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Analysis of COVID-19 using a modified SLIR model with nonlinear incidence

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**ABSTRACT**

Infectious diseases kill millions of people each year, and they are the major public health problem in the world. This paper presents a modified Susceptible-Latent-Infected-Removed (SLIR) compartmental model of disease transmission with nonlinear incidence. We have obtained a threshold value of basic reproduction number ($R_0$) and shown that only a disease-free equilibrium exists when $R_0 < 1$ and endemic equilibrium when $R_0 > 1$. With the help of the Lyapunov-LaSalle Invariance Principle, we have shown that disease-free equilibrium and endemic equilibrium are both globally asymptotically stable. The study has also provided the model calibration to estimate parameters with month wise coronavirus (COVID-19) data, i.e. reported cases by worldometer from March 2020 to May 2021 and provides prediction until December 2021 in China. The Partial Rank Correlation Coefficient (PRCC) method was used to investigate how the model parameters' variation impact the model outcomes. We observed that the most important parameter is transmission rate which had the most significant impact on COVID-19 cases. We also discuss the epidemiology of COVID-19 cases and several control policies and make recommendations for controlling this disease in China.

**Introduction**

COVID-19 is an infectious disease that establishes a persistent and vital public-health problem over the world. In 2020, it was estimated that there were approximately 153,523 cases of COVID-19, and 5,736 persons died due to the disease infection [1]. The COVID-19 virus is easily transmitted from an infected person to another person and creates respiratory droplets via close contact when an infected person sneezes or coughs. Once infected, the person will initially experience a period without visible clinical signs, called latent (asymptomatic) COVID-19 infection, and after a particular time, the latent (asymptomatic) COVID-19 infection progress to active (symptomatic) COVID-19 infection.

COVID-19 is a potentially contagious disease that causes several deaths in 2020 [1,2]. It is a worldwide viral disease, has been spreading and affecting people of all ages [2]. The illness can be more severe if it occurs in teenagers with a weakened immune system [3]. The COVID-19 spreads undoubtedly from a person with an active COVID-19 patient to another person when the infectious person coughs, conjunctivitis, and a susceptible person comes into physical contact [4]. The spread of COVID-19 depends on the duration of exposure of susceptible people to the infected person [5]. In turn, this depends on many factors, such as whether there were a crowded environment, the prevailing climatic conditions and the immune status of the individual [6].

Mathematical models can improve our understanding of the epidemiology of infectious disease and those components that are significant to infectious disease diagnosis and treatment [7–12]. Mathematical modelling tools can be used to study some factors, e.g. service factors, disease-related factors, environmental factors, and sociological factors, and to describe the underlying physical mechanisms, inspiring researchers to eliminate trial and error methods and visible them towards rational model-based manner [13–17].

In this paper, we exploit the Lyapunov direct method [18] to study the global stability analysis on the transmission dynamics of a modified SLIR compartmental model with nonlinear incidence. The incidence rate is an essential component for infectious disease transmission in a mathematical model [19]. In the epidemiology perception, incidence rate measures the number of new cases of a disease within a specified time duration as a percentage of the number of persons at threat for the disease [20].

The stability analysis reveals that the disease-free equilibrium is globally asymptotically stable when the basic reproduction number $R_0$ is
less than one; otherwise, it is unstable. Further, the disease-endemic equilibrium is globally asymptotically stable when $R_0$ is greater than one and $\alpha$ is sufficiently large or $\alpha$ is sufficiently small. To estimate the values of the parameters, the model calibrates and makes forecasts about the number of COVID-19 cases in China. Sensitivity analysis also carries out to recognize the crucial model parameters that could probably assist policymakers in preventing the COVID-19 epidemic in China. The results that we generate from this model can be effective in other settings dealing with the high burden of COVID-19 cases.

The rest of the paper is structured as follows: “Model description and analysis” section presents model descriptions, estimates the basic reproduction number $R_0$ and equilibrium solutions. In “Global stability analysis” section, perform the global stability analysis. “Estimation of model parameters” and “Sensitivity analysis” sections performed the model calibration and sensitivity analysis. Finally, in “Numerical simulations” section, we provide numerical simulations to support analytic results. A brief discussion and concluding remarks finalize the paper.

