



Dynamical Analysis of a Mathematical Model of COVID-19 Spreading on Networks

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In this article, an *SEAIRS* model of COVID-19 epidemic on networks is established and analyzed. Following the method of the next-generation matrix, we derive the basic reproduction number R_0 , and it shows that the asymptomatic infector plays an important role in disease spreading. We analytically show that the disease-free equilibrium E_0 is asymptotically stable if $R_0 \leq 1$; moreover, the effects of various quarantine strategies are investigated and compared by numerical simulations. The results obtained are informative for us to further understand the asymptomatic infector in COVID-19 propagation and get some effective strategies to control the disease.

Keywords: COVID-19 epidemic, networks, asymptomatic, the basic reproduction number, quarantine

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1 INTRODUCTION

Coronaviruses are a group of enveloped viruses with a positive-sense, single-stranded RNA and viral particles resembling a crown from which the name derives. They belong to the order of Nidovirales, family of Coronaviridae, and subfamily of Orthocoronavirinae. A recent coronavirus outbreak has started since December 2019. So far, the new virus has infected more than 25 million people and killed at least 842,000 of them [1–3].

In order to better study the spread and control of infectious diseases, several classical mathematical models have been discussed to understand the transmission mechanism and dynamics of the disease, such as the SI model, SIR model, SEIR model, and SEIRS model [4, 5]. Different factors are considered in different models, and the purpose of these models is finding the transmission mechanism and dynamics of the diseases for controlling the diseases. This process needs a contact between them. It is found that the best pattern of contact is contact in the networks [6–11], and study epidemic model on networks has been a hot field in mathematical biology [6–20,35].

To find the transmission mechanism, we have to know who is the infector [27]. An infector is an individual who carries the virus and can export the virus. Sometimes, an individual with the infected state may not show any symptoms of disease, and the state can be defined as asymptotically infected. An individual with the infected state who shows symptoms of disease can be defined as symptomatically infected. Exposed state and asymptotically infected state are more dangerous than symptomatically infected state. When a susceptible individual contacts a symptomatic one, he/she will usually do something possible to prevent the disease spreading.

In the disease spreading process, a symptomatic infector will be restricted by individual behavior or public behavior, but a symptomatic one has a higher efficiency than an asymptomatic one. A class of works studied the role of symptomatic infectors and

asymptomatic infectors in diseases infections, such as dengue virus [21, 22], malaria [23], and *Norovirus* [24–27]. The results of these works all show that the asymptomatic cases cannot be neglected. Recently, several mathematic models within asymptomatic infection have been studied, such as SAIR model [21], CI model [28], SEIADR model [29], and SEII_cQR model [30]. But mathematical models within asymptomatic infection on networks are still relatively rare. When the COVID-19 pandemic begin to outbreak, quarantining certain individuals may be the most efficient way to stop the outbreak of disease, but the choice of which is an important step in the control process, and thus we also discuss the effect of different quarantine strategies.

In this article, we consider an SEAIRS epidemic model on scale-free networks to study the spread of COVID-19. Further, we consider the effect of the asymptomatic state changing into the symptomatic state. By studying the dynamics of the model, we try to find what role the three infectors, E, A, and I, play in disease spreading, especially for the asymptomatic infectors, E and A. Next, in Section 2, we establish an SEAIRS COVID-19 model on network. In Section 3, we study the dynamics of the model. In Section 4, we study and compare the effects of various control strategies, and carry out numerical simulations to illustrate the theoretical results in Section 5. Finally, conclusions and some discussions are given in Section 6.

2 THE MODEL

In the spread process, each node has five states: susceptible (S); exposed (E); asymptotically infected (A), those who are infectious but have no symptoms; symptomatically infected (I), those who are infectious but have symptoms; and removed (R). The disease transmission flow is depicted in Figure 1. A susceptible individual can be infected by contact at rate β_1 if there are infected individuals with A in its neighbors, or be infected by contact at rate β_2 if there are symptomatically infected individuals I. The exposed individual will become A and I with rate $p\alpha$ and $(1-p)\alpha$, and A will become I individual at rate q . The infected A and I will become a recovered individual at rate μ_1 and μ_2 due to treatment, respectively. A recovered individual will become S at rate δ . All parameters are nonnegative.

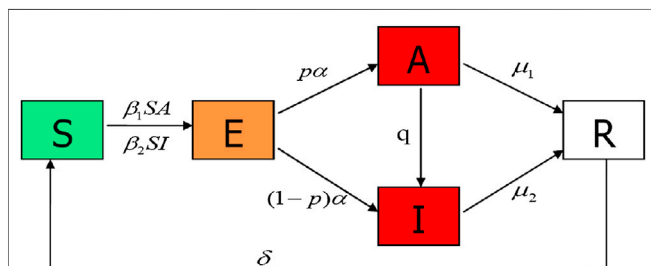


FIGURE 1 | State-transition rules of the SEAIRS model of COVID-19.

