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A Novel Metric to Quantify the Real-Time Robustness of Complex Networks With Respect to Epidemic Models

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Spread velocity, epidemic threshold, and infection density at steady state are three non-negligible features describing the spread of epidemics. Combining these three features together, a new network robustness metric with respect to epidemics was proposed in this paper. The real-time robustness of the network was defined and analyzed. By using the susceptible–infected (SI) and susceptible–infected–susceptible (SIS) epidemic models, the robustness of different networks was analyzed based on the proposed network robustness metric. The simulation results showed that homogeneous networks present stronger robustness than do heterogeneous networks at the early stage of the epidemic, and the robustness of the heterogeneous networks becomes stronger than that of the homogeneous ones with the progress of the epidemic. Moreover, the irregularity of the degree distribution decreases the network robustness in homogeneous networks. The network becomes more vulnerable as the average degree grows in both homogeneous and heterogeneous networks.

Keywords: real-time robustness, epidemic spread, spread velocity, complex network, robustness metrics

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INTRODUCTION

Nowadays, various dynamic phenomena exist in real networks, many of which are harmful and bring great damage to real life. Especially, the threat of infectious diseases is growing increasingly due to the increasing complexity of modern social networks in all facets of human endeavor [1–5]. For example, as reported by the WHO on October 29, 2021, there have been more than 245 million confirmed cases of coronavirus disease 2019 (COVID-19) globally, including almost 5 million deaths (https://covid19.who.int/). Also, other fields like economy, politics, and culture have suffered extensive damages during the outbreak of COVID-19. Since network structures show a great impact on the propagation dynamics [6–9, 11], it is crucial to assess the robustness of different network structures with respect to the spread.

Epidemic propagation models have been recently used to analyze network robustness against virus attacks, and the robustness of different networks has been studied [10–14]. By modeling and analyzing the epidemic propagation, the descriptive features of the propagation process are often used to measure network robustness against the epidemic spread. For example, the epidemic threshold and the final infection rate at the steady state have been used to measure the robustness of the network against virus attacks individually or jointly [13]. The results of network robustness with

respect to epidemics can help in understanding and further improving network robustness against epidemics.

Although the existing measurements have been proven to be effective for network robustness when it comes to the spread of epidemics, some inherent challenges have been overlooked. Firstly, the existing measurements have mainly focused on the robustness of the network at the steady state. To our knowledge, the real-time robustness of complex networks with respect to epidemics has not been widely studied, i.e., the changes in the network robustness over time in different network structures have not been explored. Secondly, it is not accurate to measure network robustness without considering the spread velocity, which is an important factor in measuring the spread of epidemics. Therefore, the robustness of the network against epidemics can be comprehensively and accurately measured by considering the spread velocity. Furthermore, the spread velocity describes the changes in the propagation over time, which is very suitable for measuring the real-time network robustness with respect to epidemics [17].

In this paper, combining spread velocity, infection density at steady state, and the epidemic threshold, a novel metric was proposed to measure real-time robustness with respect to epidemics in complex networks. Network robustness with respect to the spread of the susceptible–infected (SI) [15] and susceptible–infected–susceptible (SIS) models [16] was analyzed based on the new metric, and some interesting results are presented in our paper. Firstly, the results confirmed that the irregularity of the degree distribution strengthens the network's vulnerability with respect to the epidemic in homogeneous networks. However, the simulation results on the real-time robustness of the different networks showed that the robustness of the Barabási–Albert (BA) scale-free network [19] is not always stronger than that of the Watts–Strogatz (WS) network [20] at any time, which was different from the results of existing studies. At the early stage of the epidemic, the BA network is more fragile than the WS network. As the infection rate worsens, the BA network becomes more robust than the WS network. Moreover, the simulation results showed that the network becomes more vulnerable to the epidemic as the average degree grows in both homogeneous and heterogeneous networks.

The rest of this paper is organized as follows. *Related Work* presents the literature review and related works. In *Network Robustness With Respect to Epidemic Models*, we analyze the necessity of proposing the new metric to measure the network robustness against diseases. In *The Novel Metric to Quantify Network Robustness*, the novel metric to quantify the network robustness with respect to the SI/SIS epidemic spread is proposed. The simulation results in different networks are presented and analyzed in *Results*, and the main conclusions and the direction for future studies are summarized in *Discussion*.

