Research on Nonlinear Infectious Disease Models Influenced by Media Factors and Optimal Control

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Abstract

In this article, a mathematical model is developed to specify a specific function to describe the degree of disease control by mediating factors. The Lambert W function is used to convert the system defined by implicit functions into explicit functions. We analyze the dynamics of the defined segmented smooth system and verify the correctness of the theoretical analysis through numerical simulation. Through research, it is found that media influences can delay the peak of the epidemic and lead to a reduction in the scale of the epidemic. Then, we find that different control have a certain effect on the scale of the epidemic, our analysis shows that implementing dual is the most effective approach in limiting the spread of diseases.

Keywords: COVID-19, SIR epidemic model, optimal control

1. Introduction

2019 coronavirus disease (COVID-19) originates from a new type of severe acute respiratory syndrome coronavirus \[1\]. According to existing case data, some patients may experience respiratory and digestive symptoms such as nasal congestion, runny nose, diarrhea \[2,3\]. In severe cases, they may rapidly progress to acute respiratory distress syndrome, septic shock, difficult to correct metabolic acidosis, coagulation disorders, and multiple organ failure \[3\]. It should be noted that elderly people and those with chronic underlying diseases have a poorer prognosis, and the symptoms of youngster cases are relatively mild \[4,5\]. During the sudden outbreak of the epidemic, for avoiding cross infection of patients
with multiple strains [6], it is important to practice self-protection, wash hands frequently, maintain hygiene habits, raise health awareness, correctly wear disposable masks, avoid contact with infected individuals, and avoid going to dangerous crowded and enclosed places [7].

Media factors play a crucial role in the outbreak of an epidemic [8] [9]. They can detect and report on the epidemic early on, provide a warning, and use public opinion to take effective measures to eliminate potential crises before they escalate [10] [11]. By disseminating information about the epidemic, the media can help the public understand its severity and better control its development [12]. During the initial phases of the SARS outbreak in 2003, speculations circulated amongst the general public, causing widespread social unrest due to media coverage and insufficient dissemination of information regarding SARS prevention and treatment [13] [14]. In the wake of COVID-19, the media disseminated information to assist medical personnel and warn the public to prioritise personal protection, contributing significantly to guiding public perception. This statement highlights the need to explore how media coverage impacts the transmission and management of infectious diseases.

Mathematical models have the ability to predict disease development trends through dynamic analysis [15] [16] [17] [18] [19]. Currently, numerous studies have established mathematical models to simulate the dynamics of COVID-19 [20] [21] [22]. Jia and his colleagues proposed a dynamic model of COVID-19 based on official data to analyze the impact of non-pharmaceutical interventions on transmission dynamics during the COVID-19 pandemic [20]. Huang et al. established a COVID-19 mathematical model to analyze how spontaneous social distance and public social distance can increase the outbreak threshold of asymptomatic infections [21]. Nyaberi et al. through theoretical analysis and numerical simulation reveal that social distance has a significant impact on reducing the spread of COVID-19 [22]. Mathematical models enable the simulation of the impact of various factors on diseases and offer optimal control strategies for infectious diseases, thus providing valuable ideas for disease control.
The remaining structure of this article is as follows: in the second section, a SIR model is proposed, which includes media related factors. Using the properties of the Lambert W function to convert the system into an explicitly defined system through an implicit function. This system is a piecewise smooth system that can analyze the dynamic of the system. In the third section, we study the dynamics of piecewise smooth system, as well as the existence and local stability of endemic equilibrium points. Finally, the impact of various control methods on disease prevalence is modeled using optimal control theory. The numerical simulation results indicates that verifying the theoretical analysis results, and discovering that the vector function caused by infected individuals and infection rates can affect the scale of epidemic outbreaks. And our analysis shows that implementing dual measures is the most effective way to limit the spread of diseases, this may provide clues for disease control.