In this article, we consider an SEAIRS epidemic model on scale-free networks as follows:

$$\begin{cases} \frac{dS_k(t)}{dt} = -\beta_1 k S_k(t) \theta_1(t) - \beta_2 k S_k(t) \theta_2(t) + \delta R_k(t), \\ \frac{dE_k(t)}{dt} = \beta_1 k S_k(t) \theta_1(t) + \beta_2 k S_k(t) \theta_2(t) - \alpha E_k(t), \\ \frac{dA_k(t)}{dt} = p\alpha E_k(t) - q A_k(t) - \mu_1 A_k(t), \\ \frac{dI_k(t)}{dt} = (1-p)\alpha E_k(t) + q A_k(t) - \mu_2 I_k(t), \\ \frac{dR_k(t)}{dt} = \mu_1 A_k(t) + \mu_2 I_k(t) - \delta R_k(t). \end{cases} \quad (2.1)$$

Following [6], $\langle k \rangle = \sum_k k P_k$ describes the average degree and $P_k (k = 1, 2, \dots, n)$ describes the degree distribution; the probability $\theta_i (i = 1, 2, 3)$ is a link pointing to an exposed individual, asymptomatic infected individual, and symptomatic infected individual. Here, N_k is a constant that stands for the number of nodes with degree k . Then, $N_k = S_k(t) + E_k(t) + A_k(t) + I_k(t) + R_k(t)$, $(k = 1, 2, \dots, n)$ and $\sum_k N_k = N$. We have

$$\theta_1(t) = \frac{1}{\langle k \rangle} \sum_k k P_k \frac{A_k(t)}{N_k}, \quad (2.2)$$

$$\theta_2(t) = \frac{1}{\langle k \rangle} \sum_k k P_k \frac{I_k(t)}{N_k}. \quad (2.3)$$

For the practice, the initial condition for model (2.1) satisfies the following:

$$\begin{cases} 0 \leq S_k(0), E_k(0), A_k(0), I_k(0), R_k(0) \leq N_k, \\ S_k(0) + E_k(0) + A_k(0) + I_k(0) + R_k(0) = N_k, \end{cases} \quad (k = 1, 2, \dots, n). \quad (2.4)$$

3 DYNAMICAL BEHAVIORS OF THE MODEL

In this section, we study the stability of disease-free equilibrium of model (2.1). First, we derive the basic reproduction number of the model.

3.1 Equilibria and Basic Reproduction Number

Lemma 3.1. Suppose that $S_k(t), E_k(t), A_k(t), I_k(t), R_k(t)$ is a solution of model (2.1) satisfying initial conditions of Eq. (2.4), then

$$\Omega = \{(S_k, E_k, A_k, I_k, R_k) | 0 \leq S_k, E_k, A_k, I_k, R_k \leq N_k, k = 1, 2, \dots, n\}$$

is a positive invariant for model (2.1).

The proof of this lemma is similar to that in [31]. We ignore it here. The stability analysis and numerical simulations are investigated in the positive invariant Ω .

It is easy to find that model (2.1) has a disease-free equilibrium E_0 , it is the only susceptible state. $E_0 = (S_1^0, 0, 0, 0, 0, S_2^0, 0, 0, 0, 0, \dots, S_n^0, 0, 0, 0, 0)$, where $S_k^0 = N_k$, $k = 1, 2, \dots, n$. Following the method of the next-generation matrix [32], the basic reproduction number can be calculated

by $R_0 = \rho(FV^{-1})$. Here, F is the matrix of the rate of generation of new infections, and V is the matrix of the rate of transfer of individuals out of the two compartments. F is given by

$$F = \begin{pmatrix} A_{11} & A_{12} & \cdots & A_{1n} \\ A_{21} & A_{22} & \cdots & A_{2n} \\ \vdots & \vdots & \ddots & \vdots \\ A_{n1} & A_{n2} & \cdots & A_{nn} \end{pmatrix}_{3n \times 3n}$$

and

$$A_{ij} = \begin{pmatrix} 0 & \frac{\beta_1 S_i^0 P_j}{\langle k \rangle N_j} ij & \frac{\beta_2 S_i^0 P_j}{\langle k \rangle N_j} ij \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

In addition, $V = \text{diag}(B_1, B_2, \dots, B_n)$, where

$$B_k = \begin{pmatrix} \alpha & 0 & 0 \\ -p\alpha & q + \mu_1 & 0 \\ -(1-p)\alpha & -q & \mu_2 \end{pmatrix}$$

and

$$B_k^{-1} = \begin{pmatrix} \frac{1}{\alpha} & 0 & 0 \\ \frac{p}{q + \mu_1} & \frac{1}{q + \mu_1} & 0 \\ \frac{q + (1-p)\mu_1}{\mu_2(q + \mu_1)} & \frac{q}{\mu_2(q + \mu_1)} & \frac{1}{\mu_2} \end{pmatrix}$$

so, $V^{-1} = \text{diag}(B_1^{-1}, B_2^{-1}, \dots, B_n^{-1})$, and the basic reproduction number R_0 can be obtained:

$$R_0 = \rho(FV^{-1}) = \frac{\mu_2 p \beta_1 + [q + (1-p)\mu_1] \beta_2}{\mu_2(q + \mu_1)} \frac{\langle k^2 \rangle}{\langle k \rangle}. \tag{3.1}$$

Let J_0 be the Jacobian matrix of the middle three equations of model (2.1), then $J_0 = F - V$ and $s(J_0) = \max \{ \text{Re} \lambda : \lambda \text{ is an eigenvalue of } J_0 \}$, $R_0 < 1 \Leftrightarrow s(J_0) < 0$, $R_0 > 1 \Leftrightarrow s(J_0) > 0$. [34]

Remark 3.1. Let $R_{0,a} = \frac{p\beta_1}{q + \mu_1} \frac{\langle k^2 \rangle}{\langle k \rangle}$, $R_{0,d} = \frac{[q + (1-p)\mu_1]\beta_2}{\mu_2(q + \mu_1)} \frac{\langle k^2 \rangle}{\langle k \rangle}$, then $R_0 = R_{0,a} + R_{0,d}$, if $q = 0$, this result is corresponding to the result in [27].

