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Forecast and evaluation of asymptomatic COVID-19 patients spreading in China

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A R T I C L E   I N F O

MSC:
37B25
37G10
37N25

Keywords:
Disease COVID-19
Population mobility
Asymptomatic patients
Transcritical bifurcation
Global stability

A B S T R A C T

In this paper, we propose a new SAIR model to depict the transmission dynamics of a novel coronavirus in China. We focus on the ability of asymptomatic COVID-19 patients to transmit and the potential impact of population movements on renewed outbreak transmission. Qualitative analysis of the model shows that when the basic reproductive number \( R_0 > 1 \), the system will stabilize towards a unique endemic equilibrium and pass through a transcritical bifurcation around its disease-free equilibrium. Furthermore, by constructing an appropriate Lyapunov function, we prove that the disease-free equilibrium and endemic equilibrium are globally asymptotically stable under appropriate parameter conditions. Finally, some important results have been verified by numerical simulations.

Introduction

Since the first case of COVID-19 was diagnosed, the epidemic of COVID-19 has been one of the focuses of people’s attention. Unfortunately, as of December 20, 2021, the world hit a grim coronavirus milestone with 5.36 million confirmed deaths and close to 275.6 million confirmed cases. The number of deaths has greatly exceeded the other two coronaviruses (severe acute respiratory syndrome coronavirus, SARS-CoV, and Middle East respiratory syndrome coronavirus, MERS-CoV) [1,2]. The global epidemic is still ongoing, which has seriously impacted the global public health and economics. Despite the joint efforts of the whole country, the coronary epidemic in China has been well controlled. However, the impact of overseas imports and population mobility (especially for asymptomatic patients) on epidemic control cannot be ignored.

The establishment of the infectious disease model as a powerful tool to study the mechanism of infectious disease control and transmission is of great significance to control infectious diseases and reduce the occurrence of infectious diseases. At the beginning of the discovery of COVID-19, domestic and foreign scholars combined with epidemiological data or dynamic models to predict the peak time and scale of the epidemic, and the effective times of COVID-19 reproduction, etc [3–7]. Some scholars also build models to infer the turning point of the outbreak in Hubei Province and even the whole country or predict the impact of resumption of work on the development of the epidemic [8–11].

But the early studies lacked sufficient raw data. And the understanding of novel coronavirus pneumonia is still in the exploratory stage. Therefore, most of the predictions of the epidemic situation in these studies deviated from the real situation. More importantly, these studies did not take into account the high transmission capacity of new coronavirus in the incubation period, asymptomatic infection, and the impact of population mobility on the transmission of the epidemic. Intensive research on novel coronavirus pneumonia, we have learned that both patients with latent period and asymptomatic infection are possible sources of transmission. Thus, in subsequent studies, the transmission characteristic of patients with a latent period or asymptomatic infections have been incorporated into novel coronavirus pneumonia models by most scholars [12–16]. For instance, Manotosh Mandal et al. formulated a model introducing a quarantine class and governmental intervention. The study revealed that reducing exposure to exposed and susceptible individuals is the most critical factor in achieving disease control [12]. Khan and Atangana et al. formulated a new mathematical model for the dynamics of COVID-19 with quarantine and isolation [17]. Some scholars also put forward mathematical models to study the spread and transmission of COVID-19 in the population, especially the role of asymptomatic infected people. In Ref. [14], the...
authors propose a compartmental mathematical model for the spread of the COVID-19 disease to focus the infectiousness of super-spreaders individuals. But in this model, only the infectiousness of exposed persons is considered, and the infectiousness of asymptomatic with infections is not considered. In fact, studies using data from China’s early reports combined with Bayesian inference analysis indicated that asymptomatic infections accelerated the spread of the epidemic [18]. Besides, a large number of mathematical models or researches have been developed to focus on the COVID-19 and other related topics [19–26]. However, it is rare to establish a model that considers the impact of both the infection characteristics of asymptomatic infected persons and population mobility on the COVID-19 spread.

