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A class of delay SIQR-V models considering quarantine and vaccination: Validation based on the COVID-19 perspective

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\textbf{ABSTRACT}

To contain the novel SARS-CoV-2 (COVID-19) spreading worldwide, governments generally adopt two measures: quarantining the infected people and vaccinating the susceptible people. To investigate the disease latency’s influence on the transmission characteristics of the system, we establish a new SIQR-V (susceptible-infective-quarantined-recovered-vaccinated) dynamic model that focuses on the effectiveness of quarantine and vaccination measures in the scale-free network. We use theoretical analysis and numerical simulation to explore the evolution trend of different nodes and factors influencing the system stability. The study shows that both the complexity of the network and latency delay can affect the evolution trend of the infected nodes in the system. Still, only latency delay can destroy the stability of the system. In addition, through the parameter sensitivity analysis of the basic reproduction number, we find that the effect of the vaccination parameter $\alpha$ on the basic reproduction number $R_0$ is more significant than that of transmission rate $\beta$ and quarantine parameter $\sigma$. It shows that vaccination is one of the most effective public policies to prevent infectious diseases’ spread. Finally, we calculate the basic reproduction numbers that are greater than one for Germany and Pakistan under COVID-19 and validate the model’s effectiveness based on the disease data of COVID-19 in Germany. The results show that the changing trend of the infected population in Germany based on the SIQR-V model is roughly the same as that reflected by the actual epidemic data in Germany. Therefore, providing suggestions and guidance for treating infectious diseases based on this model can effectively reduce the harm caused by the outbreak of contagious diseases.

\textbf{Introduction}

Applying dynamic models to study the transmission mechanism of infectious diseases in the host population can reveal the law of disease transmission, predict the time of disease outbreak, and evaluate the best strategy for disease control. According to the different states of individuals, researchers construct different dynamic models consequently and split the established dynamic models into two standard models: SIR (susceptible-infected-recovered) and SIS (susceptible-infected-susceptible) models. They further researched infectious diseases’ outbreak mode and transmission law \cite{1-3}. For example, Venkatasen et al. \cite{4} used the SIR model and actual COVID-19 data published by the government to predict the outbreak rule of COVID-19 in India. They concluded that public health policies in countries with a high incidence of COVID-19 could control its spread. Based on the classical SIR model, Chekroun et al. \cite{5} studied the global asymptotic behavior of the SIR epidemic model with the age structure of infection. Arfan et al. \cite{6} established an improved SI model to predict the spread of COVID-19 in Pakistan. They argue that increasing quarantine parameters will lead to an exponential decline in the virus following strict precautions. Han et al. \cite{7} proposed a stochastic SIS epidemic model with a saturated incidence rate to describe the transmission of HIV/AIDS. On this basis, they were seeking effective ways to prevent AIDS. These studies provide some useful references for government departments to grasp the characteristics of disease transmission.

To make the mathematical model create realistically, the researchers further extend the standard model in the scale-free network. Given the complex nature of the spread of infectious diseases, to prevent the further spread of the epidemic, infected people are often placed in designated locations and temporarily avoid contact with surrounding...
people. This method facilitates the treatment of patients and reduces the chance of transmission of infectious diseases. Therefore, in establishing the transmission dynamics model, scholars gradually begin to focus on the intervention procedures that affect the transmission of infectious diseases. Such as Ruschef et al. [8] used quarantine measures to control the spread of infectious diseases, mainly studied the best duration SIQR model in quarantine. Cao et al. [9] used the SIQR model with quarantine to calculate the system’s different transmission thresholds to regulate the disease dynamics. Liu et al. [10] proposed the stochastic multi-population SIQR model and pointed out that the system had random weak stability, which provided sufficient conditions for disease outbreaks. Zuzek et al. [11] focused on the effects of failed or delayed quarantine measures on human lives. Huang et al. [12-15] also paid attention to the SIQR model with quarantine and explored the law of infectious disease outbreaks on this basis. Moreover, Sahoo et al. [16] took COVID-19 as the research object, regarded quarantine as an effective preventive measure in the absence of a vaccine, and proposed a mathematical model based on quarantine to describe disease transmission dynamics. Memon et al. [17] created a class of SEIQR model and verified the model with COVID-19 epidemic data in Pakistan. They believed that the contact between individuals and the extension of the quarantine period was the most effective strategies to combat infectious diseases. Quarantine measures play an essential role in preventing human diseases and epidemics, such as smallpox, tuberculosis, SARS, and the current outbreak of COVID-19.

In addition to measures such as quarantine of patients for infectious diseases that are difficult to prevent and highly transmissible, vaccination methods represented by vaccination are also effective measures widely adopted by various countries. Vaccination gives the recipient immunity against a specific or vaccine-similar pathogen, making the recipient more resistant to the disease. We can control the spread of many infectious diseases through effective vaccination. Therefore, vaccination is one of the most effective public policies to prevent the spread of infectious diseases [18]. Peng et al. [19] built the SIV model, which combined public vaccination with personal protection, and provided a more comprehensive approach to eradicate infectious diseases. Lv et al. [20] established a SIVS model based on the variability of population structure and calculated the optimal control strategy for vaccination. Hosseini et al. [21] introduced vaccination into the field of computers, proposed the SEIRS-V model of network malware transmission and analyzed the effectiveness of anti-virus software. Some researchers combined vaccination and quarantine to create a more realistic model and studied the global dynamics of the system [22-27].

The perfection of the infectious disease model above mainly focuses on improving the individual state. However, we need to consider more factors. For example, when the susceptible individuals are transformed into infected individuals through contact, due to the differences in infectious diseases and personal physical conditions, the susceptible individuals will be infected at different times, and there will be a time delay. More and more scholars introduce the time delay factor into the propagation dynamics model to solve this problem in recent years. For example, Li et al. [28] proposed a new vector bias model with a time delay to study the law of malaria transmission. The vector bias was the difference between the probability of the virus choosing the infectious host. They proved that the time delay has a significant influence on the direction and stability of Hopf bifurcation. Liu et al. [29] believed that time delay existed in the transportation process and proposed a periodic SIRS model with time delay and the result is that the disease will be eradicated. They concluded that the basic reproduction number did not necessarily increase with the increase of time delay. Li et al. [30] proposed a class of SEIQR infective disease model, in which the time lag between the latent state and the infected state was considered as the incubation period of the virus. Huang et al. [31,32] also further proved that time delay significantly influences the system properties. Therefore, adding time delay to the propagation model can more fully fit the actual scenario, which is helpful for epidemic supervisors to analyze the propagation law of infectious diseases in the real world. It could also make recommendations for curbing outbreaks of infectious diseases and keeping people safe.

