DYNAMICAL ANALYSIS OF COVID-19 EPIDEMIC MODEL WITH INDIVIDUAL MOBILITY

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Abstract. COVID-19 (Corona Virus Disease 2019) differs from previous epidemics in two ways: one is that the exposed do not show symptoms but are still capable of infecting others, and the other is that SARS-CoV can be indirectly transmitted through environment. The truth indicated that the infected and the exposed have the ability to spread the disease farther afield, gives rise to a reasonable research of the impact of individual mobility. In this paper, based on uncompleted enclosure and isolation lead to infected individual and virus would spread different regions, we establish an SEIVR (Susceptible-Exposed-Infected-Environment-Remove) epidemic model with individual mobility and analysis the dynamical properties of model. We show that the unique endemic equilibrium is always existent and globally asymptotically stable. Numerical simulations of the impact of infected individual mobility suggests that customs inspection is very effective in preventing high-risk group move.

Keywords: COVID-19; epidemic model; individual mobility; global stability.

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1. INTRODUCTION

Epidemic diseases are caused by the invasion of various pathogens (microorganisms, parasites, etc.) and it can spread from humans-to-human, animals-to-animals or humans-to-animals.
For instance, tuberculosis, Ebola virus, Zika virus, influenza and etc. may bring many disasters to public security and individual health.

The occurrence of infection usually have three parts, the source of infection, route of transmission, susceptible population respectively. One of the parts breaks, we can treat it or prevent the occurrence or development of the diseases. Therefore, the research of the pathogenesis, transmission mechanism, development trend and control strategy is realistic. It is a great practical significance to analyze the spread of diseases and propose appropriate control strategies by using mathematical models and dynamic properties.

The existence of the incubation period and latent period are important characteristics in the epidemiology of infectious diseases. The incubation period refers to the period between the moment of being infected and the moment of becoming infectious and the latent period between the moment of being infected and the moment of becoming infectious. By definition, the incubation period is longer than the latent period.

In late November or early December 2019, several hospitals in Wuhan reported clusters of severe pneumonia of unknown cause, and the Chinese government notified the World Health Organization (WHO) after verification. On January 31, 2020, WHO declares the Novel coronavirus outbreak a Public Health Emergency of international Concern (PHEIC) [2]. Then came the March 11, 2020, the WHO have therefore made the assessment that COVID-19 can be characterized as a pandemic[3]. It is the first time that a pandemic sparked by a coronavirus. The severe acute respiratory syndrome coronavirus (SARS-CoV) in 2002 and the Middle East respiratory syndrome coronavirus (MERS-CoV) in 2012 were far less serious than it is now.

Meanwhile, recent comparisons of the genetic sequences of this virus and bat coronaviruses show a 96 similarity. In a world, SARS-CoV-2 virus as a novel coronavirus also is a classic respiratory syndrome coronavirus, and its pathological process and immune response also follow the typical respiratory infection process. SARS-CoV-2 is a completely new virus which may have a number of unclear peculiarities. For example, SARS-CoV-2 has been found as the most complicated point of coronavirus, that is, it can be asymptomatic transmission, and patients infected through asymptomatic infected or symptomatic infected individuals [5, 6]. Asymptomatic transmission is thought to be the main ingredient why the virus was able to cause such
a pandemic in such a short period of time. Clinical evidence shows that the incubation period of this disease ranges from 2 to 14 days. During this period of time, infected individuals may not develop any symptoms and may not be aware of their infection, yet they are capable of transmitting the disease to other people[7]. Meanwhile, COVID-19 outbreak coincided with college students on their winter vacation and migrant workers returning home for Spring Festival. The seriousness of the disease was not perceived until New Year’s Day. Large population movements increase the transmission rates and contribute to the progression of disease outbreaks.

Moreover, there is preliminary evidence being detected in ocular secretions from some patients [11, 12]. Maybe the survival of SARS-CoV-2 would result very low with temperatures higher than 20°C, and the inactivation rate of cronavirus is usually higher than other examined viruses. These results lead to an augmented transmission risk of COVID-19.

In addition, unprecedented research and attention has been devoted to this disease, which will bring about the constant discovery and reporting of many rare infections and special cases, changing people’s understanding of viruses, infectious diseases, immunology and epidemiology and public health. Hence, it has become an urgent and realistic problem to study the pathogenesis, transmission mechanism, development trend and control strategy of infectious diseases.

