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Research Article

Global Stability of a Variation Epidemic Spreading Model on Complex Networks

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Epidemic spreading on networks becomes a hot issue of nonlinear systems, which has attracted many researchers' attention in recent years. A novel epidemic spreading model with variant factors in complex networks is proposed and investigated in this paper. One main feature of this model is that virus variation is investigated in the process of epidemic dynamical spreading. The global dynamics of this model involving an endemic equilibrium and a disease-free equilibrium are, respectively, discussed. Some sufficient conditions are given for the existence of the endemic equilibrium. In addition, the global asymptotic stability problems of the disease-free equilibrium and the endemic equilibrium are also investigated by the Routh-Hurwitz stability criterion and Lyapunov stability criterion. And the uniform persistence condition of the new system is studied. Finally, numerical simulations are provided to illustrate obtained theoretical results.

1. Introduction

The research of infectious disease has always been a hot issue of nonlinear systems with applications. The popular dynamics on complex network is the epidemic spreading, which describes how infections spread throughout a network [1]. In recent years, much research work has been done about the viral dynamics of epidemic spreading [2, 3]. These results are helpful for preventing and controlling most emerging infectious diseases like SARS, HIV/AIDS, H5N1, and H1N1. They are also meaningful to provide important information for the research in the field of rumor spreading [4–7], traffic dynamics [8–10], computer viruses [11, 12], biology mechanism [13], and medicine developing [14–16].

In real world, the population size is large enough such that the mixing of individuals can be considered to be homogeneous. Social and biological systems can be properly described as complex networks with nodes representing individuals and links mimicking the interactions [17, 18]. Suitable mathematical models of the infectious disease spreading in complex homogeneous networks are of great practical value to analyze the detailed spreading process. Because epidemic spreading usually brings great harm to society, it is very urgent to establish accurate propagation models considering the infection contagion spreading problems. In past decades, complicated *SIR* models were formulated from different perspectives of epidemiology [19, 20]. In these models, S, I, and R denote, respectively, the number of individuals susceptible to the disease, the number of infectious individuals, and the number of individuals who are recovered from being infectious. The process of epidemic spreading can be further modeled with differential equations, such as *SIS*, *SIR*, and *SIRS* model [21–26]. In these research works the network topological structure is simplified presumptively to regular network or sufficient mixing homogeneous network, where the relationship between the network structure and the epidemic spreading is discussed. Kephart et al. [27] established a virus spreading model based on a homogeneous network by characterizing the average degree as the network metrics and obtained a virus spreading threshold $\tau_c = 1/\langle k \rangle$, while Pastor-Satorras [28–31] studied the epidemic outbreaks in complex heterogeneous network, which chose the degree and average degree as the network metrics and obtained the epidemic spreading threshold $\tau_c = \langle k \rangle / \langle k^2 \rangle$. Moore and Newman [32] applied the percolation theory to analyze the epidemic spreading behaviors in small-world network and

showed the differences of spreading action between small-world network and regular network. These research results illustrate that the different network topological structure can affect the epidemic spreading.

Apart from the network topological structure, one of the most important characteristics of epidemic spreading models is the dynamical stability which can reflect the development of the spreading behaviors of infectious disease. Hence, the stability problems of these epidemic models need to be investigated. Kuniya [21] applied a discretization method to prove the global asymptotic stability of the *SIR* model with the age structure. Zhang and Feng [22] deal with the global analysis of a dynamical model describing the spread of tuberculosis with isolation and incomplete treatment. Lahrouz et al. [23] studied a nonlinear *SIRS* model with saturated birth and death rates, and the global asymptotic stability of the model is also discussed. Xu et al. [24] analyzed a time-delayed *SIRS* model with temporary immunity, and some conditions for the globally asymptotically stability of the disease-free equilibrium and the endemic equilibrium are given. Besides, for the epidemic model with time-delay Kang and Fu [25] presented a new *SIS* model with an infective vector on scale-free networks and the global stability of equilibrium is proved. The influences of treatment and vaccination efforts on a dynamic disease model in presence of incubation delays and relapse are studied and sufficient conditions for the local stability of the equilibrium are derived [26].

