Modeling the dynamics of epidemic spreading on homogenous and heterogeneous networks

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Modeling the dynamics of epidemic spreading on homogenous and heterogeneous networks

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This paper proposes two modified susceptible-infected-recovered-susceptible models on homogenous and heterogeneous networks, respectively. In the study of the homogenous network model, it is proved that if the basic reproduction number $R_0$ of the model is less than one, then the disease-free equilibrium is locally asymptotically stable and becomes globally asymptotically stable under the condition that the threshold value $R_1$ is less than one. Otherwise, if $R_0$ is more than one, the endemic equilibrium is locally asymptotically stable and becomes globally asymptotically stable under the assumption that the total population $N$ will tend to a specific plane. In the study of the heterogeneous network model, this paper discusses the existences of the disease-free equilibrium and endemic equilibrium of the model. It is proved that if the threshold value $R_0$ is less than one, then the disease-free equilibrium is globally asymptotically stable. Otherwise, if $R_0$ is more than one, the system is permanent. A series of numerical experiments are given to illustrate the theoretical results. We also numerically predict the effect of vaccination ratio on the size of HBV-infected mainland Chinese population.

Keywords: SIRS model; homogenous network; heterogeneous network; local stability; global stability

AMS Subject Classifications: 92B05; 91D30

1. Introduction

During the past decade, the research based on mathematical modeling of infectious disease spreading on complex networks has received increasing attention.[1–4] Complex network has also penetrated into the society,[5] economy,[6] biological science,[7] and other research areas.[8–10] A typical network is composed of nodes which represent individuals or organizations and links which mimic the interactions or connections among them.[9,11–14] Recently, the dynamic behaviors of the susceptible-infected-susceptible (SIS) model, the susceptible-infected-recovered (SIR) model, and the susceptible-infected-recovered-susceptible (SIRS) model have been widely studied on complex networks.[7,15–22]

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The studies of the SIS models and the SIR models on homogenous networks (e.g. small-world network [15] and random rules [23]) showed that there exists a threshold: below this threshold, a disease will eventually disappear; otherwise, the disease will become endemic.

In most of the studies of epidemic spreading on complex networks, the population dynamics factors are not considered. Liu et al. [22] have introduced a modified epidemic model with birth and death on homogeneous and heterogeneous networks. Through mean-field analysis, they found that on homogeneous network, there is an epidemic threshold $\lambda_c$, while for a heterogeneous network, the epidemic threshold is absent in the thermodynamic limit. The result is the same as that of the standard SIS model.

However, there is a connotation that the threshold values of those models are dependent on the total size of population, which means that the more the total number of the population, the easier the disease outbreaks. This is not supported by practical observations.

We first introduce a modified SIRS on homogenous network model with vaccinated population, consider different death rates for susceptible individuals and infected individuals, and study the stabilities of the disease-free equilibrium and the endemic equilibrium of the model. Secondly, we propose a SIRS model on heterogeneous network, and discuss the dynamic behaviors of the model. The threshold values of the two models are independent of the total size of population. Illustrative results of numerical simulation on the two models are given.

The rest of this paper is organized as follows. Section 2 describes the transition rules among three states. Section 3 introduces a SIRS model on homogenous networks, presents the global epidemic dynamics and numerical simulation results. Section 4 describes the SIRS model on heterogeneous networks, formulates the epidemic threshold, establishes the globally asymptotically stability of disease-free equilibrium, and presents illustrative results of numerical simulations. Section 5 concludes this paper.

2. Transition rules

For the SIRS model, each individual can be in three states. $S$, $I$, and $R$ represent the susceptible, infected, and recovered or vaccinated individuals, respectively. Transition among these states is governed by the following rules.[7] First, a susceptible individual can acquire infection from an infected neighbor at rate $\beta$. Then, an infected individual is cured at rate $\kappa$ and becomes susceptible again at rate $\gamma$. At the same time, the susceptible individual is vaccinated at rate $\nu$. The constant $\lambda$ is the recruitment rate of susceptibles corresponding to births and immigration, $\mu$ is the natural death rate of population. And $\alpha$ is the disease-related death rate. One assumes that all the newborns are susceptible individuals. The transmission sketch is described in Figure 1, where all the parameters are positive constants.

3. SIRS model on homogenous networks

Different from previous works, we propose a more realistic model on homogenous networks, considering vaccination factors. In order to establish the SIRS model on homogenous networks, we consider two hypotheses:

(1) Homogeneity: The network is homogeneous. For simplicity, assume that each node has degree $k_i \approx \langle k \rangle$, $\langle k \rangle$ is the average connectivity in the network neglecting the heterogeneity of the node degrees.
(2) Homogenous mixing: The infection strength is proportional to the population density.

Combining the above hypotheses, the proposed novel SIRS model is formulated by the following system:

\[
\begin{align*}
    \frac{dS}{dt} &= \lambda - \beta \langle k \rangle SI S + I + R + \gamma R - (\nu + \mu) S, \\
    \frac{dI}{dt} &= \beta \langle k \rangle SI S + I + R - (\alpha + \kappa + \mu) I, \\
    \frac{dR}{dt} &= \kappa I - (\mu + \gamma) R + \nu S.
\end{align*}
\]

(3.1)

We denote the total population as \( N = S + I + R \). Adding all equations in system (3.1) gives the rate of changes of the total population:

\[
\frac{dN}{dt} = \frac{d}{dt}(S + I + R) = \lambda - \mu N - \alpha I,
\]

(3.2)

It is easy to see that the set \( \Gamma \) is a positive invariant set of (3.1):

\[
\Gamma = \left\{ (S, I, R) \in \mathbb{R}_+^3 : S \geq 0, \ I \geq 0, \ R \geq 0, \ S + I + R \leq \lambda / \mu \right\}
\]

3.1. The necessary condition of disease spreading

If disease spreading occurs, then \( I(t)_{|t=0} > 0 \). This implies that \( \beta \langle k \rangle S(0)I(0)/N(0) - (\alpha + \kappa + \mu) I(0) > 0 \). Solving this inequality gives the following,

**Theorem 3.1** If the initial fraction of susceptible individual \( S(0)/N(0) \) in system (3.1) satisfies the following inequality

\[
\frac{S(0)}{N(0)} > \frac{\alpha + \kappa + \mu}{\beta \langle k \rangle},
\]

(3.3)

then a disease transmission will occur.
Whether a disease appears is related to the average degree of network $\langle k \rangle$. When $\langle k \rangle$ is small, a disease transmission can hardly appear. Otherwise, it will be relatively easy to occur. Reduction of the propagation rate can also lead to the possibility of the decrease in disease transmission. Therefore, to prevent the transmission of a disease, we can take some practical approaches, such as consciously reducing interpersonal contacts ($\beta \langle k \rangle$), especially with those infected ones. Moreover, high disease-related death rate ($\alpha$) and cure rate ($\kappa$) of infected individuals are also important factors to prevent the spreading of a pandemic disease.