At the same time, to prove the model’s effectiveness in solving real problems after mathematical modeling based on the law of infectious disease transmission, some scholars chose real scenarios to verify the model. Using data on HIV-1 positive cases reported by the Turkish Ministry of Health in 2016, Farouk et al. [33] calculated the basic reproduction number based on the model created and concluded that HIV transmission in Turkey would continue. Fatima et al. [34] focused on the influence of information on the transmission and control of MERS-COV in the Middle East. They concluded that, to some extent, the more media reports in a specific population, the fewer people will be infected. At present, as the most representative infectious disease, COVID-19 is spreading widely almost everywhere globally and causing great panic among people. To explore the complex dynamics of COVID-19, scholars employed different models and research methods and conducted simulations using COVID-19 statistics. Rahim et al. [35] used the SIR model to study the impact of migration rates on the COVID-19 epidemic, using accurate data from Wuhan, China, in 2020 to generate two different sets of migration parameter values to simulate the results. The authors believed that avoiding unnecessary movement of people will significantly help to reduce or control the spread of the epidemic. Moussa et al. [36] proposed a new nonlinear fractional model in the sense of Caputo, which divided infected persons into detected and undetected categories, and took Algeria as an example to study and simulate the dynamics of COVID-19. Khan et al. [37] studied the existence results of the fractional COVID-19 model using fixed point theorem and conducted stability analysis using the HUS method. Bastos et al. [38] argued that existing data on COVID-19 in Brazil were underestimated mainly due to a lack of large-scale testing.. Therefore, they used the SIR model to include underreporting and population responses to public health policies explicitly. Nie et al. [39] paid attention to information changes and established a new dynamic SEIR model with information entropy to simulate the epidemic situation in China. They confirmed that the improved model could effectively predict the peak and scale of the COVID-19 outbreak using COVID-19 epidemic data. In addition, according to the modeling results, Cleo et al. [40–44] verified the model based on the accurate infectious disease data and predicted the actual transmission trend.

The existing studies show that although propagation dynamics on complex networks have made significant progress, there are also several shortcomings. 1) The existing communication dynamics model is improved on the original standard model. The researchers begin to consider introducing people with quarantine or vaccination status in the problem of individual status classification. However, few models present both states at the same time. With the continuous change of climate and environment, there are more and more types of infectious diseases. The model of introducing these two states can more accurately explore the general law of infectious disease transmission. 2) Some scholars have introduced the time delay factor into the standard model considering the latent period of infectious diseases. However, due to the model’s simplicity, the conclusions drawn from this model tend to deviate from the actual situation. Introducing time delay into a more realistic model can provide a practical reference for government departments to formulate preventive measures. 3) At present, most scholars have begun to verify the infectious disease model based on actual data. However, the dynamic model in such studies is often in the state of hypothesis and deriving the theoretical part. Hence, it is necessary to strengthen the theoretical proof and verify its validity based on actual data.

People have more and more frequent contact with economic globalization, enabling infectious diseases to spread rapidly worldwide. Since Barabasi [45] discovered the scale-free nature of social networks, many infectious disease models have been extended to networks with scale-free properties further to understand the transmission behavior in the real world. Zhu et al. [46] established the SIS model on a scale-free
network and discussed the stability of the system’s equilibrium by using the Lyapunov function. Yang et al. [47] believed that the infection behavior caused by contact between populations was not uniform, which was as heterogeneous as the spread of epidemic disease, so they introduced the idea of the complex network into the mathematical model. Li et al. [48] further explored the influence of network structure on time-varying control. Descriptions from the above literature show that the real-world network is scale-free. The scale-free network with power-law degree distribution is more consistent with the transmission law of infectious diseases in the real world. Therefore, in this context, the study of system dynamics is more meaningful.

Given this, to better reflect the reality, we introduce two-node states of quarantine and vaccination into the SIR epidemic model on scale-free networks and create a class of SIQR-V model. To better understand the dynamics of the spread of infectious diseases, we consider realistic incidence and time lag factors in the model. Employing the Lyapunov function, we prove the system’s stability with a critical threshold $R_0$ as the boundary condition and use numerical simulation to verify the correctness of the theoretical research. Numerical simulation is applied to verify the validity of the theoretical study, and the model’s validity is proved based on the COVID-19 epidemic data in Germany in 2021.

**SIQR-V model**

In the model, we divide the total population $N$ into $n$ subgroups according to the degree of nodes. Namely, $N = N_1 + N_2 + \ldots + N_n$. Furthermore, each subgroup is divided into five compartments, $S_k(t)$, $I_k(t)$, $Q_k(t)$, $R_k(t)$ and $V_k(t)$ represent the density of susceptible, infective, quarantined, recovered, vaccinated nodes with degree $k$ at time $t$, respectively. Among them, we want to emphasize that the recovered compartment $R_k(t)$ represents the population that produces antibodies in the body, that is, the people who recover after treatment and those who make antibodies stably after vaccination. Compartment $V_k(t)$ represents people who have been vaccinated but have not yet developed antibodies. The relationship between $N_k(t)$ and the five existing states of the system is as follows.

$$N_k(t) = S_k(t) + I_k(t) + Q_k(t) + R_k(t) + V_k(t)$$

where $k$ is defined as the number of nodes connect to nodes in the network, positive integer $n$ is the maximum degree on the scale-free network, where $k = 1, 2, \ldots, n$. Fig. 1 shows the state transition diagram of the SIQR-V model.

From the state transition diagram, we can obtain the following ordinary differential equations.

$$\begin{align*}
\frac{dS_k(t)}{dt} &= \Lambda - \beta_k S_k(t) \frac{\Theta(t - \tau)}{1 + \beta \Theta(t - \tau)} - \mu S_k(t) - \alpha S_k(t) \\
\frac{dI_k(t)}{dt} &= \beta_k S_k(t) \frac{\Theta(t - \tau)}{1 + \beta \Theta(t - \tau)} - \mu I_k(t) - \sigma I_k(t) \\
\frac{dQ_k(t)}{dt} &= \sigma I_k(t) - \epsilon Q_k(t) - \mu Q_k(t) \\
\frac{dR_k(t)}{dt} &= \epsilon Q_k(t) + \gamma V_k(t) - \mu R_k(t) \\
\frac{dV_k(t)}{dt} &= \alpha S_k(t) - \nu V_k(t) - \mu V_k(t)
\end{align*}$$

(2.2)

The parameters $\Lambda, \beta, \eta, \alpha, \sigma, \delta, \gamma, \epsilon$ are all positive constants. Based on this model, we consider $\Lambda$ is the constant birth rate and suppose the newborns are all susceptible, $\mu$ is the death rate. $\beta$ is the transmission rate of contact infections. $\alpha$ is the vaccination rate. Suppose the susceptible individual is related to the infected individual. In that case, the susceptible nodes are infected at the rate of $\beta$ to become infected nodes and at the rate of $\alpha$ to be transformed into vaccination nodes. Infected nodes are quarantined at a rate of $\sigma$. The quarantine nodes are healed at the rate of $\epsilon$. The vaccination nodes are transformed into the rehabilitation nodes at the rate of $\gamma$. Due to the difference in the time taken by each susceptible person to be infected, we introduce the concept of time delay and use parameter $\tau$ to describe the average infection delay of infectious diseases. $\Theta(t - \tau)$ represents the average number of density of connections between an individual from one node to another infected node. $\lambda$ represents the strength of an individual’s intention to avoid contact due to psychological factors during infectious diseases. We can express $\Theta(t - \tau)$ as

$$\Theta(t - \tau) = \sum_{j=1}^{n} \frac{p(j|k)}{\langle k \rangle} \lambda \frac{\kappa}{\mu}, \quad k = 1, 2, \ldots, n.$$  

(2.3)

where $p(j|k)$ is the conditional probability of a node of degree $k$ connecting to a point of degree $j$ and

$$p(j|k) = \frac{\kappa j}{\langle k \rangle}, \quad j = 1, 2, \ldots, n.$$  

(2.4)