RELATED WORK

Epidemics in social networks can be theoretically described using biological epidemic models, through which the spread

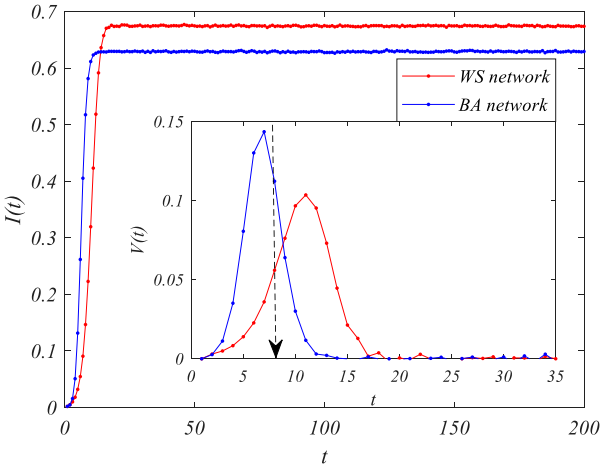

FIGURE 1 | The susceptible–infected–susceptible (SIS) epidemic spreading process in different networks, $\beta = \delta = 0.3$. $I(t)$ represents the fraction of infected nodes at time t and $V(t)$ the growth rate of infection at time t.

mechanism of viruses can be described and analyzed. For example, the SI and SIS epidemic models are often used to model the spread of epidemics [13–17]. In the SI model, the S-state nodes can pass to the infected state through contagion by the infected ones, and the rate of an S-state node being infected by a single infected neighbor is β. In the SIS model, the I-state node recovered to the S state at the rate δ in the SIS model, and the ratio between β and δ is denoted the effective infection rate τ. The time evolution of the different states of the nodes can be described using differential equations from which the relevant conclusions of epidemics can be derived.

In the complex network theory, three important features describing the epidemic spread were introduced into the epidemic models. Firstly, the epidemic threshold τ_c, as a function of the basic reproductive number R_0, was used to determine the outbreak of the epidemic [21]. When the effective infection rate τ is higher than τ_c, i.e., $R_0 > 1$, the epidemic spreads in the population, but when the effective infection rate τ is lower than τ_c, the epidemic dies out. With the outbreak of the epidemic, the states of the nodes in the network change with time, and the changing rate can be measured by the spread velocity. When the network reaches a stable state, the density of each state in the network becomes stable, and the final infection rate at the steady state can be used to measure the scale of the spread.

Therefore, the epidemic threshold, spread velocity, and the final infection rate at the steady state can comprehensively describe the propagation mechanism and can also be used as a measure of network robustness with respect to epidemics. As one of the most prominent features, the epidemic threshold is the first and commonly used measure of network robustness with respect to the epidemic spread [22, 23]. The larger the threshold, the more difficult it is to spread the virus, i.e., the more robust a network is against the virus attack [13]. Studies have found that the threshold cannot fully measure network robustness. For example, the Erdős–Rényi (ER) network [18] and the BA

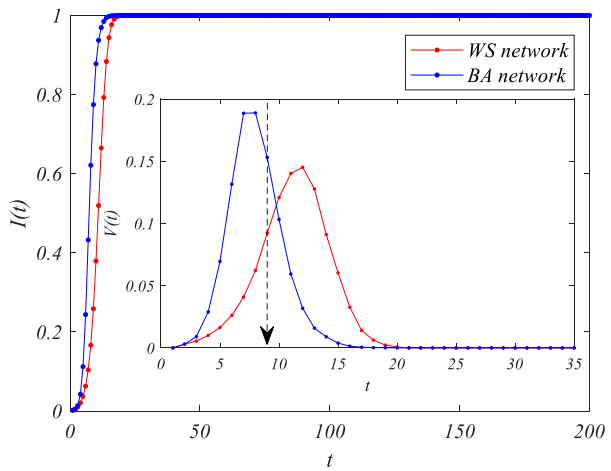

FIGURE 2 | The susceptible–infected (SI) epidemic spreading process in different networks, $\beta = 0.3$. $I(t)$ represents the fraction of infected nodes at time t and $V(t)$ the growth rate of infection at time t.

network [19] have two opposing features—the epidemic threshold and the steady-state infection rate—to measure their robustness. In [13], a new measure incorporating the fraction of infected nodes at the steady state and the epidemic threshold to assess the robustness of the complex networks with respect to the spread of epidemic has been proposed and proven to be effective in modeling epidemics with different final infection densities.

NETWORK ROBUSTNESS WITH RESPECT TO EPIDEMIC MODELS

In existing studies, the epidemic threshold and the steady-state infection rate have been bound together to measure network robustness against epidemics since it has been proven that the results are inaccurate when only one feature is considered. Besides the epidemic threshold and the steady-state infection rate, the spread velocity is another widely discussed variable that should not be ignored in the study of network robustness. For example, **Figure 1** shows the infection rate at the nodes at time t due to the SIS epidemic spreading process in the WS and BA networks, where the average degree of the two networks is the same. We can differentiate between the propagation processes in the two networks from the curve in **Figure 1**. Firstly, the final infection density in the BA network (I_{BA}) is smaller than that in the WS network (I_{WS}), i.e., $I_{WS} > I_{BA}$. Based solely on the infection scale at the steady state, we can conclude that the BA network is more robust than the WS network.