3.2. Local stability of equilibria

In this subsection, we discuss the local stability of disease-free equilibrium and endemic equilibrium of system (3.1) by analyzing the corresponding characteristic equations. The system (3.1) always has a disease-free equilibrium

$$E_1 = \left( \frac{\lambda(\gamma + \mu)}{\mu(\gamma + \mu + \nu)}, 0, \frac{\lambda \nu}{\mu(\gamma + \mu + \nu)} \right) \equiv (S^0, 0, R^0).$$

(3.4)

Define

$$R_0 = \frac{\beta \langle k \rangle (\gamma + \mu)}{(\alpha + \kappa + \mu)(\gamma + \mu + \nu)},$$

(3.5)

$$A = \alpha + \kappa + \mu, \ B = \gamma + \mu + \nu.$$

(3.6)

Then, if $R_0 > 1$, system (3.1) has a unique endemic equilibrium

$$E_2 = (S^*, I^*, R^*),$$

(3.7)

where

$$S^* = \frac{\lambda AB}{AB[\mu R_0 + \alpha(R_0 - 1)] + \beta \langle k \rangle \mu \kappa},$$

$$I^* = \frac{\lambda AB(R_0 - 1)}{AB[\mu R_0 + \alpha(R_0 - 1)] + \beta \langle k \rangle \mu \kappa},$$

$$R^* = \frac{\kappa I^* + \nu S^*}{\mu + \gamma}.$$

$R_0$ is called the basic reproduction number of system (3.1). One important feature of SIRS epidemic model (3.1) is that $R_0$ does not depend on the total size $N$ of population.

Theorem 3.2 For system (3.1),

(i) If $R_0 < 1$, then the disease-free equilibrium $E_1$ in $\Gamma$ is locally asymptotically stable.

(ii) If $R_0 > 1$, then the endemic equilibrium $E_2$ in $\Gamma$ is locally asymptotically stable.
Proof  At a point \( E(S, I, R) \) of system (3.1), the Jacobian matrix is

\[
J = \begin{bmatrix}
\frac{\beta(k)SI}{N^2} - \nu - \frac{\beta(k)I}{N}\beta(k)SI - \nu - \frac{\mu}{\kappa} - \beta(k)SI - \frac{\beta(k)S}{N} - \frac{\beta(k)SI}{N} - \frac{\gamma + \beta(k)SI}{N^2} - \frac{\beta(k)SI}{N} - \frac{(\alpha + \mu + \kappa)}{\kappa}
\end{bmatrix}.
\]

(i) The Jacobian matrix at \( E_1 \) of the vector field corresponding to system (3.1) is

\[
J(E_1) = \begin{bmatrix}
-\nu - \mu & -\frac{\beta(k)(\gamma + \mu)}{N^2} & \gamma \\
0 & \frac{\beta(k)(\gamma + \mu)}{\gamma + \mu + \nu - (\alpha + \mu + \kappa)} & 0 \\
\nu & \frac{\beta(k)(\gamma + \mu)}{\kappa} & -(\gamma + \mu)
\end{bmatrix}.
\]

Solving the corresponding eigenequation gives three eigenvalues,

\[x_1 = -\mu, \quad x_2 = -(\gamma + \mu + \nu), \quad x_3 = -(\alpha + \mu + \kappa)(1 - R_0)\]

Observe that \( x_1 < 0 \) and \( x_2 < 0, x_3 < 0 \) if \( R_0 < 1 \) and \( x_3 > 0 \) if \( R_0 > 1 \). Hence, \( E_1 \) is locally asymptotically stable if \( R_0 < 1 \), and it is unstable if \( R_0 > 1 \).

(ii) As \( E_2 = (S^*, I^*, R^*) \) is an equilibrium of system (3.1), where \( S^*, I^*, R^* > 0 \), we get

\[
\frac{\beta(k)S^*I^*}{N^*} - (\alpha + \mu + \kappa)I^* = 0,
\]

in which \( N^* = S^* + I^* + R^* \). To facilitate the analysis, let

\[
p = \frac{I^*}{S^*}, \quad \tau = \frac{I^*}{N^*}, \quad w = (\alpha + \mu + \kappa) = \frac{\beta(k)S^*}{N^*}.
\]

Furthermore, we have

\[
\frac{\beta(k)S^*I^*}{(N^*)^2} = w\tau, \quad \frac{\beta(k)I^*}{N^*} = wp, \quad \frac{\beta(k)S^*}{N^*} = w, \quad p - \tau > 0.
\]

With those facilitations, we get

\[
det(\lambda E - J(E_2)) = \begin{vmatrix}
\lambda + \mu + \nu + w(p - \tau) & w(1 - \tau) & -\gamma - w\tau \\
-w(p - \tau) & \lambda + \nu \tau & w \tau \\
-\nu & -\kappa & \lambda + (\gamma + \mu)
\end{vmatrix}
\[
= \lambda^3 + a_1\lambda^2 + a_2\lambda + a_3,
\]
where \( J(E_2) \) is the Jacobian matrix of system (3.1) at \( E_2 \). By calculating straightforwardly, we obtain that

\[
\begin{align*}
\alpha_1 &= \gamma + 2\mu + v + pw > 0, \\
\alpha_2 &= (p - \tau)w^2 + (\gamma p + \kappa \tau + p\mu + \tau\mu)w + \gamma\mu + \mu v + \mu^2 > 0, \\
\alpha_3 &= [p(\gamma + \mu) - \tau(\mu + v + \gamma)]w^2 + [\tau(\mu + v + \gamma)(\mu + \kappa) - \gamma\kappa p]w, \\
\alpha_1\alpha_2 - \alpha_3 &= p(p - \tau)w^3 + [p(\gamma p + \kappa \tau + p\mu + \tau\mu) + \mu(p - \tau) + pv]w^2 \\
&\quad + [p(\gamma \mu + \mu v + \mu^2) + (\gamma + 2\mu + v)(\gamma p + \kappa \tau + p\mu + \tau\mu) \\
&\quad + \tau\mu(\kappa + \mu) + \gamma\kappa p]w \\
&\quad + (\gamma + 2\mu + v)(\gamma \mu + \mu v + \mu^2) > 0.
\end{align*}
\]