2. Model description

We consider the dynamics in susceptible population \( S(t) \), infected population \( I(t) \) and convalescent population \( R(t) \). The exponential decreasing factor of \( I(t) \) is used to express the reducing effect of media influence on the infection rate of infectious diseases, similar to reference [12], the function is defined as \( f(I) = be^{-N(I, \frac{dI}{dt})} \), where \( N(I, \frac{dI}{dt}) \) is shown as follows

\[
N(I, \frac{dI}{dt}) = \max \left\{ 0, m_1 I(t) + m_2 \frac{dI(t)}{dt} \right\}.
\]

Among them, \( m_1 \) and \( m_2 \) are non-negative parameters, which are used to represent the impact of media factors on media coverage cases and change rates. The \( N(t) = N(I, \frac{dI}{dt}) \) function is guaranteed to be nonnegative. For the \( R(t) \) population after recovery, they will no longer impose risk on susceptible individuals. In most studies, the model is established by assuming that the total population is constant or satisfies exponential growth [23, 24]. A new epidemic
dynamic model is obtained as follows

\[
\begin{align*}
\frac{dS}{dt} &= aS(1 - \frac{S}{k}) - e^{-N(1, \frac{dI}{dt})}bIS, \\
\frac{dI}{dt} &= e^{-N(1, \frac{dI}{dt})}bIS - \alpha I - \beta I - \gamma I, \\
\frac{dR}{dt} &= \gamma I - \mu R.
\end{align*}
\]

(1)

In model (1), all parameters are positive based on theoretical facts, and the meaning of each parameter is as follows: \(a\) is the inherent growth rate of population, \(k\) is the population carrying capacity of a given region. We hypothesis \(b\) is the basic propagation coefficient, \(f(I) = be^{-M(I, \frac{dI}{dt})}\) is the term of contact and transmission, which measures the spread of the virus from an infected person to a susceptible individual. \(\alpha\) is the mortality rate associate with the disease, \(\beta\) is the natural death rate, or death caused by the sequelae of disease recovery, \(\gamma\) is the rate of recovery from infection. To simplify, let \(n = \alpha + \beta + \gamma\), and \(N_1(t) = m_1 I(t) + m_2 \frac{dI}{dt}\), when \(N_1(t) > 0\), then \(N_1(t) = N(t)\). It from the second equation in (1), we can get

\[
m_2(\frac{dI}{dt} + nI)e^{m_2(nI + \frac{dI}{dt})} = m_2bSIe^{-m_1I + m_2nI}.
\]

(2)

It can be observed that the form of equation (2) is quite complex, then we introduce the definition and properties of Lambert W function, which is

**Definition 1.**[25] The Lambert W function is the inverse of the function \(f(z) = ze^z\) and satisfies the following conditions

\[
\text{Lambert W}(z) \cdot \exp(\text{Lambert W}(z)) = z.
\]

By definition, we have

\[
\text{Lambert W}(z) = \frac{\text{Lambert W}(z)}{z + (\text{Lambert W}(z))}.
\]

Using the definition of Lambert W function, we can obtain

\[
\frac{dI}{dt} = \frac{1}{m_2}W[m_2bSIe^{-m_1I + m_2nI}] - nI.
\]

(3)

Therefore \(N(t)\) reads

\[
N(t) = N_1(t) = m_1 I + m_2 \frac{dI}{dt}
\]

\[
=W[m_2bSIe^{-m_1I + m_2nI}] - (-m_1I + m_2nI).
\]

(4)
Then, we study that $N_1(t)$ is greater than zero, for this we consider is $N_1(t)$ equal to 0, then

$$W[m_2bSe^{-m_1I+m_2nI}] - (-m_1I + m_2nI) = 0,$$

we use the properties of Lambert W function, then

$$(-m_1I + m_2nI)e^{-m_1I+m_2nI} = m_2bSe^{-m_1I+m_2nI},$$

we obtain

$$S = \frac{m_2n - m_1}{bm_2} = S_q,$$

Because $N_1(t) > 0$ is strictly monotone for $S$, which yields, $N_1(t) > 0$ is equivalent to $S > S_q$.