However, few papers are available in the literature to consider variant factors in the epidemic spreading from a systematic framework. In real world, certain variants exist in the infectious disease transmitting, resulting from some factors including gene mutation and cell division environment. Viruses evolve rapidly because they have strong ability of propensity for genetic variation and short generation time, which leads to evading human immunization response and obtaining drug resistance. For example, influenza viruses can be classified into three major types (A, B, and C). There are many different virus forms because of mutation; type A infects many animal species including humans, while type B and type C viruses are mainly human pathogens. If individuals are affected by viruses, not all infected individuals can be recovered. Some of them may suffer from other diseases because virus variation or the infectious individuals contacted with variants. In fact, some infectious persons, who may be infected by some diseases, would have certain probability to become variant members of another group. It is necessary to propose a new model considering this condition. How to build models with variant factors in the epidemics spreading becomes a challenge. Therefore, the paper presents a novel *SIVRS* epidemic spreading model considering variant factors, where S stands for the susceptible and I, V, and R stand for the infectious, the variant, and the recovered, respectively.

Given the mechanism of the *SIVRS* model in a homogeneous network, which is only composed by blank nodes initially, the entire population can be divided into four groups described by the symbols of S, I, V, and R, respectively. They denote four epidemiological statuses: susceptible, infectious, variant, and recovered. All new individuals are supposed to be blank nodes in complex networks. When a susceptible individual contacts the other infected individual, this individual may be infectious with certain probability. Then an infectious individual would have only three states including infectious, variant, and recovered. The infectious individuals would become the variants with certain probability affected by some factors such as gene mutation and the indeterminacy of cell division. Similarly, an infectious person may become a variant with certain probability after contacting with a variant. Usually, human body can be protected by one's immune system. Some infectious individuals with recovery probability may become the recovered, while others will keep the infectious status. We assume that the four groups have the same mortality rate.

In this paper, a novel *SIVRS* epidemic spreading model with virus variation in complex homogeneous network is proposed and investigated. The rest of this paper is organized as follows. In Section 2, the propagation mechanism of the *SIVRS* model in complex networks is presented, and mean-field equations are used to describe the dynamics of epidemic spreading model with virus variation. The existence of endemic equilibrium is considered. Section 3 is devoted to discuss the global stability of the disease-free equilibrium, which is followed by the discussion of the system uniform persistence in Section 4. Then the proofs of global stability of an endemic equilibrium are presented in Section 5. In Section 6, numerical simulations are performed to illustrate obtained theoretical results. Finally, conclusions are given in Section 7.

2. Epidemic Spreading Model and Its Property

2.1. The SIVRS Model. As described above, in the paper a new *SIVRS* model is established. The model involves a new variant group which is caused by the infectious variation. Assume the number of nodes is N in a closed complex network, which includes four statuses susceptible S, infectious I, variant V, and recovered V as well as some initial blank nodes. All new nodes produced from blank nodes are susceptible.

The flow chart of epidemic spreading is shown in Figure 1.

Assume in a homogeneous network only composed by blank nodes initially the susceptible individuals are produced by the blank nodes; the probability is characterized as δ. Others come from the recovered group with the probability ϕ. A susceptible individual will become infectious with probability α if he/she contacts the infected individual. Then an infectious individual may perhaps become one variant when he/she has tight relation with variants or is affected by other factors. We assume that the variant probability is γ when contacting with a variant. In the process of epidemic spreading, an infectious individual may become the variant with internal probability η. Some infectious individuals with recovery probability β may recover and others will keep the infectious status. In this paper, the four groups are supposed to have the same mortality rate μ. Then the *SIVRS* epidemic spreading rules can be summarized as follows.

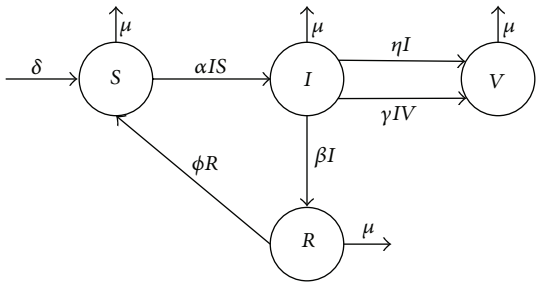


Figure 1: A schematic representation of *SIVRS* epidemic spreading model.

(1) Apart from the four groups in a closed network, there are blank nodes which exist in initial network. The blank nodes may become susceptible ones with probability δ, namely, crude birth rate.

(2) The susceptible individual becomes infectious with probability α when contacting with an infectious one, namely, infection rate.

(3) An infectious individual can be recovered with probability β, namely, recovery rate.

(4) The variants coming from some of the infectious nodes at a variation rate η (internal variation rate) can reflect the variation factors. When an infectious individual contacts with a variant, the individual may become a variant with probability γ (contact variant rate).

