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Research article

Analysis of the COVID-19 model with self-protection and isolation measures affected by the environment

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Abstract: Since the global outbreak of COVID-19, the virus has continuously mutated and can survive in the air for long periods of time. This paper establishes and analyzes a model of COVID-19 with self-protection and quarantine measures affected by viruses in the environment to investigate the influence of viruses in the environment on the spread of the outbreak, as well as to develop a rational prevention and control measure to control the spread of the outbreak. The basic reproduction number was calculated and Lyapunov functions were constructed to discuss the stability of the model equilibrium points. The disease-free equilibrium point was proven to be globally asymptotically stable when $R_0 < 1$, and the endemic equilibrium point was globally asymptotically stable when $R_0 > 1$. The model was fitted using data from COVID-19 cases in Chongqing between November 1 to November 25, 2022. Based on the numerical analysis, the following conclusion was obtained: clearing the virus in the environment and strengthening the isolation measures for infected people can control the epidemic to a certain extent, but enhancing the self-protection of individuals can be more effective in reducing the risk of being infected and controlling the transmission of the epidemic, which is more conducive to the practical application.

Keywords: COVID-19; stability analysis; isolation measures; self-protection

1. Introduction

COVID-19 [1, 2] is an infectious disease caused by the SARS-CoV-2 coronavirus, which was first detected in December 2019 in Wuhan, Hubei, China; since then, it has been found in other parts of the country and in other countries. The virus is highly contagious and causes respiratory infections that can lead to difficulty breathing, pneumonia, and other serious complications; the elderly and those with chronic illnesses are more susceptible to infections and serious conditions that can even lead to death [3, 4]. In an effort to contain the outbreak, countries have implemented embargo measures

and restrictions, thus leading to the disruption of business activities, business closures, and increased unemployment. Disruptions in global supply chains and reduced consumption have slowed economic growth, thus leading to a severe recession [5]. Since the outbreak, scientists around the world have been actively researching information on the biological properties, epidemiological characteristics, clinical manifestations, and therapeutic strategies of the virus in order to better respond to the outbreak [6]. The development of vaccines has played a key role in protecting people's health and reducing the risk of the virus. However, the diversity and variability of SARS-CoV-2 prevent the vaccine from completely helping people avoid the risk of infection. Therefore, other measures are needed to either reduce the risk of infection or to inhibit the spread of the disease.

1.1. COVID-19 model with isolation measures.

Isolation measures are an important prevention and control tool that are widely used in disease control and prevention. They can curb the spread of disease by separating patients from healthy people, thus reducing the chances of disease transmission and minimizing the risks resulting from interpersonal contact. Additionally, they facilitate early diagnoses and interventions, thus improving the cure and survival rates. At the same time, isolation measures also help to track and investigate the transmission pathways of diseases, thereby providing important data and information for the development of appropriate prevention and control strategies. Therefore, many scholars have investigated the importance of isolation measures for COVID-19 control in mathematical models [7–12]. Zhang et al. [13] studied the effect of soft isolation of susceptible populations on the spread of COVID-19 based on case data from Wuhan. Numerical simulations showed that when the isolation rate of susceptible populations was larger or the exposure rate of susceptible populations was smaller, the number of infected people reached the peak value earlier, the peak value was lower, the duration of the epidemic was smaller, and the final size of the infected people was significantly smaller. Tang et al. [14] designed a deterministic house model based on the clinical progression of the disease, the epidemiologic status of various individuals, and interventions; estimates based on likelihood and modeling analyses indicated that the number of controlled reproductions could be as high as 6.47 (95% CI 5.71–7.23). Sensitivity analyses indicated that interventions, such as close contact tracing followed by isolation and quarantine, could be effective in reducing the number of controlled reproductions and the risk of transmission. Yuan et al. [15] considered a model of infectious disease with asymptomatic infection and isolation, and numerically analyzed the data statistics of the number of infected people in India; the study concluded that the greater the recovery rate of the isolates, the shorter the treatment period, and the faster the disease disappears. Therefore, the study of potent drugs is important. Moreover, when the isolation rate was 0.3519–0.5411, asymptomatic infected people affected the spread of infectious diseases. Isolation of symptomatic people is very important to control and eliminate the disease. Maryam et al. [16] proposed a mathematical model for the deterministic delineation of neococcal pneumonia with different isolation and segregation types, thereby dividing segregation compartments into short-term and long-term segregation compartments, and into detected and non-detected home segregated individuals and institutional segregated individuals. Numerical simulations were performed using the Omani dataset to demonstrate the impact of some model parameters associated with the different types of isolation and segregation on the dynamics of disease transmission. In the face of infectious diseases, the rational implementation of quarantine measures is crucial. In order to make an effective and reasonable plan to prevent the spread of variant COVID-19, Guo et al. [17] established a new vaccination population

epidemic model. By fitting the data of the Delta strain epidemic in Jiangsu Province, China, and analyzing the parameters, it was found that the best control measure was to dynamically adjust three control measures (vaccination, isolation and nucleic acid testing) to achieve the lowest number of infections at the lowest cost.

1.2. COVID-19 model with self-protection measures.

Although vaccines can reduce and against the risk to human beings, it cannot completely protect them from being infected. Therefore, in times of epidemics, people need to take greater protective measures for themselves to either avoid contact with patients or reduce interpersonal contact, thus lowering the risk of infection and protecting their own health and the health of others. Self-protection measures [18–20] can reduce the number of people who become ill, thereby reducing the burden on the health-care system and allowing health-care resources to be better utilized when treating patients in need of urgent care. The literature [21] described a reaction-diffusion SEIR model for self-protection of susceptible individuals, and treatment of infected individuals, using numerical simulations to demonstrate the existence of traveling waves and to show that self-protection and treatment reduced the rate of transmission of infectious diseases. Using epidemiological data on COVID-19 and anonymized data on human mobility, Lai et al. [22] constructed a modeling framework to simulate different outbreaks and intervention scenarios across China using daily travel networks. It was found that early detection and isolation of cases prevented more infections than travel restriction and contact reduction; however, non-pharmacological interventions had the strongest and most rapid combined effect. Hu et al. [23] constructed a kinetic model that described the spread of COVID-19 in wildlife, human populations, and the environment, which also introduced asymptomatically infected populations and susceptible populations with self-protection awareness. Numerical simulations showed that stopping all human migration, increasing treatment intensity, enhancing personal protection awareness, and especially improving the ability to recognize asymptomatically infected people were indispensable control tools to control the spread of the disease in the early stages of a malignant outbreak.