In order to study the properties of the epidemic model, we remove the dynamics of the individual $R(t)$, and system (1) is transformed into system (8)

$$\begin{cases} 
\frac{dS}{dt} = aS(1 - \frac{S}{k}) - e^{-\theta M_1(t)}bIS, \\
\frac{dI}{dt} = e^{-\theta M_1(t)}bIS - \alpha I - \beta I - \gamma I.
\end{cases}$$

with

$$\theta = \begin{cases} 
0, S - S_q \leq 0, \\
1, S - S_q \geq 0,
\end{cases}$$

Equations (8) and (9) indicate that the system has a susceptibility threshold, and there is no influence of media factors below the threshold. Above the threshold, the media has a certain role in reducing the spread of disease, so the media has an impact on the disease to a certain extent. The susceptibility threshold is analyzed in the following content.

We set $P(Z) = S - S_q$ with $X = (S, I)^T$, then

$$P_{A_1}(Z) = (aS(1 - \frac{S}{k}) - bIS, bIS - \alpha I - \beta I - \gamma I)^T,$$

$$P_{A_2}(Z) = (aS(1 - \frac{S}{k}) - e^{-\theta M_1(t)}bIS, e^{-\theta M_1(t)}bIS - \alpha I - \beta I - \gamma I)^T,$$
The equations (8) and (9) become a non-smooth system

\[
\dot{X}(t) = \begin{cases} 
  P_{A_1}(X), & X \in A_1, \\
  P_{A_2}(X), & X \in A_2,
\end{cases}
\]

with

\[
A_1 = \{ X \in \mathbb{R}_+^2 : J(X) \leq 0 \}
\]

\[
A_2 = \{ X \in \mathbb{R}_+^2 : J(X) > 0 \}
\]

and the system (1) invariant set is \( R^2_+ = \{ X = (S,I), S \geq 0, I \geq 0 \} \). If \(-m_1 + m_2n > 0\) holds, we have \( S_q > 0 \); if \( S_q < 0 \), the set \( A_1 \) becomes an empty set. Then the non-smooth system (10) becomes smooth system \( \dot{X}(t) = P_{A_2}(X) \).

The switching line is defined by \( \Xi \) is

\[
\Xi = \{ X \in \mathbb{R}^2 : P(X) = 0 \}. 
\]

Therefore, the system (10) is located in region \( A_1 \) or \( A_2 \) record as \( S_{A_1} \) or \( S_{A_2} \), respectively. We use the properties of the Lambert W function to transform the implicitly defined function system (1) into a piecewise smooth (PWS) system [26]. The equilibrium point of a piecewise smooth system (10) as follows:

**Theorem 1.** If \( P_{A_1}(X^*) = 0, J(X^*) \leq 0 \), or \( P_{A_2}(Z^*) = 0, J(X^*) > 0 \), the point \( X^* \) is regular equilibrium of system (10); if \( P_{A_1}(X^*) = 0, J(X^*) > 0 \), or \( P_{A_2}(X^*) = 0, J(X^*) \leq 0 \), \( X^* \) is the virtual equilibrium point.

### 3. The dynamics of piecewise smooth system

In this part, we analyze the different areas \( S_{A_1} \) and \( S_{A_2} \) of the system to study the global dynamics of the system (10).

**The dynamics of \( S_{A_1} \)**

The system dynamics of \( S_{A_1} \) has a disease-free equilibrium point \( E_{0^*} \), which is \((k, 0)\), then

\[
k - S_q = \frac{m_2(R_0 - 1) + m_1}{bm_2},
\]
When $R_0 = kb/n > -\frac{m_1}{m_2} + 1$, the point $E_0^*$ in the area $A_2$, when $R_0 < -\frac{m_1}{m_2} + 1$, the equilibrium point $E_0^*$ is locally stable in the system of $S_{A_1}$. The interior equilibrium point $\tilde{E}_1 = (\tilde{S}_1, \tilde{I}_1)$ of $S_{A_1}$, which is exists only if $R_0 > 1$, and

$$\tilde{S}_1 = \frac{n}{b}, \tilde{I}_1 = \frac{a}{b} (kb/n - 1) = \frac{a}{b} (R_0 - 1),$$

note that $\tilde{S}_1 > S_q$ holds, which means that equilibrium $\tilde{E}_1$ is located in region $A_2$, so it is a virtual equilibrium.

