to the previous numerical experiment, we start our numerical experiment by finding the initial equilibrium point. By substituting the parameter values above, we find that the COVID-19 endemic equilibrium given by

$$\mathcal{E}_* = (S_*, E_*, I_*, R_*) = (4.71, 0.05, 0.13, 0.18),$$

is stable, while the COVID-19 free equilibrium

$$\mathcal{E}_0 = (S_0, E_0, I_0, R_0) = (5.15, 0, 0, 0),$$

is unstable. Using AUTO, we seek for the branch of equilibrium points using β as the bifurcation parameter, and the results are depicted in Figure 10.

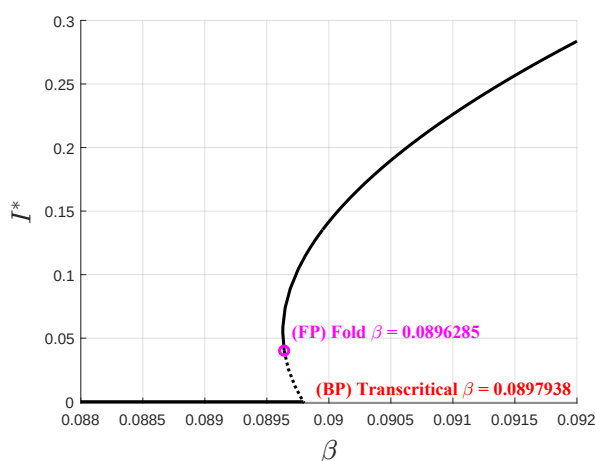


Figure 10. Backward bifurcation occurs at $\mathcal{R}_0 = 1$, with the branching point (BP) located at $\beta = 0.089793793$ and the fold point (FP) at $\beta = 0.0896248703$.

A backward bifurcation arises when the system exhibits multiple equilibria around the threshold where $\mathcal{R}_0 = 1$, leading to the coexistence of a stable COVID-19-free equilibrium ($\mathcal{E}_0$) and a stable COVID-19 endemic equilibrium ($\mathcal{E}_*$) for certain values of β as the chosen bifurcation parameter. In this context, Figure 10 illustrates a backward bifurcation diagram of System (2.1). The BP occurs at $\beta = 0.089793793$, where the endemic branch emerges from the COVID-19-free equilibrium, and

a fold (or saddle-node) bifurcation appears at $\beta = 0.0896248703$, and we denote this specific value of β as fold point (FP). For $\beta < BP$, $\mathcal{E}_0$ remains locally asymptotically stable, while $\mathcal{E}_*$ is stable when β is larger than FP. Within the interval $\beta \in (FP, BP)$, both $\mathcal{E}_0$ and $\mathcal{E}_*$ are locally stable, leading to a bistable region in which the long-term outcome depends on the initial condition. This structure implies that simply reducing β below the threshold BP may not be sufficient to eliminate the disease; instead, control measures must be strong enough to bring β below the FP. The presence of backward bifurcation highlights the importance of achieving a more aggressive intervention strategy to ensure disease eradication.

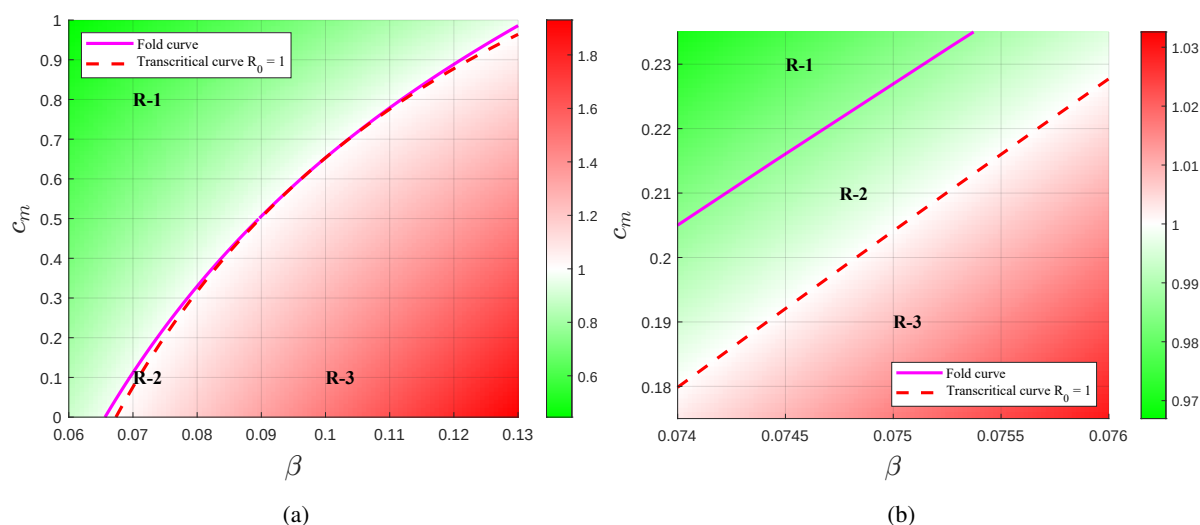


Figure 11. Codimension-2 bifurcation diagram in the plane of $\beta - c_m$ when backward bifurcation appears.

Figure 11(a) presents a codimension-2 bifurcation diagram using β and c_m as the bifurcation parameters, highlighting the case of a backward bifurcation. This scenario is illustrated in Figure 11(a) with the fold curve shown by the solid magenta line and the transcritical curve represented by the dashed red line. The fold curve consistently lies above the transcritical curve, as demonstrated in the magnified view in Figure 11(b). This indicates that for a fixed parameter c_m , the dynamics of System (2.1) are similar to those depicted in Figure 10. The diagram reveals that different combinations of β and c_m give rise to three distinct regions. In Region R-1, where $\mathcal{R}_0 < 0.99811$ (with $\mathcal{R}_0 = 0.99811$ marking the value at which the fold bifurcation curve), the system admits a unique and locally asymptotically stable COVID-19-free equilibrium. In the narrow Region R-2, the system exhibits bistability, with two endemic equilibria (one stable and one unstable) coexisting alongside a stable COVID-19-free equilibrium. In this region, the long-term outcome depends sensitively on the initial conditions. Finally, in Region R-3, the system admits a unique and stable endemic equilibrium, while the COVID-19-free equilibrium becomes unstable. This bifurcation structure highlights the critical role of behavior-related parameters, such as c_m , in shaping disease persistence. Even small changes in parameter values can shift the system between elimination and persistence, emphasizing the need for carefully designed and sustained interventions.