In view of the above-mentioned literature, we also present a simpler mathematical model that can be used to study the impact of population mobility and the movement of asymptomatic infectious individuals on the development of the COVID-19. Different from the existing models mentioned above, we consider the following assumptions. Firstly, considering that symptomatic infected persons have obvious diseases, they will be isolated and treated in time once they are found out. It means that the rate infection from symptomatic infections to susceptible individuals are quite low. Hence, we ignored the infection rate from symptomatic infections to susceptible individuals. Second, the epidemic situation in the provinces has basically stabilized since May. This also indicates that both medical conditions and treatment techniques have been basically stable in China. Therefore, the constant cure rate is considered in our model. However, as people step into normal life, the potential risk of re-outbreak of the epidemic situation cannot be ignored due to the floating population, especially the asymptomatic individuals. Then, we focus on the impact of population mobility on the further development of the epidemic. In addition, patients who have been cured, relapse cases are very rare. Thus, we assumed that there is no transfer from the recovery of individuals to susceptible class.

To sum up, we study the dynamics of a SAIR epidemic model with nonlinear incidence rate, constant input rate, and constant treatment rate. The whole paper is organized as follows, we introduce the SAIR model used for our analysis in the model formation section. Some model basic properties and the existence and uniqueness of equilibrium are also presented in this section. The analysis of the stability and bifurcation of equilibrium are given in Section “On the Stability and bifurcation of equilibrium”. The corresponding numerical simulations of some important theorems are given in Section “Numerical Simulations”. A brief summary and further discussion of these results are presented in Section “Conclusion”.

Model formation

In this section, we consider a mathematical model SAIR that describes the dynamic transmission of COVID-19 in mobile populations. The total population size \(N(t)\) is divided into four compartments, namely susceptible individuals (S(t)), asymptomatic individuals (A(t)), symptomatic individuals (I(t)) and recovered individuals (R(t)). The flow between different compartments of the model is shown in Fig. 1.

The system of ordinary differential equations as follows

\[
\begin{align*}
\frac{dS(t)}{dt} &= \lambda - \beta SA(t)N(t) - \mu S(t), \\
\frac{dA(t)}{dt} &= \frac{\beta SA(t)N(t)}{N(t)} - \mu A(t) - p_1A(t) - p_2I(t), \\
\frac{dI(t)}{dt} &= p_1A(t) - \mu I(t) - dI(t) - \gamma I(t), \\
\frac{dR(t)}{dt} &= p_2A(t) + \gamma I(t) - \mu R(t),
\end{align*}
\]

where \(S(0) > 0, A(0) \geq 0, I(0) \geq 0, R(0) \geq 0\) are the initial state.

Here, the parameter \(\lambda > 0\) is the comprehensive input rate and \(\mu > 0\) represents the natural death rate. \(d > 0\) denotes the death rate due to disease. In our model, vertical transmission is not considered, i.e. all newborns are susceptible. According to the results of clinical practice, asymptomatic infections generally do not need treatment, but this group of people is a strong source of infection. Therefore, the incidence rate is, where indicates the effective per capita contact rate of asymptomatic infections. Some asymptomatic infected people in the fourteen-day isolation period, will show obvious signs of infection, that is, diagnosed. The parameter \(p_1\) is the rate at which the asymptomatic individuals become symptomatic individuals. If the asymptomatic infected person is asymptomatic during the fourteen-day isolation period and the nucleic acid test is negative for two times, the isolation can be released. We hypothesized that the asymptomatic infected patients who were released from isolation were transformed into recovered individuals after they eliminated the new coronavirus through autoimmune resistance. The parameter \(p_2 > 0\) is the rate at which the asymptomatic individuals become recovered individuals. \(\gamma > 0\) is the recovery rate of symptomatic individuals. Clinically, relapse is rare in cured patients. Thus, in our model, it is assumed that there is no transfer from the recovery of individuals to the susceptible class. Based on the above assumptions, we formulated a dynamical system (1) consisting of four differential equations to depict the flow diagram of COVID-19.

Model basic properties

In this section, we present some basic model properties that will be useful in the rest of this paper.

**Theorem 1.** If \(S(0) > 0, A(0) \geq 0, I(0) \geq 0\) and \(R(0) \geq 0\), the solutions \(S(t), A(t), I(t)\) and \(R(t)\) of system (1) are positive for all \(t \geq 0\).