(5) The recovered node turns into susceptible with probability ϕ after a period of time due to the loss of immunity. For the four groups in the network, all individuals will become blank with probability μ, namely, natural mortality rate.

A closed and homogeneous network consisting of N individuals is investigated in this paper. Individuals in the network can be represented with nodes and the contact between different individuals can be denoted by edges. Then the network can be described by an undirected graph $G = (V, E)$, where V and E denote the set of nodes and edges, respectively. Therefore, a differential equation model is derived based on the aforementioned rules and the basic assumptions:

$$\begin{aligned}
\frac{dS}{dt} &= \delta(1-N) - \alpha\langle k\rangle SI + \phi R - \mu S,\\
\frac{dI}{dt} &= \alpha\langle k\rangle SI - \gamma\langle k\rangle IV - (\eta+\beta+\mu)I,\\
\frac{dV}{dt} &= \gamma\langle k\rangle IV + \eta I - \mu V,\\
\frac{dR}{dt} &= \beta I - \phi R - \mu R,
\end{aligned} \tag{1}$$

where $\langle k\rangle$ denotes the average degree of the network.

The total population satisfies $N = S + I + V + R$, and the following equation is obtained:

$$\frac{dN}{dt} = \delta - (\delta+\mu)N \tag{2}$$

which is derived by adding the four equations in (1).

In (2) N will eventually tend to $N_0 = \delta/(\delta+\mu)$ with the exponential decay. Therefore, assume that $N(0) = N_0$. The closed and positively invariant set for (1) is $\Sigma = \{(S, I, V, R) \in \mathbb{R}_+^4 : 0 \le S + I + V + R = N_0 \le 1\}$, where $\mathbb{R}_+^4$ denotes the nonnegative cone of $\mathbb{R}^4$ with its lower dimensional faces. Use $\partial\Sigma$ and Σ° to denote the boundary and interior of Σ in $\mathbb{R}_+^4$, respectively.

2.2. Existence of Equilibrium. The system (1) has a disease-free equilibrium (DFE) E_0, where

$$E_0 = (S_0, I_0, V_0, R_0) = \left(\frac{\delta}{\delta+\mu}, 0, 0, 0\right). \tag{3}$$

Denote the basic reproduction number parameter as

$$R_0 = \frac{\alpha\langle k\rangle S_0 - \gamma\langle k\rangle V_0}{\eta+\beta+\mu} = \frac{\alpha\langle k\rangle\delta}{(\delta+\mu)(\eta+\beta+\mu)}. \tag{4}$$

The following theorem summarizes the parameter restrictions on the existence of equilibrium.

Theorem 1. *If $R_0 > 1$ and the inequality*

$$1 > \eta > \frac{\alpha(1-\gamma\langle k\rangle)}{\alpha+\gamma} \tag{5}$$

is satisfied, there are two endemic equilibria for system (1).

Proof. Assume that $E^* = (S^*, I^*, V^*, R^*)$ is an endemic equilibrium (EE) of system (1). According to system (1), we have

$$\begin{aligned}
\delta(1-N^*) - \alpha\langle k\rangle S^*I^* + \phi R^* - \mu S^* &= 0,\\
\alpha\langle k\rangle S^*I^* - \gamma\langle k\rangle I^*V^* - (\eta+\beta+\mu)I^* &= 0,\\
\gamma\langle k\rangle I^*V^* + \eta I^* - \mu V^* &= 0,\\
\beta I^* - \phi R^* - \mu R^* &= 0.
\end{aligned} \tag{6}$$

For (6) a straightforward calculation leads to

$$\begin{aligned}
S^* &= \frac{\eta+\beta+\mu}{\alpha\langle k\rangle} + \frac{\gamma\eta I^*}{\alpha(\mu-\gamma\langle k\rangle I^*)},\\
V^* &= \frac{\eta I^*}{\mu-\gamma\langle k\rangle I^*},\\
R^* &= \frac{\beta}{\mu+\phi}I^*.
\end{aligned} \tag{7}$$

From (7) $\mu - \gamma\langle k\rangle I^* > 0$, which implies that

$$0 < I^* < \min\left\{1, \frac{\mu}{(\gamma\langle k\rangle)}\right\}, \tag{8}$$

the component I^* is a positive solution of

$$p(I^*) = AI^{*2} + BI^* + C = 0, \tag{9}$$

where

$$\begin{aligned} A &= \alpha\gamma \langle k\rangle^2 (\mu + \phi + \beta), \\ B &= -\eta \langle k\rangle (\mu + \phi)(\alpha + \gamma) - \alpha \langle k\rangle \mu (\mu + \phi + \beta) \\ &\quad + (\mu + \phi)(\eta + \beta + \mu)(1 - \gamma \langle k\rangle R_0), \\ C &= \mu (\eta + \beta + \mu)(\mu + \phi)(R_0 - 1). \end{aligned} \tag{10}$$

Therefore, consider the following:

(1) If $R_0 < 1$, we have $C < 0$; (9) has only one positive solution.