Remark 3.2. If we do not consider the asymptomatic infected state, that is $\beta_1 = 0$, $\mu_1 = \mu_2 = \mu$, $p = q = 0$, then $R_0 = \frac{\beta_2}{\mu} \frac{\langle k^2 \rangle}{\langle k \rangle}$, which is corresponding to the result in [6, 33].

Lemma 3.2. If and only if $R_0 > 1$, there is a unique endemic equilibrium E_+ .

Proof. We consider the right side of the model (2.1) to be equal to zero. Then, we obtain

$$\begin{cases} -\beta_1 k S_k \theta_1 - \beta_2 k S_k \theta_2 + \delta R_k = 0, \\ \beta_1 k S_k \theta_1 + \beta_2 k S_k \theta_2 - \alpha E_k = 0, \\ p\alpha E_k - qA_k - \mu_1 A_k = 0, \\ (1-p)\alpha E_k + qA_k - \mu_2 I_k = 0, \\ \mu_1 A_k + \mu_2 I_k - \delta R_k = 0. \end{cases}$$

Following the above equation, using $\theta = \beta_1 \theta_1^* + \beta_2 \theta_2^*$, we can find

$$E_k^* = \frac{k\theta}{\alpha} S_k^*, A_k^* = \frac{pk\theta}{q + \mu_1} S_k^*, I_k^* = \frac{[q + (1-p)\mu_1]k\theta}{\mu_2(q + \mu_1)} S_k^*, R_k^* = \frac{k\theta}{\delta} S_k^*. \tag{3.2}$$

According to the condition $N_k^* = S_k^* + E_k^* + A_k^* + I_k^* + R_k^*$, we can get

$$S_k^* = \frac{\mu_2 \alpha (q + \mu_1) \delta N_k^*}{\delta \mu_2 \alpha (q + \mu_1) + \{ \mu_2 \alpha (q + \mu_1) + \delta \mu_2 (q + \mu_1) + \delta \mu_2 p \alpha + \delta \alpha [q + (1-p)\mu_1] \} k \theta}. \tag{3.3}$$

Substituting E_k^* , A_k^* , and I_k^* of Eq.(3.2) and Eq.(3.3) into θ , we can obtain $\theta f(\theta) = 0$, where

$$f(\theta) = 1 - \frac{A_1}{\langle k \rangle} \sum_k \frac{k^2 P_k}{A_2 + A_3 k \theta^2}$$

and $A_1 = \delta \alpha \{ \mu_2 p \beta_1 + [q + (1-p)\mu_1] \beta_2 \}$, $A_2 = \delta \mu_2 (q + \mu_1) \alpha$, $A_3 = \mu_2 (q + \mu_1) \alpha + \delta \{ \mu_2 (q + \mu_1) + \mu_2 p \alpha + \alpha [q + (1-p)\mu_1] \}$. It is easy to find that $\theta f(\theta) = 0$ has a trivial solution with no condition, and model (2.1) has a disease-free equilibrium. In addition,

$$\frac{df(\theta)}{d\theta} = \frac{A_1 A_3}{\langle k \rangle} \sum_k \frac{k^3 P_k}{(A_2 + A_3 k \theta^2)^2} > 0,$$

$$f(\beta_1 + \beta_2) = 1 - \frac{A_1}{\langle k \rangle} \sum_k \frac{k^2 P_k}{A_2 + A_3 k (\beta_1 + \beta_2)}$$

$$> 1 - \frac{A_1}{\langle k \rangle} \sum_k \frac{k^2 P(k)}{A_3 k (\beta_1 + \beta_2)} = 1 - \frac{A_1}{A_3 (\beta_1 + \beta_2)}$$

$$= 1 - \frac{\delta \alpha \{ \mu_2 p \beta_1 + [q + (1-p)\mu_1] \beta_2 \}}{\mu_2 (q + \mu_1) \alpha + \delta \{ \mu_2 (q + \mu_1) + \mu_2 p \alpha + \alpha [q + (1-p)\mu_1] \}} \frac{1}{\beta_1 + \beta_2}$$

$$> 1 - \frac{\delta \alpha \{ \mu_2 p \beta_1 + [q + (1-p)\mu_1] \beta_2 \}}{\mu_2 (q + \mu_1) \alpha + \delta \{ \mu_2 (q + \mu_1) + \mu_2 p \alpha + \alpha [q + (1-p)\mu_1] \}} > 0.$$

Thus, $\theta f(\theta) = 0$ has a nontrivial solution if and only if $f(0) < 0$, that is if and only if $R_0 > 1$, there is a unique endemic equilibrium.

3.2 Stability of Disease-free and Endemic Equilibrium

Theorem 3.1. If $R_0 \leq 1$, E_0 of model (2.1) is locally asymptotically stable; if $R_0 > 1$, E_0 is unstable, and there is a unique endemic equilibrium E_+ , and E_+ is locally asymptotically stable.