**Proof.** It follows from the first equation of system (1) that

\[
\frac{dS(t)}{dt} = \lambda - \beta S(t) \cdot \frac{A(t)}{N(t)} - \mu S(t),
\]

hence, one has

\[
\frac{dS(t)}{dt} + S(t) \left(\mu + \frac{\beta A(t)}{N(t)}\right) \geq 0.
\]

Both sides in last inequality are multiplied by \(e^{\int_0^t F(s)ds}\), here \(F(t) = \mu + \frac{\beta A(t)}{N(t)}\). We can obtain \(e^{\int_0^t F(s)ds} \frac{dS(t)}{dt} + e^{\int_0^t F(s)ds} F(t)S(t) \geq 0\), then

\[
\frac{d}{dt} \left[e^{\int_0^t F(s)ds} S(t)\right] \geq 0.
\]

Integrating this inequality from 0 to \(t\) gives

\[
\int_0^t \frac{d}{ds} \left[e^{\int_0^s F(r)dr} S(r)\right] ds \geq 0.
\]

Then,

\[
S(t) \geq S(0) \exp \left(\int_0^t - \left(\mu + \frac{\beta A(r)}{N(t)}\right) dr\right) > 0.
\]
Similarly, we have

\[ A(t) \geq A(0) \exp \left( \int_0^t (\mu + p_1 + p_2) \, ds \right) \geq 0, \]

\[ I(t) \geq I(0) \exp \left( \int_0^t (-\gamma + d + \mu) \, ds \right) \geq 0, \]

\[ R(t) \geq R(0) \exp \left( \int_0^t (-\mu) \, ds \right) \geq 0. \]

Therefore, we prove that the solutions \( S(t), A(t), I(t) \) and \( R(t) \) of the system (1) are positive for all \( t \geq 0 \). This completes the proof. \( \square \)

**Theorem 2.** The set \( \Omega = \left\{ (S, A, I, R) \in \mathbb{R}^4_+ \mid 0 < S + A + I + R \leq \frac{1}{\mu} \right\} \) is a positively invariant set for the system (1) with initial conditions, \( S(0) \geq 0, A(0) \geq 0, I(0) \geq 0 \) and \( R(0) \geq 0 \).

**Proof.** By adding the four equations of the systems (1), we can obtain

\[ \frac{dN(t)}{dt} = \mu N(t) - dI(t) \]

\[ < \lambda - \mu N(t). \]

Then, \( N(t) \leq \frac{d}{\lambda - \mu N(0)} \exp(-\mu t) \). If we take \( t \to \infty \) we have \( 0 \leq N(t) \leq \frac{d}{\lambda - \mu} \). It implies that the region \( \Omega \) is a positive invariant set for the system (1). \( \square \)

In epidemiological models, the basic reproduction number \( R_0 \) is a key epidemiological parameter to determine the nature of a disease. This parameter \( R_0 \) determines whether there will be a risk of an outbreak of disease when an infected person moves among the population. Several techniques have been used to assess the basic reproduction number for an epidemic transmission \([27,28]\). In our present paper, we will use the next-generation matrix approach to calculate the expected outcome of COVID-19 spread. In the absence of confusion, we omit the independent variable \( t \) in the remainder of this article. Following the article Manotosh Mandal \([29]\), we can rewrite the system (1) to

\[ X' = \phi(X) - \psi(X), \]

where \( X = \begin{bmatrix} S \\ A \\ I \\ R \end{bmatrix}, \phi(X) = \begin{bmatrix} 0 \\ \frac{\beta \Lambda S}{N} \\ 0 \\ 0 \end{bmatrix}, \psi(X) = \begin{bmatrix} -\lambda - \frac{\beta \Lambda S}{N} + \mu S \\ \mu A + p_1 A + p_2 A \\ -p_1 A + \mu I + d + \gamma I \\ -p_2 A - \gamma I + \mu R \end{bmatrix} \)

Obviously, the system (1) always exits the disease-free equilibrium \( P_0(\frac{1}{\mu}, 0, 0, 0) \). Then, the Jacobi matrix of \( \phi(X) \) and \( \psi(X) \) at the disease-free equilibrium are respectively given by

\[ J(\phi \mid P_0) = \begin{bmatrix} 0 & \beta & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}, J(\psi \mid P_0) = \begin{bmatrix} \mu & 0 & 0 \\ 0 & Q_1 & 0 \\ 0 & -p_1 & Q_2 \\ 0 & -p_2 & -\gamma & \mu \end{bmatrix}. \]

where \( Q_1 = \mu + p_1 + p_2, Q_2 = \mu + d + \gamma. \)

Then,

\[ \mathcal{L} = \begin{bmatrix} 0 \\ 0 \\ \beta \\ 0 \end{bmatrix}, \mathcal{V} = \begin{bmatrix} \mu \\ 0 \\ \beta \\ Q_1 \end{bmatrix}. \]

The basic reproduction number \( R_0 \) for the model (8) is the spectral radius of the matrix \( \mathcal{FV}^{-1} \). Hence, \( R_0 = \frac{\beta}{\mu} \).