Forward bifurcation with hysteresis

For the next numerical experiment, we will show the forward bifurcation with hysteresis phenomena from our model. To do this, we use the following parameter values.

$$\begin{aligned} \Lambda = 3.5, \beta = 0.001024560829, \epsilon_{m_1} = 0.5, \epsilon_{m_2} = 0.5, c_m = 0.1, \eta = 0.0001, \\ \mu = 0.009, \nu = 0.8, \theta = \frac{1}{7}, \delta = 0.014, \gamma = \frac{1}{14}, \rho = 0.2, \kappa = 0.05, \zeta = 0.0006. \end{aligned} \quad (6.2)$$

Substituting all parameters except for β and c_m into Eq (4.5) yields an implicit formula for the fold curves in the (β, c_m) plane, denoted as

$$\begin{aligned} 0 = & 7.894605173 \times 10^{-25} \beta c_m^2 - 1.037139819 \times 10^{-24} \beta c_m + 2.181465719 \times 10^{-32} c_m^4 \\ & + 1.154244537 \times 10^{-30} c_m^3 - 6.534103979 \times 10^{-30} c_m^2 + 8.997369586 \times 10^{-24} c_m^6 \beta^2 \\ & + 2.173744767 \times 10^{-22} c_m^5 \beta^2 - 1.291988476 \times 10^{-17} c_m^4 \beta^2 + 7.279873440 \times 10^{-28} c_m^5 \beta \\ & + 1.033489718 \times 10^{-16} c_m^3 \beta^2 - 3.100360896 \times 10^{-16} \beta^2 c_m^2 + 4.133738928 \times 10^{-16} \beta^2 c_m \\ & + 4.892650368 \times 10^{-20} c_m^7 \beta^3 + 4.449694293 \times 10^{-19} c_m^6 \beta^3 - 7.176701818 \times 10^{-15} c_m^5 \beta^3 \\ & + 7.172867350 \times 10^{-14} c_m^4 \beta^3 - 2.868593335 \times 10^{-13} c_m^3 \beta^3 + 5.736643112 \times 10^{-13} \beta^3 c_m^2 \\ & - 5.736340025 \times 10^{-13} \beta^3 c_m + 9.894342520 \times 10^{-17} c_m^8 \beta^4 - 5.596607179 \times 10^{-16} c_m^7 \beta^4 \\ & - 7.47342182 \times 10^{-16} c_m^6 \beta^4 + 1.383319125 \times 10^{-14} c_m^5 \beta^4 - 4.701787168 \times 10^{-14} c_m^4 \beta^4 \\ & + 7.849345589 \times 10^{-14} c_m^3 \beta^4 - 7.103960832 \times 10^{-14} \beta^4 c_m^2 + 3.305318791 \times 10^{-14} \beta^4 c_m \\ & + 2.840608120 \times 10^{-26} c_m^4 \beta - 2.573279864 \times 10^{-25} c_m^3 \beta - 6.621399780 \times 10^{-30} \\ & - 6.134808834 \times 10^{-15} \beta^4 + 2.294463698 \times 10^{-13} \beta^3 - 2.066845765 \times 10^{-16} \beta^2 \\ & + 4.972685650 \times 10^{-25} \beta + 1.158741245 \times 10^{-29} c_m. \end{aligned} \quad (6.3)$$

There are two curve solutions to (6.3) in the (β, c_m) plane, which intersect tangentially at $(\beta, c_m) = (0.0010323970842, 0.18805359354)$ and denote as the cusp bifurcation point. We illustrate these fold curves along with the transcritical curve in Figure 12(a), which divides the parameter space into three distinct domains, each representing qualitatively different dynamics of the disease.

This cusp point is confirmed by results obtained using a similar approach to that of the previous steps. After identifying the fold and Hopf bifurcation points while varying the parameter β , we conduct our continuation using AUTO, starting at each bifurcation point and varying the parameter c_m . As a result, we produce a complete bifurcation diagram of co-dimension 2, illustrated in Figure 12(b), with respect to the parameters β and c_m . As we get the codimension-2 bifurcations, i.e., cusp bifurcation point at $(\beta, c_m) = (0.00103239, 0.188053)$ and Generalized Hopf bifurcation point or Bautin bifurcation point at $(\beta, c_m) = (0.00151068, 0.390359)$, then the dynamics of the system are complex for each pair of (β, c_m) .

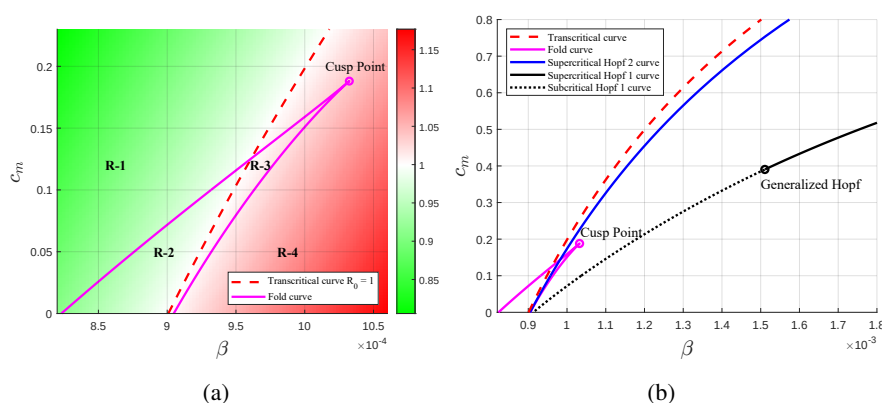


Figure 12. Codimension-2 bifurcation diagram in the plane of $\beta - c_m$ when forward bifurcation with hysteresis.

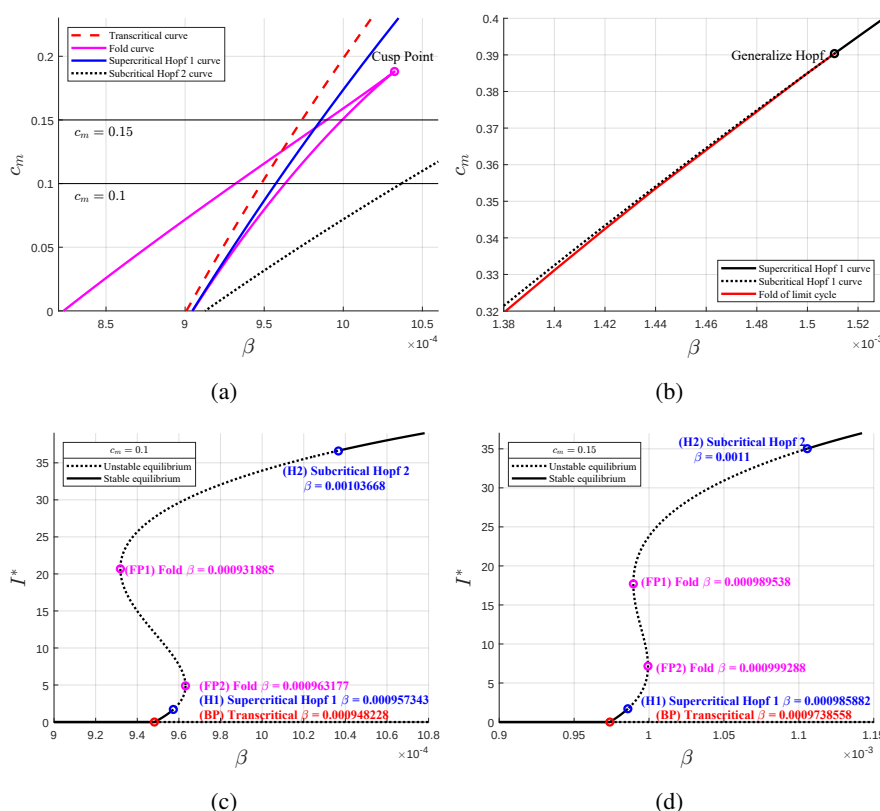


Figure 13. (a) Codimension-2 bifurcation diagram of the nontrivial equilibrium near the cusp point. (b) Codimension-2 bifurcation diagram of the nontrivial equilibrium near the generalized Hopf bifurcation. (c) Forward bifurcation with hysteresis appears from System (2.1). The I^* -coordinate of the branch of continuation equilibrium as a variation of parameter β for $c_m = 0.1$. (d) Forward bifurcation with no hysteresis appears from System (2.1). The I^* -coordinate of the branch of continuation equilibrium as a variation of parameter β for $c_m = 0.15$.