However, the performance of spread velocity is more interesting than that of the final propagation scale. In our simulation, we first described the spread velocity as the growth of the infection rate, i.e., $V(t) = I(t+1) - I(t)$. As shown in **Figure 1**, when $t < 9$, the spread velocity of the epidemic in the WS network is slower than that of the BA network, i.e., $V(t)_{BA} > V(t)_{WS}$. However, when $t \geq 9$, the spread velocity of the epidemic in the WS network is faster than that of the BA network, i.e., $V(t)_{BA} < V(t)_{WS}$. Especially, when the final infection densities at the steady state are the same, such as in the SI model shown in **Figure 2**, we can hardly conclude which network shows stronger robustness based solely on the spread velocity. Therefore, estimating the robustness of different networks based solely on the spread velocity is different, which is one of the reasons to study real-time network robustness. In addition, since the spread velocity describes the dynamics of the propagation process before attaining the steady state, it is essential in measuring real-time network robustness.

Moreover, we also measured the moment at which the steady state of the infection first arrives [$T(i\_\max)$] under different infection rates β in the BA and WS networks. **Figure 3** shows that the $T(i\_\max)$ in the WS network was larger than that in the BA network under the same β, especially when β was very small. Therefore, we can conclude that one single feature may fail to comprehensively measure the robustness of the network. Besides the epidemic threshold, the infection rate at the steady state and the spread velocity are also very important in measuring network robustness with respect to epidemics. Therefore, we proposed a novel metric with multiple features to quantify network robustness against the spread of the epidemic in this paper.

NOVEL METRIC TO QUANTIFY NETWORK ROBUSTNESS

We proposed a multi-indicator-based measurement to quantify network robustness against the epidemic by combining the epidemic threshold, the infection density at steady state, and the spread velocity. Suppose that, in the SIS epidemic model, the rate of a susceptible node being infected by a single infected neighbor is β and the infected node recovered at the rate δ in the SIS model. When $\delta = 0$, the SIS model is transformed into the SI model. In the SIS model, the effective recovery rate can be defined as $s = 1/\tau = \delta/\beta$, $s \in (0, \lambda_{\max})$. The density of the infected nodes

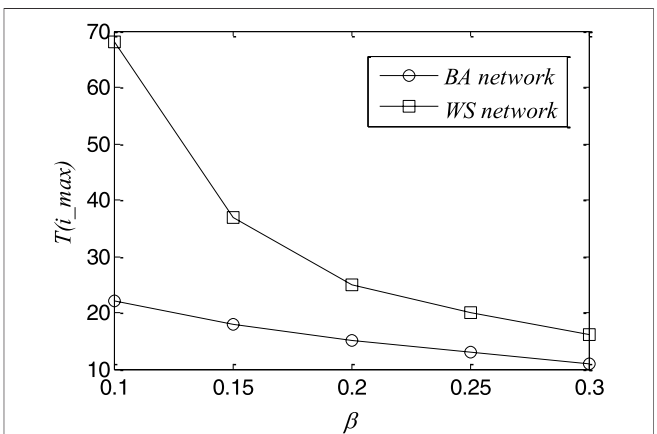

FIGURE 3 | The time of reaching the steady states of the epidemic spread under different infection rates in the Barabási–Albert (BA) and Watts–Strogatz (WS) networks.

at time t is described as $i(t)$, and the steady state of the infection under the effective infection rate τ can be written as $i_\infty(\tau)$. Considering the infection at the steady state and the spread velocity, we define $i^A(T)$ as the average infection rate of the network after time T:

$$i^A(T) = \frac{1}{T}\int_0^T i(t)dts. \tag{1}$$

The network robustness with respect to the epidemic spread can be written as

$$R_G = \int_0^{\lambda_{\max}} i^A(s)ds. \tag{2}$$

Equation 2 shows that the greater the value of R_G, the more fragile is the network, i.e., the weaker is its robustness.

The real-time robustness of network G can be written as

$$R_G(T) = \frac{1}{T}\int_0^{\lambda_{\max}}\int_0^T i(t,s)dtds. \tag{3}$$

Especially, when $T \to \infty$,

$$i^A(T) = \frac{1}{T}\int_0^T i(t)dt = \frac{1}{T}\left(\int_0^{T_s} i(t)dt + \int_{T_s}^{\infty} i_\infty dt\right) \approx i_\infty, \tag{4}$$

where T_s represents the moment when the infection reaches a steady state for the first time. Then, the network robustness can be written as

$$R_G = \int_0^{\lambda\ \max} i_\infty(s)ds, \tag{5}$$

which is the viral conductance proposed in **Eq. 13**.

Based on the SI and SIS epidemic models, we can further write the robustness of the network with respect to the spread of the SI and SIS epidemic models.