### Model description and analysis

We developed a modified SLIR transmission dynamics compartmental model framework with a nonlinear incidence between the following mutually exclusive compartments: $S(t)$- susceptible persons; $L(t)$- latent persons who have not yet shown symptoms and consider to asymptomatic infected population; $I(t)$- symptomatic infected persons who are capable to infectious and infected, and $R(t)$- recovered persons who are before infected but effectively recovered from the disease. In evaluating the model construction below, we remark that COVID-19 virus infections with purifying immunity do not naturally present infection. In this aspect, our model is reasonable for COVID-19 that are commonly modeled as SLIR type in China’s actual situation, many populations are moving in and out of each city and epidemic-associated deaths.

It should be noted that while people are moving from one city to another city, there exists a higher incidence of COVID-19 in congested areas. Therefore, considering the nonlinear incidence rate is reasonable due to the influential contacts between infective and susceptible persons may saturate at high levels through crowding of infective persons. A typical SLIR model diagram is described in Fig. 1.

Here, we assumed that susceptible persons are recruited at a fixed rate $\Lambda$, and they can be affected at a time-dependent rate $\beta(t)\frac{S(t)L(t)}{1+\alpha}$ which is the saturated level of incidence rate due to the large values of $I$. Here, $\beta(t)$ contains the force of infection due to the disease is starting an entirely susceptible population, and $\frac{1}{1+\alpha}$ contains the inhibition impact from the susceptible persons due to the behaviour change through their number rise or from the impact of contributing factors such as high population density area of the infective persons with $\alpha$ regulates the saturates level of the force of infection. People in various classes experience the same constant natural death rate $\mu$. All infected people progress to the latently (asymptomatic) infected compartment,$L(t)$. The latently (asymptomatic) infected population progress to the active (symptomatic) infected compartment due to the progression rate $\omega$. A proportion of the infected (symptomatic) peoples moves to the recovered compartment $R(t)$ due to the treatment and natural recovery rate $\gamma$.

In this way, the model can be designated as the following system of differential equations:

$$\frac{dS}{dt} = \Lambda - \frac{\beta S I}{1+\alpha} - \mu S \tag{1}$$

$$\frac{dL}{dt} = \frac{\beta S I}{1+\alpha} - (\omega + \mu)L \tag{2}$$

$$\frac{dI}{dt} = \omega L - (\gamma + \mu)I \tag{3}$$

$$\frac{dR}{dt} = \gamma I - \mu R. \tag{4}$$

With non-negative initial conditions for the system of equations above, it is easy to express that each of the state variables remains non-negative for all $t > 0$. Moreover, from the Eqs. (1) to (4), we find that the total population size, $N(t)$, satisfies

$$\frac{dN(t)}{dt} = \Lambda - \mu N$$

Integrating this equation, we find

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

It reveals that the total population size $N(t)$ is bounded, and it follows that each compartment states (i.e. $S$, $I$, and $L$) are also bounded.

### Basic reproduction number and existence of equilibria

The basic reproduction number is well-defined as the expected number of secondary cases created by a single infectious case introduced into a totally susceptible population. The disease can spread in a population only if the basic reproduction number is greater than one. An epidemic occurs when an infection spreads through and infects a significant proportion of a population. A disease-free population is possible when the basic reproduction number is less than one, which means that the disease naturally fades out [21,22].

There are four states in the modelling system in which two are infected states, i.e. $L$ and $I$, and two are uninfected states, i.e. $S$ and $R$. At the infection-free steady state $L = I = R = 0$, hence $S^0 = \frac{\Lambda}{\mu}$. The Eqs. (2) and (3) are closed in that they do not involve the derivation of $S$ from steady-state value. Also, $R$ does not appear in Eqs. (2) and (3), and for $(L, I)$ we have the following equations:

$$\frac{dL}{dt} = \frac{\beta S I}{1+\alpha} - (\omega + \mu)L. \tag{5}$$
\[ \frac{dl}{dt} = \omega L \left(1 - \frac{l}{L}\right). \]  

Here, these Ordinary Differential Equations (ODEs) in (5) and (6) are referred to as the infection subsystem, as they only describe the production of newly infected individuals and changes in the states of already infected individuals.