1.3. COVID-19 model with environmental factor.

According to the World Health Organization's scientific briefing on the spread of COVID-19, the mode of transmission of SARS-CoV-2 included contaminated environments in addition to close contact [24]. Therefore, we should also pay attention to the impact of viruses in the environment on the spread of the epidemic. Wang et al. [25] designed a two-strain, nonlinear kinetic model that considered the simultaneous transmission of mutated and wild strains, environmental transmission, and implementation of vaccination in the context of a shortage of basic healthcare resources. Findings suggest that reducing the prevalence of the mutant strain, increasing viral clearance in the environment, and enhancing the ability to treat infected individuals are critical to mitigate and control the spread of COVID-19, especially in resource-limited areas. Musa et al. [26] proposed a new dynamic model to study the spread of COVID-19. The model incorporated possible recurrence, reinfection, and environmental contributions to assess their combined impact on the overall transmission dynamics of SARS-CoV-2. The model was numerically simulated using standard Eulerian techniques to elucidate the impact of some key parameters on the overall transmission dynamics. The results of the study indicated that exposure, hospitalization, and reactivation rates were essential parameters that required

special attention for pandemic prevention, mitigation, and control.

During outbreaks in countries such as China, a large number of researchers have studied the effectiveness of isolation measures and analysed the impact of the virus on the environment. However, few papers have considered site-specific isolation measures, as well as the simultaneous isolation of confirmed and asymptomatic patients. Numberous papers considered the importance of vaccinations, though they neglected the importance of individual self-protection.

Motivated by the aforementioned literature, in order to reveal the impact of isolation measures and self-protection on the spread of COVID-19, we will establish an epidemic model that includes the aforementioned factors and conduct in-depth study. In addition, considering the influence factors of viruses in the environment is also a highlight of this article, which can further analyze the spread of COVID-19. This article will analyze the reliability of the model based on actual data from Chongqing, China. Chongqing is one of the municipalities directly under the central government in China, and the outbreak started on November 1, 2022. The outbreak rapidly spread and the situation was critical, which was mainly caused by Omicron BA5.2, which can be transmitted by aerosol, respiratory droplets, and close contact. Chongqing is a mountainous city with a lot of water mist, which makes it easy for virus aerosols to be suspended in the air and spread. Moreover, the large population and the different living patterns from other cities, with old high-rise buildings densely packed and poorly sealed, make it easy for the virus to spread and propagate. Based on the above studies and the actual situation in Chongqing, the model combines self-protection, isolation measures, and measures to remove the virus from the environment to assess their combined impact on the overall transmission dynamics of SARS-CoV-2. The main points of this paper are as follows: (1) the stability of the equilibrium point is theoretically demonstrated; (2) the accuracy of the model is verified by fitting it to the actual data; and (3) parametric analyses illustrate the validity of self-protection and the necessity of isolation measures for both diagnosed and asymptomatic patients.

2. Mathematical model

In this paper, we categorize susceptible individuals into unconscious susceptible individuals and conscious susceptible individuals, where conscious susceptible individuals are those who will take personal protective measures to reduce the probability of infection when a disease is prevalent. When infected with SARS-CoV-2, the infected person is often accompanied by fever, cough, sputum, shortness of breath, fatigue, and other symptoms; additionally, confirmed infected individuals have positive nucleic acid test results. As SARS-CoV-2 continues to mutate, there are infected individuals who do not have any clinical symptoms but have a positive nucleic acid test results. Moreover, these infected individuals are at risk of harboring and transmitting the virus, and are referred to as asymptomatic infected individuals [27]. Therefore, the dynamical model is described as follows:

$$\begin{cases} \frac{dS_1}{dt} = \Lambda - \beta_1 S_1 I_1 - \beta_2 S_1 I_2 - \beta_W S_1 W - \rho S_1 - dS_1, \\ \frac{dS_2}{dt} = \rho S_1 - \beta_1 \sigma S_2 I_1 - \beta_2 \sigma S_2 I_2 - \beta_W \sigma S_2 W - dS_2, \\ \frac{dE}{dt} = \beta_1 S_1 I_1 + \beta_2 S_1 I_2 + \beta_W S_1 W + \beta_1 \sigma S_2 I_1 + \beta_2 \sigma S_2 I_2 + \beta_W \sigma S_2 W - \tau E - dE, \\ \frac{dI_1}{dt} = \tau p E - \gamma_1 I_1 - \mu I_1 - dI_1 - k_1 I_1, \\ \frac{dI_2}{dt} = \tau(1-p) E - \gamma_2 I_2 - \mu I_2 - dI_2 - k_2 I_2, \\ \frac{dQ}{dt} = k_1 I_1 + k_2 I_2 - \mu Q - dQ - \phi Q, \\ \frac{dR}{dt} = \gamma_1 I_1 + \gamma_2 I_2 + \phi Q - dR, \\ \frac{dW}{dt} = \lambda_1 I_1 + \lambda_2 I_2 - \delta W, \end{cases} \tag{2.1}$$

where $S_1(t), S_2(t), E(t), I_1(t), I_2(t), Q(t)$, and $R(t)$ represent the number of the unconscious susceptible individuals, conscious susceptible individuals, exposed, confirmed infected, asymptomatic infected, quarantined, and the recovered population at time t, respectively. The viral spread in the environment is denoted as $W(t)$. $\Lambda > 0$ describes the annual birth rate, and ρ is the migration rate from S_1 to S_2. σ is the rate at which susceptible individuals with self-protection reduce contacts with infectious. β_1 is a constant, β_2 represent the infection rates, and β_W represents the infection rates of the virus in the environment. $\frac{1}{\tau}$ represents the mean incubation period. p is the proportion of the confirmed infected. k_1 and k_2 are the quarantine rates. μ is the disease-induced death rate. γ_1 and γ_2 are the recovery rates of infected persons. ϕ is the recovery rate after treatment. d is the natural human mortality rates. δ is the clearance rate of viruses in the environment. λ_1 and λ_2 are the virus released rates.

Define $N(t) = S_1(t) + S_2(t) + E(t) + I_1(t) + I_2(t) + Q(t) + R(t) \geq 0$ and $W(t) \geq 0$, from system (2.1), we know that

$$\begin{aligned} \frac{dN}{dt} =& \Lambda - dS_1 - dS_2 - dE - dI_1 - \mu I_1 - dI_2 - \mu I_2 - dQ - \mu Q - dR \\ =& \Lambda - dN - \mu(I_1 + I_2 + Q) \\ \leq& \Lambda - dN, \end{aligned}$$

and

$$\frac{dW}{dt} = \lambda_1 I_1 + \lambda_2 I_2 - \delta W \leq (\lambda_1 + \lambda_2) N - \delta W,$$

so, $\lim\limits_{t \to +\infty} sup(N(t)) \leq \frac{\Lambda}{d}$ and $\lim\limits_{t \to +\infty} sup(W(t)) \leq \frac{\Lambda(\lambda_1 + \lambda_2)}{d\delta}$.

This is implies that the solutions of system (2.1) are bounded and the region

$$\begin{aligned} X = \Big\{ &(S_1(t), S_2(t), E(t), I_1(t), I_2(t), Q(t), R(t), W(t)) \in \mathbb{R}_+^8 : 0 \leq S_1(t) + S_2(t) \\ &+ E(t) + I_1(t) + I_2(t) + Q(t) + R(t) \leq \frac{\Lambda}{d}, 0 \leq W(t) \leq \frac{\Lambda(\lambda_1 + \lambda_2)}{d\delta} \Big\} \end{aligned}$$

is a positively invariant with respect to system (2.1).