(2) If $R_0 = 1$, we have $C = 0$; (9) has only one positive solution $-B/A$.

(3) If $R_0 > 1$, we have $C > 0$:

(i) if $B > 0$, the positive solution of (9) does not exist;

(ii) if $B < 0$ and $B > -2\sqrt{AC}$, the solution of (9) does not exist;

(iii) if $B < 0$ and $B = -2\sqrt{AC}$, (9) has only one positive solution;

(iv) if $B < 0$ and $B < -2\sqrt{AC}$, (9) has two positive solutions.

Therefore, if $R_0 > 1$, the solution of (9) exists only when $B \le -2\sqrt{AC}$.

According to the inequality $a + b \ge 2\sqrt{ab}$, $\forall a, b \in Z^+$, assume $R_0 > 1$, choose $a = \alpha\langle k\rangle\mu(\mu + \phi + \beta)$, $b = \gamma\langle k\rangle(\eta + \beta + \mu)(\mu + \phi)(R_0 - 1)$, and then

$$\begin{aligned} &2\sqrt{AC} \\ &= 2\sqrt{\alpha\gamma \langle k\rangle^2 \mu (\mu + \phi + \beta)(\eta + \beta + \mu)(\mu + \phi)(R_0 - 1)} \\ &< \alpha \langle k\rangle \mu (\mu + \phi + \beta) \\ &\quad + \gamma \langle k\rangle (\eta + \beta + \mu)(\mu + \phi)(R_0 - 1) \\ &= \alpha \langle k\rangle \mu (\mu + \phi + \beta) + \gamma \langle k\rangle R_0 (\eta + \beta + \mu)(\mu + \phi) \\ &\quad - \gamma \langle k\rangle (\eta + \beta + \mu)(\mu + \phi). \end{aligned} \tag{11}$$

If the inequality $\gamma\langle k\rangle(\eta + \beta + \mu)(\mu + \phi) > (\mu + \phi)[\eta + \beta + \mu - \eta\langle k\rangle(\alpha + \gamma)]$ is satisfied, we have

$$\eta + \beta + \mu < \frac{\eta \langle k\rangle (\alpha + \gamma)}{1 - \gamma \langle k\rangle}. \tag{12}$$

Then the following equation is obtained:

$$\begin{aligned} 2\sqrt{AC} &< \alpha \langle k\rangle \mu (\mu + \phi + \beta) \\ &\quad + \gamma \langle k\rangle R_0 (\eta + \beta + \mu)(\mu + \phi) \\ &\quad - \gamma \langle k\rangle (\eta + \beta + \mu)(\mu + \phi) \\ &< \alpha \langle k\rangle \mu (\mu + \phi + \beta) \\ &\quad + \gamma \langle k\rangle R_0 (\eta + \beta + \mu)(\mu + \phi) \\ &\quad - [\eta + \beta + \mu - \eta \langle k\rangle (\alpha + \gamma)](\mu + \phi) \\ &< \alpha \langle k\rangle \mu (\mu + \phi + \beta) \\ &\quad + (\eta + \beta + \mu)(\mu + \phi)(\gamma \langle k\rangle R_0 - 1) \\ &\quad + \eta \langle k\rangle (\mu + \phi)(\alpha + \gamma) < -B \end{aligned} \tag{13}$$

which implies system (1) has two endemic equilibria.

When $R_0 > 1$,

$$\frac{\alpha \langle k\rangle}{\eta + \beta + \mu} > R_0 = \frac{\alpha \langle k\rangle \delta}{(\delta + \mu)(\eta + \beta + \mu)} > 1 \tag{14}$$

which can transfer to inequality $\alpha\langle k\rangle > \eta + \beta + \mu$.