The dynamics of $S_{A_2}$

For the smooth system $S_{A_2}$, the equation is as follows

$$\begin{cases}
\frac{dS}{dt} = aS(1 - \frac{S}{k}) - e^{-N_1(t)}bIS, \\
\frac{dI}{dt} = e^{-N_1(t)}bIS - \alpha I - \beta I - \gamma I,
\end{cases}$$

$S > S_c$. (11)

Due to $N_1(t)$ contains the Lambert W function, which is difficult to study its dynamics through theoretical analysis. The disease-free equilibrium of system (11) is $E_0^* = (k, 0)$, which is consistent with the corresponding of system $S_{A_1}$.

Existence of endemic equilibrium

Lemma 1. The interior equilibrium point $\hat{E}_2 = (S^*, I^*)$ is located in the area of $A_2$ and it is a regular equilibrium, where

$$S^* = \frac{n}{b} e^{m_1 I^*}, I^* = \frac{a}{b} e^{m_1 I^*} (1 - \frac{n}{kb} e^{m_1 I^*})$$

Proof: In the function $N_1(t)$, we define

$$G_1(S, I) \triangleq m_2 bSI e^{-m_1 I + m_2 n I}, G_2(I) \triangleq -m_1 I + m_2 n I,$$

then $N_1(t) = W(G_1(S, I)) - G_2(I)$, by using the properties of Lambert W function, we can get

$$\exp(-N_1(t)) = \exp(-W(G_1(S, I)) + G_2(I)) = \frac{W(G_1(S, I))}{G_1(S, I)} \exp(G_2(I)) = \frac{W(G_1(S, I))}{m_2 bSI}. \quad (12)$$

Substituting equation (12) into model (11), we have

$$\begin{cases}
\frac{dS}{dt} = aS(1 - \frac{S}{k}) - \frac{W(G_1(S, I))}{m_2}, \\
\frac{dI}{dt} = \frac{W(G_1(S, I))}{m_2} - nI,
\end{cases} \quad S > S_c. \quad (13)$$
If the second of the above formulas is equal to zero, then there is \( W(G_1(S, I)) = m_2 n I \), utilizing the properties of Lambert W function, we can obtain

\[
S^* = \frac{n}{b} e^{m_1 I^*},
\]

(14)

Substituting (14) into the first formula in (13) and combining with \( W(G_1(S, I)) = m_2 n I \), we get

\[
\frac{an}{b} e^{m_1 I^*}(1 - \frac{n}{kb} e^{m_1 I^*}) = n I^*,
\]

(15)

\[
I^* = \frac{a}{b} e^{m_1 I^*}(1 - \frac{n}{kb} e^{m_1 I^*}),
\]

(16)

We consider \( I^* > 0 \), and the parameters are all positive, that is

\[
(1 - \frac{n}{kb} e^{m_1 I^*}) > 0,
\]

(17)

which is equivalent to

\[
R_0 = \frac{kb}{n} > exp(m_1 I^*) > 1,
\]

(18)

If \( R_0 > 1 \), the interior equilibria \( \tilde{E}_2 = (S^*, I^*) \) is satisfies condition \( S^* > S_q \), which means that \( \tilde{E}_2 \) is located in the area of \( A_2 \) and it is a regular equilibrium. \( \square \)

**Local stability of the endemic equilibrium \( \tilde{E}_2 \)**

**Lemma 2.** If the parameters satisfy the following relationship

\[
-T_2 \frac{n}{m_2} + n + \frac{T_1}{m_2} - a + \frac{2aS^*}{k} > 0,
\]

\[
\frac{nT_1}{m_2} + \frac{aT_2}{m_2} - \frac{2aT_2S^*}{m_2 k} - a + \frac{2aS^*}{k} > 0,
\]

\[
(-\frac{T_2}{m_2} + n + \frac{T_1}{m_2} - a + \frac{2aS^*}{k})^2 - 4(\frac{nT_1}{m_2} + \frac{aT_2}{m_2} - \frac{2aT_2S^*}{m_2 k} - a + \frac{2aS^*}{k}) > 0,
\]

then the system (13) has the point \( \tilde{E}_2 \) is locally asymptotically stable.