The rest of this paper is organized as follows. In Section 2, we formulate a COVID-19 epidemic model with individual mobility and show that the existence of the unique endemic equilibrium. In Section 3, global stability of the positive equilibrium is also established. In Section 4, Some numerical simulations are given to verify the obtained results. Finally, we present conclusions and discussions in Section 5.

2. Statement of the Problem

In this section, ground on the typical SEIR infectious disease model, we further consider the amount of the coronavirus in the environment(V) and obtain the following mathematical compartment diagram.

Here we consider that asymptomatic infected of COVID-19 can also infect susceptible. The population is divided into four classes: the susceptible (S), the exposed (E), the infected (I), and the recovered (R). SARS-CoV-2 can be spread not only by person-to-person contact, but also by item-to-person contact. The infected are the individuals have the disease and be able
to transmit it. The exposed are the individuals in the incubation period who are infected with disease but not yet infectious, they do not show symptoms but are still capable of infecting others. Thus, another interpretation of the E and I compartments in our model is that they contain asymptomatic infected and symptomatic infected individuals separately. The parameters in the equations are explained as below. \( \Lambda \) represents the population influx. \( W_E, W_I \) respectively mean the number of exposed individual mobility and infected individual mobility, \( W_V \) is represent the number of SARS-CoV-2 from anywhere else. The parameter \( d_i \) is the death rate of the susceptible, the exposed, the infected, the recovered and the amount of the coronavirus in the environment respectively. The parameter \( \beta_1, \beta_2 \) and \( \beta_3 \) represent the transmission rates between susceptible individuals and the exposed, the infected and the the amount of the coronavirus in the environment, separately. \( p^{-1} \) is the incubation period between the infection and the onset of symptoms. The recover rate from infection is \( \alpha \). Of course, all the parameters are positive.
Based on the above compartment diagram, we can construct the following epidemic model to describe the transmission mechanism of COVID-19,

\[
\begin{align*}
\frac{dS}{dt} &= \Lambda - \beta_1 SE - \beta_2 SI - \beta_3 SV - d_1 S, \\
\frac{dE}{dt} &= WE + \beta_1 SE + \beta_2 SI + \beta_3 SV - d_2 E - pE, \\
\frac{dI}{dt} &= W_I + pE - d_3 I - \alpha I, \\
\frac{dV}{dt} &= W_V + \gamma_1 E + \gamma_2 I - d_4 V, \\
\frac{dR}{dt} &= W_R + \alpha I - d_5 R.
\end{align*}
\]

(1)

The initial conditions of system (1) are given as

\[
S(t) = S_0 > 0, E(t) = E_0 \geq 0, I(t) = I_0 \geq 0, V(t) = V_0 \geq 0, R(t) = R_0 \geq 0,
\]

After of these specifications about model (1), we focus on dynamical analysis and stability analysis of system (1) in below subtitles. First of all, look that the variable \(R(t)\) does not appear in other remainder equations, we can only analyze the following subsystem,

\[
\begin{align*}
\frac{dS}{dt} &= \Lambda - \beta_1 SE - \beta_2 SI - \beta_3 SV - d_1 S, \\
\frac{dE}{dt} &= WE + \beta_1 SE + \beta_2 SI + \beta_3 SV - d_2 E - pE, \\
\frac{dI}{dt} &= W_I + pE - d_3 I - \alpha I, \\
\frac{dV}{dt} &= W_V + \gamma_1 E + \gamma_2 I - d_4 V.
\end{align*}
\]

(2)

We now establish that all solutions of (2) with initial conditions are positive. Obviously, \(\frac{dS}{dt} = \Lambda > 0\) when \(S(t) = 0\), that means \(S(t)\) will increase to be larger then 0. And if \(I(t)\) equals zero, then \(\frac{dI}{dt} = W_I + pE > 0\). Similarly, \(\frac{dE}{dt} = WE + \beta_2 SI > 0\), and \(\frac{dV}{dt} = W_V + \gamma_1 E + \gamma_2 I > 0\). Among \(E(t), I(t), V(t)\) will increase to be larger than 0. Then \(S(t), E(t), I(t), V(t) > 0\) become apparent.