By setting \( T = (L, I) \), where the prime denotes transpose, the infection subsystem can be written in the following form:

\[ \dot{x} = (T + \Sigma)x. \]  

The matrix \( T \) corresponds to transmission (arrival of susceptibles into the infected compartments \( L \) and \( I \)) and the matrix \( \Sigma \) to transitions. Removal through death is included in the transition to keep the notation simple. All epidemiological events that lead to new infections are incorporated in the model via \( T \) and other events via \( \Sigma \). If the infected states are indicated with \( I \) and \( j \) with \( i, j \in 1, 2 \), then the entry \( T_{ij} \) is the rate at which individuals in infected state \( j \) give rise to individuals in infected state \( i \).

Regarding the subsystem Eqs. in (5) and (6), we obtain

\[ T = \begin{pmatrix} 0 & \beta S^0 \\ 0 & 0 \end{pmatrix} \quad \text{and} \quad \Sigma = \begin{pmatrix} -(\omega + \mu) & 0 \\ \omega & -(\gamma + \mu) \end{pmatrix}, \]

\[ -\Sigma^{-1} = \begin{pmatrix} 1 & 0 \\ \omega & (\omega + \mu)(\gamma + \mu) \end{pmatrix}. \]

Here, the element of \( (\Sigma^{-1}) \) is the expected time spent in state \( i \) for an individual currently in state \( j \) during its entire life. Also, it has the components described as below:

The term \( \frac{1}{\omega(\gamma + \mu)} \) refers to the time spent in \( L \) compartment; the term \( \frac{\mu}{\omega(\gamma + \mu)} \) indicates the expected life of spent in \( I \) compartment starting from \( L \) compartment with the factor \( \frac{\mu}{\omega(\gamma + \mu)} \) representing the probability of transitioning from compartment \( L \) to compartment \( I \), and the term \( \frac{1}{\gamma + \mu} \) intimates the time spent in \( I \) compartment starting from \( I \).

The next-generation matrix (NGM) method is used to evaluate the basic reproduction number for a compartmental model of the spread of infectious diseases. Two related matrices exist, which we define as the NGM with the large domain and a small domain. NGM with the large domain is always the matrix with the highest dimension, incorporating all infected states in the model. The small domain of NGM has a lower dimension and only incorporates infectious states. A small domain matrix will exist and can be used to define the NGM if there are fewer states of infectiousness than states at infection [21].

For this analysis, NGM with the large domain is denoted by \( K_L \). A latency state and infectious state are both infected states, but the variation from latency to infectiousness does not contain a new infection but an already established infection moving to a different infection stage. It has led to confusion as other researchers have tried to reconcile the appealing linear algebra approach [21]. To make the distinction clear and remove confusion, we will call the matrix NGM (K_{L*}), and the \( K_L \) can be expressed as

\[ K_L = -T\Sigma^{-1} = T(-\Sigma^{-1}) = \begin{pmatrix} 0 & \beta S^0 \\ 0 & 0 \end{pmatrix} \begin{pmatrix} 1 & 0 \\ \omega & (\omega + \mu)(\gamma + \mu) \end{pmatrix} \begin{pmatrix} \beta S^0 \omega \\ \omega \end{pmatrix} \begin{pmatrix} S^0 \beta \\ (\omega + \mu)(\gamma + \mu) \end{pmatrix}. \]

i.e., \( K_L = \begin{pmatrix} \beta S^0 \omega \\ \omega \end{pmatrix} \begin{pmatrix} S^0 \beta \\ (\omega + \mu)(\gamma + \mu) \end{pmatrix}. \)

The dominant eigenvalue is the basic reproduction number of the disease. It represents the average number of infections produced by one infected individual.

Now the characteristic equation is \( |K_L - \lambda I| = 0 \), here \( \lambda \) represents the identity matrix.

\[ \begin{vmatrix} \frac{\beta S^0 \omega}{(\omega + \mu)(\gamma + \mu)} - \lambda & S^0 \beta \\ 0 & 0 - \lambda \end{vmatrix} = 0, \]

\[ \lambda^2 - \lambda \left( \frac{\beta S^0 \omega}{\omega(\gamma + \mu)} \right) = 0. \]

\[ \lambda_1 = 0 \text{ and } \lambda_2 = \frac{\beta S^0 \omega}{\omega(\gamma + \mu)}. \]

Hence, the basic reproduction number is \( R_0 = \frac{\beta S^0 \omega}{\omega(\gamma + \mu)} \).