The parameter space shown in Figure 12(a) is divided into four regions (R-1 to R-4), each corresponding to a distinct combination of β and c_m . These combinations lead to qualitatively different dynamics of System (2.1). In Region R-1, where $\mathcal{R}_0 < 1$, there is one equilibrium point—the COVID-19-free equilibrium—which is locally asymptotically stable. In Region R-2, the COVID-19-free equilibrium remains asymptotically stable, but two new endemic equilibria emerge due to a fold bifurcation. These two endemic equilibria are unstable. In Region R-3, the COVID-19-free equilibrium becomes unstable, while a new COVID-19 endemic equilibrium emerges that is locally asymptotically stable. Consequently, there are three endemic equilibria in this region. In Region R-4, the COVID-19-free equilibrium remains unstable, and two of the endemic equilibria coalesce. Thus, in this region, there is one COVID-19-free equilibrium and one endemic equilibrium. For larger values of β in Region R-4, the endemic equilibrium undergoes a Hopf bifurcation, altering its stability so that the endemic equilibrium becomes stable.

To better understand the dynamics near the cusp point, we take two cross-sections, specifically for $c_m = \{0.1, 0.15\}$ (see Figure 13(a),(b)). We can compare these two cross-sections in Figure 13(c),(d). When $c_m = 0.1$, the BP occurs between the two FPs, indicating that we can have two endemic equilibria before the BP. Conversely, when $c_m = 0.15$, the BP occurs before the two fold points, resulting in three endemic equilibria after the branching point. In Figure 14, we provide details of the three-dimensional bifurcation diagram, including stable and unstable limit cycles for $c_m = 0.1$, where forward bifurcation with hysteresis appears from System (2.1).

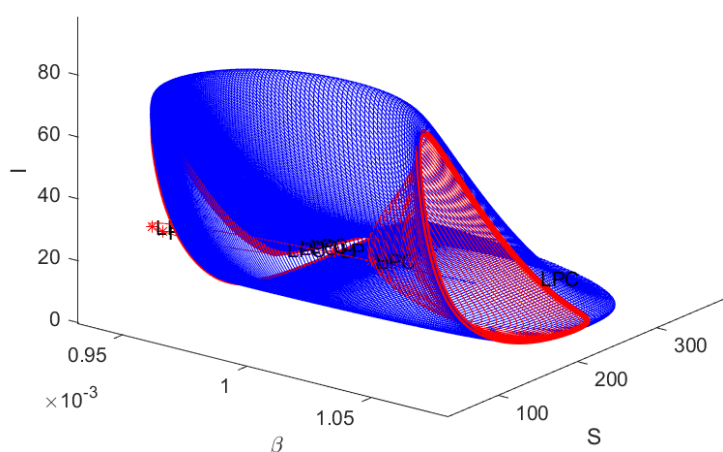


Figure 14. A forward bifurcation with hysteresis and double limit cycles in three-dimensional space. The red and blue curves represent the unstable and stable families of limit cycles, respectively.

The generalized Hopf bifurcation point separates the supercritical and subcritical Hopf bifurcations. The supercritical Hopf bifurcation corresponds to a stable limit cycle, while the

subcritical Hopf bifurcation corresponds to an unstable limit cycle. Consequently, we observe a fold of limit cycles where the stable limit cycle collides with the unstable limit cycle, as seen in Figure 14. In Figure 13(c),(d), for $0.06 < c_m < 0.15$, we have one limit cycle. Meanwhile, for $0.0289635 < c_m < 0.06$, we observe two limit cycles: A stable limit cycle and an unstable limit cycle. At $c_m = 0.0289635$, these limit cycles collide and disappear. Details of the impact of the proportion of infected individuals who use masks on both the maximum outbreak size and the period of limit cycles are shown in Figure 15. Furthermore, Figure 15(a) illustrates that a higher proportion of mask usage lengthens the period between successive outbreaks. At the same time, increasing c_m reduces the maximum peak of infection as shown in Figure 15(b), indicating that widespread mask use not only delays the occurrence of outbreaks but also mitigates their severity.

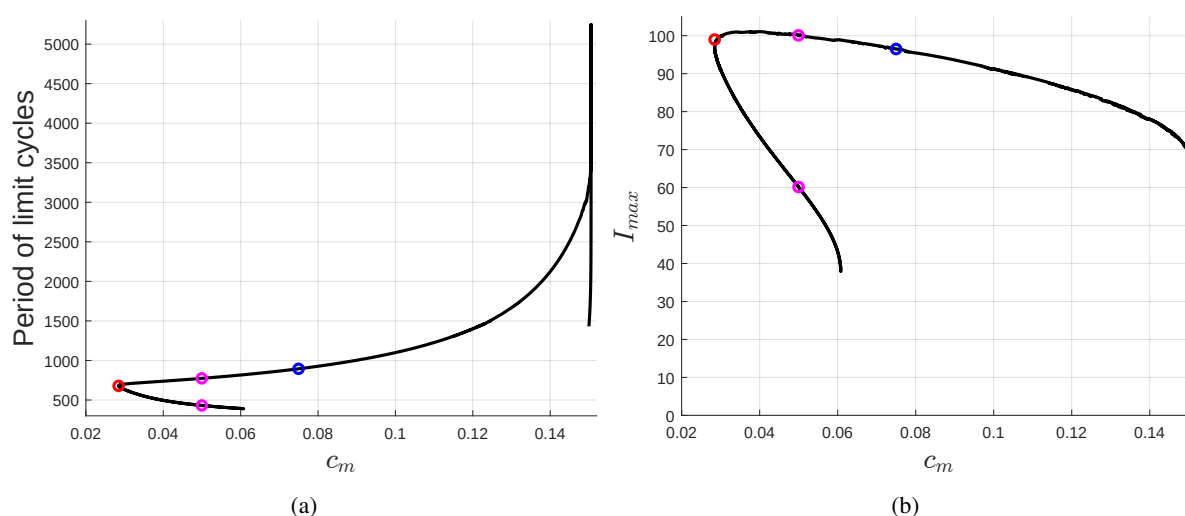


Figure 15. The branch of the periodic solution of Hopf 1 at $\beta = 0.000985882$ with variation in parameter c_m .

To illustrate the dynamics, we provide four examples as shown Figure 16.

- (1) At $c_m = 0.075$, all trajectories converge to a stable limit cycle (the solid blue limit cycle in Figure 16(a)), while the endemic equilibrium acts as an unstable spiral point.
- (2) At $c_m = 0.05$, all trajectories near the endemic equilibrium converge to it. However, trajectories outside the unstable limit cycle (the dashed magenta limit cycle in Figure 16(b)) will converge to a stable limit cycle (the solid magenta limit cycle in Figure 16(b)).
- (3) At $c_m = 0.0289635$ (the fold of the limit cycle point), we observe a semistable limit cycle (the dashed red dot limit cycle in Figure 16(c)) and a stable endemic equilibrium. Trajectories outside the limit cycle converge to the limit cycle, while those from inside converge to the endemic equilibrium.
- (4) At $c_m = 0.02$ as shown in Figure 16(d), the limit cycle disappears and all trajectories converge to the endemic equilibrium.