Denoting \( B^* = \frac{\beta\langle k \rangle S^* I^*}{N^*} > 0 \), we obtain the following equations

\[
\begin{align*}
0 &= \lambda - B^* + \gamma R^* - (\mu + v)S^*, \\
0 &= B^* - (\alpha + \kappa + \mu)I^*, \\
0 &= \kappa I^* - (\mu + \gamma)R^* + vS^*, \\
0 &= \lambda - \mu N^* - \alpha I^*.
\end{align*}
\]

It follows that

\[
\begin{align*}
S^* &= \frac{1}{\mu + v + \gamma} \left[ \lambda - B^* + \gamma \left( \frac{\lambda}{\mu} - \frac{\alpha B^*}{\mu(\alpha + \kappa + \mu)} - I^* \right) \right], \\
I^* &= \frac{1}{\alpha + \kappa + \mu}, \\
R^* &= \frac{1}{\mu + \gamma} (\kappa I^* + v S^*), \\
N^* &= \frac{1}{\mu} \left( \lambda - \frac{\alpha B^*}{\alpha + \kappa + \mu} \right).
\end{align*}
\]

Using (3.10), we have

\[
\begin{align*}
\frac{\tau}{p} &= \frac{\lambda - B^* + \gamma \left( \frac{\lambda}{\mu} - \frac{\alpha B^*}{\mu(\alpha + \kappa + \mu)} - I^* \right)}{\mu + v + \gamma} \times \frac{\mu}{\lambda - \frac{\alpha B^*}{\alpha + \mu + \kappa}} \\
&= \left[ (\mu + \gamma) - \frac{\mu B^* + \mu \gamma I^* - \frac{\mu \alpha B^*}{\alpha + \mu + \kappa}}{\lambda - \frac{\alpha B^*}{\alpha + \mu + \kappa}} \right] \times \frac{1}{\mu + v + \gamma}, \\
\frac{\tau}{p} &< \frac{\mu + \gamma}{\mu + v + \gamma}.
\end{align*}
\]
Noticing that \( w = \alpha + \mu + \kappa \), we can rewrite \( a_3 \) as
\[
\begin{align*}
    a_3 &= [p(\gamma + \mu) - \tau(\mu + v + \gamma)]w^2 + [\tau(\mu + v + \gamma)(\mu + \kappa) - \gamma \kappa \rho]w \\
&= w[p(\gamma + \mu) - \gamma \kappa \rho] + w \tau[(\mu + v + \gamma)(\mu + \kappa) - w(\mu + v + \gamma)] \\
&= w[(\alpha + \mu + \kappa)p(\gamma + \mu) - \gamma \kappa \rho] + w \tau[(\mu + v + \gamma)(\mu + \kappa) - w(\mu + v + \gamma)] \\
&= w[(\alpha + \mu + \kappa)p(\gamma + \mu) - \gamma \kappa \rho] - w \tau \alpha(\mu + v + \gamma) \\
&= wp \left[ (\alpha + \mu + \kappa)(\gamma + \mu) - \gamma \kappa - \frac{\tau}{p} \alpha(\mu + v + \gamma) \right].
\end{align*}
\]
Using (3.11) yields
\[
\begin{align*}
a_3 &> wp \left[ (\alpha + \mu + \kappa)(\gamma + \mu) - \gamma \kappa - \frac{\mu + \gamma}{\mu + v + \gamma} \alpha(\mu + v + \gamma) \right] \\
&= wp[\mu(\gamma + \mu) + \kappa \mu] > 0.
\end{align*}
\]
Since \( a_1 > 0, a_2 > 0 \), and \( a_1a_2 - a_3 > 0 \), all inequalities of the Routh-Hurwitz criterion are satisfied. Therefore, the endemic equilibrium \( E_2 \) is locally asymptotically stable if \( R_0 > 1 \). This completes the proof. \( \square \)

### 3.3. Globally asymptotical stability

In this subsection, we will discuss the global asymptotical stability of the disease-free equilibrium \( E_1 \) of system (3.1).

**Theorem 3.3** Define
\[
R_1 = \frac{\beta(k)}{\alpha + \kappa + \mu}. \tag{3.12}
\]
If \( R_1 < 1 \), then the disease-free equilibrium \( E_1 \) of system (3.2) is globally asymptotically stable in \( \Gamma \).

**Proof** If \( R_1 < 1 \), then we have
\[
I' = \frac{\beta(k)SI}{S + I + R} - (\alpha + \kappa + \mu)I = (\alpha + \kappa + \mu)I \left( R_1 \frac{S}{S + I + R} - 1 \right) \leq (\alpha + \kappa + \mu)I(R_1 - 1) \leq 0.
\]
It follows that \( \lim_{t \to \infty} I(t) = 0 \).

Next, we shall show that \( \lim_{t \to \infty}(S(t), R(t)) = (S^o, R^o) \). To this end, we shall apply Thieme’s result [24] which states that bounded solutions of asymptotically autonomous systems, in our case system (3.1), converge to the \( \omega \)-limit set of a reduced system which resulted from the system of (3.1) by taking \( I(t) = 0 \). In other words, the \( S \) and \( R \) components of any bounded solution \((S(t), I(t), R(t))\) of system (3.1) converge to the \( S \) and \( R \) components of the \( \omega \)-limit set of the following system
\[
\begin{align*}
\frac{dS}{dt} &= \lambda + \gamma R - (v + \mu)S = \gamma(R - R^o) - (v + \mu)(S - S^o), \\
\frac{dR}{dt} &= vS - (\mu + \gamma)R = v(S - S^o) - (\mu + \gamma)(R - R^o). \tag{3.13}
\end{align*}
\]
Let $X = S - S^o$, $Y = R - R^o$, and $V(S, R) = [v(S - S^o)^2 + \gamma(R - R^o)^2]$. Then

$$
\frac{dV}{dt} = 2\nu X(\gamma Y - (\nu + \mu)X) + 2\gamma Y(\nu X - (\mu + \gamma)Y)
$$

$$
= -2(\nu X - \gamma Y)^2 - 2\nu \mu X^2 - 2\gamma \mu Y^2
$$

Clearly, all solutions of the reduced system (3.13) tend to $(S^o, R^o)$ which implies that the \(\omega\)-limit set of system (3.13) is $(S^o, R^o)$. Therefore, $\lim_{t \to \infty} (S(t), R(t)) = (S^o, R^o)$. This proves the theorem. \(\square\)

Remark 1 By vast simulations, we have found that when $R_0 < 1$, the trajectory of system (3.1) always converges to the disease-free equilibrium $E_1$. We guess that under the condition $R_0 < 1$, the disease-free equilibrium $E_1$ may be also globally asymptotically stable.