**Existence and uniqueness of equilibrium**

From biological considerations, we will study the system (1) in the set \( \Omega \). In Theorem 2, we have verified that \( \Omega \) is a positive invariant set concerning (1). The following theorem presents the existence and uniqueness of the possible equilibrium.

**Theorem 3.** The system (1) always has a disease-free equilibrium \( P_0 \) which exists for all parameter values. On the other hand, if \( R_0 > 1 \), then the system (1) also admits a unique endemic equilibrium \( P^* (S^*, A^*, I^*, R^*) \).

**Proof.** A steady-state of the model (1) satisfying the following equations,

\[ 0 = \lambda - \frac{\beta \Lambda S}{N} - \mu S, \]

\[ 0 = \mu A - p_1 A - p_2 A. \]

\[ 0 = p_1 A - \mu I - dI - \gamma I, \]

\[ 0 = p_2 A + \gamma I - \mu R. \]

If \( I = 0 \), then \( A = R = 0 \) and \( S = \frac{1}{\mu} \). Therefore, the disease-free equilibrium \( P_0 \) exists for all parameters values. Furthermore, from the above equations, we can obtain \( A = \frac{Q_2}{p_1}, I = \frac{Q_2 \beta S + p_1 Q_2 + p_2 I}{\mu p_1} \) and \( (R_0 - 1) S = \frac{\mu Q_2 + p_2 Q_2 + p_1 I}{\mu p_1} \).

If \( R_0 > 1 \), then \( S = \frac{Q_2 (\mu Q_2 + p_2 Q_2 + p_1 I)}{\mu p_1 (R_0 - 1)} > 0 \). Besides, \( I \) also satisfies the following equation,

\[ \lambda - \mu \left( \frac{\beta \Lambda S}{N} + \mu S \right) - \mu A - p_1 A - p_2 A, \]

\[ 0 = p_1 A - \mu I - dI - \gamma I, \]

\[ 0 = p_2 A + \gamma I - \mu R. \]

when \( R_0 > 1 \), \( I^* = \left( (R_0 - 1) S, \frac{Q_2 (\mu Q_2 + p_2 Q_2 + p_1 I)}{\mu p_1 (R_0 - 1)} \right) \) is the only positive root of the above equation. Further, we can get

\[ A^* = \frac{Q_2}{p_1} I^*, \]

\[ R^* = \frac{Q_2 p_1 + p_2}{p_1} I^*, \]

\[ S^* = \frac{\mu Q_2 + p_2 Q_2 + p_1 I^*}{\mu p_1 (R_0 - 1)} I^*, \]

\[ R_0 = \frac{\beta}{\mu}. \]

Thus, only when \( R_0 > 1 \), the system (1) exists unique endemic equilibrium \( P^* (S^*, A^*, I^*, R^*) \) in the set \( \Omega \). \( \square \)

**On the stability and bifurcation of equilibrium**

In this section, we mainly investigate the stability and bifurcation of equilibrium. Firstly, we study the linear stability of \( P_0(\frac{1}{\mu}, 0, 0, 0) \) by Jacobian matrix

\[ J = \begin{bmatrix} \frac{\beta \Lambda S}{N} - \frac{\beta \Lambda S}{N^2} & -\frac{\beta \Lambda S}{N} + \frac{\beta \Lambda S}{N^2} & \frac{\beta \Lambda S}{N} - \frac{\beta \Lambda S}{N^2} & \frac{\beta \Lambda S}{N} - \frac{\beta \Lambda S}{N^2} \\ \frac{\beta \Lambda S}{N} - \frac{\beta \Lambda S}{N^2} & -\frac{\beta \Lambda S}{N} + \frac{\beta \Lambda S}{N^2} & \frac{\beta \Lambda S}{N} - \frac{\beta \Lambda S}{N^2} & \frac{\beta \Lambda S}{N} - \frac{\beta \Lambda S}{N^2} \\ 0 & p_1 & -Q_2 & 0 \\ 0 & p_2 & \gamma & -\mu \end{bmatrix}. \]

**Theorem 4.** \( P_0(\frac{1}{\mu}, 0, 0, 0) \) is locally unstable whenever \( R_0 > 1 \), and it is locally stable whenever \( R_0 < 1 \).