If $\alpha < \eta(\alpha+\gamma)/(1-\gamma\langle k\rangle)$, we have $1 > \eta > \alpha(1-\gamma\langle k\rangle)/(\alpha+\gamma)$. Then inequality (12) is satisfied. □

Remark 2. According to this theorem, if the reproduction number parameter is above the threshold, then the endemic equilibrium is globally asymptotically stable, which will be discussed further in Section 5.

3. Global Stability of the Disease-Free Equilibrium

Definition 3. If the equilibrium is stable under the meaning of Lyapunov, for $\delta(\varepsilon, t_0)$ and $\forall\mu > 0$, there is real number $T(\mu, \delta, t_0) > 0$ which makes any initial value x_0 of inequality $\|x_0 - x_e\| \le \delta(\varepsilon, t_0)$, $t \ge t_0$, satisfy the following inequality:

$$\left\|\phi(t; x_0, t_0 - x_e)\right\| \le \mu, \quad \forall t \ge t_0 + T(\mu, \delta, t_0); \tag{15}$$

then the equilibrium is asymptotically stable.

The Jacobian matrix at the disease-free equilibrium E_0 of system (1) is

$$J(E_0) = \begin{pmatrix} -\delta - \mu & -\delta - \alpha\langle k\rangle S_0 & -\delta & -\delta + \phi \\ 0 & \alpha\langle k\rangle S_0 - (\eta + \beta + \mu) & 0 & 0 \\ 0 & \eta & -\mu & 0 \\ 0 & \beta & 0 & -\mu - \phi \end{pmatrix}. \tag{16}$$

Obviously, if $R_0 < 1$, all eigenvalues of matrix (16) are negative. Then the disease-free equilibrium E_0 is locally asymptotically stable in Σ. Moreover, if $R_0 > 1$, there is one positive eigenvalue and E_0 is unstable.

Theorem 4. *If $R_0 < 1$, the disease-free equilibrium (DFE) E_0 is globally asymptotically stable in Σ and if $R_0 > 1$, the disease-free equilibrium (DFE) E_0 is unstable in Σ.*

Proof. Let $L(S, I, V, R) = I > 0$ as a Lyapunov function; then $L(E_0) = 0$. When $R_0 < 1$

$$\begin{aligned}\frac{dL}{dt}(S, I, V, R) &= \alpha \langle k \rangle SI - \gamma \langle k \rangle IV - (\eta + \beta + \mu) I \\ &< \alpha \langle k \rangle SI - (\eta + \beta + \mu) I \\ &< I(\alpha \langle k \rangle N_0 - (\eta + \beta + \mu)) \\ &< I(\eta + \beta + \mu)(R_0 - 1) < 0.\end{aligned} \tag{17}$$

L is positive definite and $\dot{L}$ is negative definite. Therefore, the disease-free equilibrium (DFE) E_0 is globally asymptotically stable in Σ; the following result can be given. □

4. Uniform Persistence

In this section, the uniform persistence of system (1) will be discussed when the basic reproduction number $R_0 > 1$.

Definition 5 (see [33]). System (1) is said to be uniformly persistent if there exists a constant $0 < c < 1$, which makes any solution $(S(t), I(t), V(t), R(t))$ with $(S(0), I(0), V(0), R(0)) \in \Sigma^\circ$ satisfy

$$\begin{aligned}&\min\left\{\lim_{t\to\infty}\inf S(t), \lim_{t\to\infty}\inf I(t), \lim_{t\to\infty}\inf V(t), \lim_{t\to\infty}\inf R(t)\right\} \\ &\geq c.\end{aligned} \tag{18}$$

Let X be a locally compact metric space with metric ∂ and let Γ be a closed nonempty subset of X with boundary $\partial\Gamma$ and interior Γ°. Obviously, $\partial\Gamma$ is a closed subset of Γ. Let Φ_t be a dynamical system defined on Γ. A set B in X is said to be invariant if $\Phi_t(B, t) = B$. Define $M_\partial := \{x \in \partial\Gamma : \Phi_t x \in \partial\Gamma,\ \forall t \geq 0\}$.

Lemma 6 (see [34]). *Assume the following:*

(H1) *Φ_t has a global attractor.*

(H2) *There exists an $M = \{M_1, \ldots, M_k\}$ of pair-wise disjoint, compact, and isolated invariant set on $\partial\Gamma$ such that*

(a) *$\bigcup_{x\in M_\partial} \omega(x) \subset \bigcup_{j=1}^{k} M_j$;*

(b) *no subsets of M form a cycle on $\partial\Gamma$;*

(c) *each M_j is also isolated in Γ;*

(d) *$W^s(M_j) \cap \Gamma^\circ = \phi$ for each $1 \leq j \leq k$, where $W^s(M_j)$ is the stable manifold of M_j. Then Φ_t is uniformly persistent with respect to Γ°.*

According to Lemma 6, the following result is obtained.