3. Equilibrium points analysis

In this section, we show that the system (2.1) has a unique disease-free equilibrium P_0. Then, we derive the basic reproduction number R_0 using the next generation matrix method. Finally, we prove system (2.1) has a unique endemic equilibrium P_* when $R_0 > 1$.

3.1. Disease-free equilibrium point and basic reproduction number

Making the right end of the system (2.1) equal to 0 gives the only disease-free equilibrium point $P_0 = (S_1^0, S_2^0, 0, 0, 0, 0, 0, 0)$ of the system, and

$$S_1^0 = \frac{\Lambda}{\rho + d}, \quad S_2^0 = \frac{\rho\Lambda}{d(\rho + d)}.$$

Using the next generation matrix formulated in Diekmam et al. [28] and van den Driessche and Watmough [29], we define the basic reproduction number by the following:

$$R_0 = R_1 + R_2 + R_3,$$

where,

$$\begin{aligned}
R_1 &= \frac{\tau p}{(k_1 + \gamma_1 + d + \mu)(\tau + d)}(\beta_1 S_1^0 + \sigma\beta_1 S_2^0),\\
R_2 &= \frac{\tau(1-p)}{(k_2 + \gamma_2 + d + \mu)(\tau + d)}(\beta_2 S_1^0 + \sigma\beta_2 S_2^0),\\
R_3 &= (\frac{\tau p\lambda_1}{\delta(k_1 + \gamma_1 + d + \mu)(\tau + d)} + \frac{\tau(1-p)\lambda_2}{\delta(k_2 + \gamma_2 + d + \mu)(\tau + d)})(\beta_W S_1^0 + \sigma\beta_W S_2^0).
\end{aligned}$$

Here, R_1 is the average number of infections transmitted by confirmed infected individuals during their illness period, R_2 is the average number of asymptomatic infections transmitted during the disease period, and R_3 is the average number of infections caused by viruses in the environment. Thus, we have the following theorem.

3.2. Existence of an endemic equilibrium point

Theorem 1. *System* (2.1) *has a unique endemic equilibrium P_* if and only if $R_0 > 1$ holds.*

Proof. By the equilibrium equations of system (2.1), we can conclude that

$$\begin{cases}
\Lambda - \beta_1 S_1 I_1 - \beta_2 S_1 I_2 - \beta_W S_1 W - \rho S_1 - d S_1 = 0,\\
\rho S_1 - \beta_1\sigma S_2 I_1 - \beta_2\sigma S_2 I_2 - \beta_W\sigma S_2 W - d S_2 = 0,\\
\beta_1 S_1 I_1 + \beta_2 S_1 I_2 + \beta_W S_1 W + \beta_1\sigma S_2 I_1 + \beta_2\sigma S_2 I_2 + \beta_W\sigma S_2 W - \tau E - dE = 0,\\
\tau p E - \gamma_1 I_1 - \mu I_1 - d I_1 - k_1 I_1 = 0,\\
\tau(1-p) E - \gamma_2 I_2 - \mu I_2 - d I_2 - k_2 I_2 = 0,\\
k_1 I_1 + k_2 I_2 - \mu Q - d Q - \phi Q = 0,\\
\gamma_1 I_1 + \gamma_2 I_2 + \phi Q - d R = 0,\\
\lambda_1 I_1 + \lambda_2 I_2 - \delta W = 0.
\end{cases} \tag{3.1}$$

Combining with the equations in system (3.1), one can obtain the following:

$$\begin{aligned}
S_1 = \frac{\Lambda}{\beta_1 I_1 + \beta_2 I_2 + \beta_W W + \rho + d}, \quad S_2 = \frac{\rho S_1}{\sigma\beta_1 I_1 + \sigma\beta_2 I_2 + \sigma\beta_W W + d},\\
E = \frac{(\mu + d + k_1 + \gamma_1) I_1}{\tau p}, \quad I_2 = \frac{\tau(1-p)E}{\mu + d + k_2 + \gamma_2}, \quad W = \frac{\lambda_1 I_1 + \lambda_2 I_2}{\delta}.
\end{aligned} \tag{3.2}$$

Because $I_1 \neq 0$, it can be obtained through (3.2) that

$$\begin{aligned} S_1 + \sigma S_2 =& \frac{\Lambda}{\beta_1 I_1 + \beta_2 I_2 + \beta_W W + \rho + d} + \frac{\sigma\rho S_1}{\sigma\beta_1 I_1 + \sigma\beta_2 I_2 + \sigma\beta_W W + d} \\ =& \frac{\Lambda\delta pB}{MI_1 + (\rho + d)\delta pB} \frac{\sigma MI_1 + (d + \sigma\rho)\delta pB}{\sigma MI_1 + d\delta pB}. \end{aligned} \tag{3.3}$$

By the third equations of the system (3.1), we can conclude that

$$S_1 + \sigma S_2 = \frac{(\tau + d)E}{\beta_1 I_1 + \beta_2 I_2 + \beta_W W} = \frac{\delta(\tau + d)AB}{\tau M}; \tag{3.4}$$

here, we denote $A = k_1+\gamma_1+d+\mu, B = k_2+\gamma_2+d+\mu$ and $M = \delta pB\beta_1+\delta(1-p)A\beta_2+(\lambda_1 pB+\lambda_2(1-p)A)\beta_W$. Combining (3.3) and (3.4), we have

$$F(I_1) = \frac{\delta pB}{MI_1 + (\rho + d)\delta pB} \frac{\sigma MI_1 + (d + \sigma\rho)\delta pB}{\sigma MI_1 + d\delta pB} - \frac{\delta(\tau + d)AB}{\tau M\Lambda}$$

and

$$\frac{dF(I_1)}{dt} = \frac{F_1(I_1)}{(\sigma MI_1 + d\delta pB)^2[MI_1 + (\rho + d)\delta pB]^2}$$

and

$$\begin{aligned} F_1(I_1) =& -\delta pBM(\sigma^2 M^2 (I_1)^2 + 2BM\delta p\rho\sigma^2 I_1 + 2BMd\delta p\sigma I_1 + B^2 d^2 p^2 \delta^2 \\ &+ B^2 d\delta^2 p^2 \rho\sigma^2 + B^2 d\delta^2 p^2 \rho\sigma + B^2 \delta^2 p^2 \rho^2 \sigma^2) < 0. \end{aligned}$$

Therefore, we obtain the derivative $\frac{dF(I_1)}{dt} < 0$. Thus, $F(I_1)$ is a monotone decreasing function.