Now we break down the various components that make up the basic reproduction number of the model.

Here, the term \( \frac{\beta S^0 \omega}{\omega(\gamma + \mu)} \) is the probability of becoming infectious once infected (i.e. the probability of transitioning from state \( L \) to state \( I \)) and \( \frac{1}{\gamma + \mu} \) represents mean infectious period.

**Positivity and boundedness of solutions**

For the above system (1)–(4), we find a region of attraction which is given by lemma 1.

Lemma 1: The set \( \Omega = \{(S, L, I, R) \in \mathbb{R}^4 : 0 < S + L + I + R \leq \frac{\Lambda}{\mu} \} \) is a positive invariant region of system (1)–(4).

Proof. Let \( N = S + L + I + R \) then

\[ \frac{dN}{dt} = \frac{\delta \Lambda}{t^2} + \frac{\delta \Lambda}{t^2} + \frac{\delta \Lambda}{t^2} + \frac{\delta \Lambda}{t^2}, \]

\[ \frac{d\Lambda}{dt} = -\mu N, \] and

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

This shows that solutions of system (1)–(4) point toward \( \Omega \). Hence, \( \Omega \) is positively invariant, and solutions (6)–(8) are bounded. The above lemma 1 shows that all solutions of the model are non-negative and bounded.

**Existence of equilibria**

Clearly, Eqs. (1)–(4) always have a disease free equilibrium

\[ E^0 = (S^0, L^0, I^0, R^0) = \left( \frac{\Lambda}{\mu}, 0, 0, 0 \right). \]

From Eqs. (1) to (4) we can also derive the endemic equilibrium \( E^* = (S^*, L^*, I^*, R^*) \), where

\[ S^* = \frac{\Lambda \omega \mu + \mu (\omega + \mu)(\gamma + \mu)}{\mu \omega (\beta + \alpha)}, \]

\[ L^* = \frac{\mu (R_0 - 1)}{\omega \beta + \alpha}, \]

\[ I^* = \frac{\mu (R_0 - 1)}{\beta + \alpha}, \]

\[ R^* = \frac{\gamma (R_0 - 1)}{\beta + \alpha}. \]

which shows that if \( R_0 > 1 \) then the endemic equilibrium \( E^* = (S^*, L^*, I^*, R^*) \in \Omega \).

**Global stability analysis**

In this section, we discuss the global stability analysis of the disease-free and disease-endemic equilibriums. The stability analysis will identify in which conditions the disease is stable or unstable. Hence, it will help policymakers make decisions for intervention strategies to achieve a disease-free population. Here, we consider the global stability analysis for the disease-free equilibrium \( E^0(S^0, L^0, I^0, R^0) \) and disease-endemic
equilibrium \( E^* (S^*, L^*, \Gamma^*, R^*) \) follow by the Lyapunov-LaSalle Invariance Principle [23,24].

**Theorem 3.1.** The disease-free equilibrium \( E^0 (S^0, L^0, \Gamma^0, R^0) \) is globally asymptotically stable if \( R_0 < 1 \).

**Proof.** Consider the following Lyapunov function,

\[
V = (S - S^0) \ln S + L + I + C
\]

where, \( C = -S^0 + S^0 \ln S^0 \)

\[
V = \left( 1 - \frac{S^0}{S} \right) \left( \Lambda - \frac{\beta S}{1 + \alpha L} - \mu S \right) + \frac{\beta S}{1 + \alpha L} (\omega + \mu) L + (\gamma + \mu) I
\]

\[
= \left( 1 - \frac{S^0}{S} \right) \left( \frac{\beta S}{1 + \alpha L} - (\omega + \mu) L + \mu L + (\gamma + \mu) I \right)
\]

\[
= \left( 1 - \frac{S^0}{S} \right) \left( \frac{\beta S}{1 + \alpha L} - (\omega + \mu) L + \mu L + (\gamma + \mu) I \right)
\]

\[
= \mu S^0 - \frac{\beta S}{1 + \alpha L} - \mu S - \frac{\beta S^0}{1 + \alpha L} + \mu S + \frac{\beta S}{1 + \alpha L} - (\gamma + \mu) I - \mu L
\]

\[
= 2 \mu S^0 - \frac{\beta S^0}{1 + \alpha L} - \mu S - \frac{\beta S^0}{1 + \alpha L} + \mu S + \frac{\beta S}{1 + \alpha L} - (\gamma + \mu) I - \mu L
\]