Theorem 7. *When $R_0 > 1$, system (1) is uniformly persistent.*

Proof. Let

$$\begin{aligned}\Gamma &= \Sigma = \left\{(S, I, V, R) \in \mathbb{R}_4^+ \mid 0 \leq S + I + V + R \leq 1\right\}, \\ \Gamma^\circ &= \{(S, I, V, R) \in E : I, V > 0\}, \\ \partial\Gamma &= \frac{\Gamma}{\Gamma^\circ}.\end{aligned} \tag{19}$$

Obviously, $M_\partial = \partial\Gamma$.

Choose $M = \{E_0\}$, $\omega(x) = \{E_0\}$ for all $x \in M_\partial$. On $\partial\Gamma$, system (1) reduces to $S' = \delta - (\delta + \mu)S$, in which $S(t) \to \delta/(\delta + \mu)$ as $t \to \infty$. It is concluded that $M = \{E_0\}$, $\omega(x) = \{E_0\}$ for all $x \in M_\partial$, which indicates that hypotheses (a) and (b) hold. When $R_0 > 1$, the disease-free equilibrium E_0 is unstable according to Theorem 4 $W^s(M) = \partial\Gamma$. Hypotheses (c) and (d) are then satisfied. Due to the ultimate boundedness of all solutions of system (1), there is a global attractor, making (H1) true. □

5. Global Dynamics of Endemic Equilibrium

From the previous analysis, the disease dies out when $R_0 > 1$; then the disease becomes endemic. In this section, Lyapunov asymptotic stability theorem is used to investigate the globally asymptotic stability of the endemic equilibrium E^* when $R_0 > 1$.

Theorem 8. *The endemic equilibrium E^* is globally asymptotically stable in Σ, whenever $R_0 > 1$.*

Proof. Consider the following function:

$$\begin{aligned}V_1 = \ln\big[&(S - S^*) + (I - I^*) + (V - V^*) + (R - R^*) \\ &+ 1\big].\end{aligned} \tag{20}$$

Then the derivative of V_1 along the solution of (1) is given by

$$\begin{aligned}\dot{V}_1 &= \frac{\partial V_1}{\partial S}\frac{dS}{dt} + \frac{\partial V_1}{\partial I}\frac{dI}{dt} + \frac{\partial V_1}{\partial V}\frac{dV}{dt} + \frac{\partial V_1}{\partial R}\frac{dR}{dt} \\ &= \frac{(dS + dI + dV + dR)(1/dt)}{(S - S^*) + (I - I^*) + (V - V^*) + (R - R^*) + 1}.\end{aligned} \tag{21}$$

From (2), all solutions of (6) satisfy the equality

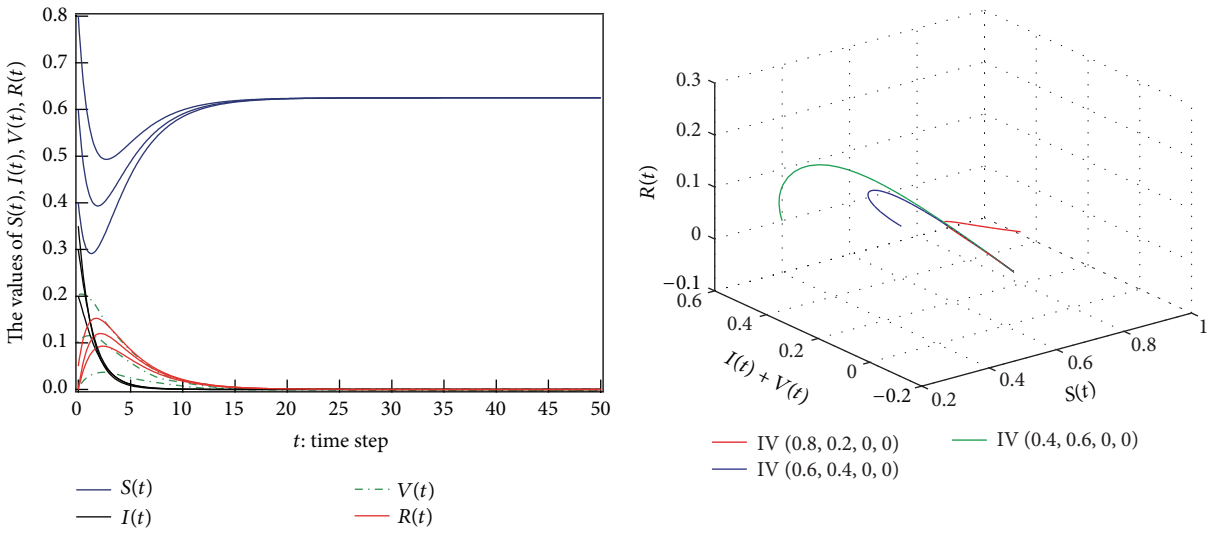
$$N^* = S^* + I^* + V^* + R^* = \frac{\delta}{\delta + \mu} \tag{22}$$