Proof. From the results of Lemma 3.1, Lemma 3.2, and the basic reproduction number, we only need to prove the case of $R_0 = 1$. We consider the following model:

$$\begin{cases} \frac{dS_k}{dt} = -\beta_1 k S_k \theta_1 - \beta_2 k S_k \theta_2 + \delta R_k, \\ \frac{dE_k}{dt} = \beta_1 k S_k \theta_1 + \beta_2 k S_k \theta_2 - \alpha E_k, \\ \frac{dA_k}{dt} = p\alpha E_k - qA_k - \mu_1 A_k, \\ \frac{dI_k}{dt} = (1-p)\alpha E_k + qA_k - \mu_2 I_k. \end{cases} \tag{3.4}$$

The Jacobian matrix of model (3.4) at disease-free equilibrium is given by

$$J = \begin{pmatrix} J_{11} & J_{12} & \cdots & J_{1n} \\ J_{21} & J_{22} & \cdots & J_{2n} \\ \vdots & \vdots & \ddots & \vdots \\ J_{n1} & J_{n2} & \cdots & J_{nn} \end{pmatrix}_{4n \times 4n}$$

with

$$J_{ij} = \begin{pmatrix} -\delta & -\delta & -\frac{\beta_1 P_j N_i}{\langle k \rangle N_j} ij - \delta & -\frac{\beta_2 P_j N_i}{\langle k \rangle N_j} ij - \delta \\ 0 & -\alpha & \frac{\beta_1 P_j N_i}{\langle k \rangle N_j} ij & \frac{\beta_2 P_j N_i}{\langle k \rangle N_j} ij \\ 0 & p\alpha & -q - \mu_1 & 0 \\ 0 & (1-p)\alpha & q & -\mu_2 \end{pmatrix}$$

The characteristic equation of the disease-free equilibrium is

$$(\lambda + \mu_2)^{n-1} (\lambda + q + \mu_1)^{n-1} (\lambda + \delta)^n (\lambda + \alpha)^{n-1} (\lambda^3 + H_2 \lambda^2 + H_1 \lambda + H_0) = 0,$$

where

$$H_2 = \alpha + q + \mu_1 + \mu_2 > 0,$$

$$H_1 = \alpha(q + \mu_1) + \alpha\mu_2 + \mu_2(q + \mu_1) - [p\alpha\beta_1 + (1-p)\alpha\beta_2] \frac{\langle k^2 \rangle}{\langle k \rangle},$$

$$H_0 = \mu_2\alpha(q + \mu_1) - \alpha\{\mu_2 p\beta_1 + [q + (1-p)\mu_1]\beta_2\} \frac{\langle k^2 \rangle}{\langle k \rangle},$$

when $R_0 = 1$, $H_0 = 0$, and

$$\alpha\mu_2 p\beta_1 \frac{\langle k^2 \rangle}{\langle k \rangle} < \mu_2\alpha(q + \mu_1) \Rightarrow \alpha p\beta_1 \frac{\langle k^2 \rangle}{\langle k \rangle} < \alpha(q + \mu_1),$$

$$(1-p)\alpha\mu_1\beta_2 \frac{\langle k^2 \rangle}{\langle k \rangle} < \mu_2\alpha(q + \mu_1) \Rightarrow (1-p)\alpha\beta_2 \frac{\langle k^2 \rangle}{\langle k \rangle} < \mu_2(\alpha + q + \mu_1).$$

Then, we obtain $H_1 > 0$, and the characteristic equation can be written as

$$\lambda(\lambda + \mu_2)^{n-1} (\lambda + q + \mu_1)^{n-1} (\lambda + \delta)^n (\lambda + \alpha)^{n-1} (\lambda^2 + H_2\lambda + H_1) = 0,$$

where the eigenvalues of J are all negative except zero eigenvalue when $R_0 = 1$.

4 QUARANTINE STRATEGIES

Quarantine is helpful in controlling diseases. In this section, we will discuss three different quarantine strategies for COVID-19 disease model on networks.

4.1 Proportional Quarantine

In this case, for fixed spreading rates β_1 and β_2 , let ω ($0 < \omega < 1$) is the density of quarantine nodes in the network. At the mean-field level, the presence of proportional quarantine will effectively reduce

the spreading rate. Thus, we can approximately use $\beta_1(1 - \omega)$ to substitute β_1 and use $\beta_2(1 - \omega)$ to substitute β_2 ; thus, the system (2.1) becomes

$$\begin{cases} \frac{dS_k(t)}{dt} = -\beta_1(1 - \omega)kS_k(t)\theta_1(t) - \beta_2(1 - \omega)kS_k(t)\theta_2(t) + \delta R_k(t) \\ \frac{dE_k(t)}{dt} = \beta_1(1 - \omega)kS_k(t)\theta_1(t) + \beta_2(1 - \omega)kS_k(t)\theta_2(t) - \alpha E_k(t) \\ \frac{dA_k(t)}{dt} = p\alpha E_k(t) - qA_k(t) - \mu_1 A_k(t) \\ \frac{dI_k(t)}{dt} = (1-p)\alpha E_k(t) + qI_k(t) - \mu_2 I_k(t) \\ \frac{dR_k(t)}{dt} = \mu_1 A_k(t) + \mu_2 I_k(t) - \delta R_k(t) \end{cases} \quad (4.1)$$

Using the same method in Section 3, we obtain a self-consistency equation as follows:

$$\theta = \frac{\delta\alpha(1 - \omega)A_1}{\langle k \rangle} \sum_k \frac{\langle k^2 \rangle P_k}{A_2 + A_3 k \theta} \theta \equiv \tilde{f}(\theta).$$