**Proof:** We use the Jacobian matrix to analyze the stability of equilibrium point \( \tilde{E}_2 \), set \( c = -m_1 + m_2 n \) and

\[
D_1(S, I) = aS(1 - \frac{S}{k}) - \frac{W(G_1(S, I))}{m_2},
\]

\[
D_2(S, I) = \frac{W(G_1(S, I))}{m_2} - n I,
\]

(19)
the Jacobian matrix is as follow
\[
J = \begin{pmatrix}
\frac{\partial D_1}{\partial S} & \frac{\partial D_1}{\partial I} \\
\frac{\partial D_2}{\partial S} & \frac{\partial D_2}{\partial I}
\end{pmatrix} = \begin{pmatrix}
-\frac{1}{m_2} T_1 + a - \frac{2aS}{k} & -\frac{1}{m_2} T_2 \\
-\frac{1}{m_2} T_1 & \frac{1}{m_2} T_2 - n
\end{pmatrix}, \quad (20)
\]

Where \( T_1 \) and \( T_2 \) are defined as
\[
T_1 = \frac{\partial W(G_1(S, I))}{\partial S} = \frac{W(G_1(S, I))}{G_1(S, I)(1 + W(G_1(S, I)))} \frac{\partial G_1}{\partial S},
\]
\[
T_2 = \frac{\partial W(G_1(S, I))}{\partial I} = \frac{W(G_1(S, I))}{G_1(S, I)(1 + W(G_1(S, I)))} \frac{\partial G_1}{\partial I},
\]

with \( \frac{\partial G_1}{\partial S} = m_2 b I e^{c_I}, \frac{\partial G_1}{\partial I} = m_2 b I e^{c_I} (1 + c_I) \). In order to simplify the calculation at the equilibrium point \( \tilde{E}_2 \), we can get
\[
T_1 = \frac{m_2 n I^*}{G_1(S^*, I^*)(1 + m_2 n I^*)} m_2 b I^* e^{c_I^*} = \frac{m_2 n I^*}{S^*(1 + m_2 n I^*)},
\]
\[
T_2 = \frac{m_2 n I^*}{G_1(S^*, I^*)(1 + m_2 n I^*)} m_2 b S^* e^{c_I^*} (1 + c_I^*)
\]
\[
= \frac{m_2 n (1 + c_I^*)}{S^*(1 + m_2 n I^*)},
\]

Thus, the characteristic equation at point \( \tilde{E}_2 \) is
\[
\lambda^2 + \left( -\frac{T_2}{m_2} + n + \frac{T_1}{m_2} - a - \frac{2aS^*}{k} \right) \lambda + \frac{n T_1}{m_2} + \frac{a T_2}{m_2} - \frac{2a T_2 S^*}{m_2 k} - a + \frac{2a S^*}{k} = 0
\]
\[
(22)
\]

Then if the characteristic equation satisfies the following conditions
\[
-\frac{T_2}{m_2} + n + \frac{T_1}{m_2} - a + \frac{2a S^*}{k} > 0,
\]
\[
\frac{n T_1}{m_2} + \frac{a T_2}{m_2} - \frac{2a T_2 S^*}{m_2 k} - a + \frac{2a S^*}{k} > 0,
\]
\[
\left( -\frac{T_2}{m_2} + n + \frac{T_1}{m_2} - a + \frac{2a S^*}{k} \right)^2 - 4 \left( \frac{n T_1}{m_2} + \frac{a T_2}{m_2} - \frac{2a T_2 S^*}{m_2 k} - a + \frac{2a S^*}{k} \right) > 0,
\]

it can be obtained that the characteristic equation has two negative roots in the region \( A_2 \), which means that the point \( \tilde{E}_2 \) is locally asymptotically stable.\( \square \)