Then,

$$\begin{aligned} F(0) =& \frac{\delta pB}{(\rho + d)\delta pB} \frac{(d + \sigma\rho)\delta pB}{d\delta pB} - \frac{\delta(\tau + d)AB}{\tau M\Lambda} \\ =& \frac{S_1^0 + \sigma S_2^0}{\Lambda} - \frac{\delta(\tau + d)AB}{\tau M\Lambda} \\ =& \frac{\delta(\tau + d)AB}{\tau M\Lambda} \frac{\tau M(S_1^0 + \sigma S_2^0)}{\delta(\tau + d)AB} - \frac{\delta(\tau + d)AB}{\tau M\Lambda} \\ =& \frac{\delta(\tau + d)AB}{\tau M\Lambda}(R_0 - 1). \end{aligned}$$

Because the inequality $[MI_1 + \delta pB(\rho + d)](\sigma MI_1 + \delta dpB) > MI_1[\sigma MI_1 + (d + \sigma\rho)\delta pB]$, we can obtain the following:

$$\begin{aligned} F(\frac{\Lambda}{d}) =& \frac{\delta pB}{M\frac{\Lambda}{d} + (\rho + d)\delta pB} \frac{\sigma M\frac{\Lambda}{d} + (d + \sigma\rho)\delta pB}{\sigma M\frac{\Lambda}{d} + d\delta pB} - \frac{\delta(\tau + d)AB}{\tau M\Lambda} \\ <& \frac{\delta pBd}{\Lambda M} - \frac{\delta(\tau + d)AB}{\tau\Lambda M} \leq \frac{\delta Bd}{\Lambda M} - \frac{\delta(\tau + d)AB}{\tau\Lambda M} < 0. \end{aligned}$$

According to the monotonicity of the function of $F(I_1)$, when $R_0 > 1$, the equation has a unique positive root on the interval of $(0, \frac{\Lambda}{d})$. When $R_0 < 1$, the equation has no positive root on the interval of $(0, \frac{\Lambda}{d})$.

Therefore, if $R_0 > 1$, then the system (2.1) has an endemic equilibrium $P_* = (S_1^*, S_2^*, E^*, I_1^*, I_2^*, Q^*, R^*, W^*)$.

4. Global stability of the equilibrium points

In this section, we will prove the global stability of the equilibrium points by constructing the Lyapunov function. Now, we provide the following two key Theorems to provide the global stability.

4.1. Global stability of disease-free equilibrium point

Theorem 2. *If $R_0 < 1$, then the disease-free equilibrium P_0 of the system* (2.1) *is global asymptotically stable.*

Proof. Define the Lyapunov function as follows:

$$\begin{aligned} V =& \frac{E}{\tau + d} + \frac{(\beta_1 S_1^0 + \beta_1 \sigma S_2^0) I_1}{(\tau + d)(\mu + d + k_1 + \gamma_1)} + \frac{(\beta_2 S_1^0 + \beta_2 \sigma S_2^0) I_2}{(\tau + d)(\mu + d + k_2 + \gamma_2)} \\ &+ \frac{(\beta_W S_1^0 + \beta_W \sigma S_2^0)\lambda_1 I_1}{(\tau + d)(\mu + d + k_1 + \gamma_1)\delta} + \frac{(\beta_W S_1^0 + \beta_W \sigma S_2^0)\lambda_2 I_2}{(\tau + d)(\mu + d + k_2 + \gamma_2)\delta} + \frac{(\beta_W S_1^0 + \beta_W \sigma S_2^0) W}{(\tau + d)\delta}. \end{aligned}$$

For all $t > 0$, the derivative of $V(t)$ is as follows:

$$\begin{aligned} \frac{dV}{dt} =& \frac{1}{\tau + d}(\beta_1 S_1 I_1 + \beta_2 S_1 I_2 + \beta_W S_1 W + \beta_1 \sigma S_2 I_1 + \beta_2 \sigma S_2 I_2 + \beta_W \sigma S_2 W - \tau E - dE) \\ &+ \frac{(\beta_1 S_1^0 + \beta_1 \sigma S_2^0)}{(\tau + d)(\mu + d + k_1 + \gamma_1)}(\tau p E - \gamma_1 I_1 - \mu I_1 - d I_1 - k_1 I_1) \\ &+ \frac{(\beta_2 S_1^0 + \beta_2 \sigma S_2^0)}{(\tau + d)(\mu + d + k_2 + \gamma_2)}(\tau(1 - p)E - \gamma_2 I_2 - \mu I_2 - d I_2 - k_2 I_2) \\ &+ \frac{(\beta_W S_1^0 + \beta_W \sigma S_2^0)\lambda_1}{(\tau + d)(\mu + d + k_1 + \gamma_1)\delta}(\tau p E - \gamma_1 I_1 - \mu I_1 - d I_1 - k_1 I_1) \\ &+ \frac{(\beta_W S_1^0 + \beta_W \sigma S_2^0)\lambda_2}{(\tau + d)(\mu + d + k_2 + \gamma_2)\delta}(\tau(1 - p)E - \gamma_2 I_2 - \mu I_2 - d I_2 - k_2 I_2) \\ &+ \frac{(\beta_W S_1^0 + \beta_W \sigma S_2^0)}{(\tau + d)\delta}(\lambda_1 I_1 + \lambda_2 I_2 - \delta W) \\ \leq& \frac{(\beta_1 S_1^0 + \beta_1 \sigma S_2^0)}{\tau + d} I_1 + \frac{(\beta_2 S_1^0 + \beta_2 \sigma S_2^0)}{\tau + d} I_2 + \frac{(\beta_W S_1^0 + \beta_W \sigma S_2^0)}{\tau + d} W - E \\ &+ \frac{\tau p(\beta_1 S_1^0 + \beta_1 \sigma S_2^0)}{(\tau + d)(\mu + d + k_1 + \gamma_1)} E - \frac{(\beta_1 S_1^0 + \beta_1 \sigma S_2^0)}{\tau + d} I_1 + \frac{\tau(1 - p)(\beta_2 S_1^0 + \beta_2 \sigma S_2^0)}{(\tau + d)(\mu + d + k_2 + \gamma_2)} E \\ &+ \frac{\tau p \lambda_1 (\beta_W S_1^0 + \beta_W \sigma S_2^0)}{(\tau + d)(\mu + d + k_1 + \gamma_1)\delta} E - \frac{(\beta_W S_1^0 + \beta_W \sigma S_2^0)}{(\tau + d)\delta}\lambda_1 I_1 + \frac{\tau(1 - p)\lambda_2(\beta_W S_1^0 + \beta_W \sigma S_2^0)}{(\tau + d)(\mu + d + k_2 + \gamma_2)\delta} E \\ &- \frac{(\beta_W S_1^0 + \beta_{2W} \sigma S_2^0)}{(\tau + d)\delta}\lambda_2 I_2 + \frac{(\beta_W S_1^0 + \beta_W \sigma S_2^0)}{(\tau + d)\delta}\lambda_1 I_1 + \frac{(\beta_W S_1^0 + \beta_W \sigma S_2^0)}{(\tau + d)\delta}\lambda_2 I_2 \\ &- \frac{(\beta_2 S_1^0 + \beta_2 \sigma S_2^0)}{\tau + d} I_2 - \frac{(\beta_W S_1^0 + \beta_W \sigma S_2^0)}{\tau + d} W \\ =& E(R_0 - 1). \end{aligned}$$

Thus, $\frac{dV}{dt} < 0$ if $R_0 < 1$. Using LaSalle's [30] extension to the Lyapunov's method, the disease-free equilibrium P_0 is globally asymptotically stable.