Let \(d = \min\{d_1, d_2 + p, d_3 + \alpha\}\), \(W = \Lambda + W_E + W_I\), since

\[
\frac{d(S + E + I)}{dt} = W - d_1 S - (d_2 + p)E - (d_3 + \alpha)I \\
\leq W - d(S + E + I),
\]

Therefore, the subsystem (2) is uniformly persistent, and has the unique globally asymptotically stable positive equilibrium \((S^*, E^*, I^*, V^*, R^*)\). After this, we can analyze the stability of the equilibrium. If \(R_0 < 1\), then the disease-free equilibrium \((S^*, E^*, I^*, V^*, R^*)\) is globally asymptotically stable; if \(R_0 > 1\), then the disease-free equilibrium \((S^*, E^*, I^*, V^*, R^*)\) is unstable; if \(R_0 = 1\), then the disease-free equilibrium \((S^*, E^*, I^*, V^*, R^*)\) is locally asymptotically stable and may be unstable.
we have that \( \limsup (S + E + I) \leq \frac{W}{d} \) and \( 0 \leq V \leq \frac{W + W(\gamma_1 + \gamma_2)}{d_4 d} \).

Thus, it leads to a biologically feasible domain

\[
\Omega = \left\{ (S, E, I, V) \in \mathbb{R}_+^4 \mid 0 < S + E + I \leq \frac{W}{d}, 0 \leq V \leq \frac{W + W(\gamma_1 + \gamma_2)}{d_4 d} \right\}.
\]

It can be seen from the fact that \( \frac{dS}{dt} > 0, \frac{dE}{dt} > 0, \frac{dI}{dt} > 0 \) and \( \frac{dV}{dt} > 0 \). System (2) has not a disease free equilibrium and hence there is no the basic reproduction number. Next we show that system (2) has a unique endemic equilibrium.

**Theorem 1.** System (2) has a unique endemic equilibrium \( X^*(S^*, E^*, I^*, V^*) \).

**Proof.** The equilibria of (2) satisfy the following system of algebraic equations,

\[
\begin{align*}
\Lambda - \beta_1 SE - \beta_2 SI - \beta_3 SV - d_1 S &= 0, \\
W E + \beta_1 SE + \beta_2 SI + \beta_3 SV - d_2 E - pE &= 0, \\
W I + pE - d_3 I - \alpha I &= 0, \\
W V + \gamma_1 E + \gamma_2 I - d_4 V &= 0.
\end{align*}
\]

Solving it gives

\[
\begin{align*}
S^* &= \frac{1}{d_1} (\Lambda + W E - (d_2 + p)E^*) = \frac{\Lambda}{\beta_1 E^* + \beta_2 I^* + \beta_3 V^* + d_1}, \\
I^* &= \frac{W I + pE^*}{d_3 + \alpha}, \\
V^* &= \frac{1}{d_4} (W V + \frac{\gamma_2 W I}{d_3 + \alpha} + \gamma_1 E^* + \frac{\gamma_2 pE^*}{d_3 + \alpha}),
\end{align*}
\]

and \( E^* \) is the solution to the univariate quadratic equation

\[
aE^2 + bE + c = 0,
\]
where

\[
a = (d_2 + p) \left( \beta_1 + \frac{\beta_2 p}{d_3 + \alpha} + \frac{\beta_3}{d_4} \left( \gamma_1 + \frac{\gamma_2 p}{d_3 + \alpha} \right) \right),
\]

\[
b = (d_2 + p) \left( d_1 + \frac{\beta_2 W_I}{d_3 + \alpha} + \frac{\beta_3}{d_4} \left( W_V + \frac{\gamma_2 W_I}{d_3 + \alpha} \right) \right)
- (\Lambda + W_E) \left( \beta_1 + \frac{\beta_2 p}{d_3 + \alpha} + \frac{\beta_3}{d_4} \left( \gamma_1 + \frac{\gamma_2 p}{d_3 + \alpha} \right) \right),
\]

\[
c = - \left( \frac{\beta_2 W_I}{d_3 + \alpha} + \frac{\beta_3}{d_4} \left( W_V + \frac{\gamma_2 W_I}{d_3 + \alpha} \right) \right) (\Lambda + W_E) - d_1 W_E.
\]

According to Hurwitz criterion, \( aE^2 + bE + c = 0 \) exists only one positive root. Formula of quadratic root of one variable imply that

\[
E^* = \frac{-b + \sqrt{b^2 - 4ac}}{2a}.
\]

Then we know \( S^* > 0, E^* > 0, I^* > 0, V^* > 0 \). System (2) has a unique endemic equilibrium \( X^*(S^*, E^*, I^*, V^*) \).