Now, we will discuss, under an assumption, the globally asymptotically stability of $E_2$ in $\Gamma$, when $R_0 > 1$. First, we define

$$
X = \frac{\mu S}{\lambda}, \quad Y = \frac{\mu I}{\lambda}, \quad Z = \frac{\mu R}{\lambda}.
$$

Furthermore, let

$$
\tilde{i} = \mu t, \quad \tilde{\beta} = \frac{\beta}{\mu}, \quad \tilde{\alpha} = \frac{\alpha}{\mu}, \quad \tilde{\gamma} = \frac{\gamma}{\mu}, \quad \tilde{v} = \frac{v}{\mu}, \quad \tilde{k} = \frac{k}{\mu},
$$

and

$$
\tilde{N} = X + Y + Z = \frac{\mu N}{\lambda}.
$$

Then, we can rewrite system (3.1) as

$$
\begin{cases}
X'(\tilde{i}) = 1 - \frac{\tilde{\beta}(k)XY}{\tilde{N}} + \tilde{\gamma} Z - (1 + \tilde{v})X, \\
Y'(\tilde{i}) = \frac{\tilde{\beta}(k)XY}{\tilde{N}} - (1 + \tilde{\alpha} + \tilde{k})Y, \\
Z'(\tilde{i}) = \tilde{k} Y - (1 + \tilde{\gamma})Z + \tilde{v} X.
\end{cases}
$$

(3.14)

Hence, the equation of $N$ becomes $\tilde{N}'(\tilde{i}) = 1 - \tilde{N} - \tilde{\alpha} Y$, and the invariant set $\Gamma$ become

$$
\bar{\Gamma} = \{(X, Y, Z)|X \geq 0, \ Y \geq 0, \ Z \geq 0, \ X + Y + Z \leq 1\}.
$$

(3.15)

Corresponding to formulas (3.4) and (3.7), we denote the disease-free equilibrium and the endemic equilibrium of system (3.14) by $\tilde{E}_1$ and $\tilde{E}_2$, respectively.

Define

$$
D = \{(X, Y, Z)|X \geq 0, \ Y \geq 0, \ Z \geq 0, \ X + Y + Z = 1\},
$$

(3.16)

$$
D_1 = \left\{ \left(\tilde{N}, Y, Z\right) \in \bar{\Gamma} : \tilde{N} = 1 - \tilde{\alpha} Y \right\}
$$

$\quad= \left\{ (X, Y, Z) \in \bar{\Gamma} : X + (1 + \tilde{\alpha})Y + Z = 1 \right\}.
$$

(3.17)
Then, $\tilde{N}' = 0$ in set $D_1$, $\tilde{N}' < 0$ if $\tilde{N} > 1 - \bar{a}Y$ and $\tilde{N}' > 0$ if $\tilde{N} < 1 - \bar{a}Y$. Furthermore, vast simulation results show that the flow of system (3.14) always tends to the plane $D_1$ with initial values in $\tilde{\Gamma}$ (see Figure 2). This motivates us to give the following proposition under an assumption which seems to be evident but has not been proved by us. First, we need the following:

**Lemma 3.4** Let $\tilde{\Gamma}$ and $D_1$ be defined by (3.15) and (3.17), respectively. Then System (3.14) has no periodic solution in the invariant domain $D_1$.

**Proof** Obviously, the boundary curve of the domain $D_1$ cannot form the periodic solution of system (3.14). We consider the following in the interior of $D_1$. Assuming that system (3.14) has periodic solution $\Phi(t) = (X(t), Y(t), Z(t))$, the image $H$ of $\Phi(t)$ is the boundary of a plane domain $\Pi$ which is in the interior of domain $D_1$.

Let $f_1, f_2, f_3$ denote the functions of the right-hand side in system (3.14), respectively. Let $f = (f_1, f_2, f_3)^T$ ($T$ denotes transpose), $g(X, Y, Z) = (1/XYZ) \cdot r \times f$ (where $r = (X, Y, Z)^T$), then $g \cdot f = 0$. By calculating straightforwardly, we get in the interior of domain $D_1$

$$(\text{curl } g) \cdot (1, 1 + \bar{a}, 1)^T = -\left(\frac{\bar{v}}{YZ^2} + \frac{1 + \bar{a}}{X^2Z} + \frac{1 + \bar{v}}{X^2Y}\right) < 0.$$ 

If we choose the direction of plane domain $\Pi$ upward, the direction of the image $D_1$ conforms to the right-hand rule with the direction of plane domain $\Pi$. Vector $(1, 1 + \bar{a}, 1)$ is the normal vector of plane domain $\Pi$, then we get by Stoker’s theorem

$$\frac{1}{\sqrt{3 + 2\bar{a} + \bar{a}^2}} \int \int \int (\text{curl } g) \cdot (1, 1 + \bar{a}, 1)^T dS = \oint_H \frac{g \cdot f}{|f|} ds.$$ 

This is in contradiction with the calculation above, $g \cdot f = 0$ and $(\text{curl } g) \cdot (1, 1 + \bar{a}, 1)^T < 0$. This completes the proof. \qed

Now, we give the following:

**Proposition 3.5** If system (3.14) satisfies the following:

**Assumption I** For any initial value $(X_0, Y_0, Z_0)$ in the domain $\Psi_1$ surrounded by planes $D_1$, $D_1$, $X > 0$, $Y > 0$, and $Z > 0$, or in the domain $\Psi_2$ surrounded by planes $D_1$, $X > 0$, $Y > 0$, and $Z > 0$, the flow in $\tilde{\Gamma}$ of system (3.14), tends to $D_1$ when $t \to \infty$ (see Figure 2).

Then when the threshold value

$$R_0 = \frac{\beta(k)(\gamma + \mu)}{(\alpha + \kappa + \mu)(\gamma + \mu + \nu)} = \frac{\bar{\beta}(k)(1 + \bar{v})}{(1 + \bar{a} + \bar{\kappa})(1 + \bar{v} + \bar{\mu})} > 1,$$

the endemic equilibrium $\tilde{E}_2$ is globally asymptotically stable in

$$\tilde{\Gamma}^* = \tilde{\Gamma} - \{ (X, Y, Z) \in \tilde{\Gamma} : Y = 0 \},$$

and all the solutions in $\{ (X, Y, Z) \in \tilde{\Gamma} : Y = 0 \}$ tend to $\tilde{E}_1$. 

Figure 2. Domain $\Psi$ surrounded by planes $D_1$, $X > 0$, $Y > 0$, and $Z > 0$. Assume that plane $D_1$ is an attractive set for any trajectory with an initial condition in $\tilde{\Gamma}$.