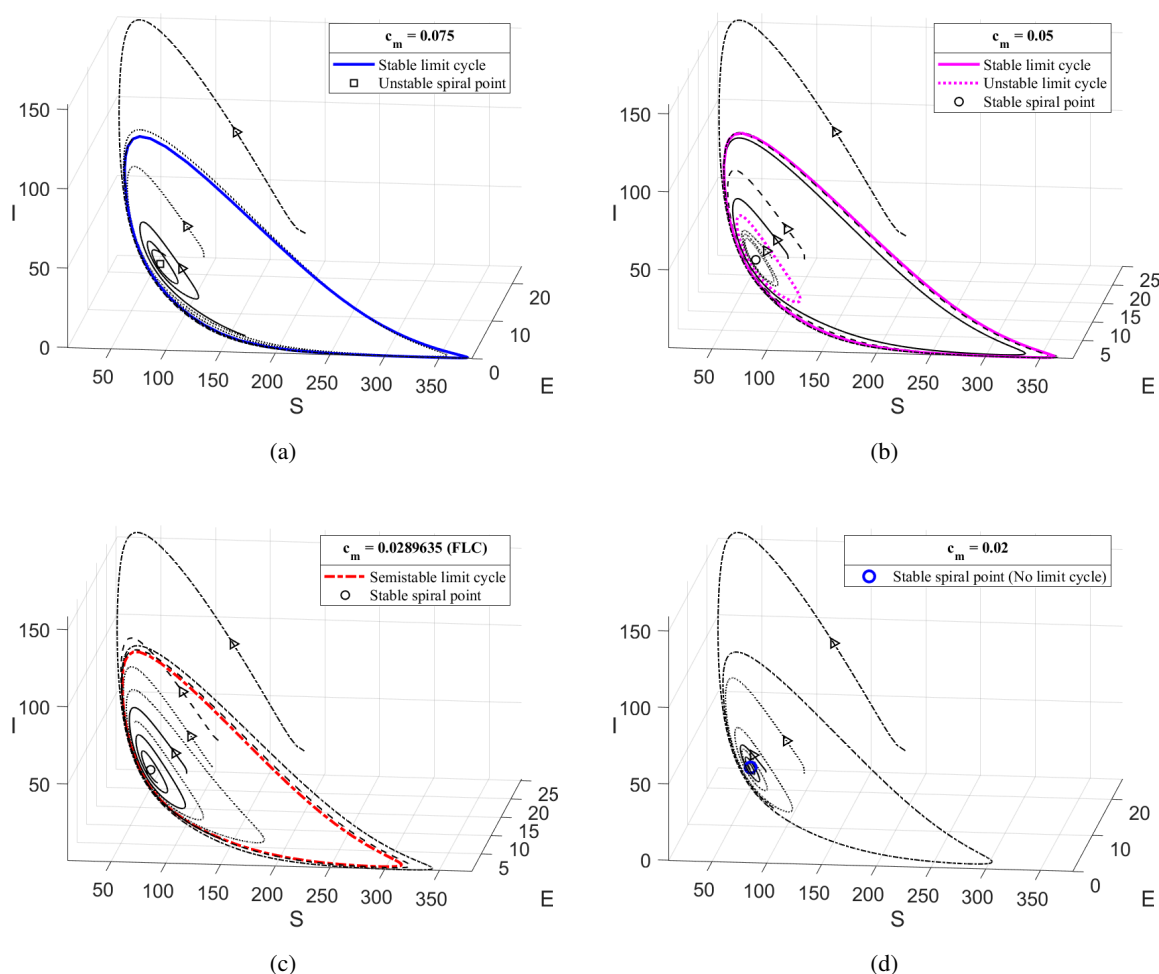


Figure 16. (a) One stable limit cycle in the range $c_m = (0.060734, 0.150201)$. (b) One stable and one unstable limit cycles in the range $c_m = (0.0289635, 0.060734]$. (c) By numerical observation, the fold of limit cycles occurs at $c_m = 0.0289635$. (d) No limit cycle in the range $c_m = [0, 0.0289635) \cup (0.150201, 1]$.

6.4. Linking continuation results with epidemic outcomes

The numerical experiments on the bifurcation diagram, especially on the impact of the infection rate β and proportion of infected individuals who use a medical mask c_m provide useful insights for epidemic control strategies. The infection parameter β can be interpreted as the effective transmission rate, which is influenced by factors such as contact behavior, mask use, vaccination coverage, and many other factors. When β or c_m falls within the oscillatory range, the population experiences recurrent waves of COVID-19. Increasing infection rate leads to more frequent epidemic waves, but not necessarily larger ones, since the peak size of infection is maximized at an intermediate value of β . On the other hand, increasing the proportion of medical mask usage extends the period between outbreaks but, at the same time, produces larger outbreak peaks.

From a policy perspective, this implies that partial interventions which only slightly reduce β may

have unintended consequences: They may shift the system into the region where the outbreak peak is largest. For example, moderate reductions in contact rates or incomplete vaccination coverage could inadvertently lead to larger outbreaks, even though transmission is somewhat reduced. In particular, caution is needed when implementing mask interventions, since our results show that although greater mask use increases the period between outbreaks, it is consistently accompanied by higher outbreak peaks. This trade-off underscores the importance of combining mask use with other complementary measures, such as vaccination and early detection, to ensure that interventions effectively reduce both the frequency and the severity of outbreaks.

Therefore, epidemic control should be designed with two possible objectives in mind.

- (1) To reduce the infection rate below the oscillatory threshold so that recurrent outbreaks disappear entirely, or
- (2) If full elimination is not feasible, to push the infection rate sufficiently away from the parameter region where outbreak peaks are maximized. In practice, this means that layered interventions—such as combining vaccination, mask mandates, public awareness campaigns, and mobility regulation—are likely required to both reduce outbreak frequency and avoid excessively high epidemic peaks. These results emphasize that epidemic control should not only aim at lowering transmission but also at avoiding parameter ranges where the epidemic burden becomes most severe.

7. Optimal control problem

7.1. Model extension incorporating optimal control

Next, we extend our problem into an optimal control model to handle the limited budget for face mask intervention. For this purpose, let $c_m = u(t)$ represent the time-dependent proportion of infected individuals who use face mask. With this assumption, then the model now reads as

$$\frac{dS}{dt} = \Lambda - (1 - u(t)\epsilon_{m_1})\beta SI + \eta R - \mu S, \quad (7.1a)$$

$$\frac{dE}{dt} = (1 - u(t)\epsilon_{m_1})\beta SI - \nu E - \theta E - \mu E + (1 - u(t)\epsilon_{m_2})\zeta RI, \quad (7.1b)$$

$$\frac{dI}{dt} = \nu E - \delta I - \mu I - \left(\gamma + \frac{\rho}{1 + \kappa I}\right)I, \quad (7.1c)$$

$$\frac{dR}{dt} = \theta E + \left(\gamma + \frac{\rho}{1 + \kappa I}\right)I - \eta R - \mu R - (1 - u(t)\epsilon_{m_2})\zeta RI. \quad (7.1d)$$

Our aim is to minimize the number of infected individuals E and I by implementing the most effective strategy of face mask use $u(t)$. This aim is represented by minimizing the following cost function:

$$\mathcal{J} = \int_0^T \left[c_1 u(t)^2 + c_2 E(t) + c_3 I(t) \right] dt, \quad (7.2)$$

where c_1 represent the weight cost for the control variables $u(t)$, while c_2 and c_3 represent the weight parameter for E and I , respectively, and T is the final day of simulation.