The proof is completed. \( \square \)

**Remark 1:** Meanwhile, we find that if \( c = 0 \) in Equ.(5), there exist two equilibria which the other is trivial solution. The condition \( c = 0 \) is equivalent of \( W_E = W_I = W_V = 0 \). In this case, it means that the regions take policy of completed enclosure and isolation and there is not exist individual mobility, the related researches could be found in [5]. In this paper, we focus on individual mobility and discuss the impact of uncompleted enclosure and isolation. As long as there is the phenomenon of individual mobility, the pandemic will always persistent.

### 3. Dynamical Properties of System (2)

Actually, when one or all of \( W_E, W_I, W_V \) are positive, system (2) only has an endemic equilibrium \( X^*(S^*, E^*, I^*, V^*) \). Next, we establish global stability of the endemic equilibrium \( X^*(S^*, E^*, I^*, V^*) \) of (2) and study the impact of the individual mobility.

**Theorem 2.** The endemic equilibrium \( X^*(S^*, E^*, I^*, V^*) \) is globally asymptotically stable.
Proof. Define

$$L_S = S^* \left( \frac{S}{S^*} - 1 - \ln \frac{S}{S^*} \right),$$

$$L_E = E^* \left( \frac{E}{E^*} - 1 - \ln \frac{E}{E^*} \right),$$

noting that

$$\frac{dL_S}{dt} = \left(1 - \frac{S^*}{S} \right) \frac{dS}{dt} = \left(1 - \frac{S^*}{S} \right) \left( \beta_1 S^* E^* + \beta_2 S^* I^* + \beta_3 S^* V^* + d_1 S^* - \beta_1 S^* E - \beta_2 S^* I - \beta_3 S^* V - d_1 S \right)$$

$$= d_1 S^* \left( 2 - \frac{S}{S^*} - \frac{S^*}{S} \right) + \beta_1 S^* E^* \left( 1 - \frac{S^*}{S} + \frac{E}{E^*} - \frac{ES}{E^* S^*} \right)$$

$$+ \beta_2 S^* I^* \left( 1 - \frac{S^*}{S} + \frac{I}{I^*} - \frac{IS}{I^* S^*} \right) + \beta_3 S^* V^* \left( 1 - \frac{S^*}{S} + \frac{V}{V^*} - \frac{VS}{V^* S^*} \right),$$

and

$$\frac{dL_E}{dt} = \left(1 - \frac{E^*}{E} \right) \frac{dE}{dt} = \left(1 - \frac{E^*}{E} \right) \left( W_E + \beta_1 S^* E + \beta_2 S^* I + \beta_3 S^* V - (d_2 + p) E^* \frac{E}{E^*} \right)$$

$$= W_E \left( 2 - \frac{E}{E^*} - \frac{E^*}{E} \right) + \beta_1 S^* E^* \left( 1 - \frac{S}{S^*} - \frac{E}{E^*} + \frac{ES}{E^* S^*} \right) + \beta_2 S^* I^* \left( 1 - \frac{E}{E^*} + \frac{SI}{S^* I^*} - \frac{ISE^*}{I^* S^* E} \right)$$

$$+ \beta_3 S^* V^* \left( 1 - \frac{E}{E^*} + \frac{SV}{S^* V^*} - \frac{VSE^*}{V^* S^* E} \right).$$

Similarly, one can verify that

$$L_I = E^* \left( \frac{I}{I^*} - 1 - \ln \frac{I}{I^*} \right).$$

we have

$$\frac{dL_I}{dt} = \left(1 - \frac{I^*}{I} \right) \frac{dI}{dt} = \left(1 - \frac{I^*}{I} \right) \left( W_I + p E - (d_3 + \alpha) I^* \frac{I}{I^*} \right)$$

$$= W_I \left( 2 - \frac{I}{I^*} - \frac{I^*}{I} \right) + p E^* \left( 1 + \frac{E}{E^*} - \frac{I}{I^*} - \frac{EI^*}{E^* I} \right).$$