and $N = e^{-(\delta+\mu)t+C} + \delta/(\delta + \mu) \leq \delta/(\delta + \mu)$, where C is the value that makes $N_0 = \delta/(\delta + \mu)$ satisfied.

Hence $V_1 = \ln(N - N^* + 1) \geq 0$; then

$$\begin{aligned}\dot{V}_1 &= \frac{1}{N - \delta/(\delta + \mu) + 1}\frac{dN}{dt} \\ &= \frac{\delta + \mu}{N - \delta/(\delta + \mu) + 1}\left(\frac{\delta}{\delta + \mu} - N\right) \leq 0.\end{aligned} \tag{23}$$

If and only if $N = \delta/(\delta + \mu)$, $V_1 = 0$ and $\dot{V}_1 = 0$ are satisfied.



(a) The states of disease-free equilibrium

(b) The evolution for the disease-free system states

FIGURE 2: (a) and (b) showed that the disease-free equilibrium E_0 of the system (1) is globally asymptotically stable with different initial conditions $(0.8, 0.2, 0, 0)$, $(0.6, 0.3, 0.1, 0)$, and $(0.4, 0.35, 0.2, 0.05)$ and the parameters $\langle k \rangle = 10$, $\alpha = 0.8$, $\beta = 0.5$, $\eta = 0.2$, $\delta = 0.5$, $\gamma = 0.2$, $\phi = 0.01$, and $\mu = 0.3$; $R_0 = 0.5 < 1$. The value of DFE is $E_0 = (0.625, 0, 0, 0)$.

V_1 is positive definite and $\dot{V}_1$ is negative definite. Therefore, the function V_1 is a Lyapunov function for system (1) and the endemic equilibrium E^* is globally asymptotically stable by Lyapunov asymptotic stability theorem [35]. The proof is completed. □

6. Numerical Simulation

To demonstrate the theoretical results obtained in this paper, some numerical simulations will be discussed. In this paper, the hypothetical set of initial values (IV) and parameter values will be given as follows.

Consider the initial values of $(S(0), I(0), V(0), R(0))$ are set as $(0.8, 0.2, 0, 0)$, $(0.6, 0.3, 0.1, 0)$, and $(0.4, 0.35, 0.2, 0.05)$, respectively.

(1) The disease-free equilibrium: Set $\langle k \rangle = 10$, $\alpha = 0.08$, $\beta = 0.5$, $\eta = 0.2$, $\delta = 0.5$, $\gamma = 0.02$, $\phi = 0.01$, and $\mu = 0.3$. $R_0 = 0.5 < 1$ and the disease-free equilibrium $E_0 = (0.625, 0, 0, 0)$ from the parameter values above through the calculation. According to Theorem 4, the disease-free equilibrium E_0 of system (1) is globally asymptotically stable in Σ in this case. The simulation results are shown in Figures 2(a) and 2(b).