Since the arithmetic mean exceeds the geometric mean, it follows that

\[
\frac{S}{S^0} + \frac{S^0}{S} \geq 2. \text{ Therefore, } \frac{V}{S} \leq 0 \text{ for } R_0 < 1.
\]

Hence it follows by the Lyapunov-LaSalle invariance principle, the disease-free equilibrium \( E^0 \) is globally asymptotically stable when \( R_0 < 1 \).

**Theorem 3.2.** The disease endemic equilibrium \( E^* (S^*, L^*, \Gamma^*, R^*) \) is globally asymptotically stable if \( R_0 > 1 \).

**Proof.** Let us consider the function

\[
f(x) = \frac{x}{1 + ax}
\]

In order to simplify many of the expressions which follows evaluation both sides of (1), (2) and (3), we get

\[
\Lambda = \mu S^* + \beta S^* f(\Gamma^*)
\]

\[
(\omega + \mu) L^* = \beta S^* f(\Gamma^*)
\]

and

\[
\omega L^* = (\gamma + \mu) \Gamma^*
\]

which will be used as substitutions in the calculations below.

Let, \( g(x) = x - 1 - \ln(x) \) and

\[
V_l(t) = g \left( \frac{S(t)}{S} \right)
\]

\[
V_l(t) = g \left( \frac{L(t)}{L} \right)
\]

\[
V_l(t) = \frac{1}{\beta f(\Gamma)} V_s + \frac{L^*}{\beta S^* f(\Gamma)} V_l + \frac{1}{(\gamma + \mu) V_1}
\]

We will study the behaviour of the Lyapunov function

\[ V(t) = \frac{1}{\beta f(\Gamma)} V_s + \frac{L^*}{\beta S^* f(\Gamma)} V_l + \frac{1}{(\gamma + \mu) V_1} \] (11)

We note that \( g \) has the strict global minimum \( g(1) = 0 \). Thus, \( V(t) \geq 0 \) with equality if and only if \( \frac{S}{S^0} = \frac{x}{x^0} = \frac{S^0}{S} = 1 \). For clarity, the derivatives of \( V_s, V_l \) and \( V_1 \) will be calculated separately and then combined to obtain \( V_l(t) \).

Now,

\[
V_l(t) = \frac{S}{S} - 1 - \ln \left( \frac{S}{S^0} \right)
\]

\[
\frac{dV_s}{dt} = \frac{1}{S} \left( S - S^0 \right) \frac{dS}{dt}
\]

\[
= \frac{1}{S} \left( 1 - \frac{S}{S^0} \right) \frac{dS}{dt}
\]

\[
= \frac{1}{S} \left( 1 - \frac{S}{S^0} \right) \left( \Lambda - \mu S - \beta S f(\Gamma) \right)
\]

\[
= \frac{1}{S} \left( 1 - \frac{S}{S^0} \right) \left( \mu S^* + \beta S^* f(\Gamma^*) - \mu S - \beta S f(\Gamma) \right)
\]

\[
= \frac{1}{S} \left( 1 - \frac{S}{S^0} \right) \left[ \mu (S^* - S) + \beta (S^* f(\Gamma^*) - S f(\Gamma)) \right]
\]

\[
= \mu \left( \frac{S - S^*}{S^0} \right)^2 + \beta f(\Gamma) \left( 1 - \frac{S^0}{S} \right) \left[ 1 - \frac{S f(\Gamma)}{S^0 f(\Gamma)} \right]
\]

Let, \( x = \frac{S^0}{S}, y = \frac{L^*}{L}, z = \frac{\Gamma^*}{\Gamma} \) and \( F(x) = \frac{e^x - 1}{x} \)

\[
\frac{dV_l}{dt} = \frac{\mu (S^0 - S^*)^2}{S^0 S} + \beta f(\Gamma) \left( 1 - \frac{S^0}{S} \right) \left\{ 1 - x F(x) - \frac{1}{x} F(x) \right\}
\]