4.2. Global stability of the equilibrium point of endemic diseases

Theorem 3. *The equilibrium point P_* of system* (2.1) *is globally asymptotically stable if and only if* $R_0 > 1$.

Proof. Since the equilibrium point P_* of endemic diseases is determined by system (2.1), we have the following:

$$\begin{cases} \rho + d = \frac{\Lambda}{S_1^*} - \beta_1 I_1^* - \beta_2 I_2^* - \beta_W W^*, \\ d = \frac{\rho S_1^*}{S_2^*} - \sigma\beta_1 I_1^* - \sigma\beta_2 I_2^* - \sigma\beta_W W^*, \\ d + \tau = \frac{\beta_1 S_1^* I_1^*}{E^*} + \frac{\beta_2 S_1^* I_2^*}{E^*} + \frac{\beta_W S_1^* W^*}{E^*} + \frac{\sigma\beta_1 S_2^* I_1^*}{E^*} + \frac{\sigma\beta_2 S_2^* I_2^*}{E^*} + \frac{\sigma\beta_W S_2^* W^*}{E^*}, \\ k_1 + \gamma_1 + d + \mu = \frac{\tau p E^*}{I_1^*}, \\ k_2 + \gamma_2 + d + \mu = \frac{\tau(1-p)E^*}{I_2^*}, \\ d + \phi + \mu = \frac{k_1 I_1^*}{Q^*} + \frac{k_2 I_2^*}{Q^*}, \\ d = \frac{\gamma_1 I_1^*}{R^*} + \frac{\gamma_2 I_2^*}{R^*} + \frac{\phi Q^*}{R^*}, \\ \delta = \frac{\lambda_1 I_1^*}{W^*} + \frac{\lambda_2 I_2^*}{W^*}. \end{cases} \tag{4.1}$$

We denote

$$x = \frac{S_1}{S_1^*}, \quad y = \frac{S_2}{S_2^*}, \quad e = \frac{E}{E^*}, \quad a = \frac{I_1}{I_1^*}, \quad b = \frac{I_2}{I_2^*}, \quad c = \frac{W}{W^*}.$$

Define the Lyapunov function as follows:

$$\begin{aligned} V =& S_1^*(x - 1 - lnx) + S_2^*(y - 1 - lny) + E^*(e - 1 - lne) + nI_1^*(a - 1 - lna) \\ &+ mI_2^*(b - 1 - lnb) + fW^*(c - 1 - lnc). \end{aligned}$$

The derivative of $V(t)$ is as follows:

$$\begin{aligned} \frac{dV}{dt} =& (1 - \frac{1}{x})\frac{dS_1}{dt} + (1 - \frac{1}{y})\frac{dS_2}{dt} + (1 - \frac{1}{e})\frac{dE}{dt} + n(1 - \frac{1}{a})\frac{dI_1}{dt} + m(1 - \frac{1}{b})\frac{dI_2}{dt} + f(1 - \frac{1}{c})\frac{dW}{dt} \\ =& (1 - \frac{1}{x})[\Lambda(1 - x) - \beta_1 S_1^* I_1^*(a - 1)x - \beta_2 S_1^* I_2^*(b - 1)x - \beta_W S_1^* W^*(c - 1)x] \\ &+ (1 - \frac{1}{y})[\rho S_1^*(x - y) - \sigma\beta_1 S_2^* I_1^*(a - 1)y - \sigma\beta_2 S_2^* I_2^*(b - 1)y - \sigma\beta_W S_2^* W^*(c - 1)y] \\ &+ (1 - \frac{1}{e})[\beta_1 I_1^* S_1^*(xa - e) + \beta_2 I_2^* S_1^*(xb - e) + \beta_W S_1^* W^*(cx - e) \\ &+ \sigma\beta_1 S_2^* I_1^*(ya - e) + \sigma\beta_2 S_2^* I_2^*(yb - e) + \sigma\beta_W S_2^* W^*(cy - e)] \\ &+ n(1 - \frac{1}{a})[\tau p E^*(e - a)] + m(1 - \frac{1}{b})[\tau(1 - p)E^*(e - b)] \\ &+ f(1 - \frac{1}{c})[\lambda_1 I_1^*(a - c) + \lambda_2 I_2^*(b - c)] \\ =& \beta_1 S_1^* I_1^*(3 - \frac{1}{x} - \frac{xa}{e} - \frac{e}{a}) + \beta_2 S_1^* I_2^*(3 - \frac{1}{x} - \frac{xb}{e} - \frac{e}{b}) + \beta_W S_1^* W^*(4 - \frac{1}{x} - \frac{cx}{e} - \frac{e}{a} - \frac{a}{c}) \\ &+ \sigma\beta_1 S_2^* I_1^*(4 - \frac{1}{x} - \frac{ya}{e} - \frac{e}{a} - \frac{x}{y}) + \sigma\beta_2 S_2^* I_2^*(4 - \frac{1}{x} - \frac{yb}{e} - \frac{e}{b} - \frac{x}{y}) \\ &+ \sigma\beta_W S_2^* W^*(5 - \frac{1}{x} - \frac{x}{y} - \frac{cy}{e} - \frac{e}{b} - \frac{b}{c}) + dS_1^*(2 - x - \frac{1}{x}) + dS_2^*(3 - y - \frac{1}{x} - \frac{x}{y}), \end{aligned}$$

where the constants $n = \frac{\beta_1 S_1^* I_1^* + \sigma\beta_1 S_2^* I_1^* + \beta_W S_1^* W^*}{\tau p E^*}, m = \frac{\beta_2 S_1^* I_2^* + \sigma\beta_2 S_2^* I_2^* + \sigma\beta_W S_2^* W^*}{\tau(1-p)E^*}, f = \frac{\beta_W S_1^* W^*}{\lambda_1 I_1^*} = \frac{\sigma\beta_W S_2^* W^*}{\lambda_2 I_2^*}$.

According to the relationship of inequality, we have $3-\frac{1}{x}-\frac{xa}{e}-\frac{e}{a} \leq 0, 3-\frac{1}{x}-\frac{xb}{e}-\frac{e}{b} \leq 0, 4-\frac{1}{x}-\frac{cx}{e}-\frac{e}{a}-\frac{a}{c} \leq 0, 4-\frac{1}{x}-\frac{ya}{e}-\frac{e}{a}-\frac{x}{y} \leq 0, 4-\frac{1}{x}-\frac{yb}{e}-\frac{e}{b}-\frac{x}{y} \leq 0, 5-\frac{1}{x}-\frac{x}{y}-\frac{cy}{e}-\frac{e}{b}-\frac{b}{c} \leq 0, 2-x-\frac{1}{x} \leq 0, 3-y-\frac{1}{x}-\frac{x}{y} \leq 0$, so, the derivative of $\frac{dV}{dt} \leq 0$. Additionally, $\frac{dV}{dt} = 0$ if and only if $x = y = 1, a = b = c = e$. Using the LaSalle's [30] extension to Lyapunov's method, the equilibrium P_* is globally asymptotically stable.