**Proof** Under assumption I, $D_1$ is an attracting invariant set. Similar to Theorem 3.2, we get that $\tilde{E}_1$ is unstable if $R_0 > 1$ and the unique positive equilibrium (denoted by $\tilde{E}_2$) of system (3.14) is locally asymptotically stable. Considering $\tilde{E}_1$ is unstable and Lemma 3.4, all solutions contained in any neighborhood of $\tilde{E}_1$ in $\tilde{\Gamma}^*$ will leave the field when $t$ become large enough.

Therefore, the trivial equilibrium, $\tilde{E}_1$, is not an attractor in $\tilde{\Gamma}^*$. On the other hand, $\tilde{E}_2$ is locally asymptotically stable, and system (3.14) has no periodic solution in $D_1$. Under assumption I, it follows that the unique positive equilibrium $\tilde{E}_2$ is a global attractor.

As system (3.1) and system (3.14) are equivalent, we get the following:

**Proposition 3.6** If

$$R_0 = \frac{\beta(k)(\gamma + \mu)}{(\alpha + \kappa + \mu)(\gamma + \mu + \nu)} > 1.$$  

Then under assumption I, the endemic equilibrium $E_2$ of system (3.1) is globally stable in $\Gamma^* = \Gamma - \{(S, I, R) \in \Gamma : I = 0\}$, and all the solutions in $\{(S, I, R) \in \Gamma : I = 0\}$ tend to the disease-free equilibrium $E_1$.

### 3.4. Numerical simulations

In this section, we will give two numerical examples with different system parameters to illustrate the theoretical results.
Example 1 Firstly, we select the following parameters in system (3.1).

(1) $\langle k \rangle = 6$, $\lambda = 1$, $\mu = 0.001$, $\alpha = 0.00087$, $\nu = 0.3$, $\beta = 0.2$, $\gamma = 0.1$, $\kappa = 0.5$. From formula (3.5) and (3.12), the basic reproduction number $R_0 = 0.6022 < 1$ and the threshold value $R_1 = 2.3911 > 1$.

(2) $\langle k \rangle = 6$, $\lambda = 1$, $\mu = 0.001$, $\alpha = 0.00087$, $\nu = 0.005$, $\beta = 0.2$, $\gamma = 0.1$, $\kappa = 0.5$, then the basic reproduction number $R_0 = 2.2783 > 1$.

We select two different initial states as follows:

(3) $S(0) = 450$, $I(0) = 550$, $R(0) = 0$.
(4) $S(0) = 800$, $I(0) = 200$, $R(0) = 0$.

Figure 3(a) shows the dynamic trajectories of system (3.1) when we select the system parameters defined by (1) with initial condition (3). In this case, the basic reproduction number $R_0 < 1$. Observe that when $R_0 < 1$, even if for a large fraction of the infected individuals at the initial time, the disease will eventually disappear.

Figure 3(b) shows the dynamic trajectories of system (3.1) when one selects the system parameters defined by (2) with initial condition (4). In this case, the basic reproduction number $R_0 > 1$. Observe that, the disease will converge to a positive stationary level, even if for a small fraction of the infected individuals at the initial time. This means that the endemic state is globally attractive.

Example 2 Now, we give an example based on Hepatitis B virus (HBV) infection data of the mainland Chinese collected from the Internet.

According to the 2010 Chinese Census,[25] the total size of population of Mainland China was 1,339,724,852 persons. An increase of 73,899,804 persons from the previous census conducted in 2000 represented an average annual growth rate of 0.57%. Therefore, we estimate that the total size of population of Mainland China in 2006 is about $1.3096 \times 10^9$, because 86.74% of the population was aged between 1 and 59.[25] Multiplying that population by 86.74%, we can get that the total size of population of Mainland China, aged between 1 and 59, is about $1.136 \times 10^9$ in 2006.
Table 1. Parameters used for simulation.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
<th>Data source</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\lambda$</td>
<td>13552480</td>
<td>[27]</td>
</tr>
<tr>
<td>$\mu$</td>
<td>0.00714</td>
<td>[27]</td>
</tr>
<tr>
<td>$\alpha$</td>
<td>0.0007</td>
<td>[28]</td>
</tr>
<tr>
<td>$\beta$</td>
<td>0.13</td>
<td>Estimated</td>
</tr>
<tr>
<td>$\gamma$</td>
<td>0.01</td>
<td>Estimated</td>
</tr>
<tr>
<td>$\kappa$</td>
<td>0.073</td>
<td>Estimated</td>
</tr>
<tr>
<td>$\langle k \rangle$</td>
<td>6</td>
<td>Estimated</td>
</tr>
<tr>
<td>$\nu$</td>
<td>0.2707</td>
<td>[29]</td>
</tr>
</tbody>
</table>

Figure 4. The dynamic trajectories of system (3.1) with parameters in Table 1.

According to the serum epidemiological survey of Hepatitis B virus from 1992 to 1995 in China, the rate of infection of HBV in China was 57.6%.[26] Calculating $1.136 \times 10^9 \times 57.6\%$, we can estimate that the population of the recovered individuals of the year 2006, aged between 1 to 59, is $6.54 \times 10^8$. The positive rate of HBsAg of the population aged between 1 and 59 was 7.18%. Calculating $1.136 \times 10^9 \times 7.18\%$ gives that the population of the HBV-infected individuals are about $8.56 \times 10^7$. Then the rest of the population aged between 1 and 59 in 2006, about $4.0 \times 10^8$, are the susceptible. Therefore, we obtain the initial status:

$$S(0) = 4.0 \times 10^8, \quad I(0) = 8.56 \times 10^7, \quad R(0) = 6.54 \times 10^8.$$  

The selected parameter values of system (3.1) are summarized in Table 1. It follows formula (3.5) that $R_0 = 0.5746 < 1$. 

The dynamic trajectories of system (3.1) are shown in Figure 4. The simulated data show that the size of the infected population in 2010 is about 120.36 million, which is in agreement with the practical HBV-infected population – 12 million of China in 2010.[30]

The long-term simulation predicts that the ratio of the HBV-infected individuals will decrease to 0.5% until 2104, which is the level of the HBV-infected population ratio in the Northern America.[31]
If increasing the percentage ($\nu$) of the vaccinated population to $\nu = 0.5$ and $\nu = 0.9$, the required time that reduces to 0.5% HBV-infected population will decrease from 98 (2104) to 58 (2064) and 45 (2051) years, respectively. Figure 5(a) and (b) shows the dynamic evolutions of the SIRS system (3.1), respectively.

It can be concluded that increasing the percentage ($\nu$) of the vaccinated population is a very effective way for the HBV spread control.