Again,

\[
V_l(t) = \frac{L}{L^*} - 1 - \ln \left( \frac{L}{L^*} \right)
\]

\[
\frac{dV_l}{dt} = \frac{1}{L} \frac{dL}{dt} \left( \frac{L}{L^*} - 1 \right) \frac{dL}{dt}
\]

\[
= \frac{1}{L} \left( \frac{L}{L^*} - 1 \right) \frac{dL}{dt}
\]

\[
= \frac{1}{L} \left( \frac{L}{L^*} - 1 \right) \left( \beta S f(\Gamma) - (\omega + \mu) L^* \right)
\]

\[
= \frac{1}{L} \left( \frac{L}{L^*} - 1 \right) \left( \beta S f(\Gamma) - (\omega + \mu) L^* \right)
\] (12)
\[ V_i(t) = \frac{1}{\Gamma} - 1 - \ln \left( \frac{1}{\Gamma} \right) \]

\[ \frac{dV_i}{dt} = \frac{1}{\Gamma} \frac{df}{dt} - \frac{1}{\Gamma} \frac{d\Gamma}{dt} \]

\[ \frac{dV_i}{dt} = \frac{1}{\Gamma} \left( 1 - \frac{\Gamma}{T} \right) \frac{d\Gamma}{dt} \]

\[ = \frac{1}{\Gamma} \left( 1 - \frac{\Gamma}{T} \right) \left( \omega L - (\gamma + \mu) I \right) \]
Combining Eqs. (12)–(14), multiplying appropriately by coefficients determined by (11), we obtain

\[
\frac{dV_i}{dt} = \left(\gamma + \mu\right) \left(1 + \frac{1}{\Gamma^2}\right) \left(1 - \frac{1}{\Gamma}\right) \left(y - z\right)
\]

\[
\frac{dV_s}{dt} = \frac{1}{\beta S \Gamma} \frac{dV_s}{dt} + \frac{1}{\beta I \Gamma} \frac{dV_s}{dt} + \frac{1}{\beta S \Gamma} \frac{dV_s}{dt} + \frac{1}{\gamma + \mu} \frac{dV_s}{dt}
\]

\[
\frac{dV_l}{dt} = \left(\gamma + \mu\right) \left(1 + \frac{1}{\Gamma^2}\right) \left(1 - \frac{1}{\Gamma}\right) \left(y - z\right)
\]

\[
\frac{dV_i}{dt} = \left(\gamma + \mu\right) \left(1 + \frac{1}{\Gamma^2}\right) \left(1 - \frac{1}{\Gamma}\right) \left(y - z\right)
\]

\[
\frac{dV_s}{dt} = \frac{1}{\beta S \Gamma} \frac{dV_s}{dt} + \frac{1}{\beta I \Gamma} \frac{dV_s}{dt} + \frac{1}{\beta S \Gamma} \frac{dV_s}{dt} + \frac{1}{\gamma + \mu} \frac{dV_s}{dt}
\]

\[
\frac{dV_l}{dt} = \left(\gamma + \mu\right) \left(1 + \frac{1}{\Gamma^2}\right) \left(1 - \frac{1}{\Gamma}\right) \left(y - z\right)
\]
\[ \frac{dV}{dt} = -\frac{\mu(S-S^*)^2}{\beta(I)SS^*} - g(\frac{1}{x}) - g\left(\frac{xF(z)}{y}\right) + F(z) - z - \ln\left(\frac{1}{x}\right) - \ln\left(\frac{y}{z}\right) - \ln\left(\frac{xF(z)}{y}\right) \]
Fig. 5. Measured and predicted number of cumulative COVID-19 cases from March 2020 to December 2021 (red dot) and the corresponding model (blue solid curve) with the 95% confidence interval (CI) measure in the blue shaded limits. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

Fig. 6. Impact of saturation infection ($\alpha$) on the dynamics of COVID-19 cases in China.
It is simple to show that
\[ F(z) = -z - \ln\left(\frac{1}{z}\right) - \ln\left(\frac{\alpha z}{\beta}\right) - \ln\left(\frac{\gamma z}{\mu}\right) \leq 0 \]
with equality only if \( x = y = z = 1 \). Combining that with \( g \geq 0 \) with equality only if the argument is 1, we see that \( \frac{dV}{dt} \leq 0 \).