**Proof.** The Jacobian matrix \( J(P_0) \) of system (1) at the disease free equilibrium point \( P_0 \) is given by

\[ J(P_0) = \begin{bmatrix} -\mu & -\beta & 0 & 0 \\ 0 & -Q_1 & \beta & 0 \\ 0 & -Q_2 & p_1 & 0 \\ 0 & \gamma & p_2 & -\mu \end{bmatrix}. \]

The characteristic equation at the disease-free equilibrium \( P_0 \) is given by

\[ (A + \mu - Q_1 - \beta)(A + Q_2) = 0, \]

where the four eigenvalues of the characteristic equation are \( -\mu - \beta - Q_1 \). Since \( \beta - Q_1 > 0 \), it is easy to show that (13) has a real positive root when \( R_0 > 1 \). Hence, \( P_0 \) is unstable when \( R_0 > 1 \). Conversely, for \( \beta - Q_1 < 0 \), it means that \( R_0 = \frac{\beta}{\mu} < 1 \), and it is easy to show that (13) has four real negative roots. Hence, \( P_0 \) is locally asymptotically stable when \( R_0 < 1 \). \( \square \)
The endemic equilibrium point

Theorem 6. The endemic equilibrium point $P^*$ of system (1) at the endemic equilibrium point $P^*$ is given by

$$ J(P^*) = \begin{bmatrix} -\mu - \left( \frac{\beta S}{N} - \frac{\beta S A}{N^2} \right) & -Q_1 + \frac{\beta S A}{N^2} & \frac{\beta S A}{N^2} & \frac{\beta S A}{N^2} \\ 0 & p_1 & -Q_2 & 0 \\ 0 & p_2 & -Q_2 & 0 \\ \gamma & -\mu & 0 & -\mu \end{bmatrix}, $$

then the characteristic equation at the endemic equilibrium $P^*$ is given by

$$ |\lambda E - J(P^*)| = 0. $$

By further simplification, we can obtain the equivalent equation of the above characteristic equation,

$$ (\lambda + \mu)(\lambda^3 + B_1 \lambda^2 + B_2 \lambda + B_3) = 0, $$

where

$$ B_1 = \mu + Q_2 + A_{11} + (Q_1 - A_{12}), $$
$$ B_2 = A_{11}(Q_1 + Q_2) + (Q_1 - A_{12})Q_2 + \mu(Q_1 + Q_2) + A_{13}p_1 + A_{13}p_2, $$
$$ B_3 = A_{11}Q_1 + \mu(Q_1 - A_{12})Q_2 + A_{13}p_1 + A_{13}p_2 + A_{13}p_2, $$

and

$$ A_{11} = \frac{(\beta - Q_1) A^*}{N^*}, $$
$$ A_{12} = \frac{\beta(\mu Q_3 + \mu p_1 + p_2 Q_2 + p_1 \gamma)}{R_0} \cdot \frac{A^*}{N^*} \cdot \frac{Q_1 A^*}{N^*} \cdot \frac{Q_1 A^*}{N^*} + (Q_1 + Q_2)Q_2, $$
$$ A_{13} = Q_1 A^*. $$

Since $R_0 > 1$, it is easy to show that $B_2 > 0$ and $B_3 > 0$. Further, we can easily obtain that

$$ B_1 B_2 - B_3 = (\mu + Q_2 + A_{11} + (Q_1 - A_{12})) \cdot \mu^2(Q_1 + Q_2) + \mu(Q_1 + Q_2)^2 + A_{11}^2 Q_1 p_1 (Q_1 + Q_2 - \gamma) + (\mu + Q_2) A_{11}^2 Q_1 p_2 + (Q_1 + Q_2)Q_2 > 0. $$

In conclusion, when $R_0 > 1$, we have the local stability of $P^*$ by the Hurwitz's criterion. □

Now we study the global asymptotic stability of the two equilibria.

Theorem 7. If $R_0 \leq 1$, then the disease free equilibrium $P_0$ is globally asymptotically stable.