Further, if defining

$$L_V = V^* \left( \frac{V}{V^*} - 1 - \ln \frac{V}{V^*} \right),$$
we get

\[
\frac{dL}{dt} = (1 - \frac{V^*}{V}) \frac{dV}{dt} = (1 - \frac{V^*}{V})(W_V + \eta E + \gamma I - d_V \frac{V^*}{V^*})
\]

\[
=W_V(2 - \frac{V^*}{V} - \frac{V}{V^*}) + \eta E^*(1 + \frac{E}{E^*} - \frac{V}{V^*} - EV^*) + \gamma I^*(1 + \frac{I}{I^*} - \frac{V}{V^*} - IV^*).
\]

Then we consider the following Lyapunov function

\[
L(t) = S^*(\frac{S}{S^*} - 1 - \ln \frac{S}{S^*}) + E^*(\frac{E}{E^*} - 1 - \ln \frac{E}{E^*})
\]

\[
+c_1 I^*(\frac{I}{I^*} - 1 - \ln \frac{I}{I^*}) + c_2 V^*(\frac{V}{V^*} - 1 - \ln \frac{V}{V^*}),
\]

(6)

where

\[
c_1 = \frac{\beta_2 S^* I^*}{p E^*} + \frac{\beta_3 S^* V^* \gamma_2 I^*}{(\eta E^* + \gamma_2 I^*)p E^*}, \quad c_2 = \frac{\beta_3 S^* V^*}{\eta E^* + \gamma_2 I^*}.
\]

Since the function \(h(x) = x - 1 - \ln x\) and the constant \(c_1, c_2\) are always positive, it’s obvious that \(L(t)\) is nonnegative at any time \(t\). Then the derivative of \(L(t)\) along the solution of (2) is given by

\[
\frac{dL}{dt} = (1 - \frac{S^*}{S}) \frac{dS}{dt} + (1 - \frac{E^*}{E}) \frac{dE}{dt} + c_1(1 - \frac{I^*}{I}) \frac{dI}{dt} + c_2(1 - \frac{V^*}{V}) \frac{dV}{dt}
\]

\[
=(d_1 S^* + W_E + \beta_1 S^* E^*) \frac{2 - E}{E^*} + c_1 W_I (2 - \frac{I^*}{I} - \frac{I}{I^*}) + c_2 W_V (2 - \frac{V^*}{V} - \frac{V}{V^*})
\]

\[
+ \beta_2 S^* I^* \frac{2 - E}{E^*} + \frac{I}{I^*} - \frac{1}{\gamma_2}, \quad \beta_3 S^* V^* \frac{2 - E}{E^*} + \frac{V}{V^*} - \frac{S}{S^*} - \frac{VE}{V^*} + \frac{VE}{V^*} + \frac{VE}{V^*}
\]

\[
+c_1 (p E^* (1 + \frac{E}{E^*} - \frac{I^*}{I^*} - \frac{E}{E^*} - \frac{I}{I^*})) + c_2 (\eta E^* (1 + \frac{E}{E^*} - \frac{V}{V^*} - \frac{VE}{V^*} - \frac{VE}{V^*})
\]

\[
+ \gamma_2 I^* (1 + \frac{I}{I^*} - \frac{V}{V^*} - \frac{IV}{I^* V^*}),
\]
furthermore
\[
\frac{dL(t)}{dt} = (d_1S^* + W_E + \beta_1S^*E^*)(2 - \frac{E}{E^*} - \frac{E^*}{E}) + c_1W_I(2 - \frac{I}{I^*} - \frac{I^*}{I}) + c_2W_V(2 - \frac{V}{V^*} - \frac{V^*}{V}) \\
+ (\beta_2S^*I^* + \beta_3S^*V^*)\left(1 - \frac{S}{S^*} + \frac{S^*}{S}\right) + \beta_2S^*I^*\left(1 - \frac{ISE^*}{I^*S^*E^*} + \ln \frac{ISE^*}{I^*S^*E^*}\right) \\
+ \beta_3S^*V^*\left(1 - \frac{VSE^*}{V^*S^*E^*} + \ln \frac{VSE^*}{V^*S^*E^*}\right) + c_1pE^*(1 - \frac{E}{E^*} + \ln \frac{E}{E^*}) \\
+ c_2\gamma_1E^*(1 - \frac{EV^*}{E^*V} + \ln \frac{EV^*}{E^*V}) + c_2\gamma_2I^*(1 - \frac{IV^*}{I^*V} + \ln \frac{IV^*}{I^*V}) \\
+ \left(\ln \frac{E}{E^*} - \frac{E}{E^*}\right)(\beta_2S^*I^* + \beta_3S^*V^* - c_1pE^* - c_2\gamma_1) \\
+ \left(\ln \frac{I}{I^*} - \frac{I}{I^*}\right)(-\beta_3S^*V^* + c_1pE^* - c_2\gamma_2I^*) \\
+ \left(\ln \frac{V}{V^*} - \frac{V}{V^*}\right)(-\beta_3S^*V^* + c_2\gamma_1E^* + c_2\gamma_2I^*),
\]