Case 1

The robustness of homogeneous networks with respect to the spread of the SI model is shown. The state of each node in the SI model is either infected or healthy, and the change in infected individuals over time can be described as

$$\frac{di}{dt} = \beta\langle k\rangle i(1-i). \tag{6}$$

By separating the variables, **Eq. 6** can be written as

$$\frac{di}{i(1-i)} = \beta\langle k\rangle dt, \tag{7}$$

Integrating both sides of **Eq. 7**, we can obtain

$$\ln\frac{1-i(t)}{i(t)} = -\beta\langle k\rangle t + c. \tag{8}$$

The density of the infected nodes at time t can be written as

$$i(t) = \frac{1}{1+\left(1/i_0 - 1\right)e^{-\beta\langle k\rangle t}}. \tag{9}$$

The final infection density of the SI model is equal to 1, i.e., $i_\infty = 1$. Based on **Eqs. 5** and **9**, the robustness of the homogeneous network G with respect to the spread of the SI epidemic can be written as

$$\begin{aligned} R_G^{SI}(T) &= \frac{1}{T}\int_0^1\int_0^T i(t,\beta)dtd\beta = \frac{1}{T}\int_0^1\int_0^T \frac{1}{1+\left(1/i_0-1\right)e^{-\beta\langle k\rangle t}}dtd\beta \\ &= \frac{1}{T}\int_0^1\int_0^T\left(1 - \frac{\left(1/i_0-1\right)e^{-\beta\langle k\rangle t}}{1+\left(1/i_0-1\right)e^{-\beta\langle k\rangle t}}\right)dtd\beta \end{aligned} \tag{10}$$

$$\begin{aligned} R_G^{SI}(T) &= \frac{1}{T}\int_0^1\int_0^T\left(1 - \frac{\left(1/i_0-1\right)e^{-\beta\langle k\rangle t}}{1+\left(1/i_0-1\right)e^{-\beta\langle k\rangle t}}\right)dtd\beta \\ &= \frac{1}{T}\int_0^1\left(\int_0^T 1dt - \int_0^T \frac{\left(1/i_0-1\right)e^{-\beta\langle k\rangle t}}{1+\left(1/i_0-1\right)e^{-\beta\langle k\rangle t}}dt\right)d\beta \\ &= \frac{1}{T}\int_0^1\left(t + \frac{1}{\beta\langle k\rangle}\ln\left(1+\left(1/i_0-1\right)e^{-\beta\langle k\rangle t}\right)\right)\Big|_{t=0}^{t=T}d\beta \\ &= \frac{1}{T}\int_0^1\left(T + \frac{1}{\beta\langle k\rangle}\ln\frac{\left(1+\left(1/i_0-1\right)e^{-\beta\langle k\rangle T}\right)}{1/i_0}\right)d\beta \\ &= 1 + \frac{1}{T}\int_0^1\left(\frac{1}{\beta\langle k\rangle}\ln\left(i_0 + (1-i_0)e^{-\beta\langle k\rangle T}\right)\right)d\beta. \end{aligned} \tag{11}$$

Case 2

The robustness of homogeneous networks with respect to the spread of the SIS model is calculated. Ignoring the degree of correlations in the nodes of the homogeneous networks, the density of the infected nodes at time t in the SIS epidemic model, i.e., $i(t)$, satisfies

$$\frac{di}{dt} = -\delta i + \beta\langle k\rangle i(1-i). \tag{12}$$

Integrating both sides of **Eq. 12**,

$$\int_0^t dt = \int_{i_0}^{i(t)} \frac{1}{-\delta i + \beta\langle k\rangle i(1-i)}di, \tag{13}$$

Then, **Eq. 13** can be rewritten as

$$t = \frac{1}{\beta\langle k\rangle - \delta}\int_{i_0}^{i(t)}\frac{1}{i}di + \frac{\beta\langle k\rangle}{\beta\langle k\rangle - \delta}\int_{i_0}^{i(t)}\frac{1}{\beta\langle k\rangle - \beta\langle k\rangle i - \delta}di, \tag{14}$$

We can obtain

$$e^{(\beta\langle k\rangle-\delta)t} = \frac{i(t)}{\beta\langle k\rangle - \beta\langle k\rangle i(t) - \delta}\Big/\frac{i_0}{\beta\langle k\rangle - \beta\langle k\rangle i_0 - \delta}, \tag{15}$$

After a simple combination, **Eq. 15** can be rewritten as

$$i(t)\left(\beta\langle k\rangle - \beta\langle k\rangle i_0 - \delta\right) = i_0 e^{(\beta\langle k\rangle-\delta)t}\left(\beta\langle k\rangle - \beta\langle k\rangle i(t) - \delta\right). \tag{16}$$

The density of the infected nodes at time t can be written as

$$i(t) = \frac{(\beta\langle k\rangle - \delta)i_0 e^{(\beta\langle k\rangle-\delta)t}}{\beta\langle k\rangle - \beta\langle k\rangle i_0 - \delta + i_0\beta\langle k\rangle e^{(\beta\langle k\rangle-\delta)t}}. \tag{17}$$