We note that \( \frac{dV}{dt} \) is only zero if \( x = y = z = 1 \). In particular, this requires that for any solution \( S(t) = S' \), \( L(t) = L' \), and \( I(t) = I' \) for all \( t \). Thus we see that all solutions limit to the endemic equilibrium. Therefore, by the Lyapunov-LaSalle invariance principle, we conclude that the disease endemic equilibrium \( E' \) is globally asymptotically stable.

In order to establish the nature of the infection-free equilibrium and endemic equilibrium analysis, we performed numerical simulation using the Monte Carlo method in [25] to verify the conditions by computing the fundamental part of the eigenvalues of the Jacobian matrix of infection-free equilibrium and co-existent endemic equilibrium. Since Eqs. (1)-(3) are independent of the size of the recovered population \( (R) \); therefore, if we only wish to track COVID-19 incidence and prevalence, we can focus our attention on the Eqs. (1)-(3). Hence, the Jacobian matrix is in the following form,

\[
J = \begin{pmatrix}
-\mu - \frac{\beta}{(1 + \alpha \beta)} & 0 & -\frac{\beta}{(1 + \alpha \beta)} \\
\frac{\beta}{(1 + \alpha \beta)} & -(\omega + \mu) & \frac{\beta}{(1 + \alpha \beta)} \\
0 & \omega & -(\gamma + \mu)
\end{pmatrix}
\]

According to the Jacobian matrix, the simulation results of infection-free equilibrium and endemic equilibrium are presented in Figs. 2 and 3.

Fig. 2 showed that the infection-free equilibrium is locally asymptotically stable as all the three eigenvalues (real part) are negative (i.e., \( \lambda_1, \lambda_2, \lambda_3 \leq 0 \)). Whilst Fig. 3 showed that the endemic equilibrium is unstable as one eigenvalue (real part) is positive (i.e., \( \lambda_3 > 0 \)).

Estimation of model parameters

The outbreak of COVID-19 first reported the capital of China, Wuhan, Hubei, China. The outbreak rapidly spread from Wuhan city to other Chinese cities and multiple countries, and the World Health Organization has declared the COVID-19 outbreak a global crisis [26]. Therefore, it is important to examine the spread in the early stages of China to prevent further outbreak. In this study, we consider the China COVID-19 data to estimate parameters value and identify the most significant parameter for controlling the outbreak of this disease.

Model parameters are estimated based on the available data from worldometers.info [27]. In order to estimate the model parameters including transmission rate \( \beta \), progression rate \( \omega \), recovery rate \( \gamma \), infectious saturates rate \( \alpha \), we use existing knowledge available in the literature (Table 1) and fit the model curve by minimizing the error of
the incidence data from March 2020 to May 2021 employing MATLAB routine. Fig. 4 shows the incidence data of COVID-19 (red dot) and the model fitted curve (blue solid curve).

We also present the model predictions (see Fig. 5) to prepare the China Ministry of Health and political leaders responsible for controlling COVID-19 in the future. However, if the number of infected persons follows the trend, we assume that around 100,000 patients are infected by December 2021, as shown in Fig. 5. Considering that the number of available beds in intensive care units in China is close to 51,891 [28], and assuming that half of these beds can be used for patients with COVID-19, the system will be at maximum capacity, according to the prediction, by December 2021 (see Fig. 5).

Moreover, Fig. 6 shows the effect of saturation infection on the dynamics of the COVID-19 outbreak in China. We observed that the saturation infection negatively associates with the COVID-19 outbreak, which means increasing the saturation infection will reduce COVID-19 cases. Further, decreasing saturation infection will increase the number of COVID-19 cases in China.

Data strongly recommend that the number of births is nearly fixed for the 30 years globally and is projected to continue fixed for the next 30 years. Hence, it is reasonable to consider a fixed growth rate (λ) within the timescale of an SLIR model [33].

Sensitivity analysis

It is essential to measure the degree of adequacy of our proposed model and recognize which parameters play an important role in the model outcomes. [34,35]. Here, we consider the sensitivity analysis of the partial rank correlation coefficient (PRCC) method to explore the influence of model parameters on the model outcomes. [35,36]. This study considers the model outcomes as the prevalence (I) of COVID-19 cases and the basic reproduction number R0.