By arguments similar to those in Section 3, the epidemic threshold \widetilde{R}_w is determined by the following inequality:

$$\left. \frac{d\tilde{f}(\theta)}{d\theta} \right|_{\theta=0} > 1;$$

therefore, it can be shown that

$$\widetilde{R}_w = \frac{\mu_2 p(1 - \omega)\beta_1 + [q + (1-p)\mu_1](1 - \omega)\beta_2}{\mu_2(q + \mu_1)} \frac{\langle k^2 \rangle}{\langle k \rangle}, \quad (4.2)$$

that is,

$$\widetilde{R}_w = (1 - \omega)R_0. \quad (4.3)$$

Note that in (4.3), when $\omega = 0$, that is, if no quarantine were done, then $\widetilde{R}_w = R_0$; when $0 < \omega < 1$, $\widetilde{R}_w < R_0$, that is, the quarantine scheme is effective; while as $\omega \rightarrow 1$, $\widetilde{R}_w \rightarrow 0$, that is, in the case of a full quarantine, it would be impossible for the epidemic to spread in the network.

4.2 Targeted Quarantine

While proportional quarantine schemes are effective, there may be more efficient schemes due to the heterogeneous nature of scale-free networks. We introduce an upper threshold κ such that all nodes with connectivity $k > \kappa$ are immunized, that is, we define the quarantine rate ω_k by

$$\omega_k = \begin{cases} 1, & k > \kappa, \\ c, & k = \kappa, \\ 0, & k < \kappa. \end{cases}$$

where $0 < c < 1$ and $\sum_k \omega_k P(k) = \bar{\omega}$, where $\bar{\omega}$ is the average quarantine rate. The epidemic dynamics model is

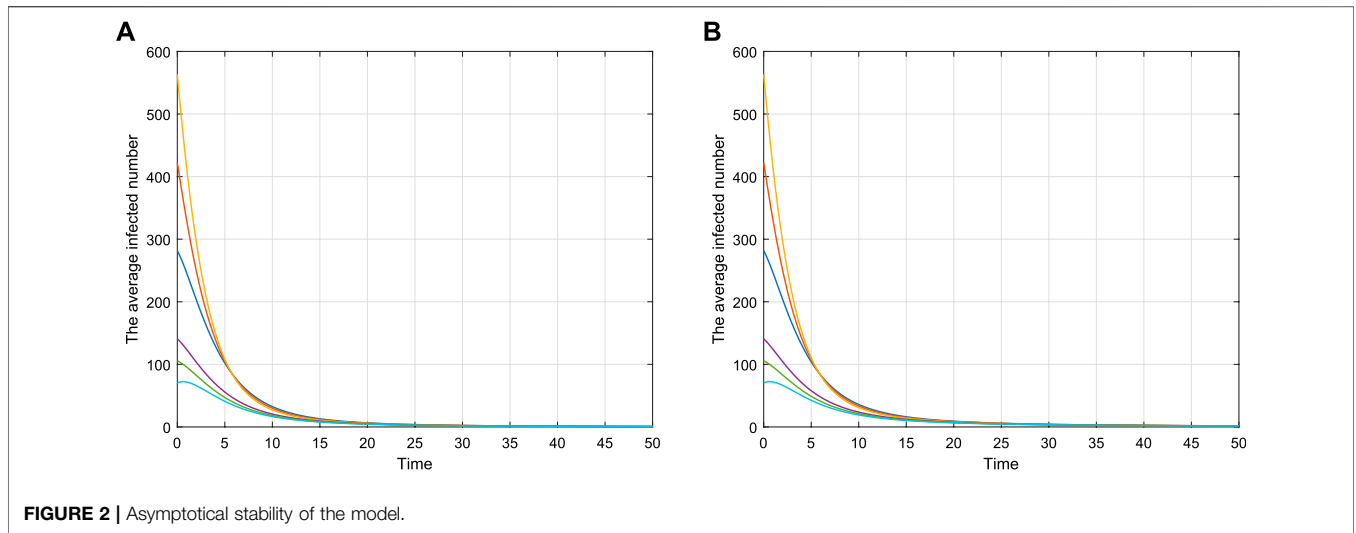


TABLE 1 | Parameters of Figure 2

Figure 2	β_1	β_2	δ	α	p	q	μ_1	μ_2	R_0
A	0.08	0.08	0.4	0.4	0.5	0.5	0.2	0.4	0.8193 < 1
B	0.0905	0.10003	0.4	0.4	0.5	0.5	0.2	0.4	1

TABLE 2 | Parameters of Figure 4

	β_1	β_2	Δ	α
Figure 4	0.0020178	0.033949	0.12178	0.1055744
Figure 4	p	q	μ_1	μ_2
Figure 4	0.40043	0.040403	0.0119,536	0.20955

$$\begin{cases}
 \frac{dS_k(t)}{dt} = -\beta_1(1-\omega_k)kS_k(t)\theta_1(t) - \beta_2(1-\omega_k)kS_k(t)\theta_2(t) + \delta R_k(t) \\
 \frac{dE_k(t)}{dt} = \beta_1(1-\omega_k)kS_k(t)\theta_1(t) + \beta_2(1-\omega_k)kS_k(t)\theta_2(t) - \alpha E_k(t) \\
 \frac{dA_k(t)}{dt} = p\alpha E_k(t) - qA_k(t) - \mu_1 A_k(t) \\
 \frac{dI_k(t)}{dt} = (1-p)\alpha E_k(t) + qI_k(t) - \mu_2 I_k(t) \\
 \frac{dR_k(t)}{dt} = \mu_1 A_k(t) + \mu_2 I_k(t) - \delta R_k(t)
 \end{cases}$$