Proof. Define a Lyapunov functional $V = \frac{A^2}{N^*}$. We have

$$ \frac{dV(t)}{dt} = A^* \frac{\beta S A}{N} - Q_1 A \leq A^* \frac{\beta S}{N} \left( S - Q_1 A - Q_1 I - Q_1 R \right) $$
$$ = \frac{A^2}{N} \left( \frac{\beta S}{N} - Q_1 A - Q_1 I - Q_1 R \right) $$
$$ = \frac{A^2}{N} \left( (R_0 - 1)S - A - I - R \right) $$
$$ \leq 0. $$

Therefore, $R_0 \leq 1$ ensures that $\frac{dV(t)}{dt} \leq 0$ for all $t \geq 0$, which holds if $(S, A, I, R) = \left( \frac{A^2}{N}, 0, 0, 0 \right)$. By LaSalle's invariant principle [32], we obtain that $P_0$ is globally asymptotically stable. □

We have the following theorem on the global asymptotic stability of the endemic equilibrium $P^*$.

Theorem 8. If $\mu > d + \gamma \frac{2d + p_1 d}{p_2} \mu > \frac{\mu}{p_2}$ and $R_0 > 1$, then the equilibrium $P^*$ is globally asymptotically stable.
Proof. Define a Lyapunov functional $V = k_1 V_1 + k_2 V_2 + k_3 V_3 + k_4 V_4$, where

\[
V_1 = \frac{(S - S^* + A - A^*)^2}{N}, \quad k_1 = 1,
\]
\[
V_2 = A - A^* - A^* \ln \frac{A}{A^*}, \quad k_2 = \frac{2N^*(Q_1 - d)(A - A^*)^2}{p_1(A^* + I^* + R^*)},
\]
\[
V_3 = \frac{(I - I^*)^2}{N}, \quad k_3 = \frac{S^*(Q_1 - d)}{p_1(A^* + I^* + R^*)},
\]
\[
V_4 = \frac{(R - R^*)^2}{N}, \quad k_4 = \frac{S^*(Q_1 - d)}{p_2(A^* + I^* + R^*)}.
\]

One has

\[
\frac{dV(t)}{dt} = \frac{(d - \mu)(S - S^*)^2 + (d + \mu - 2Q_1)(A - A^*)^2}{N} - \frac{(S - S^* + A - A^*)^2}{N^2}(\lambda + dS + dA + dR)
\]
\[
- \frac{2(Q_1 - d)S^*(A - A^*)^2}{N} - \frac{S^*(Q_1 - d)}{p_1(A^* + I^* + R^*)}\left\{ - \frac{(2Q_2 - \mu - d)(I - I^*)^2}{N} - \frac{(I - I^*)^2}{N^2}(\lambda + dS + dA + dR) \right\}
\]
\[
+ \frac{S^*(Q_1 - d)}{p_2(A^* + I^* + R^*)}\left\{ - \frac{(R - R^*)^2}{N^2}(\lambda + dS + dA + dR) + \frac{\gamma((I - I^*) - (R - R^*)^2)}{N} \right\},
\]
\[
+ \frac{(d - \mu)(S - S^*)^2}{N} - \frac{(S - S^* + A - A^*)^2}{N^2}(\lambda + dS + dA + dR)
\]
\[
- \frac{2(Q_1 - d)(A - A^*)^2}{N} - \frac{S^*(Q_1 - d)}{p_1(A^* + I^* + R^*)}\left\{ - \frac{(2Q_2 - d - \mu)S^*(I - I^*)^2}{N} - \frac{(I - I^*)^2}{N^2}(\lambda + dS + dA + dR) \right\}
\]
\[
+ \frac{S^*(Q_1 - d)}{p_2(A^* + I^* + R^*)}\left\{ - \frac{(R - R^*)^2}{N^2}(\lambda + dS + dA + dR) + \frac{\gamma((I - I^*) - (R - R^*)^2)}{N} \right\}.
\]

Because all parameters satisfy $\mu > d + \gamma, \frac{2Q_2 + \mu + d}{p_1} > \frac{1}{p_2}$ and $R_0 > 1$, we can obtain $\mu - \gamma - d > 0$.

\[
(2Q_1 - \mu - d) + 2S^*(Q_1 - d) > 0,
\]
\[
2Q_2 - \mu - d > \frac{\gamma}{p_1} > 0.
\]

Obviously, $\frac{dV(t)}{dt} \leq 0$. From the Lyapunov–LaSalle asymptotic stability [32,33], we obtain that the system (1) is globally stable around the endemic equilibrium $P^*$. □

Numerical simulations

In this section, we give some numerical simulations to support the previous theoretical analysis.