**4. Optimal control strategies**

In this section, we investigate the dynamic behaviors of the system under control variables \( u_1(t) \) and \( u_2(t) \). The first control equation \( u_1(t) \) is to enhance
prevention strategies, reduce the number of patients, vaccinate and wear masks, or increase social distance. This control can reduce the probability of illness among susceptible populations, represented by \((1 - u_1(t))\). The second control equation \(u_2(t)\) represents accelerating the recovery time of patients, enhancing medical conditions, developing specific drugs, and enhancing human immune capacity. We focus on the impact of media factor \(m_1\) on the infected individuals, while ignoring the impact of \(m_2\) on infection rates, we set \(m_1 \neq 0, m_2 = 0\), the system become

\[
\begin{align*}
\frac{dS}{dt} &= aS(1 - \frac{S}{K}) - e^{-m_1 b} IS(1 - u_1(t)), \\
\frac{dI}{dt} &= e^{-m_1 b} IS(1 - u_1(t)) - \alpha I - \beta I - (\gamma + u_2(t)) I, \\
\frac{dR}{dt} &= (\gamma + u_2(t)) I - \mu R.
\end{align*}
\]  

When \(u_i = 1, (i = 1, 2)\) indicate complete control, and \(u_i = 0\) indicate that control is ineffective. We consider the following optimal control problem to minimize the objective functional is given by

\[
J(u_1, u_2) = \min_{0 \leq u_1, u_2 \leq 1} \int_0^{T_f} \left( \omega_1 I + \frac{1}{2} \omega_2 u_1^2(t) + \omega_3 u_2^2(t) \right) dt.
\]  

The weight constant \(\omega_1\) represents the infected population, while \(\omega_2\) and \(\omega_3\) represent the weight constants of personal protection, and improvement of medical conditions, respectively. The terms \(\frac{1}{2}\omega_2 u_1^2(t)\) and \(\frac{1}{2}\omega_3 u_2^2(t)\) describe the costs associated with the corresponding intervention measures over the time interval \([0, T_f]\). Assuming that the cost is proportional to the square of the corresponding control function.

\[J(u_1^*, u_2^*) = \min\{J(u_1, u_2) : u_1, u_2 \in U\}.
\]

Where, \(U\) is defined by \(U = \{(u_1, u_2) \mid 0 \leq u_1, u_2 \leq 1\}\) for \(t \in [0, T_f]\). Therefore, in order to determine the necessary conditions that the optimal control \((u_1^*, u_2^*)\) must satisfy, Pontryagin’s maximum principle is used \([29]\) and the
Hamiltonian $\mathcal{H}$ for the control problem is defined by:

$$\mathcal{H} = \omega_1 I + \frac{1}{2}[\omega_2 u_1^2(t) + \omega_3 u_2^2(t)] + \lambda_1[aS(1 - \frac{S}{K}) - e^{-m_1 I} bIS(1 - u_1(t))],$$

$$+ \lambda_2[e^{-m_1 I} bIS(1 - u_1(t)) - \alpha I - \beta I - (\gamma + u_2(t)) I],$$

$$+ \lambda_3[(\gamma + u_2(t)) I - \mu R],$$

with the terminal (transversality) conditions

$$\lambda_i(T_f) = 0, i = 1, 2, 3.$$

Further, the optimal control double $(u_1^*, u_2^*)$ are given as follows:

$$u_1^* = \max\{0, \min\{1, \frac{e^{-m_1 I} bIS(\lambda_1 - \lambda_2)}{\omega_2}\}\},$$

$$u_2^* = \max\{0, \min\{1, \frac{I(\lambda_2 - \lambda_2)}{\omega_3}\}\}.$$

5. Numerical Simulations

In this section, we use numerical simulations to verify the rationality of the theory, observe and study the dynamic phenomena of the system.