7.2. Characterization of the optimal control problem

The following theorem provides the characterization of our optimal control problem.

Theorem 8. Consider the optimal control problem of minimizing the cost functional given in Eq (7.2) subject to the COVID-19 model in (7.1) and the initial conditions

$$S(0) > 0, E(0) \geq 0, I(0) \geq 0, R(0) \geq 0.$$

Then, there is an optimal control $u^*(t)$ together with the associated state variables (S^*, E^*, I^*, R^*) and the adjoint variables $(\lambda_1, \lambda_2, \lambda_3, \lambda_4)$, satisfying the following.

(1) The state system holds almost everywhere on $[0, T]$.

(2) The adjoint system is given by

$$\frac{d\lambda_1}{dt} = \lambda_1(\mu + (1 - u\epsilon_{m_1})\beta I) - \lambda_2(1 - u\epsilon_{m_1})\beta I, \quad (7.3a)$$

$$\frac{d\lambda_2}{dt} = -c_2 + \lambda_2(\nu + \theta + \mu) - \lambda_3\nu - \lambda_4\theta, \quad (7.3b)$$

$$\frac{d\lambda_3}{dt} = -c_3 + \lambda_1(1 - u\epsilon_{m_1})\beta S - \lambda_2(1 - u\epsilon_{m_1})\beta S + \lambda_2(1 - u\epsilon_{m_2})\zeta R \quad (7.3c)$$

$$- \lambda_3 \left(\delta + \mu + \left(\gamma + \frac{\rho}{1 + \kappa I} - \frac{\rho \kappa I}{(1 + \kappa I)^2} \right) \right) \quad (7.3d)$$

$$+ \lambda_4 \left(\gamma + \frac{\rho}{1 + \kappa I} - \frac{\rho \kappa I}{(1 + \kappa I)^2} + (1 - u\epsilon_{m_2})\zeta R \right), \quad (7.3e)$$

$$\frac{d\lambda_4}{dt} = -\lambda_1\eta + \lambda_2(1 - u\epsilon_{m_2})\zeta I - \lambda_4(\eta + \mu + (1 - u\epsilon_{m_2})\zeta I), \quad (7.3f)$$

with terminal conditions $\lambda_i(T) = 0$ for $i = 1, 2, 3, 4$,

The optimal control $u^*(t)$ is characterized almost everywhere by

$$u^*(t) = \min \left\{ \max \left\{ 0, \frac{\epsilon_{m_1}\beta S I(\lambda_1 - \lambda_2) + \epsilon_{m_2}\zeta R I(\lambda_2 - \lambda_4)}{2c_1} \right\}, 1 \right\}. \quad (7.4)$$

Proof. To proof the theorem above, we use Pontryagin's maximum principle [54, 55]. First, we define the Hamiltonian function

$$\begin{aligned} \mathcal{H} = & c_1 u^2 + c_2 E + c_3 I + \lambda_1 [\Lambda - (1 - u\epsilon_{m_1})\beta S I + \eta R - \mu S] \\ & + \lambda_2 [(1 - u\epsilon_{m_1})\beta S I - (\nu + \theta + \mu)E + (1 - u\epsilon_{m_2})\zeta R I] \\ & + \lambda_3 \left[\nu E - \delta I - \mu I - \left(\gamma + \frac{\rho}{1 + \kappa I} \right) I \right] \\ & + \lambda_4 \left[\theta E + \left(\gamma + \frac{\rho}{1 + \kappa I} \right) I - (\eta + \mu)R - (1 - u\epsilon_{m_2})\zeta R I \right]. \end{aligned}$$

The adjoint equations are derived from the Hamiltonian via

$$\frac{d\lambda_i}{dt} = -\frac{\partial \mathcal{H}}{\partial x_i}, \quad \text{for } i = 1, 2, 3, 4,$$

with transversality conditions $\lambda_i(T) = 0$.

Taking partial derivatives, we obtain the adjoint system

$$\begin{aligned}\frac{d\lambda_1}{dt} &= \lambda_1 (\mu + (1 - u\epsilon_{m_1})\beta I) - \lambda_2(1 - u\epsilon_{m_1})\beta I, \\ \frac{d\lambda_2}{dt} &= -c_2 + \lambda_2(\nu + \theta + \mu) - \lambda_3\nu - \lambda_4\theta, \\ \frac{d\lambda_3}{dt} &= -c_3 + \lambda_1(1 - u\epsilon_{m_1})\beta S - \lambda_2(1 - u\epsilon_{m_1})\beta S + \lambda_2(1 - u\epsilon_{m_2})\zeta R \\ &\quad - \lambda_3 \left(\delta + \mu + \left(\gamma + \frac{\rho}{1 + \kappa I} - \frac{\rho\kappa I}{(1 + \kappa I)^2} \right) \right) \\ &\quad + \lambda_4 \left(\gamma + \frac{\rho}{1 + \kappa I} - \frac{\rho\kappa I}{(1 + \kappa I)^2} + (1 - u\epsilon_{m_2})\zeta R \right), \\ \frac{d\lambda_4}{dt} &= -\lambda_1\eta + \lambda_2(1 - u\epsilon_{m_2})\zeta I - \lambda_4(\eta + \mu + (1 - u\epsilon_{m_2})\zeta I).\end{aligned}$$

To find the optimal control, we minimize the Hamiltonian with respect to u , and set

$$\frac{\partial \mathcal{H}}{\partial u} = 0,$$

which yields

$$\frac{\partial \mathcal{H}}{\partial u} = 2c_1 u + \epsilon_{m_1}\beta S I(\lambda_2 - \lambda_1) + \epsilon_{m_2}\zeta R I(\lambda_4 - \lambda_2) = 0.$$

Solving for u , we obtain

$$u^*(t) = \frac{\epsilon_{m_1}\beta S I(\lambda_1 - \lambda_2) + \epsilon_{m_2}\zeta R I(\lambda_2 - \lambda_4)}{2c_1}.$$

Applying the control constraint $0 \leq u \leq 1$, the optimal control is

$$u^*(t) = \min \left\{ \max \left\{ 0, \frac{\epsilon_{m_1}\beta S I(\lambda_1 - \lambda_2) + \epsilon_{m_2}\zeta R I(\lambda_2 - \lambda_4)}{2c_1} \right\}, 1 \right\}.$$

This completes the proof. $\square$

7.3. Optimal control simulations

As a recap from the previous section, our optimal control problem consists of a disease model that serves as the state system given in System (7.1), which must be solved forward in time; a cost functional in (7.2); an adjoint system with its transversality conditions in (7.3), which must be solved backward in time; and the optimal control formula given in (7.4), which must be updated iteratively until the convergence criterion is met. To solve this problem, we apply the forward-backward sweep method [54, 55]. The pseudocode is presented in Algorithm 1.