Let **Eq. 12** be equal to 0. We can obtain $-\delta i + \beta\langle k\rangle i(1-i) = 0$. When $\tau = \frac{\beta}{\delta} = \beta > \tau_c$, the infection density of the final stable state is

$$i_\infty = 1 - \frac{\delta}{\beta\langle k\rangle} = 1 - \frac{1}{\tau\langle k\rangle}. \tag{18}$$

Based on **Eqs. 5** and **17**, the robustness of the homogeneous network G with respect to the spread of the SIS epidemic can be written as

$$\begin{aligned} R_G^{SIS}(T) &= \frac{1}{T}\int_0^{\lambda_{\max}}\int_0^T i(t,s)dtds \\ &= \frac{1}{T}\int_0^{\lambda_{\max}}\int_0^T \frac{\left(\frac{\langle k\rangle}{s}-1\right)i_0 e^{\left(\frac{\langle k\rangle}{s}-1\right)t}}{\frac{\langle k\rangle}{s} - \frac{\langle k\rangle}{s}i_0 - 1 + i_0\frac{\langle k\rangle}{s}e^{\left(\frac{\langle k\rangle}{s}-1\right)t}}dtds \end{aligned} \tag{19}$$

Using simple operational processes, $R_G^{SIS}(T)$ can be rewritten as

$$\begin{aligned} R_G^{SIS}(T) &= \frac{1}{T}\int_0^{\lambda_{\max}}\int_0^T \frac{\left(\frac{\langle k\rangle}{s}-1\right)i_0 e^{\left(\frac{\langle k\rangle}{s}-1\right)t}}{\frac{\langle k\rangle}{s} - \frac{\langle k\rangle}{s}i_0 - 1 + i_0\frac{\langle k\rangle}{s}e^{\left(\frac{\langle k\rangle}{s}-1\right)t}}dtds \\ &= \int_0^{\lambda_{\max}}\frac{s}{T\langle k\rangle}\int_0^T \frac{1}{\frac{\langle k\rangle}{s} - \frac{\langle k\rangle}{s}i_0 - 1 + i_0\frac{\langle k\rangle}{s}e^{\left(\frac{\langle k\rangle}{s}-1\right)t}}d\left(\frac{\langle k\rangle}{s} - \frac{\langle k\rangle}{s}i_0 - 1 + i_0\frac{\langle k\rangle}{s}e^{\left(\frac{\langle k\rangle}{s}-1\right)t}\right)ds \\ &= \int_0^{\lambda_{\max}}\frac{s}{T\langle k\rangle}\ln\left(\frac{\langle k\rangle}{s} - \frac{\langle k\rangle}{s}i_0 - 1 + i_0\frac{\langle k\rangle}{s}e^{\left(\frac{\langle k\rangle}{s}-1\right)t}\right)\Big|_{t=0}^{t=T}ds \\ &= \int_0^{\lambda_{\max}}\frac{s}{T\langle k\rangle}\left(\ln\left(\langle k\rangle - \langle k\rangle i_0 - s + i_0\langle k\rangle e^{\left(\frac{\langle k\rangle}{s}-1\right)T}\right) - \ln(\langle k\rangle - s)\right)ds. \end{aligned} \tag{20}$$

RESULTS

Based on the new network robustness measurement we proposed, Monte Carlo simulations were performed to further explore the robustness of the different networks with respect to the spread of the epidemic. It is generally known that most of the real-world networks are characterized by a high clustering effect, a short average path length, and power law node degree distribution, i.e., small-world phenomenon and scale-free property. Therefore, WS small-world networks, BA scale-free networks, and several real-world networks were used in our simulations. All the simulation results were averaged over 500 runs.

Firstly, the BA and WS networks, with the same average degree, $\langle k\rangle = 6$, were used in our simulation to study the effect of degree distribution on the robustness of the networks. **Figure 4** shows the network robustness R_G^{SIS} at time T with respect to the spread of the SIS model. For simplicity, the recovery rate δ was set as 1. The curves in

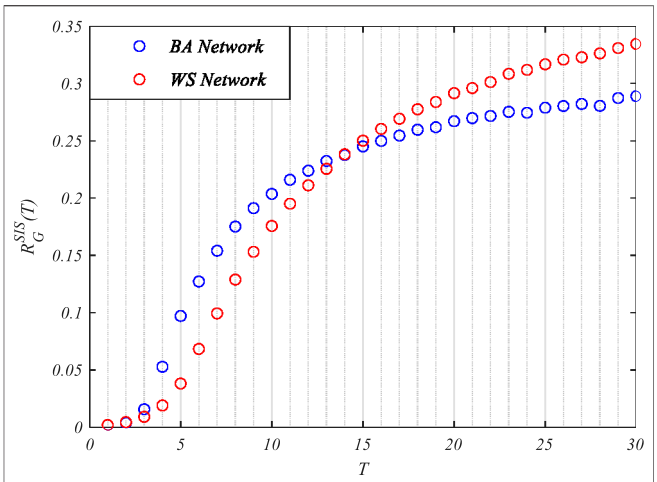

FIGURE 4 | The robustness of the Watts–Strogatz (WS) and Barabási–Albert (BA) networks with respect to the susceptible–infected–susceptible (SIS) epidemic model.