In Fig.1 and Fig.2 display the relationship between the system and $R_0$. When $R_0 < 1$, the disease-free equilibrium point $(k, 0)$ of the system is located in region $A_1$ or $A_2$, depending on the values of $m_1$ and $m_2$. This indicates that the region where the equilibrium point is located is closely related to the degree of media influence. When the equilibrium point is located in $A_1$, it is
Figure 1: The phase diagram of the SIR model under the case of $R_0 = 0.6579 < 1$. The dotted line indicates $S = S_q$, the left side of the dotted line is the $A_1$ area, and the right side is the $A_2$ area. The black point is the equilibrium point of disease elimination $E_0^*$, and the parameters are $b = 0.5, r = 1.5, \beta = 0.2, \alpha = 0.2, k = 2.5, a = 0.1, \mu = 0.1$ (a) $S(0) = 4, m_1 = 0.2, m_2 = 0.8; (b) I(0) = 0.5, m_1 = 0.6, m_2 = 0.4.$

shown in Fig.1 (a), and it can be observed that changing different initial values of system will cross the line $S = S_q$ and stabilize at the equilibrium point; when the equilibrium point is located at $A_2$, it is shown in Fig.1 (b). It can be found that the trajectory starting from $A_2$ will stabilize at the equilibrium point, while the trajectory with an initial value at $A_1$ will directly pass through $S = S_q$ to reach the equilibrium point at $A_2$.

On the other hand, when $R_0 > 1$, the system dynamics are shown in Fig.2. The trajectory with an initial value at $A_1$ will directly pass through $S = S_q$ to reach the equilibrium point at $A_2$. If the trajectory starting from region $A_2$ will cross the line $S = S_q$ and then return to the $A_2$, because its asymptotically stable equilibrium point $E_0^*$ is virtual and located in $A_2$, finally, the trajectory stabilizes at point $\tilde{E}_2$, which is regular and is globally asymptotically stable in $A_2$.

Next, we continue to analyze the impact of $R_0$ and media factors on the
Figure 2: The phase diagram of the SIR model under the case of $R_0 = 1.9737 > 1$. The dotted line indicates $S = S_q$, the left side of the dotted line is the $A_1$ area, and the right side is the $A_2$ area. The black points are the local equilibrium points $E^*_0$ and $E^*_2$, and the parameters are $b = 1.5, r = 1.5, \beta = 0.2, \alpha = 0.2, k = 2.5, a = 0.1, \mu = 0.1, m_1 = 0.2, m_2 = 0.8$.

disease, it can be seen from Fig.3 (a) and Fig.3 (b) that whether $R_0$ is greater than one has obvious impact on the disease. In Fig.3 (a), we consider the case of $m_1 = 0, m_2 = 0$. When $R_0 > 1$, the number of diseases will first increase and then decrease, and eventually asymptotically stabilize at a steady-state value. When $R_0 < 1$, the number of patients shows a downward trend, which is much lower than the number of diseases in the system when $R_0 > 1$. In Fig.3 (b), we consider media influencing factors, which is $m_1 = 0.2, m_2 = 0.8$. The situation is similar to Fig.3 (a), but when $R_0 < 1$, we change media related parameters $m_1$ and $m_2$ has no effect on the number of diseases. This shows that when the basic reproduction number $R_0 < 1$, the influence of media factors on the disease condition is less. Therefore, in the event of a disease outbreak such as COVID-19, we should use the media to have an impact on the disease at an appropriate time.

In order to explore the influence of media factors on diseases, we first select
the case when $R_0 > 1$, and change the values of media-related parameters $m_1$ and $m_2$. In Fig.3(c), we fix $m_1 = 0.2$, set $m_2 = 0, 0.4$ or 0.8, it can be found that with the increase of $m_2$ value, the peak number of patients $I$ move back, and the maximum value became smaller. In Fig.3(d), we set $m_2 = 0.4$ and change the value of $m_1 = 0, 0.4$ or 0.8, it can be observed that the peak value decreases with the increase of $m_1$, and the peak appearance time is slightly earlier. Thus, we can conclude that the impact of different media-related parameters on the disease is different, which is the same as our understanding. It shows that media
Figure 4: The influence of parameters $m_1$ and $m_2$ on $I(t)$. (a) The system fix $m_1 = 0$ and (b) $m_1 = 0.2$, (c) fix $m_2 = 0$ and (d) $m_1 = 0.4$.

reports need to choose certain methods to ensure a positive impact on disease control. This depends on the authenticity of media reports and the popularity of media reporting platforms.