4. SIRS model on heterogeneous networks

In this section, we propose a modified SIRS model on heterogeneous networks. $X_k(t)$, $Y_k(t)$, and $Z_k(t)$ are defined as in system (3.14), representing the fraction of nodes with degree $k$ that are susceptible, infectious, and recovered, respectively. To account for the heterogeneous distribution of contacted individual numbers, the following assumptions are proposed: (1) $k$ can vary as any positive integer. (2) the structured population is described by a connectivity distribution $p(k)$, which is the probability that an individual has $k$ contacts.

At the mean-field level, for undirected random-graph type of uncorrelated spare networks, the proposed SIRS model on heterogeneous networks has the following form:

$$
\begin{align*}
\frac{dX_k(t)}{dt} &= 1 - \frac{\tilde{\beta}kX_k(t)\Theta(t)}{X_k(t) + Y_k(t) + Z_k(t)} + \tilde{\gamma}Z_k(t) - (1 + \tilde{\nu})X_k(t), \\
\frac{dY_k(t)}{dt} &= \frac{\tilde{\beta}kX_k(t)\Theta(t)}{X_k(t) + Y_k(t) + Z_k(t)} - (1 + \tilde{\alpha} + \tilde{\kappa})Y_k(t), \\
\frac{dZ_k(t)}{dt} &= \tilde{\kappa}Y_k(t) - (1 + \tilde{\gamma})Z_k(t) + \tilde{\nu}X_k(t)
\end{align*}
$$

where $\Theta(t)$ denotes the probability of a contact pointing to an infected individual. It is assumed that the connectivity of nodes on this network is uncorrelated, so

$$
\Theta(t) = \frac{\sum_i ip(i)Y_i(t)}{\langle k \rangle},
$$

where $p(i)$ is the connectivity distribution, $\langle k \rangle$ is the average degree of the network, i.e. $\langle k \rangle = \sum_i ip(i)$. 

Figure 5. The dynamic trajectories of system (3.1). (a) $\nu = 0.5$. (b) $\nu = 0.9$. 

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Denote $\tilde{N}_k = X_k + Y_k + Z_k$. Then $\tilde{N}'_k(t) = 1 - \tilde{N}_k - \tilde{\alpha}Y_k$. It can be proved that the invariant set is

$$\tilde{\Gamma}_k = \{(X_k, Y_k, Z_k) | X_k \geq 0, Y_k \geq 0, X_k + Y_k + Z_k \leq 1\}.$$ 

Denote $D_k = \{(X_k, Y_k, Z_k) | X_k \geq 0, Y_k \geq 0, Z_k \geq 0, X_k + Y_k + Z_k = 1\}$,

$$D_{1k} = \{(X_k, Y_k, Z_k) | X_k \geq 0, Y_k \geq 0, Z_k \geq 0, X_k + (1 + \tilde{\alpha})Y_k + Z_k = 1\}.$$ 

Then, $\tilde{N}'_k = 0$ in set $D_{1k}$, $\tilde{N}'_k < 0$ if $\tilde{N}_k > 1 - \tilde{\alpha}Y_k$, and $\tilde{N}'_k > 0$ if $\tilde{N}_k < 1 - \tilde{\alpha}Y_k$.

Similar to the case of homogeneous network model (see Assumption I given in Proposition 3.5), we give the following:

**Assumption II** For any initial value $(X_k(0), Y_k(0), Z_k(0))$ in the domain $\Psi_{1k}$ surrounded by planes $D_k$, $D_{1k}$, $X_k > 0$, $Y_k > 0$ and $Z_k > 0$, or in the domain $\Psi_{2k}$ surrounded by planes $D_{1k}$, $X_k > 0$, $Y_k > 0$ and $Z_k > 0$, the flow in $\tilde{\Gamma}_k$ of system (4.1), tends to $D_{1k}$ when $t \to \infty$ (similar to Figure 2).

Vast simulation results demonstrate that Assumption II always holds.

Now, we will focus on the dynamics of solutions of system (4.1) in the following bounded region:

$$\Omega = \left\{(X_1, Y_1, Z_1, \ldots, X_k, Y_k, Z_k) : X_k, Y_k, Z_k \geq 0, X_k + (1 + \tilde{\alpha})Y_k + Z_k = 1, 1 \leq k \leq n\right\}.$$ (4.2)

The initial conditions will be given in $\Omega$ with $\Theta(0) > 0$. It can be verified that the region $\Omega$ is positively invariant.

### 4.1. The threshold on heterogeneous network

This subsection discusses the existence of the epidemic equilibrium solution of system (4.1). Firstly, we have the following:

**Theorem 4.1** Define the value

$$\tilde{R}_0 = \left\{\frac{k^2}{\langle k \rangle} \frac{\tilde{\beta}(1 + \tilde{\gamma})}{(1 + \tilde{\alpha} + \tilde{k})(1 + \tilde{\gamma} + \tilde{\nu})}\right\},$$ (4.3)

and $(k^2) = \sum_i i^2 p(i)$. Then

1. There always exists a disease-free equilibrium solution for system (4.1):

$$E_0 = \left\{(\frac{1 + \tilde{\gamma}}{1 + \tilde{\gamma} + \tilde{\nu}}, 0, \frac{\tilde{\nu}}{1 + \tilde{\gamma} + \tilde{\nu}})\right\}_k.$$

2. Assuming that $0 < \tilde{\alpha} \leq 1$, i.e. $\mu \geq \alpha > 0$, the system (4.1) has a unique endemic equilibrium $E^*_j = (E^*_1, E^*_2, \ldots, E^*_n)$ if and only if $\tilde{R}_0 > 1$. Here, each $E^*_j := (X^*_k, Y^*_k, Z^*_k), 1 \leq k \leq n$, satisfies
(4.1) Let

\[
\begin{align*}
X_k^* &= \frac{\tilde{A}Y_k^*(1 - \tilde{\alpha}Y_k^*)}{\bar{\beta}k\Theta^*}, \\
Y_k^* &= \frac{\tilde{A}B + (\tilde{A} + \tilde{\gamma} + \tilde{\alpha}\tilde{\gamma})\bar{\beta}k - \sqrt{C}}{\bar{\kappa}Y_k^* + \bar{\nu}X_k^*}, \\
Z_k^* &= \frac{\bar{\kappa}Y_k^* + \bar{\nu}X_k^*}{1 + \tilde{\gamma}}
\end{align*}
\]

(4.4)

with \(\tilde{A} = 1 + \tilde{\alpha} + \tilde{\kappa}\), \(\tilde{B} = 1 + \tilde{\gamma} + \tilde{\nu}\), \(C = \tilde{A}^2\tilde{B}^2 + (\tilde{A} + \tilde{\gamma} + \tilde{\alpha}\tilde{\gamma})\bar{\beta}^2k^2\Theta^* + 2[\tilde{A} + \tilde{\gamma} - 2\tilde{\alpha} - \tilde{\alpha}\tilde{\gamma}]\tilde{A}\tilde{B}\bar{\beta}k\Theta^* + \Theta^* = (k)^{-1}\sum_{j=1}^{n} f(p(j))Y_j^*.