Algorithm 1 Forward–backward sweep method for solving the optimal control problem

- 1: **Input:** Initial state values $S(0), E(0), I(0), R(0)$, initial guess $u^{(0)}(t)$, time grid $t \in [0, T]$, parameter values, stopping criteria (tol), and the maximum iteration (max_iter)
- 2: Discretize $[0, T]$ into N time steps with step size Δt
- 3: Set $k \leftarrow 0$
- 4: **repeat**
- 5: **Forward sweep:** Solve the state equations (7.1) forward in time from $t = 0$ to $t = T$ using $u^{(k)}(t)$:
- 6: Compute $S^{(k)}(t), E^{(k)}(t), I^{(k)}(t), R^{(k)}(t)$
- 7: **Backward sweep:** Set $\lambda_i(T) = 0$ for all i
- 8: Solve the adjoint equations (7.3) backward in time from $t = T$ to $t = 0$
- 9: Compute $\lambda_1^{(k)}(t), \lambda_2^{(k)}(t), \lambda_3^{(k)}(t), \lambda_4^{(k)}(t)$
- 10: **Update control:** For each t_i in $[0, T]$, update the control variable using the formula in (7.4).
- 11: **Evaluate cost functional:** Calculate the cost function using the formula in (7.2)
- 12: Compute error $e = \|u^{(k+1)} - u^{(k)}\|_1$
- 13: $k \leftarrow k + 1$
- 14: **until** $e < \text{tol}$ or $k \geq \text{max_iter}$
- 15: **Output:** Optimal control $u^*(t)$, states S, E, I, R

To begin our numerical experiment on the optimal control problem, we first declare the baseline parameters and initial conditions as follows. All numerical experiments will be based on this set up, except if it is stated differently.

$$\begin{aligned}
 \Lambda &= 3.5, \beta = 0.003601, \epsilon_{m_1} = 0.5, \epsilon_{m_2} = 0.5, c_m = 0.1, \eta = 0.0001, \\
 \mu &= 0.009, \nu = 0.8, \theta = \frac{1}{7}, \delta = 0.014, \gamma = \frac{1}{14}, \rho = 0.2, \kappa = 0.05, \zeta = 0.00013, \\
 c_1 &= 1, c_2 = 1, c_3 = 1, S(0) = 1000, E(0) = 10, I(0) = 5, R(0) = 0.
 \end{aligned} \tag{7.5}$$

With the given parameter values, the basic reproduction number is calculated to be 3.99, indicating the existence of an endemic equilibrium. Based on this setup, the numerical results are shown in Figure 17. It can be observed that the control must be implemented at its maximum level from the beginning of the simulation period; that is, 100% of infected individuals are required to use face masks. After approximately 20 days, the control effort decreases due to a reduction in the number of infected individuals. However, to keep the number of infections low, the control effort returns to its maximum around Day 70 and remains at that level until near the end of the simulation period. Under this scenario, the number of susceptible individuals is significantly higher when control is applied, while the numbers of exposed and infected individuals are notably lower. Consequently, due to the reduced number of infections, the number of removed individuals is also lower. The total cost functional for this scenario is 20,242, and the cumulative reduction in the number of infected individuals reaches 5046.

The next simulation conducted to understand the impact of the intervention's duration. In this scenario, we use the same parameter as in the previous simulation for Figure 17, except with different simulation durations from $t \in [0, 500]$ into $t \in [0, 1000]$. The numerical results are depicted in Figure 18. The simulation results reveal an interesting pattern in the implementation of the control

intervention representing mask usage, as depicted in Figure 18(e). The level of control remains relatively low—even zero—during the early phase of the simulation and then suddenly increases to its maximum level around the middle of the time horizon. Even though the implementation of the control was late, it was proved to be significantly successful in increasing the number of susceptible individuals and achieving a larger reduction in the number of infected individuals (see Figure 18(a),(c)).

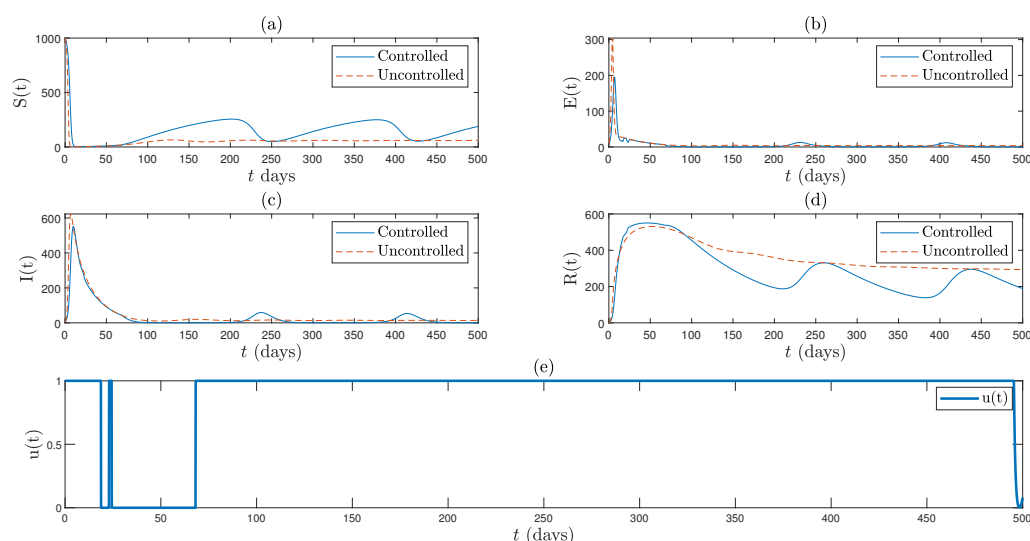


Figure 17. Numerical experiment with the baseline condition.

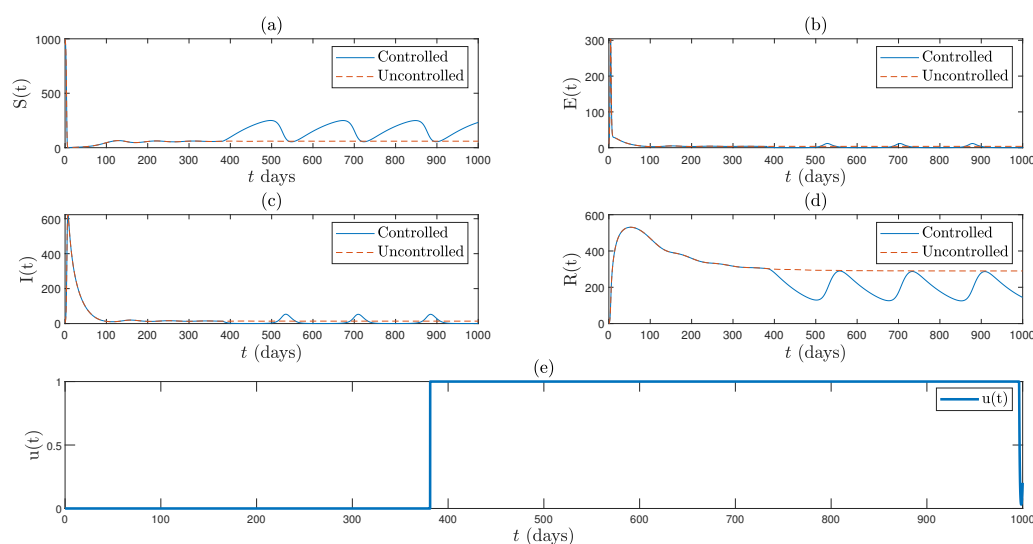


Figure 18. Numerical simulation with the baseline condition, except the duration of the intervention was extended up to 1000 days.