Therefore, by expanding (2.3), we get

$$\Theta(t - \tau) = \sum_{j=1}^{n} \frac{p(j|k) \lambda}{\langle k \rangle} \lambda \frac{\kappa}{\mu}, \quad j = 1, 2, \ldots, n.$$  

(2.5)

Suppose the initial conditions of the system (2.2) with the form

![Fig. 1. The state transition diagram of the SIQR-V model.](image-url)
\[ S_i(\eta) = \phi_{x_i}(\eta), I_i(\eta) = \phi_{y_i}(\eta), Q_i(\eta) = \phi_{z_i}(\eta), R_i(\eta) = \phi_{\omega_i}(\eta), V_i(\eta) = \phi_{\tau_i}(\eta), \phi_{x_i}(\eta) \geq 0, \eta \in (-\epsilon, 0), \phi_{y_i}(0) > 0, t \in (1, 2, 3, 4, 5), \]

(2.6)

Here, \( \phi_{x_i}, \phi_{y_i}, \phi_{z_i}, \phi_{\omega_i}, \phi_{\tau_i} \in C \), such that \( \phi_{x_i}(0) = 0, t \in (1, 2, 3, 4, 5) \) for all \( \eta \in (-\epsilon, 0) \), and \( C \) denotes the Banach space \( C((-\epsilon, 0), R^m) \) of continuous functions mapping the interval \((-\epsilon, 0)\] into \( R^m \) and designating the norm of an element \( C \) by \( \| \phi_{x_i} \| = \sup_{\eta \in (-\epsilon, 0)} |(\phi_{x_i}(\eta) - \phi_{x_i}(\eta))| \).

Positive characterization and basic reproduction number of the model

**Lemma 3.1.** If \( S_i(0) > 0, I_i(0) > 0, Q_i(0) > 0, R_i(0) > 0, V_i(0) > 0, \)

then the system (2.2) with this initial condition has a positive solution \( S_i(t), I_i(t), Q_i(t), R_i(t), V_i(t) \) under condition \( t > 0, r > 0, k = 1, 2, \ldots, n \).

**Proof.** We will prove that for any \( t > 0, k = 1, 2, \ldots, n \), there exists \( S_i(t) > 0 \). Assuming that \( S_i(t) \) is not always positive for \( t > 0, r > 0, k = 1, 2, \ldots, n \), there exists a small enough \( \epsilon > 0 \), according to the continuity of \( S_i(t) \), such that \( S_i(t) > 0 \) is true for \( t \in (0, \epsilon) \). Besides, there is \( j = 1, 2, \ldots, n, S_i(t_1) = 0 \) for \( t_2 \geq \epsilon > 0 \) and for any \( t \in (0, t_2), S_i(t) > 0 \).

(3.1)

Hence

\[ S_i(t) > S_i(0)e^{-(\mu_{I_i} + \alpha_{I_i})t}, t \in (0, t_1), 1 = 2, \ldots, n, j = 1, 2, \ldots, n. \]

(3.2)

Notice that \( I_i(0) > 0, k = 1, 2, \ldots, n \), according to the continuity of \( I_i(t) \), there is a small enough \( \epsilon > 0 \), such that \( I_i(t) > 0 \) is true for \( t \in (0, \epsilon) \), \( k = 1, 2, \ldots, n \). At the same time, for any \( j = 1, 2, \ldots, n, I_i(t_1) > 0 \) is true for \( t_2 \geq \epsilon > 0 \) and for any \( t \in (0, t_2), k = 1, 2, \ldots, n, I_i(t) > 0 \). Combine with the second formula (2.2), we reach

(3.3)

Further we get

(3.4)

Notice that \( Q_i(t) > 0, k = 1, 2, \ldots, n \), according to the continuity of \( Q_i(t) \), there is a small enough \( \epsilon > 0 \), such that \( Q_i(t) > 0 \) is true for \( t \in (0, \epsilon) \), \( k = 1, 2, \ldots, n \). Meanwhile, for any \( j = 1, 2, \ldots, n, \) \( Q_i(t_1) = 0 \) is true for \( t_2 \geq \epsilon > 0 \) and for any \( t \in (0, t_2), k = 1, 2, \ldots, n, Q_i(t) > 0 \). Combine with the third formula (2.2), we get

(3.5)

Go a step further

(3.6)

Notice that \( R_i(t) > 0, k = 1, 2, \ldots, n \), according to the continuity of \( R_i(t) \), there is a small enough \( \epsilon > 0 \), such that \( R_i(t) > 0 \) is true for \( t \in (0, \epsilon) \), \( k = 1, 2, \ldots, n \). Meanwhile, for any \( j = 1, 2, \ldots, n, R_i(t_1) = 0 \) is true for \( t_2 \geq \epsilon > 0 \) and for any \( t \in (0, t_2), k = 1, 2, \ldots, n, R_i(t) > 0 \). Combine with the fourth formula (2.2), we obtain

(3.7)

Further

(3.8)

Notice that \( V_i(t) > 0, k = 1, 2, \ldots, n \), according to the continuity of \( V_i(t) \), there is a small enough \( \epsilon > 0 \), such that \( V_i(t) > 0 \) is true for \( t \in (0, \epsilon) \), \( k = 1, 2, \ldots, n \). Meanwhile, for any \( j = 1, 2, \ldots, n, V_i(t_1) > 0 \) is true for \( t_2 \geq \epsilon > 0 \) and for any \( t \in (0, t_2), k = 1, 2, \ldots, n, V_i(t) > 0 \). Combine with the fifth formula (2.2), we reach

(3.9)

Then

(3.10)

Let \( t' = \min(t_1, t_2, t_3, t_4, t_5) \), given the continuity of \( I_i(t), Q_i(t), R_i(t), V_i(t) \), \( j = 1, 2, \ldots, n \), we know \( I_i(t'), Q_i(t'), R_i(t'), V_i(t') > 0 \) according to the first formula (2.2). Thus, \( S_i(t') < S_i(t) > 0, t \in (t' - \phi, t') \), \( j = 1, 2, \ldots, n \). Where \( \phi \) is any positive constant, the conclusion is inconsistent with the assumption, so for \( t > k = 1, 2, \ldots, n \), there is \( S_i(t) > 0 \). Similarly, \( I_i(t), Q_i(t), R_i(t), V_i(t) \) hold for any \( t > k = 1, 2, \ldots, n \), where we prove Lemma 3.1.

**Lemma 3.2.** Positive invariant for system (2.2) is

(3.11)

**Proof.** Add the five differential equations of (2.2) and we can get

(3.12)

Integrating the above formula, we obtain

(3.13)

where \( N_k(0) = 1, 2, \ldots, n \) represents the initial value of the degree value \( \kappa \) at the vertex time \( t = 0 \). So

(3.14)

Therefore, the bounded region contains all feasible solutions of the system (2.2).