Figure 4 show that, when $T < 15$, $R_{BA}^{SIS} > R_{WS}^{SIS}$, i.e., the robustness of the WS network is stronger than that of the BA network. Due to the existence of a small fraction of hub nodes, the epidemic in the BA network is more likely to break out than that in the WS network. Therefore, at the early stage of the epidemic, the BA network is more fragile than the WS network because of the higher epidemic threshold and faster spread velocity. However, when the infection rate becomes more severe, the spread gradually slows down since most of the nodes in the BA network are of a lower degree than that of the average, and the infection scale in the WS network becomes larger than that in the BA network. Therefore, when $T \geq 15$, the WS network becomes more fragile than the BA network, i.e., $R_{BA}^{SIS} < R_{WS}^{SIS}$, as shown in **Figure 4**.

Moreover, the simulations were carried out in a group of WS small-world networks, where the irregularity/randomness of the networks increases as the value of the rewiring rate p grows following the generation algorithm of the WS network. Especially, when $p = 0$, the network is a regular graph; when $p = 1$, the network is completely random. **Figure 5** shows that, as p grows, R_G^{SIS} becomes larger; that is, the network becomes more vulnerable. Therefore, the irregularity/randomness of the network weakens the robustness of the homogeneous networks. In addition, compared with the robustness of the BA network (red circle), the homogeneous networks were more robust than the BA network at the early stage of the epidemics ($T < 8$ in **Figure 5**). Also, after $T > 15$, the BA network showed better robustness than the group of homogeneous networks. The results further extend our conclusion that, in the initial stage of propagation, the homogeneous networks showed better robustness than the heterogeneous networks. As the robustness gap grew smaller with the spread of the epidemic, and finally, the heterogeneous networks became more robust than the homogenous networks.

The above simulation results indicated that it is not adequate to simply conclude which network is more robust

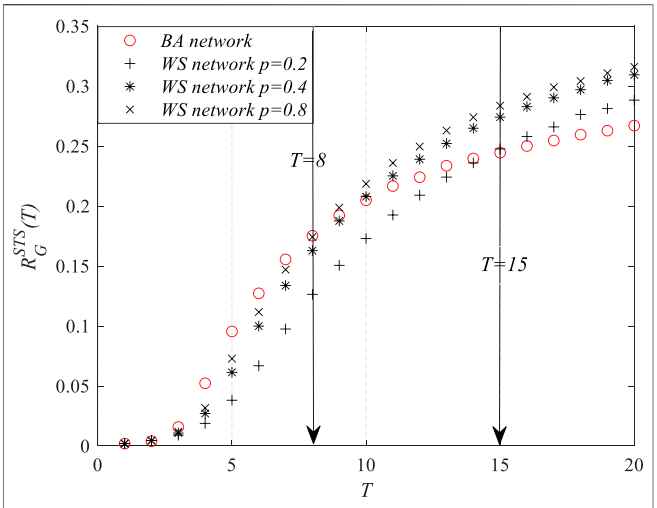

FIGURE 5 | The robustness of homogeneous networks at time T with respect to the susceptible–infected–susceptible (SIS) epidemic spread.

with respect to the epidemic. The robustness of the network changes with time, and the network does not always show a strong/weak robustness at all stages of the epidemic. During the early stage of the epidemic spread, the robustness of homogeneous networks was stronger than that of the heterogeneous networks. After the propagation reached the steady state, heterogeneous networks showed better robustness than the homogeneous networks.

To analyze the impact of the degree distribution on network robustness with respect to the epidemic, simulations were also carried out in the WS and BA networks with different average degrees, $\langle k \rangle$, as shown in **Table 1** and **Figure 6**. **Table 1** shows that, at the steady state ($T = 30$), the network became more vulnerable to virus attacks as the average degree of the network increases. The results validated the BA network as having stronger robustness than the WS network at the steady state.

Figure 6 shows the changes of network robustness as time T increased in the WS and BA networks with different average degrees, $\langle k \rangle$. The figure shows that the WS network exhibited stronger robustness than did the BA network with the same average degree at the early stage of the epidemic, and the robustness of the BA network was stronger than that of the WS network at the steady state.