according to (4), we have
\[
\frac{dL(t)}{dt} = (d_1S^* + W_E + \beta_1S^*E^*)(2 - \frac{E}{E^*} - \frac{E^*}{E}) + c_1W_I(2 - \frac{I}{I^*} - \frac{I^*}{I}) + c_2W_V(2 - \frac{V}{V^*} - \frac{V^*}{V}) \\
+ (\beta_2S^*I^* + \beta_3S^*V^*)\left(1 - \frac{S}{S^*} + \frac{S^*}{S}\right) + \beta_2S^*I^*\left(1 - \frac{ISE^*}{I^*S^*E^*} + \ln \frac{ISE^*}{I^*S^*E^*}\right) \\
+ \beta_3S^*V^*\left(1 - \frac{VSE^*}{V^*S^*E^*} + \ln \frac{VSE^*}{V^*S^*E^*}\right) + c_1pE^*(1 - \frac{E}{E^*} + \ln \frac{E}{E^*}) \\
+ c_2\gamma_1E^*(1 - \frac{EV^*}{E^*V} + \ln \frac{EV^*}{E^*V}) + c_2\gamma_2I^*(1 - \frac{IV^*}{I^*V} + \ln \frac{IV^*}{I^*V}).
\]

Therefore \(\frac{dL(t)}{dt} \leq 0\). It follows that the positive-define function \(L(t)\) has non-positive derivative \(\frac{dL(t)}{dt}\). Let \(M\) be the largest invariant set of \(\{(S(t), E(t), I(t), V(t))|\frac{dL(t)}{dt} = 0\}\). Obviously, \(\frac{dL(t)}{dt} = 0\) if and only if \(S(t) = S^*, E(t) = E^*, I(t) = I^*, V(t) = V^*\), Hence, \(M = \{X^*\}\). By the LaSalle Invariance Principle, every solution of (2) tends to the largest invariant set \(M\), which implies that \(X^*\) is globally asymptotically stable.

The proof is completed. \(\square\)

4. Influence of Individual Mobility

In this section, we give some numerical simulations of (2) to study the input parameters of the proposed model in details. Infected case imported from the rest of the region in (2) is represented as the parameters \(W_E, W_I\) and \(W_V\), where \(W_V\) is the virus input in the environment. We discuss the impact of individual mobility on the result of transmission.
4.1. Impact of completed enclosure and isolation. In the event of the total cancellation of inbound and outbound flights, there is no movement of individuals and goods, this case implies that enclosure and isolation are completed, which means \( W_E = W_I = W_V = 0 \). Model (2) reduces to the following system,

\[
\begin{align*}
\frac{dS(t)}{dt} &= \Lambda - \beta_1 SE - \beta_2 SI - \beta_3 SV - d_1 S, \\
\frac{dE(t)}{dt} &= \beta_1 SE + \beta_2 SI + \beta_3 SV - d_2 E - pE, \\
\frac{dI(t)}{dt} &= pE - d_3 I - \alpha I, \\
\frac{dV(t)}{dt} &= \gamma_1 E + \gamma_2 I - d_4 V, \\
\frac{dR(t)}{dt} &= \alpha I - d_5 R.
\end{align*}
\]

(7)

System (7) has two equilibria, a disease free equilibrium and an endemic equilibrium, detail dynamical analysis of (7) were given in [5]. Global stability of equilibria of SEIVR model (7) is derived from the basic reproduction number \( R_0 \),

\[
R_0 = \frac{\beta_1 S_0}{p + d_1} + \frac{p \beta_2 S_0}{(d_3 + \alpha)(p + d_1)} + \frac{(d_3 + \alpha) \gamma_1 + p \gamma_2) \beta_3 S_0}{d_5(d_3 + \alpha)(p + d_1)}.
\]