(4.4)

This leads to

$$\theta = \frac{\delta\alpha A_1}{\langle k \rangle} \sum_k \frac{\langle k^2 \rangle P_k (1-\omega_k)}{A_2 + A_3 k \theta} \theta \equiv \tilde{f}(\theta).$$

Therefore, the epidemic threshold

$$\widehat{R}_w = \frac{\mu_2 p \beta_1 + [q + (1-p)\mu_1] \beta_2}{\mu_2 (q + \mu_1)} \frac{\langle k^2 \rangle - \langle k^2 \omega_k \rangle}{\langle k \rangle}. \tag{4.5}$$

Note that $\langle k^2 \omega_k \rangle = \bar{\omega} \langle k^2 \rangle + \sigma'$, where $\sigma' = \langle (\omega_k - \bar{\omega}) \times [k^2 - \langle k^2 \rangle] \rangle$ is the covariance of ω_k and k^2 . There may be κ (usually big enough) where $\sigma' < 0$, but for appropriately small κ , $\omega_k - \bar{\omega}$ and $k^2 - \langle k^2 \rangle$ have the same signs except for some k where $\omega_k - \bar{\omega}$ and/or $k^2 - \langle k^2 \rangle$ is zero; Therefore $\sigma' > 0$ for appropriate κ . Then,

$$\widehat{R}_w < \frac{1-\bar{\omega}}{1-\omega} \widetilde{R}_w.$$

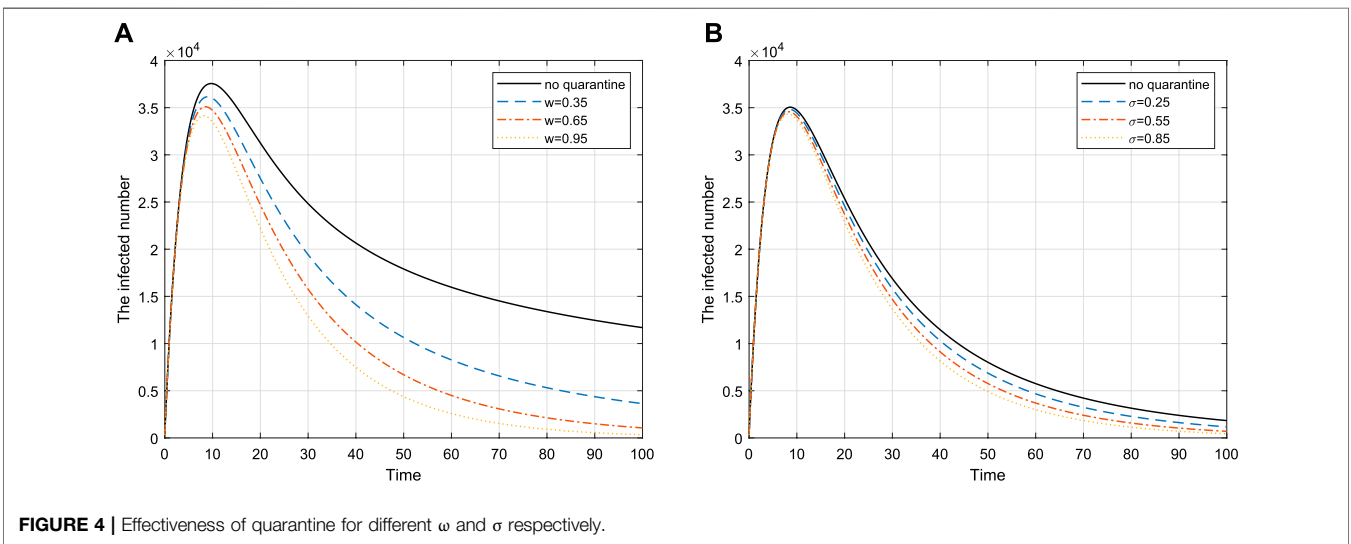
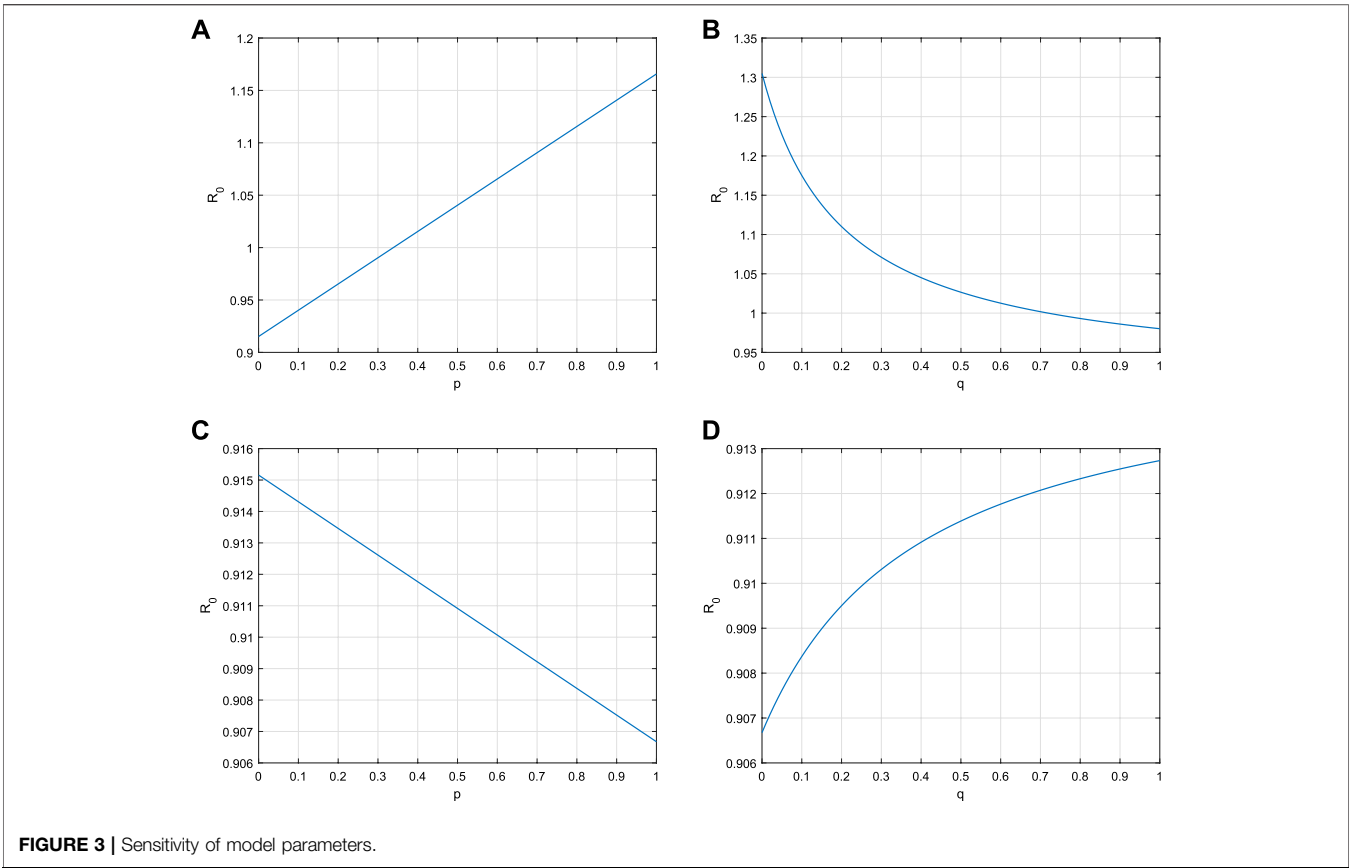
If we set $\bar{\omega} = \omega$, then,