Our study of the effects of parameters $m_1$ and $m_2$ on the infected population is shown in Fig.4, where the color bar represents the fluctuation height. It can be observed that when fix $m_1 = 0$, the system fluctuates slowly and moves backwards with the increase of $m_2$, and the fluctuation is significant at positions close to 1. When $m_1$ is magnified to 0.2, the fluctuation moves backwards very slowly. Interestingly, when we fix $m_2 = 0$, as $m_1$ increases, the second wave of $I(t)$ shifts significantly forward. When we change $m_2 = 0.4$, it can be observed that the wave decreases near 0, while the subsequent wave changes are not
Sensitivity analysis can analyze and understand the impact of different parameters on specific variables, which may help us control disease transmission or provide guidance. Referring to the definition in [27], the sensitivity index of $R_0$ for each parameter $c$ is defined as follows

$$L_c^{R_0} = \frac{\partial R_0}{\partial c} \times \frac{c}{R_0}.$$  

In Fig. 5, it can be observed that among all positive correlation factors, the population carrying capacity $k$ and basic transmission coefficient $b$ are the highest. This indicates that the value of $k$ or $b$ is positively correlated to $R_0$, with a degree of 100%. However, among all the negative correlation factors, the rate of recovery from infection $\gamma$ is the most sensitive parameter, and the value of $\gamma$ is negatively correlated to $R_0$, with a degree of 78.95%.

The numerical simulation of the optimal control system is implemented using MATLAB, and the equation is solved using the forward backward of the fourth order Runge-Kutta method in [28]. The weight constants are chosen as $\omega_1 =$
Figure 6: (a) Combined effects of optimal controls $u_1(t)$, $u_2(t)$ on the system. (b) or (c) Control profile ($u_1(t)$ or $u_2(t)$) and its effects on the system. (d), (e) and (f) Time history diagrams under different control situations of $I(t)$. 

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50, $\omega_2 = 100$, and $\omega_3 = 1$. Fig.6 (a) shows the time history of two controls $u_1$ and $u_2$ coexisting, Fig.6 (d) represents the time history of infected population $I(t)$ under dual-control, the red line represents no-control system, and the blue line represents controlled system. It can be observed that under dual-control, the disease population quickly reaches the minimum value. Fig.6 (b) and (c) show the time history of the system under a single control of $u_1$ and $u_2$, corresponding to Fig.6 (e) and (f). It can be observed that under a single control, $u_1$ has a stronger control effect on the disease population, while $u_2$ can reduce the peak of the disease population.

From the perspective of epidemics, maintaining an effective social distance under the influence of control $u_1$ reduces the probability of illness in susceptible populations and achieves the effect of controlling the total number of infectious diseases as quickly as possible in a limit period of time. Under the influence of control $u_2$, the effect of controlling the number of diseases is achieved relatively slowly in a short period of time by accelerating the recovery time of patients and improving medical conditions. Under the influence of both control strategies, the effect of controlling the number of diseases is stronger and faster, which provides ideas for the prevention of epidemics.

6. Conclusions

Inspired by COVID-19, this paper constructs the SIR model to include media factors to explore the control effects of media factors on diseases. In the theoretical analysis, the media factor uses the Lambert W function to transform the system defined by the implicit function into the displayed piecewise smooth function. We study the system dynamics and use numerical simulation to verify its correctness. Since the piecewise smooth function is closely related to the basic reproduction number $R_0$, we have studied it and found that the media factor has an obvious control effect on the epidemic situation under the basic reproduction number $R_0 > 1$, which verifies that the media can inhibit the disease to a certain extent effect.
Using sensitivity analysis, it can be found that the population carrying capacity $k$ and basic transmission coefficient $b$ are positively correlated with $R_0$, while the rate of recovery from infection $\gamma$ is negatively correlated with $R_0$. This may provide a control strategy for disease control. Finally, we used the optimal control strategy to analyze the impact of different controls on the system and found that dual-control have the best effect on disease control, and a single strategy that improved personal protection or vaccination is also have effective in disease control.

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Declarations

The authors have no conflicts of interest to declare. All co-authors have seen and agree with the contents of the manuscript.

Data availability

All data generated or analysed during this study are included in this published article.

References