(8)

Lemma 1. [5] The following statements hold for the model(7). (1) If \( R_0 \leq 1 \), the DFE of system(7) is globally asymptotically stable in \( \Omega \). (2) If \( R_0 > 1 \), the DFE of system(7) is unstable and there exists a unique endemic equilibrium. Moreover, the disease is uniformly persistent in the interior of \( \Omega = \{ X_0 = (S_0, 0, 0, 0, 0) \} \), denoted by \( \Omega^o = \{ X_* = (S_*, E_*, I_*, R_*, V_*) \} \); namely, 

\[
\lim_{x \to \infty} \inf(E(t), I(t), V(t)) > (\varepsilon, \varepsilon, \varepsilon) = (0, 0, 0)
\]

Remark 2: Here, we compare the differences of result of Theorem 2 with Lemma 1, the results indicate that complete enclosure and isolation is necessary. In system (7), as long as \( R_0 \) is less than 1, the disease will disappear from Lemma 1. However, once an individual input is present and the same parameters are selected to make \( R_0 < 1 \), the disease will persist from Theorem 2. This means that the uncompleted enclosure and isolation lead to individual mobility. Particularly, the extraneous virus carrier (exposed and influenced) and the input virus in the environment will cause the disease to be persist.
Numerically, we choose initial values as \((S_0; E_0; I_0; V_0) = (838505, 5000, 800, 31330)\), unless otherwise stated, parameter values given in Table 1 are used for the simulations. In order to get biologically plausible results, many values are taken from parameter ranges found in the literature.

**TABLE 1. Definitions and values of model parameters.**

| Parameters | Definition | Values | Reference |
|------------|------------|--------|-----------|
| \(\beta_1\) | Transmission constant between S and E | \(3.11 \times 10^{-8}\)/person/day | [8] |
| \(\beta_2\) | Transmission constant between S and I | \(1.62 \times 10^{-8}\)/person/day | [8] |
| \(\beta_3\) | Transmission constant between S and V | \(1.03 \times 10^{-8}\)/person/day | [5] |
| \(d_1\) | nature death rate | \(3.01 \times 10^{-5}\)/person/day | [5] |
| \(d_2\) | death rate of E | \(0.0317\)/person/day | [13] |
| \(d_3\) | death rate of I | \(0.0357\)/person/day | [13] |
| \(1/p\) | Incubation period | 7 days | [9] |
| \(\alpha\) | Recovery rate | \(1/13\) per day | [9] |
| \(\gamma_1\) | Virus shedding rate by exposed people | 2.3 per person per day per ml | [5] |
| \(\gamma_2\) | Virus shedding rate by infected people | 1.6 per person per day per ml | [5] |
| \(d_4\) | Removal rate of virus | 1 per day | [10] |
| \(\Lambda\) | the birth rate | 271.23 per day | estimation |

Figure 2(a) shows the numbers of cumulative confirmed cases during this period versus our fitting curve in model (7), at which time there is no infected case imported from the rest of the region. It should be clear that lines of exposed, infected and virus keep rising and falling to zero when \(R_0 < 1\). In Figure 2(a), we can note that the epidemic situation can be controlled under the condition of strict customs inspection and refusing anyone and goods to enter the region. When we consider individual mobility, we have numerical results in Figure 2(b).

Further, we choose parameters as \(W_E = 100, W_I = 200, W_V = 3000\), learned from the Figure 2(b) that as time goes on, the amount of exposed and infected is still large. We speculate that it may be related to individual mobility. If the individual mobility term about exposed and infected is reduced to zero, simulation in Figure 2(b) imply the disease still go away. Comparing Figure
When \( W_E = W_I = W_V = 0 \) and the basic reproduction number \( R_0 \) of (7) is less than 1, the disease eventually disappear in (a). However, when individual mobility exist as \( W_E = 100, W_I = 200, W_V = 3000 \), the diseases stick around in (b).

2(a) with Figure 2(b), when the individual mobility is disappear, the population of the exposed, the infected and the virus in environment has been greatly reduced. Strict customs inspection is effective and necessary for controlling the transmission of COVID-19 epidemic[13, 14].

In the following part, we carry out the detailed influence of the parameters \( W_E, W_I, W_V \).