5. Simulations

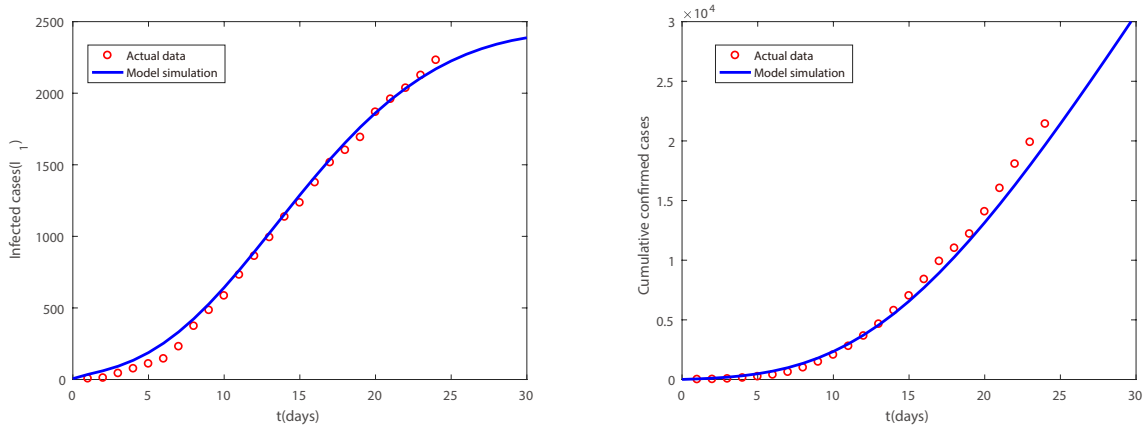
In this paper, the number of newly reported cases of COVID-19 in Chongqing between November and December 2022 was obtained from the Chongqing Municipal Health Planning Commission [31]. The Chongqing Municipal Center for Disease Control and Prevention [32] sequenced the viral genes of the infected persons, and the results showed that this Chongqing outbreak was mainly caused by the Omicron variant strain, and the incubation period of the Omicron variant virus was about 3.86 days. Because the state gradually relaxed the control of the epidemic, epidemic data were no longer collected on a daily basis in various places. In order to ensure the reasonableness of the data analysis, the data used in this paper were from November 1, 2022 to November 25, 2022. Since the epidemic mainly occurred in the main urban area of Chongqing, this paper adopted the data of the main urban area, so the total number of people was $N = 10,343,100$, the daily increase in the population was approximately 310 people, and the natural mortality rate was 2.203×10^{-5}. There was not a single case of death due to the disease in this outbreak: the case fatality rate was 0. The number of asymptomatic infected people was about 10 times of the number of diagnosed infected people; the disease could be cured in about 7 days after isolation treatment [33]. Based on the mathematical model and specific data, the model was fitted to the actual data and parameters using the least squares method. The parameters and initial values were estimated in Tables 1 and 2. Moreover, Figure 1 shows that the simulation results were consistent with the actual data, thus verifying the accuracy of the model.

Table 1. The parameters of system (2.1).

Parameter	Value	Source	Parameter	Value	Source
Λ	310	[33]	d	2.203×10^{-5}	[33]
β_1	2.4724×10^{-6}	Fitted	β_2	1.5258×10^{-7}	Fitted
β_W	1.982×10^{-9}	Fitted	ρ	0.1325	Fitted
σ	0.14	Fitted	p	0.12	[32]
μ	0	[32]	τ	1/3.86	[34]
k_1	0.6	Fitted	k_2	0.5	Fitted
γ_1	0.0714	Data	γ_2	0.0714	Data
ϕ	0.1429	Data	δ	0.5	[26]
λ_1	0.2	[26]	λ_2	0.2	[26]

Table 2. Initial values in Chongqing.

Initial values	Mean	Source
$S_1(0)$	7,240,170	Estimated
$S_2(0)$	3,102,930	Estimated
$E(0)$	1400	Fitted
$I_1(0)$	6	Data
$I_2(0)$	0	Data
$Q(0)$	0	Data
$R(0)$	0	Data
$W(0)$	0	Estimated



(a) (b)

Figure 1. The fitting results of the system (2.1) with real data from November 1, 2022 to November 25, 2022 in Chongqing.

The basic reproduction number R_0 is an indicative factor to consider whether the virus can be epidemic or not; therefore, we analyzed the sensitivity of the basic reproduction number of the system (2.1) to find out the key factors that affects the spread of COVID-19, so that effective measures can be taken to control the spread of the disease. Based on the parameter values in Table 1, we found that the basic reproduction number $R_0 = R_1 + R_2 + R_3$ was greater than 1, which meant that the disease would persist. In addition, as can be seen from Table 3, R_1 and R_2 were much larger than R_3, which implied that the probability of being infected by confirmed and asymptomatic infected persons was very high, while the probability of being infected by viruses in contaminated environments was low.

Table 3. The value of the basic reproduction number.

R_1	R_2	R_3	R_0
0.87129	0.46331	0.0026868	1.3373

We used a method based on Latin hypercube sampling to quantify the uncertainty and sensitivity of all model parameters. As shown in Figure 2, a positive partial rank correlation coefficient (PRCC) value indicated that an increase in the parameter led to an increase in R_0, while a negative value showed that an increase in the parameter decreased R_0.