Figs. 7 and 8 show the association between COVID-19 prevalence (I) and the basic reproduction number R0 with the model parameters β, o, α and γ. Results show that parameters β and o have a positive association with the model outcomes, indicating that a positive variation of these parameters will soar the value of I and R0. On the other hand, parameters α and γ have a negative association with the model outcomes, which indicates that raising these parameters will reduce the value of I and R0.

Numerical simulations

In this section, we carry out numerical simulations using the Matlab programming language to support the analytic results. Two equilibrium points were found: the disease-free equilibrium (E0) and endemic equilibrium (E∗). The Lyapunov function was used to investigate the global stability of these points. We used different initial conditions of all populations and found that if the basic reproduction number is less than one (i.e., R0 < 1), the disease-free equilibrium is globally asymptotically stable. If R0 > 1, then the disease persists in the population. Fig. 9 represents disease-free equilibrium, and we used different initial conditions for this system trajectories in the L vs I plane. In this modelling system, the disease dies out because the basic reproduction number is smaller than one. Fig. 10 displays disease-endemic equilibrium. In this case, the disease persists in the population due to the basic reproduction number being greater than one.

Discussion and concluding remarks

In this article, we performed the global qualities of a modified SLIR compartmental model with nonlinear incidence. We showed that the disease-free or disease-endemic equilibrium of the model is globally asymptotically stable depending on the basic reproduction number R0. The global stability of the disease-free equilibrium indicates that for any primary level of infection, the disease will ultimate die out from the population when the basic reproduction number R0 less than one but when the basic reproduction number R0 is greater than one, which indicates that the disease persists in the population.

Our model carried out the analytic expression of COVID-19 incidence I and other significant parameter R0, i.e. the probable number of secondary cases produced by the single infectious introduce into an entirely susceptible population. From the analytic expression, we observed that these parameters I and R0 both depend on transmission rate β, progression rate o, recovery rate γ, and saturates incidence rate α. Further, our sensitivity analysis showed that transmission rate β and recovery rate γ have the largest influence on the prevalence of COVID-19 and the basic reproduction number. Therefore, it is vital to minimize the transmission rate β and the recovery rate γ for controlling the COVID-19 in China through a viable intervention program and effective treatment strategy.

There are so many ways that we can control COVID-19 transmission (i) wash your hands regularly with soap and water or rubbing an alcohol-based sanitizer into your hands because washing your hands kills viruses that may be on your hands, (ii) avoid touching your face as much as possible because virus-containing droplets on your hands can be transferred to your eyes, mouth or nose where they can infect you, (iii) maintain at least 1.5 m distance between yourself and anyone who is coughing or sneezing because if you are too close to someone, you might breathe in the droplets they cough or sneeze, (iv) make sure you and people around you follow good respiratory hygiene. Respiratory hygiene is essential because droplets spread the virus. By following good respiratory hygiene, you catch any droplets that might be produced, and this protects the people around you from viruses, including COVID-19. (v) Must wear a mask if you are sick with symptoms that might be due to COVID-19 or looking after someone who may have COVID-19 [37-41].

Hence, we recommend that the most realistic and best strategy to control COVID-19 cases in China is to reduce the transmission rates, including suppression strategies such as immediate lockdowns in some towns at the epicentre of outbreak and prevention strategy slow down infection but not ending epidemic for reducing peak. Further, increase the treatment rate may be another option by reducing the treatment cost. Lastly, the design of the recommended model and its complementary results can be applied to other settings that are experiencing a high burden of COVID-19 cases. We propose that our future work be focused on: (i) How to shrink the transmissions of COVID-19 effectively by using the natural treatment? (ii) What policy measures would be the best option at both local and national levels to control the COVID-19 transmission? (iii) A greater community level emphasize through active social media campaigns and mass media education, individuals awareness to prevent the transmission of COVID-19.

CRediT authorship contribution statement

Md Abdul Kuddus: Data curation, Writing - original draft, Methodology, Visualization, Validation, Software, Formal analysis. Azizur Rahman: Conceptualization, Writing - review & editing, Investigation, Visualization, Validation, Supervision.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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