$$\widehat{R}_w < \widetilde{R}_w (0 < \omega < 1),$$

which means the targeted quarantine scheme is more efficient than the uniform quarantine scheme for the same average quarantine rate.

4.3 High-Risk Quarantine

If a neighbor of a susceptible individual is infected, vaccinating this susceptible individual is



called high-risk immunity [37]. We introduce the parameter σ , that is, the probability that a node is quarantined per unit time, and $\Gamma = \frac{kP_k}{\langle k \rangle N}$, which

denotes the probability that any given node is the neighbor of some specific nodes. The epidemic dynamics model is

$$\begin{cases} \frac{dS_k(t)}{dt} = -\beta_1(1-\sigma\Gamma)kS_k(t)\theta_1(t) - \beta_2(1-\sigma\Gamma)kS_k(t)\theta_2(t) + \delta R_k(t) \\ \frac{dE_k(t)}{dt} = \beta_1(1-\sigma\Gamma)kS_k(t)\theta_1(t) + \beta_2(1-\sigma\Gamma)kS_k(t)\theta_2(t) - \alpha E_k(t) \\ \frac{dA_k(t)}{dt} = p\alpha E_k(t) - qA_k(t) - \mu_1 A_k(t) \\ \frac{dI_k(t)}{dt} = (1-p)\alpha E_k(t) + qI_k(t) - \mu_2 I_k(t) \\ \frac{dR_k(t)}{dt} = \mu_1 A_k(t) + \mu_2 I_k(t) - \delta R_k(t) \end{cases} \tag{4.6}$$

This leads to

$$\theta = \frac{\delta\alpha A_1}{\langle k \rangle} \sum_k \frac{\langle k^2 \rangle P_k (1-\sigma\Gamma)}{A_2 + A_3 k \theta} \theta \equiv \bar{f}(\theta).$$

And the epidemic threshold

$$\bar{R}_0 = \frac{\mu_2 p \beta_1 + [q + (1-p)\mu_1] \beta_2}{\mu_2 (q + \mu_1)} \left(\frac{\langle k^2 \rangle}{\langle k \rangle} - \frac{\sigma \langle k^3 P_k \rangle}{\langle k \rangle^2 N} \right) < R_0.$$

This means that the implementation of high-risk quarantine is effective, and the longer the period of $\sigma \langle k^3 P_k \rangle$, the more effective it is.