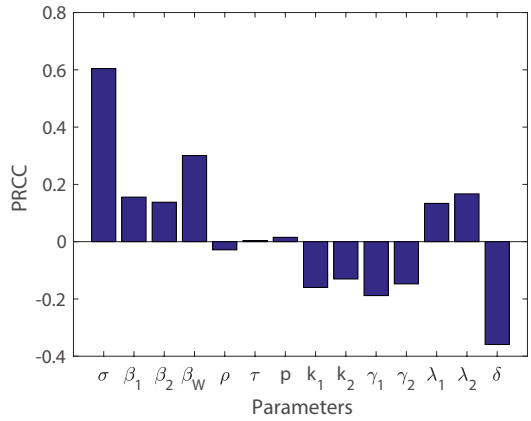
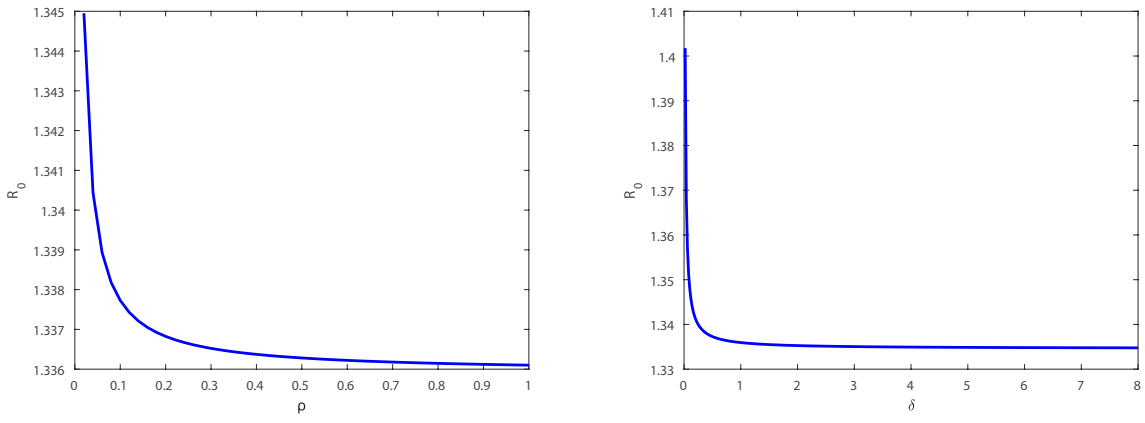


Figure 2. PRCCs of R_0 performed on model parameters.

If there were no infected individuals in a certain area for a long time, the public would relax their vigilance against the epidemic and lack self-protection measures. Once an infected person appeared, people would receive information through media channels and strengthen their prevention and control awareness, thereby transitioning from unconscious susceptible individuals to conscious susceptible individuals. Using the Matlab software, we drew a curve between the parameter ρ and the basic regeneration number R_0, as shown in Figure 3(a). It can be seen that as the immigration rate increased, the basic regeneration number R_0 decreased; however, the effect was not significant, so only having self-protection awareness and not taking effective prevention and control measures when the epidemic breaks out could not effectively control the epidemic.

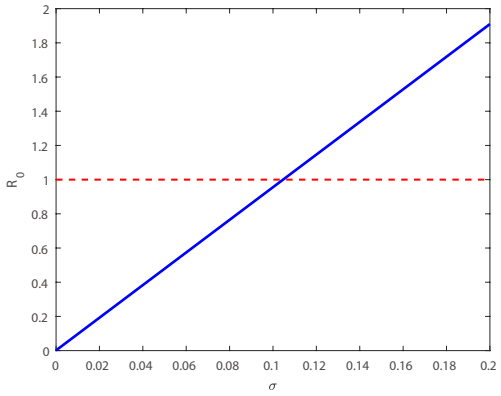


(a) (b)

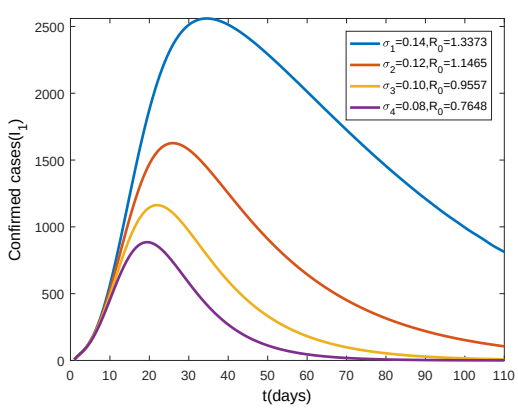
Figure 3. (a) The relationship between parameter ρ and R_0. (b) The relationship between parameter δ and R_0.

Through Figure 3(b), found that increasing the clearance rate δ of viruses in polluted environments could reduce the spread of viruses. For example, attention should be paid to hazardous areas such as sewage outlets, toilets, bathtubs in the restroom, and floor drains; if there is an infected person in the downstairs residents, the virus may spread through aerosols to the upstairs and downstairs, and it is necessary to avoid staying on the balcony for too long. Isolation areas and hospitals should strengthen infection control measures to avoid cross infection. In addition, when taking any value within the range shown in Figure 3(b), R_0 was greater than 1, which means that the disease would persist.

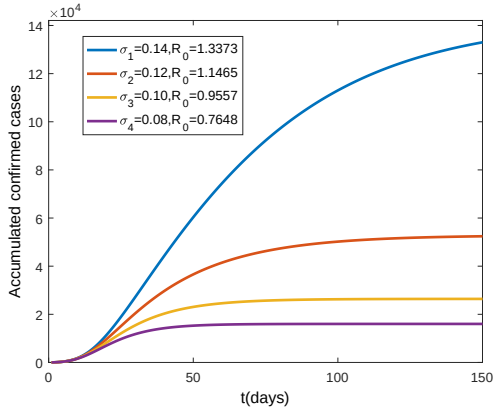
In Figure 4(a), we found that there was a positive correlation between the regulatory factors for the contact rate σ between conscious susceptible and infected individuals and the basic number of regenerations R_0. Figure 4(b),(c) show the number of confirmed infections and the number of cumulative infections for different parameters. When the moderating factor was smaller, there were fewer confirmed infections and fewer cumulative infections; when the moderating factor was larger, there were more infections and isolates. This suggests that when the moderating factor is smaller (i.e., the more effective the individual's self-protection measures are), the lower the probability of being infected, and the spread of the epidemic could be effectively controlled.



(a)



(b)



(c)

Figure 4. (a) The relationship between parameter R_0 and σ. (b) Simulations of the confirmed cases(I_1) with respect to parameter σ. (c) Simulations of the accumulated confirmed cases with respect to parameter σ.