Now to calculate the basic reproduction number of the system, let the right-hand side of the derivative of (2.2) be equal to 0.

\[
\begin{align*}
\frac{dS_i(t)}{dt} &= \Lambda - \beta k S_i(t) \frac{\Theta(t - r)}{1 + \alpha_{S_i}(t - r)} - \mu S_i(t) - \alpha S_i(t) = 0, \\
\frac{dI_i(t)}{dt} &= \beta (t - r) I_i(t) \frac{\Theta(t - r)}{1 + \alpha_{S_i}(t - r)} - \mu I_i(t) - \alpha I_i(t) = 0, \\
\frac{dQ_i(t)}{dt} &= \alpha I_i(t) - \epsilon Q_i(t) - \mu Q_i(t) = 0, \\
\frac{dR_i(t)}{dt} &= \epsilon Q_i(t) + \gamma V_i(t) - \mu R_i(t) = 0, \\
\frac{dV_i(t)}{dt} &= \alpha S_i(t) - \gamma V_i(t) - \mu V_i(t) = 0.
\end{align*}
\]

Where \( k = 1, 2, \ldots, n \), note that the disease-free equilibrium \( E^0 \) satisfies \( I_0 = 0 \). Therefore, there is a disease-free equilibrium point \( E^f = (s_i^0, r_i^0, q_i^0, r_i^0, v_i^0) \), where \( s_i^0 = \frac{\Lambda}{\mu + \alpha}, r_i^0 = \frac{\alpha \gamma}{\mu + \alpha + \gamma}, q_i^0 = \frac{\epsilon \gamma}{\mu + \alpha + \gamma}, v_i^0 = \frac{\alpha \gamma}{\mu + \alpha + \gamma} \).

(3.15)

(3.16)

At the same time, according to the initial condition \( S_i(t) + I_i(t) + Q_i(t) + R_i(t) + V_i(t) = 1 \). Various nodes in the equilibrium state have
the following relations
\[ L_i(t) = \frac{\beta k}{(\sigma + \mu)(1 + \lambda \Theta)} S_i(t), \]
\[ Q_i(t) = \sigma \frac{\beta k}{(\sigma + \mu)(1 + \lambda \Theta)} S_i(t), \]
\[ R_i(t) = \frac{\sigma \beta k}{(\sigma + \mu)(1 + \lambda \Theta)} + \frac{\gamma a}{\mu(\sigma + \mu)} S_i(t), \]
\[ V(t) = \frac{\alpha}{(\gamma + \mu)} S_i(t), \]
(3.17)

We obtain the following autonomous equation concerning the time function
\[ \Theta = \sum_{j=1}^{n} \frac{\Lambda \beta k p(j)}{(\sigma + \mu)(a + \mu)/(k)} \]
(3.18)

We define the above formula as function \( f(\Theta) \),
\[ \Theta = 0 \] is a solution to this and \( f(1) < 1 \). If there is a nontrivial solution in the interval \( 0 < \Theta < 1 \), the following inequality must be satisfied
\[ \frac{\partial f(\Theta)}{\partial \Theta} = \frac{\Lambda \beta k (\sigma + k)}{(\sigma + \mu)(a + \mu)/(k)} > 1 \]
(3.20)

Therefore, there is a disease transmission equilibrium point in system (2.2). We can calculate the disease transmission threshold, the basic reproduction number \( R_0 \)
\[ R_0 = \frac{\Lambda \beta k}{(\sigma + \mu)(a + \mu)/(k)} \]
(3.21)

In addition, the basic reproduction number of the model can also be obtained by using the next-generation matrix method:
\[ F = \begin{bmatrix} \sum_{k=1}^{n} k P(k) / (\sigma + k) & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ -\Lambda / (\mu + a) & -\Lambda / (\mu + a) & -\Lambda / (\mu + a) & -\Lambda / (\mu + a) \end{bmatrix} \]
(3.22)

As an essential parameter of the transmission of infectious diseases, the value of the basic reproduction number can determine the equilibrium state of the system. Namely, when \( R_0 < 1 \), there is disease-free equilibrium point \( E_0 \), in which the infected individual is 0, and there is no disease transmission on the network. When \( R_0 > 1 \), there is an endemic equilibrium point \( E^* \), at which time the disease tends to be stable, and an epidemic appears on the network.

**System stability**

According to the outbreak, prevalence, and degree of harm of infectious diseases, we can divide them into fixed categories: A class infectious diseases, B class infectious diseases, and C class infectious diseases. All types of infectious diseases will only develop into two final conditions or gradually disappear, where there is a disease-free equilibrium. Or develop into an epidemic, where there is an endemic equilibrium. This section will prove the stability of the system’s disease-free and endemic equilibrium point by equalizing birth and death rates.

**Theorem 4.1.** If \( R_0^* \leq 1 \), the disease-free equilibrium \( E_0 \) of the system is globally asymptotically stable.

**Proof.** When the basic reproduction number \( R_0 \) is less than 1, to prove its global stability, let \( g(k) = \frac{\beta k}{(\sigma + \mu)} \), \( k = 1, 2, \ldots, n \), define the Lyapunov function \( V(t) = V_1(t) + V_2(t) \),
\[ V_1(t) = \sum_{i=1}^{n} g(k) \left( S_i(t) - S_i^*(t) \right) \ln \frac{S_i(t)}{S_i^*(t)} + \sum_{i=1}^{n} g(k) I_i(t), \]
(4.1)

By taking the derivative of \( V_1(t) \) to \( t \), we get
\[ \frac{dV_1(t)}{dt} = \sum_{i=1}^{n} g(k) \left( \frac{S_i(t)}{S_i^*(t)} \right) \left( [\sigma + \mu] - \theta_k S_i(t) \left( 1 + \lambda \Theta \right) - \mu S_i(t) - \alpha S_i(t) \right) \]
(3.23)

Expanding (4.1) and substituting the disease-free equilibrium value into the equation can be simplified as
\[ \frac{dV_2(t)}{dt} = (\sigma + \mu) \left( \frac{\Theta(t) - \Theta(t)}{1 + \lambda \Theta(t) - \theta_k S_i(t)} - \mu I_i(t) - \alpha I_i(t) \right) \]
(4.2)

Now take the derivative of the function \( V_2(t) \) to \( t \), we get
\[ \frac{dV_2(t)}{dt} = (\sigma + \mu) \left( \frac{\Theta(t) - \Theta(t)}{1 + \lambda \Theta(t) - \theta_k S_i(t)} - \mu I_i(t) - \alpha I_i(t) \right) \]
(4.3)

Further, according to Eqn 2.5, the following relationship can be obtained
\[ \sum_{i=1}^{n} \frac{\beta k}{\lambda} (\sigma + \mu) I_i(t) = (\sigma + \mu) \Theta(t) \]
(4.4)

Since \( (\sigma + \mu) \Theta(t) < (\sigma + \mu) \Theta(t) \), the above can be contracted, we
can summarize the $V(t)$ as

$$
\frac{dV(t)}{dt} = \sum_{k=1}^{n} g(k) \left(1 - S_k(t) \right) \left(\sigma + \mu \right) \left(S_k(t) - S_k(t) \right) + \sum_{k=1}^{n} g(k) \left(\frac{\mu}{\alpha + \mu} I_k(t) \right) \frac{\Theta(t - \tau)}{1 + \lambda \Theta(t - \tau)} \\
- \left(\sigma + \mu \right) \sum_{k=1}^{n} g(k) L_k(t) + \left(\sigma + \mu \right) \frac{\Theta(t)}{1 + \lambda \Theta(t)} \left(1 - S_k(t) \right) \left(\sigma + \mu \right) \frac{\Theta(t - \tau)}{1 + \lambda \Theta(t - \tau)} \\
$$

$$
= \sum_{k=1}^{n} \left(1 - S_k(t) \right) \left(\mu S_k(t) - \mu S_k(t) \right) + \left(\sigma + \mu \right) \left(R_0 - 1 \right) \frac{\Theta(t - \tau)}{1 + \lambda \Theta(t - \tau)} + \left(\sigma + \mu \right) \frac{\Theta(t)}{1 + \lambda \Theta(t)} \left(1 - S_k(t) \right) \left(\sigma + \mu \right) \frac{\Theta(t - \tau)}{1 + \lambda \Theta(t - \tau)} \\
$$