### 4.2. Impact of the flow of symptomatic infected.

Each person is tested for nucleic acid, quarantined and returned to his or her place of origin as soon as an infected person is found. This case implies \( W_I = 0 \).

As can be seen from Figure 3, according to the customs inspection, we can identify the infected people with symptoms. The peak of the infected person’s curve over time decreased, and the value that finally stabilized decreased. Its practical significance means that preventing infected persons to enter their own cities can effectively control the spread of the epidemic and keep it to a smaller value eventually.

Meanwhile, occupational health of customs agents should not be neglected. Apart from the prevailing coronavirus disease, the customs agents are exposed to many other transmissible diseases (such as influenza etc.) which are transmitted through close work contact. Such customs
agents should be provided with effective preventive tools. It is necessary for customs agents to have nucleic acid tests after working. These procedures can be routinely undertaken during their duty hours.

4.3. **Impact of customs inspection is more stringent.** Under stricter customs inspection, individual mobility will be tested for nucleic acid and then quarantined for 14 days to monitor at any time.

After isolation, nucleic acid testing will be conducted again. Such a complex test is designed to prevent the flow of asymptomatic and asymptomatic infected individuals. This case implies $W_E = 0$. When it is clear that the virus and the infected will cause further spread, we wonder whether there is the latent impact on the epidemic.

In this part, we decrease the value of $W_E$ from 100 to 0. The other parameter values are the same as before. The result of numerical simulation is shown in Figure 4. We can notice that the density of exposed decreases to zero, which means if constant input of exposed individuals mobility are small, the exposed goes decrease in the end.

4.4. **Impact of the amount of virus in the environment.** There is evidence that people can catch COVID-19 by touching virus which is alive, so exotic items should be strictly controlled, check all items to insure that no novel Coronavirus has entered. Under the control of foreign
Figure 4. Compare the case where $W_E = 0$ with the case where $W_E > 0$, the number of the exposed has decreased markedly.

Figure 5. Compare the case where $W_V = 0$ with the case where $W_V > 0$, the number of the coronavirus in the environment has decreased markedly.

Objects, the virus in the environment only comes from the infected and latent people. Figure 5 clearly shows how the virus volume changes over time with and without control.

Similarly, if we control the individual mobility, we can also effectively control the spread of the epidemic. There are positive correlation between individual mobility and the infected, which indicates that the expand of the immigration promote the growth in infections.
In Figure 6, the black curve represent the number of people who have been infected without any action being taken. Purple represents the control of all incoming items that have been tested to ensure that there is no novel coronavirus active. The red curve means customs will not let in anyone who has shown symptoms, while the blue curve represents the number of infected and exposed people in a situation where asymptomatic and asymptomatic infected individuals are not allowed in.

In terms of the rate of growth, it is the mount of infected that has the greatest impact. And the viruses are too small to be compared with the same number of individuals. Viruses in the environment also have the least impact due to their short survival time or higher death rate. So the implication is that we need a large enough amount of virus to infect the susceptible, and a large enough amount of virus is not much for us. Raise the floating population through increase individual mobility, bring about the expand of the infected. We need to pay more attention to input virus of environment.

5. Conclusions and Discussions

In the present study, we have investigated an SEIVR model with individual mobility and analyzed global stability of the epidemic equilibrium. The model we have established can better evaluate the transmission of COVID-19. Our results suggest that: (i) Individual mobility makes
the unique positive epidemic equilibrium always exist, (ii) The unique positive equilibrium is always globally asymptotically stable as long as it exists.

From the theoretical analysis and numerical simulations, we point out that if there has a steady move of individual mobility, the disease will inevitably be present in the region. In order to control the spread of disease, the number of individual mobility should be reduced to the barest minimum. It is hard to eliminate the disease completely because the government departments are not able to remove all the individual mobility carrying the disease. It would be more reasonable that government give priority to control the number of individual mobility, including conducting nucleic acid tests on all inbound travelers and quarantining them. Once infected individuals are found, they should be quarantined for observation. We suggest that diminishing the individual mobility during the outbreak response can give rise to the increase in protection rate. In other words, avoiding unnecessary individual mobility will extremely help in reducing or controlling the spread of COVID-19.

CONFLICT OF INTERESTS

The author(s) declare that there is no conflict of interests.

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