It is crucial to note that the controlled system shows an oscillatory behavior, indicating that the system has been disrupted from a stable endemic equilibrium into a stable periodic behavior. This kind of behavior could be a result of the nonlinear relationship between the timing of the intervention and the disease spread, which indicates the difficulties of implementing the intervention consistently for a long-term period. With this scenario, the total number of reduced infections is 4005 with the total cost for the intervention being 30,268, which is higher due to a longer simulation period of intervention. In conclusion, although the optimal intervention does not act immediately, its well-timed and sustained application in the middle phase successfully suppresses the infection and shifts the system toward a lower endemic burden, demonstrating both the efficiency and strategic nature of the control implementation.

For the next simulation, we modify the scenario by doubling the infection rate β , while maintaining the other parameters as before. Hence, we have $\mathcal{R}_0$ being twice the baseline scenario. As a result of this new scenario, we can see in Figure 19 that the implementation of medical mask use should be applied at its maximum effort from the start of the simulation period, maintained for almost 480 days, then reduced for a moment, and increased again when approaching Day 500. Consequently, we can see that the number of infected individuals decreases significantly, while the number of susceptible individuals consistently remains higher from the first day of the simulation period. Although this kind of high intervention successfully reduces the infected individuals, it comes with a higher cost compared with the previous scenario. The objective function increases to $J = 19,823$, which is higher than previous scenario. Nevertheless, the number of infections is reduced by 7322 individuals, which represents a greater absolute reduction compared with the baseline scenario. This numerical experiment highlights the potential impact of medical mask implementation. Even though the initial condition is more endemic, medical masks can still be promising for controlling the spread of COVID-19, even though it comes with a much higher cost for implementation.

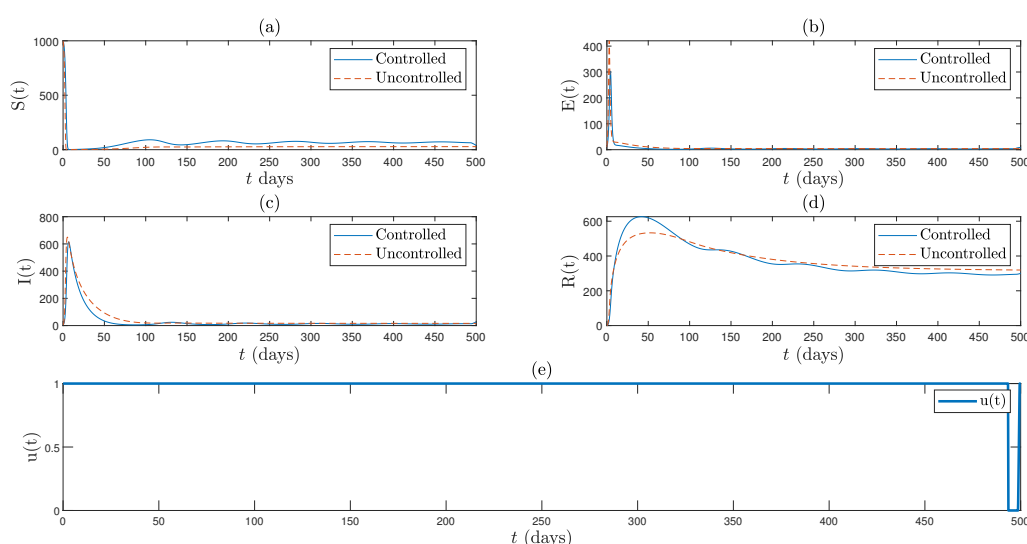


Figure 19. Numerical simulation with the baseline parameters, except with β being twice as large.

The next simulation is conducted by improving the mask efficacy from 50% to 75%, representing a better quality of medical mask that is suggested to be implemented in the population. This improvement in mask effectiveness significantly influences the structure of the optimal control profile $u(t)$ (Figure 20(e)), which begins at the maximum level, is temporarily relaxed during the middle of the simulation, and then ramps up again toward the end of the period. The impact of this improved intervention is clearly observable in the state variables. It can be seen in Figure 20(a) that although the number of susceptible also decreases in the early period of simulation, it rapidly increases after 50 days of implementing the of control, and monotonically increases to a more stable condition, which is much higher than the uncontrolled scenario. At the same time, we can see that although the number of infected individuals still experiences an outbreak, which appears on Day 10, it is much smaller than in the uncontrolled scenario, and it reduces afterward and consistently remains at a lower level compared with the uncontrolled scenario. The value of the objective functional is $J = 12,609$, substantially lower than in the baseline case. In addition, the total number of infections averted reaches 12,682, which is also higher than in the baseline scenario. This results highlight the importance of implementing a better medical mask efficacy to get better result from their implementation.

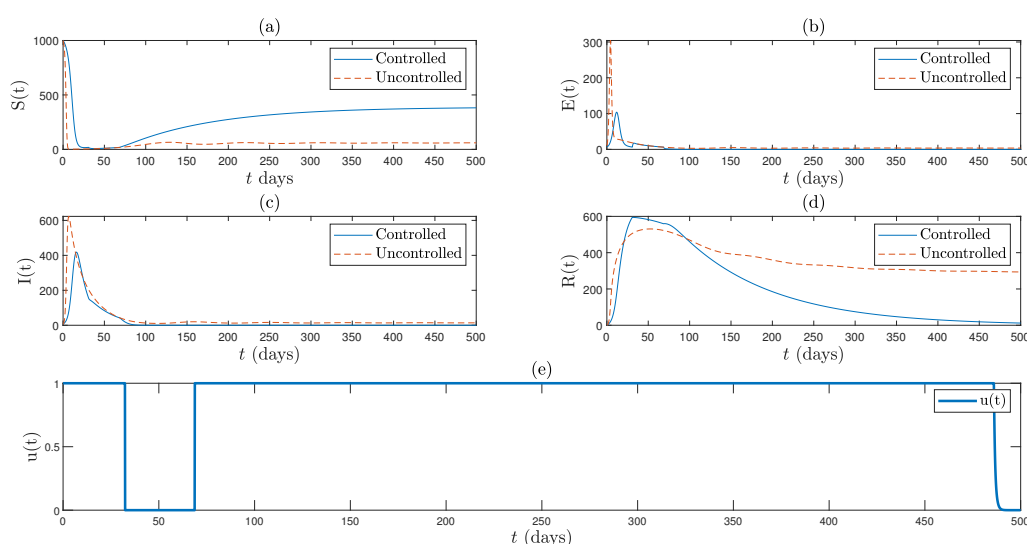


Figure 20. Numerical simulation with the baseline parameter, except the mask efficacy increases 50% to 0.75.