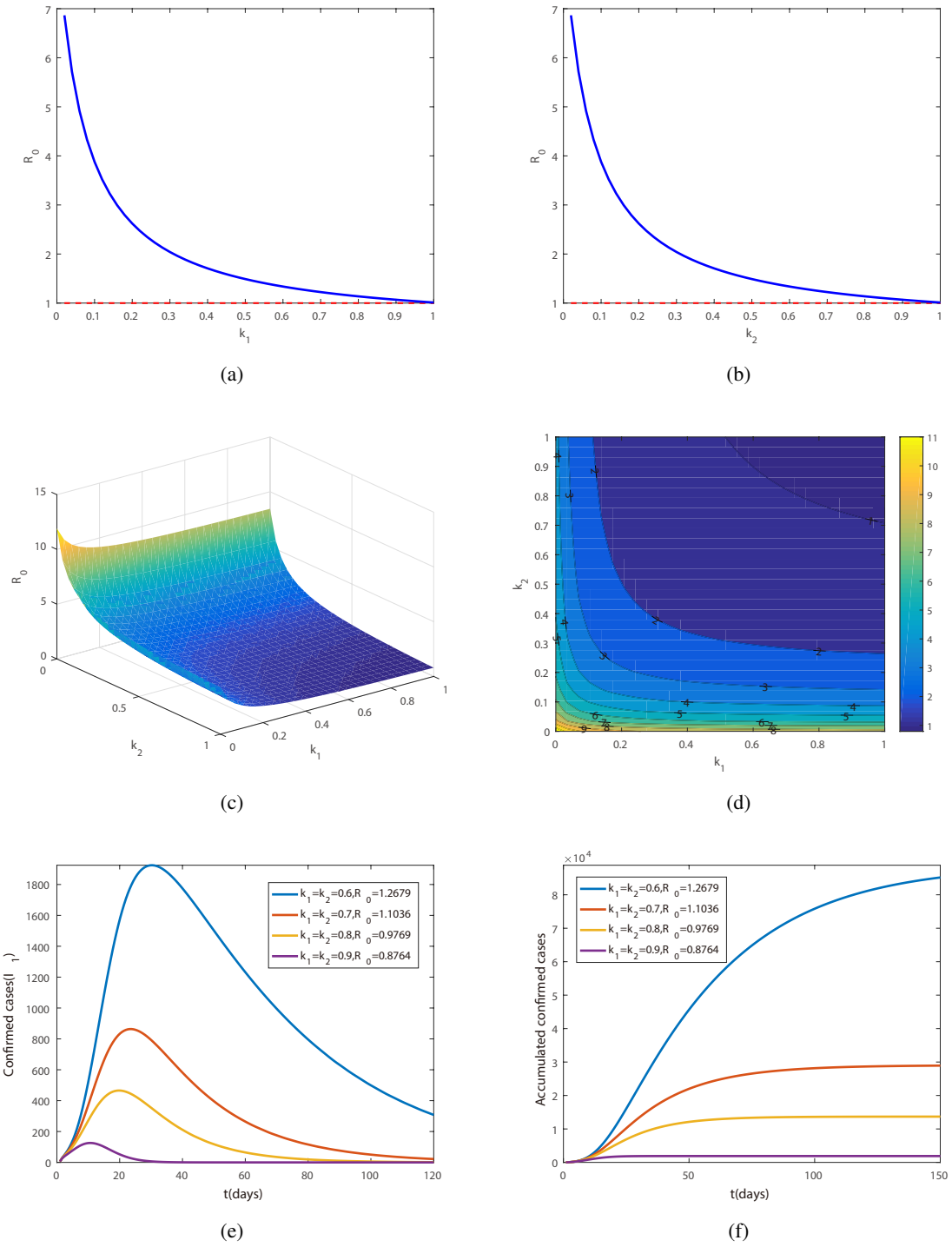
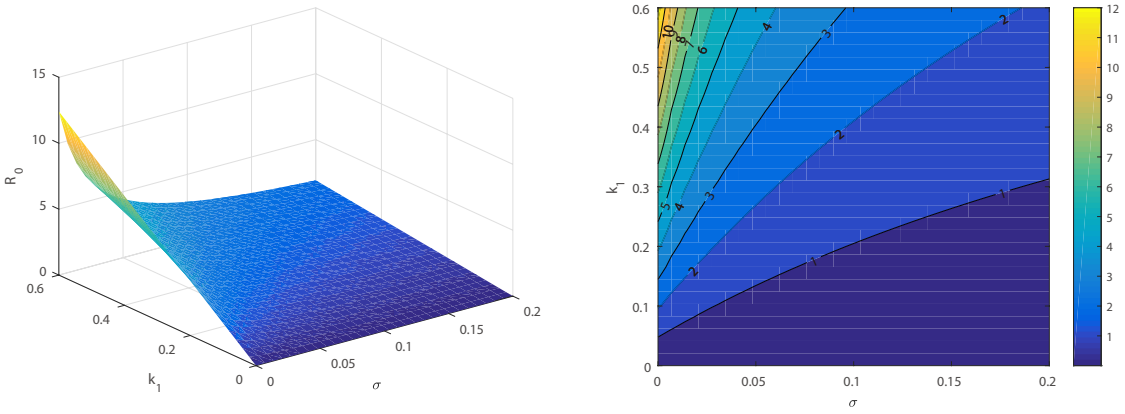


Figure 5. (a) The relationship between parameter R_0 and k_1.(b) The relationship between parameter R_0 and k_2. (c) R_0 in terms of parameters k_1 and k_2. (e) Simulations of the confirmed cases(I_1) with respect to parameter k_1 and k_2. (f) Simulations of the accumulated confirmed cases with respect to parameter k_1 and k_2.

Figure 5(a),(b) show the negative correlation between the isolation rate and the basic regeneration

number R_0, respectively. As the isolation rate increased, the R decreased, but the effect was not significant. This is because, in this outbreak, both confirmed and asymptomatic carriers were infectious and could be detected by nucleic acid antigen detection. Therefore, we should isolate both confirmed and asymptomatic patients. Through R_0 in terms of the parameters k_1 and k_2 in Figure 5(c), we can clearly show that isolating both can more effectively control the epidemic. We chose different comparison values to demonstrate the importance of implementing isolation measures for infected individuals to control the spread of the disease (in Figure 5(e),(f)).

Due to the fact that multiple measures can be taken equally when implementing specific measures, the advantages and disadvantages of using both self-protection and isolation measures were simultaneously simulated in Figure 6. From the figure, it can be seen that the combination of self-protection and isolation measures had a significant effect on controlling the epidemic.



(a) (b)

Figure 6. R_0 in terms of parameters σ and k_1.

6. Conclusions

This paper focused on a COVID-19 model with self-protection and isolation measures affected by viruses in the environment. The model extended an existing model by taking the effects of self-protection measures, isolation measures, and viruses in the contaminated environment on disease transmission into account. By calculating the basic regeneration number and the equilibrium point of the model, it was proven that the disease-free equilibrium point P_0 was globally asymptotically stable when $R_0 < 1$, and the endemic equilibrium point P_* was globally asymptotically stable when $R_0 > 1$. A numerucak analysis revealed that viruses in the environment could affect the spread of disease, but the removal of viruses in the environment alone was not effective to control the spread of epidemics. At the same time, it was found that strengthening the isolation of confirmed infected and asymptomatic infected could effectively control the spread of the epidemic. However, the actual situation is complex and variable, and it is impossible to effectively implement isolation measures. By analyzing the parameter σ, it is obvious that effective self-protection , such as correctly wearing masks, reducing activities in crowded places, and maintaining good hygiene habits, can more effectively reduce the risk of being infected and quickly contain the epidemic; at the same time, it can be more conducive to the practical application of

the epidemic, and it is an effective and low-cost prevention and control measures.

In this article, we only considered the case of being transferred to centralized isolation after infection; we did not consider the case of self-imposed home isolation. Due to the highly contagious nature of the virus, there will be a large increase in the number of patients at a given time, and we have ignored the limited availability of medical resources and the time lag in treatment. Therefore, if self-isolation and limited hospital resources can be taken into account in the model, this will make the model more complete as well as more realistic. We have taken this as future work.

Use of AI tools declaration

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

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Conflict of interest

The authors declare there is no conflict of interest.

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