**Proof**

(1) Clearly \(E_0\) is a disease-free equilibrium solution of system (4.1).

(2) Note that the equilibrium solution \(E^*\) should satisfy

\[
\begin{align*}
\bar{\beta}kX_k^*\Theta^* - (1 + \tilde{\alpha} + \tilde{\kappa})Y_k^* &= 0, \\
X_k^* + Y_k^* + Z_k^* &= 1 - \tilde{\alpha}Y_k^*, \\
\bar{\kappa}Y_k^* + (1 + \tilde{\gamma})Z_k^* + \bar{\nu}X_k^* &= 0, \\
X_k^* + (1 + \tilde{\alpha})Y_k^* + Z_k^* &= 1,
\end{align*}
\]

(4.5)

for \(k = 1, 2, \ldots, n\). Solving the last two equations, we have

\[
X_k^* + Y_k^* + Z_k^* = 1 - \tilde{\alpha}Y_k^*, \quad X_k^* = 1 - Y_k^* - \tilde{\alpha}Y_k^* - \frac{\bar{\nu} + (\tilde{\kappa} - \tilde{\nu} - \tilde{\alpha}\tilde{\nu})Y_k^*}{1 + \tilde{\gamma} + \tilde{\nu}}.
\]

Substituting the above two equations into the first equation of (4.5) gives

\[
\tilde{\alpha}(1 + \tilde{\alpha} + \tilde{\kappa})Y_k^{*2} - \left[(1 + \tilde{\alpha} + \tilde{\kappa}) + (1 + \tilde{\alpha})\bar{\beta}k\Theta^* + \frac{(\tilde{\kappa} - \tilde{\nu} - \tilde{\alpha}\tilde{\nu})\bar{\beta}k\Theta^*}{1 + \tilde{\gamma} + \tilde{\nu}}\right]Y_k^* + \frac{1 + \tilde{\gamma} + \tilde{\nu}}{1 + \tilde{\gamma} + \tilde{\nu}}\bar{\beta}k\Theta^* = 0.
\]

Thus,

\[
\tilde{\alpha}\tilde{A}\tilde{B}Y_k^{*2} - \left[\tilde{A}\tilde{B} + (\tilde{A} + \tilde{\gamma} + \tilde{\alpha}\tilde{\gamma})\bar{\beta}k\Theta^*\right]Y_k^* + (1 + \tilde{\gamma})\bar{\beta}k\Theta^* = 0.
\]

Let

\[
f(Y) \equiv \tilde{\alpha}\tilde{A}\tilde{B}Y^2 - [\tilde{A}\tilde{B} + (\tilde{A} + \tilde{\gamma} + \tilde{\alpha}\tilde{\gamma})\bar{\beta}k\Theta^*]Y + (1 + \tilde{\gamma})\bar{\beta}k\Theta^*.
\]

Then, the existence of the endemic equilibrium \(E^*\) for (4.5) is equivalent to the existence of a solution of equation \(f(Y) = 0\) in the interval \((0,1)\).

By solving this quadratic equation, \(f(Y) = 0\), we get two possible solutions:

\[
Y_1 = \frac{\tilde{A}\tilde{B} + (\tilde{A} + \tilde{\gamma} + \tilde{\alpha}\tilde{\gamma})\bar{\beta}k\Theta^* + \sqrt{D}}{2\tilde{A}\tilde{B}}, \quad Y_2 = \frac{\tilde{A}\tilde{B} + (\tilde{A} + \tilde{\gamma} + \tilde{\alpha}\tilde{\gamma})\bar{\beta}k\Theta^* - \sqrt{D}}{2\tilde{A}\tilde{B}},
\]

where

\[
\begin{align*}
D &= \tilde{A}^2\tilde{B}^2 + (\tilde{A} + \tilde{\gamma} + \tilde{\alpha}\tilde{\gamma})^2\bar{\beta}^2k^2\Theta^* + 2[(1 - \tilde{\alpha})(1 + \tilde{\gamma}) + \tilde{\kappa}]\tilde{A}\tilde{B}\bar{\beta}k\Theta^* \\
&= \tilde{A}^2\tilde{B}^2 + (\tilde{A} + \tilde{\gamma} + \tilde{\alpha}\tilde{\gamma})^2\bar{\beta}^2k^2\Theta^* + 2[\tilde{A} + \tilde{\gamma} - 2\tilde{\alpha} - \tilde{\alpha}\tilde{\gamma}]\tilde{A}\tilde{B}\bar{\beta}k\Theta > 0.
\end{align*}
\]
According to Vieta’s theorem on the relationships between the roots of a polynomial and its parameters, \( Y_1 Y_2 = \frac{(1 + \tilde{\gamma})\tilde{\beta}k\Theta}{\tilde{\alpha} \tilde{A} \tilde{B}} > 0 \). Because \( Y_1 > 0 \), we have \( 0 < Y_2 < Y_1 \). As we can see, the image of function \( u = f(Y) \) is an upward concave parabola. By calculating, we get

\[
 f(1) = \tilde{\alpha} \tilde{A} \tilde{B} - [\tilde{A} \tilde{B} + (\tilde{A} + \tilde{\gamma} + \tilde{\alpha} \tilde{\gamma})\tilde{\beta}k\Theta] + (1 + \tilde{\gamma})\tilde{\beta}k\Theta \\
= -(1 - \tilde{\alpha}) \tilde{A} \tilde{B} - (\tilde{\alpha} + \tilde{k} + \tilde{\alpha} \tilde{\gamma})\tilde{\beta}k\Theta < 0.
\]

Thus, \( 0 < Y_2 < 1 \). It can be resulted that

\[
 Y_k^* = \frac{\tilde{A} \tilde{B} + (\tilde{A} + \tilde{\gamma} + \tilde{\alpha} \tilde{\gamma})\tilde{\beta}k\Theta^* - \sqrt{C}}{2\tilde{\alpha} \tilde{A} \tilde{B}}, \tag{4.6}
\]

where \( C = \tilde{A}^2 \tilde{B}^2 + (\tilde{A} + \tilde{\gamma} + \tilde{\alpha} \tilde{\gamma})^2 \tilde{\beta}^2 k^2 \Theta^* + 2[\tilde{A} + \tilde{\gamma} - 2\tilde{\alpha} - \tilde{\alpha} \tilde{\gamma}] \tilde{A} \tilde{B} \tilde{\beta}k\Theta^* > 0 \).