We also applied the proposed metric to real-world networks where the dynamics processes can be described by epidemic models [24–27]. For example, the virus spread in e-mail networks, the information transfer in neural networks, and rumor diffusion in online social networks. In this paper, three real-world networks were used to validate our results on network models [28]: 1) e-mail network—the network of e-mail interchanges between members of the University Rovira i Virgili (Tarragona); 2) neural network—the network representing the neural network of *Caenorhabditis elegans*, which was compiled by D. Watts and S. Strogatz; and 3) Facebook network—the complete

TABLE 1 | The robustness of the Watts–Strogatz (WS) and Barabási–Albert (BA) networks with different <*k*> values at the steady state (T = 30)

	<*k*> = 4	**<*k*> = 6**	**<*k*> = 8**	**<*k*> = 10**
WS network	*0.2981*	*0.3492*	*0.3716*	*0.3842*
BA network	*0.2274*	*0.2880*	*0.3209*	*0.3488*

WS, *Watts–Strogatz;* BA, *Barabási–Albert*
(<k>) is the average degree of network.

Facebook network data (from a single-time snapshot in September 2005) of Caltech. Only intra-college links were included. The basic topological properties of these three networks are shown in **Table 2**. In order to study the impact of degree distribution on the robustness of real-world networks, new network models were created by rewiring the links in the real-world networks. After rewiring, the heterogeneity of the degree distribution of nodes was reduced, while the numbers of nodes and links remained unchanged, and the new created networks were connected graphs.

Figure 7 shows the impact of degree distribution on the robustness of real-world networks with respect to the spread of the SIS epidemic. We can see that, at the early stage of the epidemics, the robustness of the real-world networks (red circles) was worse than that of the new created network models (black circles). That is to say, the heterogeneity of the degree distributions of nodes can reduce the network robustness at the early stage of the epidemic. After the propagation reached a steady state, the robustness of the real-world networks (red circles) became stronger than that of the new created network models (black circles). The simulation results confirmed that homogeneous networks present stronger robustness than do heterogeneous networks at the early age of the epidemic, and the robustness of the heterogeneous networks becomes stronger than that of the homogeneous ones with the progress of the epidemic. In addition, we can see from **Figure 7** that the time point when the robustness of the real networks was stronger than that of the homogeneous networks was becoming earlier with the increase of the average degree (as shown in **Table 2**, the average degree was becoming larger from the e-mail network to the Facebook network, i.e., from **Figures 7A–C**).

In summary, the simulation showed different results from previous studies based on the new measures of network robustness with respect to the spread of epidemic proposed in this paper. Firstly, the robustness of the heterogeneous networks was not always better than that of the homogeneous networks. During the initial stage of propagation, the homogeneous networks showed better robustness than did the heterogeneous networks, and at the steady state, the heterogeneous networks became more robust than the homogenous networks. Furthermore, in both homogeneous and heterogeneous networks, the networks became more vulnerable as the average degree increased. In homogeneous networks, the robustness of the networks with respect to the spread of the virus decreased as p increased, i.e., the irregularity in the networks increased the vulnerability