(4.6)

Because of $S_k \leq S_k$, so $\sum_{k=1}^{n} g(k) \left(1 - S_k(t) \right) \left(\mu S_k(t) - \mu S_k(t) \right) \leq 0$. When $R_0 \leq 1$, $\left(\sigma + \mu \right) \left(R_0 - 1 \right) \frac{\Theta(t - \tau)}{1 + \lambda \Theta(t - \tau)} \leq 0$, therefore when $R_0 \leq 1$, $\frac{dV(t)}{dt} \leq 0$ exists. If and only if $S_k = S_k$, $\frac{dV(t)}{dt} = 0$. Therefore, according to LaSalle’s invariance principle of the time-delay system [49], when $R_0 \leq 1$, the disease-free equilibrium point $E_0$ of the system is globally asymptotically stable, that is, no matter what the initial density of infected persons is, the disease will eventually approach 0. There are no infected persons, which is an ideal state pursued by all researchers to prevent and treat infectious diseases.

**Theorem 4.2.** If $R_0 > 1$, the endemic equilibrium point $E^*$ for any $\tau \geq 0$ system is globally asymptotically stable.

**Proof.** When the threshold $R_0$ is greater than 1, to prove its stability, we consider that the endemic equilibrium point of the system is $E^* \left(S^*_1, I^*_1, Q^*_1, R^*_1, V^*_1, S^*_2, I^*_2, Q^*_2, R^*_2, V^*_2 \ldots, S^*_n, I^*_n, Q^*_n, R^*_n, V^*_n \right)$. Now the Lyapunov function $V_{A_1}$ and $V_{A_2}$ are defined as follows

$$
V_{A_1} = \int_{S^*_1}^{S^*_1(s)} H \left(\frac{S^*_1(s)}{Q^*_1(t)} \right) ds + I^*_1(t) - \int_{S^*_1}^{S^*_1(s)} \frac{H \left(\frac{S^*_1(s)}{Q^*_1(t)} \right)}{1 + \lambda} ds + \left(\sigma + \mu \right) \left(I^*_2(t) - \int_{S^*_2}^{S^*_2(s)} Q^*_2(t) ds \right) \\
V_{A_2} = \left(\sigma + \mu \right) I^*_1(t) \int_{t}^{h(t)} \frac{H \left(\frac{S^*_1(s)}{Q^*_1(t)} \right)}{Q^*_1(t)} ds \\
$$

(4.7)

Where $H(x) = x - 1 - \ln x$, obviously $H(x)$ is greater than 0. Thus, $V_{A_1}$ and $V_{A_2}$ are both greater than 0. Now the Lyapunov function $V_{A_1}(t)$ is defined as follows.

$$
V_{A_1}(t) = V_{A_1}(t) + V_{A_2}(t) \\
$$

(4.8)

By taking the derivative of $V_{A_1}(t)$ for $t$ and substituting the various elements of system (2.2) into it, we have

$$
\frac{dV_{A_1}(t)}{dt} = \left(1 - S^*_1(t) \right) \left(\mu \right) \frac{\Theta(t - \tau)}{1 + \lambda \Theta(t - \tau)} - \mu S^*_1(t) - \alpha S^*_1(t) + \left(\sigma + \mu \right) I^*_1(t) - \left(\sigma + \mu \right) I^*_1(t) \\
+ \left(\sigma + \mu \right) I^*_1(t) + \left(\sigma + \mu \right) \frac{I^*_1(t)}{I^*_1(t)} \left[1 - \frac{Q^*_1(t)}{Q^*_1(t)} \right] - \frac{\sigma + \mu}{\mu} \left[1 - \frac{Q^*_1(t)}{Q^*_1(t)} \right] (\sigma + \mu) Q^*_1(t) \\
$$

(4.10)

$$
= \left(1 - S^*_1(t) \right) \left(\mu + \alpha \right) \left(S^*_1(t) - S^*_1(t) \right) - \left(1 - S^*_1(t) \right) \left(\sigma + \mu \right) I^*_1(t) + \left(\sigma + \mu \right) I^*_1(t) - \left(\sigma + \mu \right) I^*_1(t) \\
$$

(4.10)
Now take the derivative of the function \( V_{k2}(t) \) with respect to \( t \). We have
\[
\frac{dV_{k2}(t)}{dt} = (\sigma + \mu)I^*_t(t) \left[ \frac{Q(t) - Q(t - t)}{Q_t(t)} + \ln Q(t - t) \right]
\]
(4.11)

Further, according to (4.8), we can obtain the expression of \( \frac{dV_{k2}(t)}{dt} = \frac{dV_k(t)}{dt} - \frac{dV_{k1}(t)}{dt} \) as
\[
\frac{dV_k(t)}{dt} = \left( 1 - \frac{S_2(t)}{S(t)} \right) (\mu + \sigma) (S_2(t) - S_1(t)) + (\sigma + \mu)I^*_t(t)
\]
\[
\left[ \frac{Q(t) - Q(t - t)}{Q(t)} - \ln Q(t - t) \right]
\]
(4.12)

where, \(-H\left(\frac{S_2(t)}{S(t)}\right), -H\left(\frac{S_2(t) - S_1(t)}{S_1(t)}\right), -H\left(\frac{S_1(t)}{S_1(t)}\right), \) and \(-H\left(\frac{Q(t)}{Q(t)}\right)\) all belong to the form of \(-H(x)\). Because \( H(x) = x - 1 - \ln x > 0 \) is always authentic, thus \(-H(x) < 0 \). That is to say, all four of these are less than 0.

Let \( \alpha_k = \beta k S'_k(t) \sum_{j \in k} I_t^j(t) G_k(Q_k(t)) = -\frac{Q(t)}{Q(t)} + \ln \frac{Q(t)}{Q(t)} \) we obtain

\[
\sum_{j \in k} I_t^j(t) G_k(Q_k(t)) = -\frac{Q(t)}{Q(t)} + \ln \frac{Q(t)}{Q(t)}
\]

(a) Time series and orbits of system (2.2) with \( R_0 < 1 \)
(b) Time series and orbits of system (2.2) with \( R_0 > 1 \)

Fig. 2. Time series and orbits of system (2.2).
Fig. 3. Time series and orbits of two kinds of nodes with $R_0 < 1$.

Fig. 4. Time series and orbits of two kinds of nodes with $R_0 > 1$.