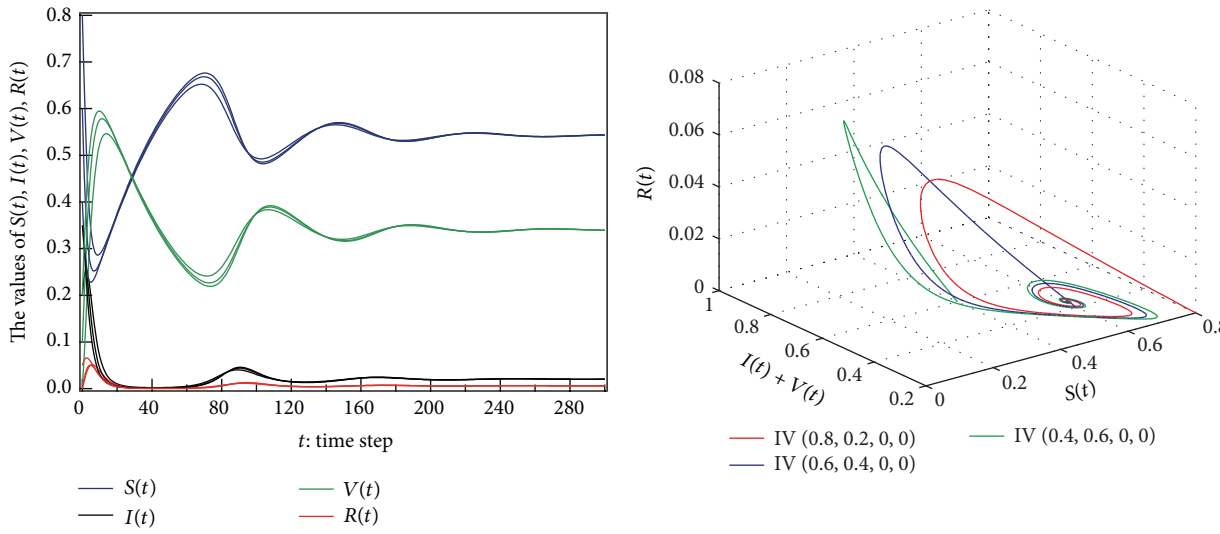
(2) The endemic equilibrium: Set $\langle k \rangle = 10$, $\alpha = 0.08$, $\beta = 0.08$, $\eta = 0.3$, $\delta = 0.2$, $\gamma = 0.01$, $\phi = 0.25$, and $\mu = 0.02$. By direct computation, $R_0 = 1.818 > 1$ and the endemic equilibrium $E^* = (0.5435, 0.0201, 0.3395, 0.00596)$ can be obtained from the parameter values above. According to Theorem 4, the positive endemic equilibrium E^* of system (1) is globally asymptotically stable in Σ°. The simulation results are shown in Figures 3(a) and 3(b).

Figure 2 shows that if $R_0 < 1$, all solutions in Σ would be attracted to the disease-free equilibrium E_0 regardless of the initial values of system (1), which illustrates the validity of Theorem 4. Similarly, it can be seen from Figure 3 that all solutions in Σ° would be attracted to the endemic equilibrium E^* regardless of the initial values of system (1) if $R_0 > 1$ and the conditions of Theorem 8 are satisfied, which is obviously the content of Theorem 4. Moreover, the relationship between the values of equilibrium can be verified as shown in (24), which is coincident with the theoretical results:

$$S_0 + I_0 + V_0 + R_0 = S^* + I^* + V^* + R^* = -\frac{\delta}{\delta + \mu}. \quad (24)$$

7. Conclusion

The stability of the *SIVRS* epidemic spreading model with virus variation in complex networks has been discussed in this paper. The model involves a new variant group which is caused by the infectious variation. By analyzing the model, the disease-free equilibrium E_0 is proved to exist when the basic reproduction number R_0 is less than 1. The analysis result reveals that the infectious disease dies out when R_0 is more than 1 and it becomes endemic. The existing conditions of endemic equilibrium related with the variation rate and the network nodes degree are obtained. Besides, the global asymptotically stability condition of the disease-free equilibrium is obtained by the Routh-Hurwitz stability criterion and the Lyapunov stability criterion. And the condition of the system uniform persistence is also given. The proof of the stability of endemic equilibrium is also illustrated. Finally, a numerical simulation is given to illustrate the correctness of the disease-free equilibrium and the endemic equilibrium results.



(a) The system states of endemic equilibrium

(b) The evolution for the endemic system states

FIGURE 3: (a) and (b) showed that the disease-free equilibrium E^* of system (1) is globally asymptotically stable with different initial conditions $(0.8, 0.2, 0, 0)$, $(0.6, 0.3, 0.1, 0)$, and $(0.4, 0.35, 0.2, 0.05)$ and the parameter values $\alpha = 0.08$, $\beta = 0.08$, $\eta = 0.3$, $\delta = 0.2$, $\gamma = 0.1$, $\phi = 0.25$, $\mu = 0.02$, and $\langle k \rangle = 10$; $R_0 = 1.818 > 1$. The value of EE is $E^* = (0.5435, 0.0201, 0.3395, 0.00596)$.

Conflict of Interests

The authors have declared that no competing interests exist.

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