Firstly, we considered the parameter set $\Omega_1 = \{\lambda, \beta, \mu, p_1, p_2, \gamma, d\}$, and these parameters are assumed with feasible value. The elements of $\Omega_1$ are chosen as follows,

\[
\lambda = 2600, \quad \beta = 0.3, \quad \mu = 0.065, \quad p_1 = 0.3, \quad p_2 = 0.1, \quad \gamma = 0.3, \quad d = 0.1,
\]

and some different initial values for each variable of state, we obtain the disease-free equilibrium $P_0(40000, 0, 0, 0)$ and $R_0 = 0.645 < 1$. In this case, the system (1) has only the disease-free equilibrium $P_0$ and it is globally asymptotically stable on the set $\Omega$ (See Fig. 3).
In this case, the system (1) has two different equilibria: one is the disease-free equilibrium $\mathcal{P}_d$ and the other is the endemic equilibrium $\mathcal{P}^*$. The elements of $\mathcal{U}$ are as follows,

$$\lambda = 2600, \beta = 0.6, \mu = 0.065, p_1 = 0.3, p_2 = 0.1, \gamma = 0.3, d = 0.1$$

and some different initial values for each variable of state, we obtain the endemic equilibrium $P^*(29915.7, 1409.6, 909.4, 6366.1)$ and $R_0 = 1.2903 > 1$ (but $\mu < \gamma + d$, i.e. $0.065 < 0.3 + 0.1$), $\frac{\mu d}{\gamma} < \frac{\gamma}{\mu}$, i.e. $2.55 < 3$.

In this case, the system (1) has two different equilibria: one is the disease-free equilibrium $P_d$ and the other is the endemic equilibrium $P^*$, and the endemic equilibrium is globally asymptotically stable (See Fig. 4).

In Fig. 4, we also use the same parameters and different initial values. When the initial value of susceptible individuals is lower than $S^* = 29915.7$, the number of susceptible individuals increases. When the initial value of susceptible individuals exceeds $\frac{1}{\mu} = 40000$, the number of susceptible individuals decreases. In the end, they all converge to $\mathcal{P}_d = 40000$ (Fig. 3A). Also, we can obvious that the number of asymptomatic individuals (infected individuals with symptomatic) decreases and get closer to zero in Fig. 3B and Fig. 3C. In Fig. 3D, the number of recovered individuals increases quickly at first, after that it decreases and approaches zero. In short, these curves are the solution curves of equilibrium point $P_d(40000, 0, 0, 0)$ corresponding to different initial values when the basic reproduction number is lower than 1. Therefore, when $R_0$ is not exceed unit 1, the trajectories starting from different initial values will eventually converge to the disease-free equilibrium point $P_d$. In fact, this has happened as the contact rate between susceptible and asymptomatic individuals (i.e. $\beta$) is relatively small.

Again for the different parameter conditions, we can prove that the endemic equilibrium $P^*$ is globally asymptotically stable in Theorem 8. As a matter of fact, we find that the endemic equilibrium is globally asymptotically stable as long as the basic regeneration number is greater than 1 (See Fig. 4). For the set of parameters $\Omega_1$, all of whose values are the same as (16) except for $\beta$. The elements of $\Omega_1$ are as follows,

$$\lambda = 2600, \beta = 0.6, \mu = 0.065, p_1 = 0.3, p_2 = 0.1, \gamma = 0.3, d = 0.1, \frac{\mu d}{\gamma} < \frac{\gamma}{\mu}$$

and some different initial values for each variable of state, we obtain the endemic equilibrium $P^*(29915.7, 1409.6, 909.4, 6366.1)$ and $R_0 = 1.2903 > 1$ (but $\mu < \gamma + d$, i.e. $0.065 < 0.3 + 0.1$), $\frac{\mu d}{\gamma} < \frac{\gamma}{\mu}$, i.e. $2.55 < 3$.