Fig. 5. Time series and orbits of two kinds of nodes with different degree $k$. 
\[ F_{ij} = G_j(Q_i(t)) - G_i(Q_j(t)) - H \left( \frac{S_j(t)}{S_i(t)} \right) - H \left( \frac{Q_j(t)}{Q_i(t)} \right) - H \left( \frac{V_j(t)}{V_i(t)} \right) \]

\[ \leq G_j(Q_i(t)) - G_i(Q_j(t)), \quad i, j = 1, 2, \ldots, n. \]

wherein, \( V_k, F_{ij}, \) and \( G_k \) satisfy the hypothesis proposed in Reference [50]. \( F_{ij} \) is the hypothesis proposed in Reference [50]. If and only if the system reaches the endemic equilibrium \( E'(S_1, I_1, Q_1, \ldots, S_n, I_n, Q_n, V_n) \), under the condition of \( R_0 > 1 \), the endemic equilibrium point \( E' \) of the system is globally asymptotically stable for any \( \tau \geq 0 \).

To sum up, according to LaSalle's invariance principle of the time-delay system [49], under the condition of \( R_0 > 1 \), the endemic disease equilibrium point exists and is globally asymptotically stable.

Numerical analysis

For the SIQR-V propagation dynamics model proposed above in (2.2), by using the contradiction, Lyapunov function, and Lasalle invariance theorem, we study the positive nature of the system, the propagation threshold (basic reproduction number), and the stability of the equilibrium. In this section, we further describe the trend of individual density and give a sensitivity analysis of critical parameters to verify the conclusions of theoretical research.

The numerical simulation is based on the scale-free network \( p(k) = a_k k^{-\gamma} \), where \( \gamma \) is the scale-free network power-law index, \( \gamma = 3 \). The constant \( a_k \) satisfies \( \sum_{k=1}^{\infty} p(k) = 1 \). The number of nodes on the scale-free network is \( n = 1000 \), and the initial values are \( S_0(0) = 0.8, I_0(0) = 0.5, Q_0(0) = 0.05, R_0(0) = 0.05, V_0(0) = 0.05 \), where \( k = 1.2, \ldots, n \).

Stability of equilibrium point

To carry out numerical verification of the conclusion of the stability above, we set the parameters \( \lambda = 0.14, \mu = 0.14, \alpha = 0.02, \beta = 0.3, \sigma = 0.9, \gamma = 0.3, \epsilon = 0.24, r = 0.5, \lambda = 0.3 \). (\( S_0, I_0, Q_0, R_0, V_0 \) = (0.8, 0.2, 0.0, 0.0)), calculate the basic reproduction number \( R_0 = 0.5649 \). Besides, we take the parameters \( \lambda = 0.14, \mu = 0.14, \alpha = 0.02, \beta = 1, \sigma = 0.1, \gamma = 0.3, \epsilon = 0.24, r = 0.5, \lambda = 0.3, \) (\( S_0, I_0, Q_0, R_0, V_0 \) = (0.8, 0.2, 0.0, 0.0)). At this point, the basic reproduction number \( R_0 = 14.5837 \). We plot the trend of five state nodes of the SIQR-V model in these two cases in Fig. 2.

As shown in the figure above, when \( R_0 < 1 \), the disease-free equilibrium point exists and is globally asymptotically stable. The proportion of susceptible nodes, recovered nodes and vaccinated nodes gradually increase and converge to a standard number. In contrast, the proportion of infected nodes and quarantined nodes gradually approaches 0. When \( R_0 > 1 \), the endemic disease equilibrium point exists and is globally asymptotically stable. The five state nodes in the system eventually converge to a standard number, in which the proportion of susceptible nodes decreases gradually. In contrast, the proportion of infected nodes, quarantined nodes, recovered nodes and vaccinated nodes increases gradually. The following focuses on the trend analysis of the susceptible state (S) and infected state (I) to further explore the law of disease transmission.

Set the parameters \( \lambda = 0.14, \mu = 0.14, \alpha = 0.02, \beta = 0.12, \sigma = 0.3, \gamma = 0.3, \epsilon = 0.24, r = 0.5, \lambda = 0.3, \) the basic reproduction number \( R_0 = 0.9546 \), which is close to 1. We plot the trend of nodes in S state and I state in these two cases in Fig. 3.

As shown in the figure above, when \( R_0 < 1 \), the density of susceptible individuals gradually increases. The proportion of infected people gradually decreases and approaches 0, which means that the infected people gradually disappear and become susceptible. The system will stabilize at the disease-free equilibrium point, and there will be no continuous transmission of disease in the network. Besides, the smaller the basic reproduction number \( R_0 \) is, the faster the proportion of susceptible individuals increases and the faster the proportion of infected individuals decreases.

\[ \text{If } \lambda = 0.14, \mu = 0.14, \alpha = 0.02, \beta = 0.5, \sigma = 0.1, \gamma = 0.3, \epsilon = 0.24, r = 0.5, \lambda = 0.3 \text{, then the basic reproduction number } R_0 = 14.5837. \] Let \( \lambda = 0.14, \mu = 0.14, \alpha = 0.02, \beta = 0.5, \sigma = 0.1, \gamma = 0.3, \epsilon = 0.24, r = 0.5, \lambda = 0.3, \) and \( R_0 = 7.2919 \). The trend of S state and I state nodes in the two cases of \( R_0 > 1 \) is shown in Fig. 4.

Figure 4 shows that when \( R_0 > 1 \), the density of susceptible individuals and infected individuals will converge to an average number, respectively. As stated in Theorem 4.2, when the basic reproduction number...
number is greater than 1, the system is globally asymptotically stable at the endemic equilibrium point $E^*$. For susceptible populations, with the increase of basic reproduction number $R_0$, the density of the susceptible population also decreases, indicating that the spread of the disease makes the proportion of the susceptible population decrease gradually. For the infected population, with the increase of basic reproduction number $R_0$, the density of infected people gradually increases, indicating that the spread of the disease makes the proportion of infected people increase gradually. Therefore, the change of $R_0$ will lead to a significant change in the trend of individuals in the system, so controlling the size of the basic reproduction number $R_0$ is also an effective way to reduce the outbreak of disease.

**The effect of degree $k$ on the spread of infectious diseases**

The spread of infectious disease depends on the source of infection and the way of infection, more the influence of the surrounding environment. Infectious diseases in prosperous areas are often more complex to control than in backward and remote areas. In this section, the degree $k$ represents the number of chains connected by neighbor nodes in the scale-free network, which also means the complexity of the network. We set the parameters $\Lambda = 0.14, \mu = 0.14, \alpha = 0.02, \beta = 0.5, \sigma = 0.4, \gamma = 0.3, r = 0.24, r = 0.5, \lambda = 0.3$ to explore the trend change of the density of susceptible and infected individuals in the system under different value ranges.

According to Fig. 5, we can observe that the change of degree value $k$ does not influence the system’s stability. As the degree value $k$ continuously increases for the susceptible population, the density of susceptible individuals gradually decreases. The valley value that the density can reach becomes larger and larger. It indicates that on a network with many neighbor nodes, the susceptible population gradually transforms into the infected population, consistent with the law of reality. The area with a dense population is more likely to be infected through frequent contact.

**The effect of time delay on the spread of infectious diseases**

In infectious disease transmission, the time delay is easy to cause the lag of intervention measures. To further study the influence of time delay on the system, we compare the disease transmission rule of the system (2.2) with different time delay parameters under $R_0 < 1$ and $R_0 > 1$. Let $\Lambda = 0.14, \mu = 0.14, \alpha = 0.01, \beta = 0.2, \gamma = 0.3, \epsilon = 0.24, \sigma = 0.9, r = 0.5, \lambda = 0.3$, we can get $R_0 = 0.7180 < 1$. In the same way, let $\Lambda = 0.14, \mu = 0.14, \alpha = 0.01, \beta = 1.7, \gamma = 0.3, x = 0.24, \sigma = 0.3, r = 0.5, \lambda = 0.3$, we can get $R_0 = 8.4851 > 1$. Take $r = 0.5, 2.5, 5, 30, 50$ respectively to study the influence of time delay $r$, and we plot the results in Fig. 6(a) and (b).