8. Discussion and conclusions

In this paper, we modified the COVID-19 model of [34] by incorporating the effect of medical masks on both the primary infection and reinfection pathways. We assumed that masks' efficacy against reinfection may differ from their efficacy against primary infection. This adjustment makes the model more realistic, since mask use can reduce not only primary transmission but also the reinfection process. Mathematical analysis of the existence and stability of equilibrium points was carried out rigorously. We derived the basic reproduction number of the model and showed that the disease-free equilibrium is locally asymptotically stable whenever $\mathcal{R}_0 < 1$ and unstable whenever

$\mathcal{R}_0 > 1$. However, due to the possibility of backward bifurcation, the condition $\mathcal{R}_0 < 1$ alone may not be sufficient to guarantee disease elimination. This result is consistent with the findings of [34] and other COVID-19 models (see, e.g., [56–58]). However, we also demonstrated analytically that the condition $\mathcal{R}_0 < 1$ does not always guarantee the eradication of COVID-19. In fact, it is still possible for an endemic equilibrium to exist and be stable even when $\mathcal{R}_0 < 1$. Thus, the proposed approach is validated by its consistency with previous COVID-19 modeling results and by the agreement between the analytical, numerical, bifurcation, sensitivity, and optimal control analyses. Moreover, our findings complement the existing literature by identifying additional dynamic scenarios that have not been fully addressed in the aforementioned studies.

Using the Castillo–Song bifurcation theorem [41], we show that a backward bifurcation can arise at $\mathcal{R}_0 = 1$ when the reinfection parameter exceeds its threshold. This implies that even though a stable COVID-19-free equilibrium exists for $\mathcal{R}_0 < 1$, a stable endemic equilibrium may also occur within a certain interval of $\mathcal{R}_0 < 1$ due to the presence of a FP. Numerical experiments also reveal a forward bifurcation with hysteresis at $\mathcal{R}_0 = 1$, where three endemic equilibria may coexist for some $\mathcal{R}_0 > 1$, two stable and one unstable, while the COVID-19-free equilibrium is unstable. In addition, Hopf bifurcation leads to periodic solutions, with either a single stable cycle or the coexistence of stable and unstable cycles together with a stable endemic equilibrium. The appearance of periodic solutions of the model suggests that the dynamics of each model variable do not tend to a stable equilibrium but instead may fluctuate over time. In practical terms, this oscillatory behavior becomes more difficult to control and is highly possible to be misunderstood, especially when we think the disease will decrease and attempt to relax the intervention. When a Hopf bifurcation phenomenon appears, then these kinds of changes happen over and over again in epidemic waves, with periods of rising and falling infections that never end. This shows how important it is to have control strategies that not only keep $\mathcal{R}_0$ below 1 but also avoid parameter regimes that can cause Hopf bifurcations. These can lead to long-term cyclical outbreaks instead of getting rid of the disease.

To complement our analytical and numerical results, we performed a global sensitivity analysis to identify the parameters that most strongly influence the model dynamics, using the PRCC method combined with LHS [53]. Our sensitivity analysis of $\mathcal{R}_0$ reveals that the infection rate is the most influential parameter that can be controlled, followed by the additional recovery rate due to treatment. Hence, reducing the probability of successful infection and/or reducing the additional recovery rate has been proven to reduce $\mathcal{R}_0$ more effectively than adjusting other parameters. In the matter of physical intervention in the field, this means that lowering the transmission rate through medical mask usage or even social distancing, like what was done at the beginning of the COVID-19 pandemic, can be considered an effective option. Furthermore, improving the recovery rate for infected individuals through treatment (hospitalization or proper home treatment) can also be considered in mitigating COVID-19. From a policy point of view, this shows how we need to balance between prevention and endemic eradication strategies. Moreover, our sensitivity analysis also shows other potential parameters that can be considered for COVID-19 eradication strategies, such as improving medical masks' efficacy. Another important piece of information that can be taken from the sensitivity analysis is that although the reinfection parameter does not appear in the expression of the basic reproduction number, this parameter still affects the dynamics of each variable, especially in the beginning of the simulation period or when the infection of COVID-19 is just at the start of its 'take-off' period. Consequently, the efficacy of medical masks in preventing reinfection exerts a

stronger influence on the early-time behavior of $I(t)$ compared with their effect on primary infection. Over longer time horizons, however, the additional recovery rate due to treatment becomes the dominant factor shaping the dynamics of $I(t)$. An increase in this parameter reduces not only the number of infected individuals $I(t)$ and exposed individuals $E(t)$, but also the removed population $R(t)$, while simultaneously increasing the susceptible population $S(t)$. This suggests that short-term control efforts should prioritize reducing reinfection, whereas long-term control depends more critically on strengthening treatment capacity and recovery rates.

To account for potential budget limitations in preventing the spread of COVID-19, we extend our model into an optimal control problem framework by defining the proportion of infected people wearing masks as a time-dependent control variable. The objective is to minimize the number of infected individuals while keeping intervention costs as low as possible. We apply Pontryagin's maximum principle (PMP) [54, 55] to characterize the optimal control problem and use the forward-backward sweep method to obtain numerical solutions. Our experimental results highlight the importance of adaptive intervention, which, in this case, depends on time, in order to control the spread of COVID-19 more effectively. Furthermore, a higher risk of COVID-19 transmission, indicated by a higher initial basic reproduction number, will require a more intense intervention.

In conclusion, our study shows that integrating reinfection, medical mask efficacy, and treatment to prevent COVID-19 transmission gives us a better understanding of how COVID-19 spreads among the population, including the possibility of backward bifurcation, hysteresis, and sustained oscillation phenomena. The analytical, numerical, and sensitivity analyses consistently demonstrate the crucial impact of reinfection, medical mask efficacy, and treatment in impacting both short-term outbreaks and long-term persistence. Optimal control results further indicate that timely implementation of adaptive mask usage can substantially decrease infections, even in the face of budget constraints. Despite its complexity, our model remains analytically tractable and numerically implementable. Moreover, the consistency of the analytical, bifurcation, sensitivity, and optimal control results supports the robustness of our main conclusions. We realize that our model has some limitations. Therefore, there are several possible future research directions that could be explored. One approach is to examine diverse mask-wearing behaviors among different population groups, such as aware and unaware individuals or children and adults, to capture disparities in compliance and accessibility. Another possible extension is to explicitly distinguish presymptomatic and asymptomatic infectious individuals, which are important characteristics of COVID-19's transmission. The model could also be expanded into a stochastic framework to account for environmental stochasticity and field uncertainties, for example, as discussed in [59]. Integrating vaccination strategies, such as newborn or adult vaccination, multiple vaccination doses, diminishing immunity, and synergistic interventions under financial limitations may offer more comprehensive guidance for policymakers. Unlike network-based epidemic models that explicitly describe individual behavioral adaptation, information dissemination, and evolving contact structures, the present study focuses on a deterministic population-level framework. Hence, extending the proposed model to include behavioral heterogeneity and evolving network structures would be an important topic for future research.

Use of AI tools declaration

The authors declare they have not used Artificial Intelligence (AI) tools in the creation of this article.

Acknowledgments

This research was supported by the Ministry of Education, Culture, Research, and Technology of the Republic of Indonesia, Directorate General of Higher Education, Research, and Technology, through Fundamental Research Grant Scheme 2026 (Grant No. PKS-182/UN2.RST/HKP.05.00/2026). The publication of this article was funded by the Indonesian Endowment Fund for Education (LPDP) on behalf of the Indonesian Ministry of Higher Education, Science and Technology and managed under the Global Reach Program (Contract No. 0791/DIRBAGA/V/2026 and 146/PKS/R/UI/2026). The funder had no role in the design of the study, data analysis, interpretation, or the decision to publish.

Conflict of interest

The authors declare there is no conflict of interest.

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