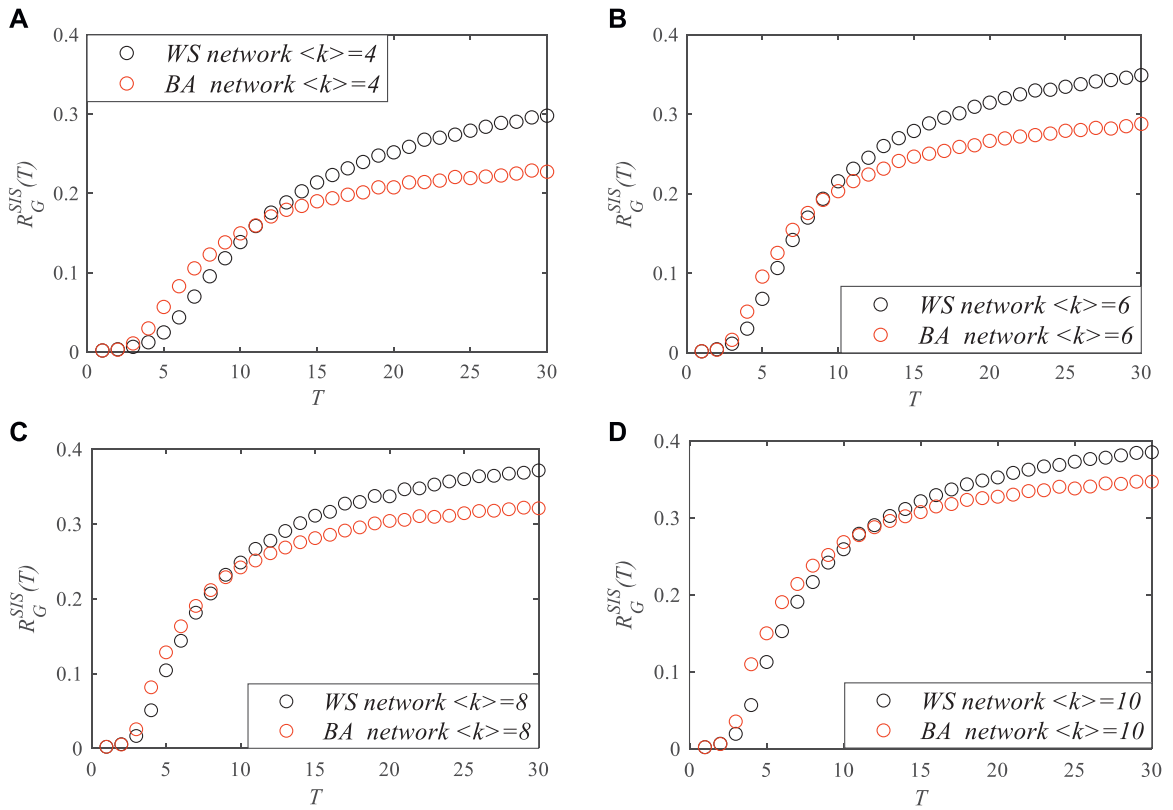

FIGURE 6 | The robustness of the Watts–Strogatz (WS) and Barabási–Albert (BA) networks with different <*k*> values at time *T* with respect to the susceptible–infected–susceptible (SIS) epidemic spread. **(A)** <*k*> = 4. **(B)** <*k*> = 6. **(C)** <*k*> = 8. **(D)** <*k*> = 10.

TABLE 2 | The real-world networks studied and their basic properties

Networks	*N*	*L*	<*k*>	*k*_max
E-mail network	1,133	5,451	9.62	71
Neural network	297	2,148	14.47	134
Facebook network	762	16,651	43.70	248

N *and* L *are the total numbers of nodes and links, respectively.* <k> *and* k_max *denote the average and the maximum degree, respectively.*

of the networks. The simulation results provided us with some ideas to enhance the network robustness with respect to the dynamic propagation processes. For example, at the beginning of the epidemic, mass gathering was harmful to improve network robustness, and after the epidemic entered a relatively stable period, avoiding small-scale clustering would help enhance the network robustness against epidemic spread.

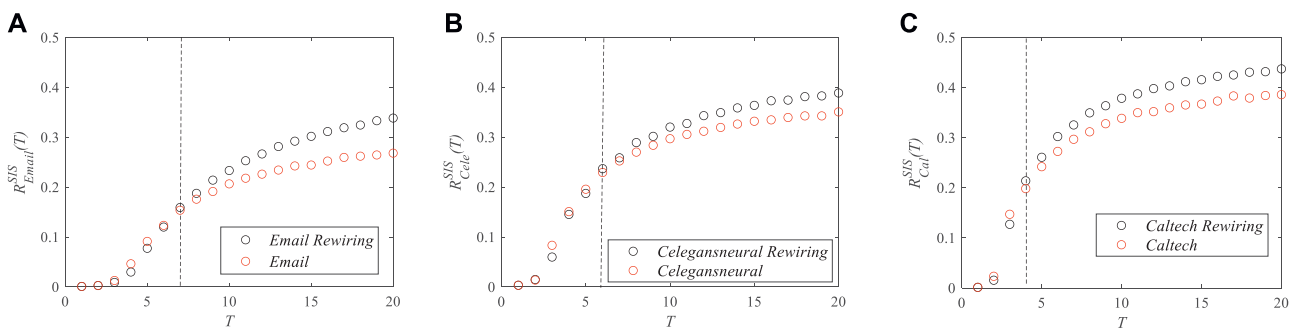

FIGURE 7 | The robustness of real-world networks at time *T* with respect to the susceptible–infected–susceptible (SIS) epidemic spread. **(A)** E-mail network. **(B)** Neural network. **(C)** Facebook network.

DISCUSSION

Considering the spread velocity, epidemic threshold, and the infection density at steady state, a novel metric to quantify network robustness with respect to epidemics was proposed in this paper. The real-time network robustness and the robustness of different networks were discussed. The simulation results showed some interesting conclusions of the impact of network structure on network robustness. The robustness of heterogeneous networks was not always stronger than that of the homogeneous networks. At the early stage of the epidemic, the homogeneous networks showed stronger robustness than did the heterogeneous networks, and at the steady state, the robustness of the heterogeneous networks was stronger than that of the homogeneous networks. In addition, the increase of irregularity and the average degree can enhance the network robustness with respect to epidemics. Our future work will explicitly focus on proposing a heuristic for computing the robustness metric for general networks. In addition, the metric proposed in our paper can be applied to network optimization to maximize network robustness with respect to different kinds of dynamic propagation processes.

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

BS was responsible for all aspects of the work. G-PJ provided the ideas for the analysis of the robustness of the real-time networks. YS and YG contributed to the analysis of the robustness of the real-time networks. JY contributed to the simulations in *Results*. XW contributed to the analysis of simulations in *Results*.

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