Figure 6(a) shows that when $R_0 < 1$, for the infected population, the larger the time delay is, the slower the rate of convergence of the proportion of the infected population is and finally approaches 0, and the longer the time for the system to reach the disease-free equilibrium point $E^0$. Fig. 6(b) shows when $R_0 > 1$, the larger the time delay is for the infected population, the slower the proportion of the infected population tends to be stable. And with the gradual increase of the time delay parameter $r$, the equilibrium point $E^*$ of endemic disease changes. On the whole, when $r$ increases to 30, the trend of susceptible population and infected population are stable for a while and then present a wave change. It indicates that due to the influence of latency time delay, there is a long battle line in preventing and controlling infectious diseases, and it is difficult to cure. Therefore, it is necessary to extend the observation time of the sick individual during treatment to prevent the disease from
Sensitivity analysis of parameters

Since the value of the basic reproduction number $R_0$ determines the equilibrium state of the system, controlling the basic reproduction number $R_0$ plays a significant role in maintaining the system’s stability and inhibiting the outbreak of disease. This section focuses on the relationship between the quarantine parameter $\sigma$, transmission rate $\beta$ and vaccination parameter $\alpha$ that have major effects on $R_0$. Think of $R_0$ as a function of these parameters, obviously

\[
\frac{dR_0}{d\sigma} = \frac{-\Lambda(\epsilon^2)}{(\epsilon + \rho)(\epsilon + \rho + \gamma)} < 0, \quad \frac{dR_0}{d\beta} = \frac{-\Lambda(\epsilon^2)}{(\epsilon + \rho)(\epsilon + \rho + \gamma)} < 0, \quad \frac{dR_0}{d\alpha} = \frac{\Lambda(\epsilon^2)}{(\epsilon + \rho)(\epsilon + \rho + \gamma)} > 0
\]

As for the transmission rate $\beta$, we plot the relationship between the quarantine parameter $\sigma$ and the vaccination parameter $\alpha$ in Fig. 7. The results shown in Fig. 7 are consistent with the theory. When the propagation rate $\beta$ increases, the basic reproduction number $R_0$ increases, and there is a positive correlation between $R_0$ and $\beta$. When the quarantine parameter $\sigma$ and the vaccination parameter $\alpha$ increase, the basic reproduction number $R_0$ decreases, and $R_0$ is negatively correlated with $\sigma$ and $\alpha$, respectively. Further, according to Fig. 7(a) and Fig. 7(c), under the same vaccination parameter, transmission rate $\beta$ has a greater tendency to increase $R_0$ than the quarantine parameter $\sigma$ does to decrease $R_0$. Therefore, transmission rate $\beta$ is more sensitive than quarantine parameter $\sigma$ to the influence of basic reproduction number $R_0$. According to Fig. 7(b) and (c), under the same quarantine parameter, the decreasing trend of $R_0$ caused by vaccination parameter $\alpha$ is more significant than that caused by transmission rate $\beta$. Therefore, in terms of the influence of vaccination parameter $\alpha$ on basic reproduction

Table 1

| Parameter | Description    | Value         | Source       |
|-----------|----------------|---------------|--------------|
| $\Lambda$ | Birth rate     | 0.0022        | Estimated    |
| $\mu$     | Mortality rate | 0.002907253   | Fitted       |
| $\beta$   | Contact rate   | 0.027122777   | Fitted       |
| $\alpha$  | Vaccination rate | 0.023805803   | Fitted       |
| $\epsilon$| Recovery rate  | 0.024210933   | Fitted       |
| $\sigma$  | Quarantine rate| 0.0035        | Assumed      |

Table 2

| Parameter | Description    | Value         | Source       |
|-----------|----------------|---------------|--------------|
| $\Lambda$ | Birth rate     | 0.0193        | Estimated    |
| $\mu$     | Mortality rate | 0.01036433    | Fitted       |
| $\beta$   | Contact rate   | 0.00290697    | Fitted       |
| $\alpha$  | Vaccination rate | 0.00043604    | Fitted       |
| $\epsilon$| Recovery rate  | 0.00250381    | Fitted       |
| $\sigma$  | Quarantine rate| 0.011         | Assumed      |

Fig. 9. COVID-19 cases in Germany in January 2021 vs. model fitting.

Fig. 10. COVID-19 cases in Pakistan in February 2021 vs. model fitting.

Fig. 11. SIQR-V model diagram based on the COVID-19 cases in Germany.
number $R_0$, it is more sensitive than transmission rate $\beta$. In general, in terms of parameter sensitivity, $a > \beta > \sigma$ indicates that the vaccination parameter can change the value range of the basic reproduction number $R_0$ more than the transmission rate and quarantine parameter. Therefore, under the same conditions, adjusting the proportion of vaccination parameters can better affect the system’s equilibrium state.

To observe the effectiveness of the three parameters in preventing the occurrence and prevalence of infectious diseases, we further analyze the influence of the three parameters on the system. Firstly, we set the parameters $\lambda = 0.14, \mu = 0.14, a = 0.01, \beta = 1, \gamma = 0.3, \tau = 0.5, \lambda = 0.3$, and set $\sigma = 0.1, 0.3, 0.5, 0.7, 0.9$ respectively to study the influence of the change of the quarantine parameter on the system. We show the results in Fig. 8(a). Then, we set the parameters $\lambda = 0.14, \mu = 0.14, a = 0.02, \sigma = 0.4, \gamma = 0.3, \tau = 0.24, \tau = 0.5, \lambda = 0.3$, and $\beta = 0.1, 0.3, 0.5, 0.7, 0.9$ respectively to study the influence of the change in the system’s infection rate. We show the results in Fig. 8(b). Finally, we set the parameters $\lambda = 0.14, \mu = 0.14, \sigma = 0.9, \beta = 0.8, \gamma = 0.3, \tau = 0.24, \tau = 0.5, \lambda = 0.3$, and $\alpha = 0.1, 0.3, 0.5, 0.7, 0.9$ respectively to study the influence of the change of the vaccination parameter on the system. We show the results in Fig. 8(c).

Figure 8(a) and (c) show that with the increase of the quarantine parameter and vaccination parameter, the infected node density gradually decreases and approaches 0. We obtain that the parameter variation within (0,1,0,3) is the most effective for the quarantine parameter. We get that the parameter variation within (0,1,0,3) is the most effective for the vaccination parameter. That shows both quarantine and vaccination can effectively inhibit the spread of disease and play an indispensable role in preventing disease outbreaks. Fig. 8 (b) shows that with the increase of transmission rate, the density of infected nodes increases gradually, and the disease will turn into an epidemic and begin to break out. To prevent this phenomenon, we can reduce the transmission rate and keep the disease in a stable state, such as controlling the source of infection and cutting off the transmission route.