5 NUMERICAL SIMULATIONS

In this section, we present numerical simulations of model (2.1), (4.1) and (4.6) to illustrate the results in Section 3 and 4. Without loss of generality, we consider the dynamical process on scale-free networks with $P_k = ck^{-\gamma}$ and $\sum_{k=1}^n P_k = 1$, $\gamma = 3$, $n = 200$. The average infected number is $A(t) + I(t) = \sum_{k=1}^n A_k(t)P_k + \sum_{k=1}^n I_k(t)P_k$. The parameters of the model are estimated by MCMC using the number of cases in Wuhan (http://wjw.wuhan.gov.cn/ztlz_28/fk/yqtb/).

Figure 2 shows the asymptotical stability of model (2.1) with different values, and the parameter values are shown in Table 1. Figures 2A,B show the time series of the average infected number $I(t)$ with different initial values, which is in accord with the asymptotical stability of the disease-free equilibrium.

The sensitivity analysis of the basic reproduction number R_0 can be performed by model parameters. It is easy to obtain that

$$\begin{aligned} \frac{\partial R_0}{\partial \beta_1} &= \frac{p}{q + \mu_1} \frac{\langle k^2 \rangle}{\langle k \rangle} > 0, & \frac{\partial R_0}{\partial \beta_2} &= \frac{q + (1-p)\mu_1}{(q + \mu_1)\mu_2} \frac{\langle k^2 \rangle}{\langle k \rangle} > 0, \\ \frac{\partial R_0}{\partial \mu_1} &= \frac{-\beta_2 p q - \beta_1 \mu_2 p}{\mu_2 (q + \mu_1)^2} \frac{\langle k^2 \rangle}{\langle k \rangle} < 0, & \frac{\partial R_0}{\partial \mu_2} &= \frac{\beta_2 [q + (1-p)\mu_1]}{\mu_2^2 (q + \mu_1)} \frac{\langle k^2 \rangle}{\langle k \rangle} < 0, \\ \frac{\partial R_0}{\partial p} &= \frac{\beta_1 \mu_2 - \beta_2 \mu_1}{\mu_2 (q + \mu_1)} \frac{\langle k^2 \rangle}{\langle k \rangle}, & \frac{\partial R_0}{\partial q} &= \frac{p(\beta_2 \mu_1 - \beta_1 \mu_2)}{\mu_2 (q + \mu_1)^2} \frac{\langle k^2 \rangle}{\langle k \rangle}. \end{aligned} \tag{5.1}$$

Equation (5.1) shows R_0 is linearly positively correlated with β_1 and β_2 ; R_0 is negatively correlated with μ_1 and μ_2 ; the correlation between R_0 and p or q is determined by $\beta_2 \mu_1 - \beta_1 \mu_2$. As can be seen from Figure 3 that the monotonicity of R_0 increases or decreases with respect to p and q , respectively. In Figure 4, the parameters of the model are estimated by MCMC using the number of cases in

Wuhan and the parameter values are shown in Table 2. Figure 4 shows the change of total infection density with time under different ω and σ . It can be seen that with the increase of ω and σ , the total infection density is becoming lower as ω and σ increase.

6 CONCLUSION AND DISCUSSION

In this article, we have generalized a traditional homogeneous epidemic model with asymptomatic infectors to a network case to study the spread of COVID-19. As one can see, homogeneous epidemic models can be considered as a special case of network epidemic models, and network epidemic models are more realistic and refined description of disease propagation in population. Considering two cases of infected state (show symptoms and show no symptoms), we investigated a SEAIRS model of COVID-19 on scale-free networks to approach the disease progress and calculated the basic reproduction number R_0 . By theoretical analysis, we obtained the asymptotical stability of the equilibria: if $R_0 \leq 1$, E_0 is asymptotically stable. Then, we analyzed the different quarantine strategies in the model. At last, numerical simulations illustrated the theoretical analysis. The results show that the asymptomatic infectors may infect health individuals as well as symptomatic infectors.

The SEAIRS model is an extension of the SEAIRS model, and it is a case of the $S \rightarrow S$ model. The $S \rightarrow S$ model, $S \rightarrow I$ model, and $S \rightarrow R$ model differ in terms of pathological mechanisms. It is difficult to say which one is better than others except introducing a special disease. Within $S \rightarrow S$ models, formally, either the SIS model or SIRS model can be a special case of the SEIRS model. The parameters β_1 and β_2 denoted in the model are constant. In fact, many infectious diseases show seasonal phenomenon, such as measles, chickenpox, and mumps [36], and each individual has a periodic order; the parameters above should have some periodic property.

At the same time, we have also discussed proportional, targeted quarantine and high-risk quarantine schemes for network models. The result of Leung et al. [27] by illustration is suitable for Norovirus and measles. Different from their work, the formulaic result seems to be unsuitable for any kind of disease, for none of the parameters in the model is assigned to a specific value. In fact, for different disease, the parameters in the model may present special values. COVID-19 is now spreading faster abroad. Susceptible people will also be infected by asymptomatic patients. If not controlled, the epidemic will spread more widely and the number of infected people will be more difficult to control. The epidemic situation in China was obviously well controlled after the implementation of the strategy of lockdown, and other quarantine strategies. Therefore, the model and quarantine strategy in this article can also be applied to other countries. And, we hope that the results will be helpful to study and control the spread of COVID-19 disease.

DATA AVAILABILITY STATEMENT

The original contributions presented in the study are included in the article/Supplementary Material; further inquiries can be directed to the corresponding author.

AUTHOR CONTRIBUTIONS

ML and YS designed the study, and WL and ML carried out the analysis and contributed to writing the article. XF and YS performed numerical simulations.

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Conflict of Interest: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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