In this case, the system (1) has two different equilibria: one is the disease-free equilibrium $P_d$ and the other is the endemic equilibrium $P^*$, and the number of symptomatic individuals decreases. In the end, they all converge to $S^* = 29915.7$ (see Fig. 4 A). Also, we can obvious that the change of the number of asymptomatic individuals occurs two oscillations of different degrees, but these solution curves get closer to $\mathcal{P}_d = 40000$ (see Fig. 4B). In Fig. 4B, the number of infected individuals with symptomatic decreases at first, after that it has two small rebounds. But these solution curves stabilizes towards $I^* = 909.4$. The changing trend of the solution curve of infected persons is also reasonable, which is related to the diagnosis process of new coronavirus infection. This is because asymptomatic individuals are diagnosed as symptomatic individuals only if they show certain clinical symptoms during the 14 day isolation period. During this period, some of the diagnosed patients may be converted to recovered individuals because of timely treatment. In Fig. 4D, it is obvious that the change of the number of recovered individuals rapidly increases at first, after that it decreases...
The restriction determines the local dynamics of the disease-free equilibrium, \( P^0 \), the basic reproduction number of the system (1). We used the stability analysis theory for nonlinear systems to study both the local and global behavior of this multidimensional model can be determined by the basic reproduction number as excepted.

Furthermore, we describe the impact of constant input rate \( \lambda \) on the final scale of an outbreak when the set \( \Omega \), all of whose values remain unchanged except \( \lambda \) and \( \beta \) (see Fig. 5). Let \( \beta = 0.6 \), we guarantee that the basic regeneration number is greater than 1, that is, the endemic disease will continue to exist without effective control. It is observed that the number of asymptomatic individuals (\( A^* \)) and symptomatic individuals (\( I^* \)) in the endemic equilibrium increases with the increase of input rate as excepted.

In addition, we display the sensitivity analysis of the system (1) with respect to the parameters \( \lambda \), and \( \beta \).

From Fig. 6, we can observe that the constant input rate \( \lambda \) is directly proportional to all variables of the system (1).

In Fig. 7, it can be stated that the number of asymptomatic, symptomatic, and recovery individuals are directly proportional to the maximum transmission rate \( \beta \), but the number of susceptible individuals is inversely proportional to \( \beta \).

It is remarkable that these simulations presented in this article should be considered from a qualitative of a view.

Conclusion

This paper focuses on the impact of large-scale population migration and the gradual increase of asymptomatic infections on the late evolution of the epidemic. Therefore, we considered a SAIR model that incorporates population mobility and natural death, as well as disease-caused death, so that the total number of population may vary in time. The incidence rate is of the nonlinear infection rate. The asymptotic behavior of this multidimensional model can be determined by the basic reproduction number of the system (1). We used the stability analysis theory for nonlinear systems to study both the local and global stability of SAIR model. The threshold parameter \( R_0 \), which completely determines the local dynamics of the disease-free equilibrium \( P_0 \) under the restriction \( R_0 \leq 1 \). On the other hand, when \( R_0 > 1 \), then the endemic equilibrium point \( P^* \) is locally asymptotically stable. If \( R_0 \leq 1 \), the global stability of the disease-free equilibrium \( P_0 \) is proved by constructing Lyapunov functions. Also, the method of constructing Lyapunov function was used to show that \( P^* \) is globally asymptotically stable under the condition of certain parameters. In fact, through numerical simulation, we found that the endemic equilibrium must be globally asymptotically stable as long as the basic reproduction number \( R_0 \) exceeds unit 1. The model is a preliminary prediction of the late development of the new coronavirus epidemic in China. The results of theoretical analysis once again alert us that the risk of re-outbreak of the COVID-19 epidemic will still exist due to the population mobility, especially the contact between susceptible and asymptomatic infected individuals. However, our model is based on certain assumptions, which will deviate from reality to a certain extent. Therefore, there may be some deviation between the preliminary results and reality. In our future work, we need to improve our model by incorporating some control strategies, for example, vaccination or isolation of asymptomatic infected individuals and so on.

CRediT authorship contribution statement

Xiaxia Kang: Conceptualization, Methodology, Software, Investigation, Writing – original draft. Ye Hu: Visualization, Methodology, Software, Writing – review & editing. Zeyu Liu: Visualization, Writing – review & editing. Shahzad Sarwar: Visualization, Writing – review & editing.

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.

Acknowledgments

The authors wish to thank the anonymous reviewers for their careful reading and providing invaluable suggestions. This work was partially supported by the Scientific and Technological Innovation Programs of Higher Education Institutions in Shanxi under grant no. 2021L563.
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