To sum up, in combination with the relationship between transmission rate $\beta$, quarantine parameter $\sigma$, and vaccination parameter $\alpha$ above, we can conclude. All things being equal, timely vaccination of vulnerable populations is more effective than cutting off transmission routes and quarantining infected people. Thus, when managers devise strategies to combat the epidemic in a situation where workforce and resources are scarce, they can prioritize vaccination first, restrict residents’ participation in gatherings to cut off transmission routes, and finally quarantine those already infected.

**Empirical analysis based on the perspective of COVID-19**

Numerical analysis of the SIQR-V model can further verify the correctness of the above theoretical results. It is also essential to verify a newly designed infectious disease model with actual data. Considering the importance of accurate data and model fitting, we use the Least-squares Curve Fitting method to optimize the observation error of the routine procedure called Curve Fitting in the toolbox through MATLAB R2016a. We use the best fitting curves of the real COVID-19 cases in Pakistan and Germany to obtain the parameters and further insert the parameters into the SIQR-V model. The validity of the model is evaluated by comparing the actual data with the predicted population density differences.

We select the data of COVID-19 infections, deaths, and vaccination population in Germany in January 2021 and estimate the parameter values according to the least-squares curve fitting method. By comparing and analyzing the fitting errors of curves with different orders, we think that the fitting effect of the sixth-order curve is more consistent with the actual situation. Fig. 9 shows the curves of first-order fitting and sixth-order fitting, respectively.

Based on the best fitting curve, we estimate the relevant parameters in the SIQR-V model, as shown in Table 1.

- As can be seen from Table 1, parameters $\mu$, $\beta$, $\alpha$, and $\epsilon$ are obtained by the best fitting method. In contrast, parameter $\lambda$ is estimated based on the total population of Germany in 2021. In addition, assume that the proportion of the quarantined population $\sigma$ is 0.0035. Using the parameter values listed in Table 1 above, we calculate that the basic reproduction number $R_0$ is about 1.5828. This moment $R_0 > 1$. It is indicated that the COVID-19 epidemic in Germany is in an epidemic period. Nevertheless, $R_0$ is only slightly greater than 1, which indicates that COVID-19 poses a threat to Germany. However, compared with the previous outbreak period, the epidemic has been basically under control at this time due to the government’s control measures on infectious diseases and the orderly launch of vaccination. It also matches the reality of Germany in 2021.

Similarly, we use the same curve-fitting and parameter estimation methods to analyze the COVID-19 situation in Pakistan in February 2021. The curve fitting diagram is shown in Fig. 10.

According to the sixth-order fitting curve in Fig. 10, we estimate the relevant parameters as shown in Table 2.

Using the parameter values in Table 2 above, we calculate basic reproduction number $R_0$ is about 1.107. At this time, $R_0$ is also slightly greater than 1, indicating that the COVID-19 outbreak in Pakistan in February 2021 was still ongoing. Therefore, only continued and aggressive quarantine and vaccination measures can ultimately reduce the basic reproduction number and achieve the effect of controlling the spread of COVID-19.

Since the basic reproduction numbers calculated for both Germany and Pakistan are slightly more than 1, we chose to verify further the validity of the SIQR-V model in the analysis and prediction of infectious diseases by using the data of COVID-19 in Germany for the prevention of wordy. According to German data, the initial values of susceptible, infected, quarantined, recovered, and vaccinated groups are 75830577, 2232327, 288027, 1967132, and 1975394. Thus, the initial value of the reproduction number $R_0$ is about 1.5828. This moment $R_0 > 1$. It is indicated that the COVID-19 epidemic in Germany is in an epidemic period. Nevertheless, $R_0$ is only slightly greater than 1, indicating that the COVID-19 outbreak in Pakistan in February 2021 was still ongoing. Therefore, only continued and aggressive quarantine and vaccination measures can ultimately reduce the basic reproduction number and achieve the effect of controlling the spread of COVID-19.

The figure above well confirms the conclusion in section 4 of this paper. That is, the endemic equilibrium exists and is globally asymptotically stable. As shown in the figure, the density of the vaccinated and recovered populations in the system will gradually increase. In contrast, the density of the susceptible and quarantined populations will gradually decrease, indicating that the COVID-19 epidemic in Germany is gradually under control after timely treatment of patients and vaccination measures, but the virus is still threatening. In Fig. 11, $t = 0$ represents the COVID-19 epidemic status in Germany in February 2021. Through observation, we find that at $t = 100$, that is, around May 2021, the increasing trend of the infected population density begins to slow

![Fig. 12. Trends in the number of COVID-19 patients in Germany.](image-url)
down, then turns into a downward trend, and finally reaches a plateau. That suggests that the COVID-19 outbreak in Germany will reach an inflection point around May, after which the outbreak will be fully contained.

To examine the validity of the SIQR-V model in predicting COVID-19 outbreaks in Germany, we draw a line chart showing daily changes in the number of new COVID-19 infections in Germany from February to August. Fig. 12 shows the number of infected people in Germany will peak in April and May, and after May, the number of newly infected people will gradually decrease and even tend to 0. It indicates that the trend of infected people in Germany based on the SIQR-V model is roughly the same as that reflected by the actual epidemic data in Germany. The validity of the SIQR-V model in predicting the law of infectious disease transmission is verified. Therefore, providing suggestions and guidance for treating infectious diseases based on this model can effectively reduce the harm caused by infectious diseases.

Conclusion

Under economic globalization, the rapid population movement makes the existing infectious diseases have strong infectivity and complex transmission processes. Therefore, the previous standard transmission dynamics model is unable to meet the needs of simulating the current transmission law of infectious diseases. To make the model more consistent with the law of reality and have a wide range of theoretical research value and practical application significance, we divide the node states into five categories: S, I, Q, R, and V, and consider quarantine and vaccination measures. At the same time, it takes some time for a disease to spread to an outbreak, and taking advantage of the time delay is crucial to contain the spread of the disease. Given this, we propose a SIQR-V propagation dynamics model with nonlinear incidence and delay over scale-free networks and conduct related studies.

First of all, according to the virus propagation problem, we establish a nonlinear delay differential equation model and calculate the basic reproduction number $R_0$. We find that the magnitude of $R_0$ is mainly affected by quarantine parameter $\alpha$, propagation rate $\beta$, and vaccination parameter $\gamma$. At the same time, the variation of latency does not determine the value of the basic reproduction number. Then, we calculate the disease-free equilibrium and endemic equilibrium and construct the Lyapunov function with time delay to study the stability of the equilibrium. That is, when $R_0<1$, the disease-free equilibrium is globally asymptotically stable; when $R_0 > 1$, the endemic equilibrium point is globally asymptotically stable. Furthermore, through numerical analysis, we find that both the complexity of the network and the variation of the latency can affect the evolution trend of the infected nodes in the system. However, only the latency can affect the stability of the system. Finally, we calculate the basic reproduction numbers of 1.583 and 1.107 for Germany and Pakistan, respectively, and determine that both countries are in the middle of COVID-19 epidemics. The SIQR-V model is validated based on a real COVID-19 case in Germany in January 2021. Our study also shows that both quarantine and vaccination measures significantly affect disease outbreaks. Adjusting the proportion of vaccination parameters has a more significant impact on the system’s equilibrium state. Therefore, vaccination plays a crucial role in preventing the occurrence and spread of infectious diseases.

Data availability statements

The data that support the findings of this study are available upon reasonable request from the authors.

Declaration of Competing Interest

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

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