From the first two equations of (4.5), we have

\[
 X_k^* = \frac{\tilde{A} Y_k^*(1 - \tilde{\alpha} Y_k^*)}{\tilde{\beta}k\Theta^*}, \quad Z_k^* = \frac{\tilde{k} Y_k^* + \tilde{\nu} X_k^*}{1 + \tilde{\gamma}}.
\]

Now, we obtain a self-consistency equation as follows:

\[
g(\Theta) \triangleq \Theta = \frac{1}{(k)} \sum_{j=1}^{n} j p(j) \frac{\tilde{A} \tilde{B} + (\tilde{A} + \tilde{\gamma} + \tilde{\alpha} \tilde{\gamma})\tilde{\beta} j \Theta - \sqrt{D}}{2\tilde{\alpha} \tilde{A} \tilde{B}}.
\]

By calculating straightforwardly, we get

\[
g(0) = \frac{1}{(k)} \sum_{j=1}^{n} j p(j) \frac{\tilde{A} \tilde{B} - \sqrt{\tilde{A}^2 \tilde{B}^2}}{2\tilde{\alpha} \tilde{A} \tilde{B}} = 0,
\]

\[
g(1) < \frac{1}{(k)} \sum_{j=1}^{n} j p(j) = 1,
\]

\[
g'(\Theta) = \frac{1}{(k)} \sum_{j=1}^{n} j p(j) \left\{ \frac{(\tilde{A} + \tilde{\gamma} + \tilde{\alpha} \tilde{\gamma})\tilde{\beta} j}{2\tilde{\alpha} \tilde{A} \tilde{B}} - \frac{D^{-1/2}[(\tilde{A} + \tilde{\gamma} + \tilde{\alpha} \tilde{\gamma})^2 \tilde{\beta}^2 j^2 \Theta + (\tilde{A} + \tilde{\gamma} - 2\tilde{\alpha} - \tilde{\alpha} \tilde{\gamma}) \tilde{A} \tilde{B} \tilde{\beta} j]}{2\tilde{\alpha} \tilde{A} \tilde{B}} \right\}
\]

\[
= \frac{1}{(k)} \sum_{j=1}^{n} j p(j) \frac{(\tilde{A} + \tilde{\gamma} + \tilde{\alpha} \tilde{\gamma})\tilde{\beta} j}{2\tilde{\alpha} \tilde{A} \tilde{B}} \left\{ 1 - \frac{1}{\sqrt{D}} \left[ (\tilde{A} + \tilde{\gamma} + \tilde{\alpha} \tilde{\gamma}) \tilde{\beta} j \Theta + \frac{(\tilde{A} + \tilde{\gamma} - 2\tilde{\alpha} - \tilde{\alpha} \tilde{\gamma}) \tilde{A} \tilde{B} \tilde{\beta} j}{\tilde{A} + \tilde{\gamma} + \tilde{\alpha} \tilde{\gamma}} \right] \right\} > 0,
\]

\[
g''(\Theta) = \frac{1}{(k)} \sum_{j=1}^{n} j p(j) \frac{-1}{2\tilde{\alpha} \tilde{A} \tilde{B}} \frac{(\tilde{A} + \tilde{\gamma} + \tilde{\alpha} \tilde{\gamma})^2 \tilde{\beta}^2 j^2}{\sqrt{D}}.
\]
Summarizing the results above, we know that \( g(\Theta) \) is a monotone increasing function and tends to a positive value as \( \Theta \to \infty \). Hence, a non-trivial solution exists if and only if \( g'(\Theta)|_{\Theta=0} > 1 \), i.e.

\[
\frac{\langle k^2 \rangle}{\langle k \rangle} \frac{\tilde{\beta}(1 + \tilde{\gamma})}{(1 + \tilde{\alpha} + \tilde{\kappa})(1 + \tilde{\gamma} + \tilde{\nu})} > 1.
\]

This yields the critical epidemic threshold \( \tilde{R}_0 \) given in (4.3). Hence, when \( \tilde{R}_0 > 1 \), one and only one epidemic equilibrium solution of system (4.1) exists. This completes the proof. \( \square \)

4.2. The stability of the equilibria

By (4.2) and let \( Z_k(t) = 1 - X_k(t) - Y_k(t) - \tilde{\alpha}Y_k(t) \) at any \( t \). Thus, system (4.1) can be reduced as the following form:

\[
\begin{align*}
\frac{dX_k(t)}{dt} &= 1 + \tilde{\gamma} - \frac{\tilde{\beta}kX_k(t)\Theta(t)}{1 - \tilde{\alpha}Y_k(t)} - \tilde{\gamma}(1 + \tilde{\alpha})Y_k(t) - (1 + \tilde{\gamma} + \tilde{\nu})X_k(t) \\
\frac{dY_k(t)}{dt} &= \frac{\tilde{\beta}kX_k(t)\Theta(t)}{1 - \tilde{\alpha}Y_k(t)} - (1 + \tilde{\alpha} + \tilde{\kappa})Y_k(t)
\end{align*}
\]

(4.7)

Since in a real system, the number of individuals is finite, it is assumed that the maximum number of contact each individual is a positive integer \( \mathbb{N} \). Hence, \( P(k) = 0 \) for all \( k > \mathbb{N} \). Let

\[
\mathcal{H} = \{(X_1, Y_1, \ldots, X_k, Y_k) \in \mathbb{R}^{2N}, 0 \leq X_k, Y_k \leq 1, X_k + Y_k \leq 1, 1 \leq k \leq \mathbb{N}\}.
\]

(4.8)

It can be verified that region \( \mathcal{H} \) is positively invariant.

The following theorem gives the criteria of globally asymptotically stability of the equilibria of model (4.7). Firstly, we need the following Lemma.

**Lemma 4.2** \textit{(Lajmanovich and York [32])} Consider the system

\[
\frac{dy}{dt} = Ay + N(y),
\]

(4.9)

where \( A \) is an \( n \times n \) matrix and \( N(y) \) is continuously differentiable in a region \( D \in \mathbb{R}^n \). Assume
Then either $y = 0$ is globally asymptotically stable in $C$, or for any $y_0 \in C - \{0\}$ the solution $\phi(t, y_0)$ of (4.9) satisfies $\liminf_{t \to \infty} \| \phi(t, y_0) \| \geq m$, where $m > 0$, independent of $y_0$. Moreover, there exists a constant solution of (4.9), $y = y^*$, $y^* \in C - 0$.

Similar to [7], one has the following:

**Theorem 4.3** Let $\tilde{R}_0$ be defined by (4.3).

(i) If $\tilde{R}_0 < 1$, the disease-free equilibrium $E_0 = \left\{ \left( \frac{1 + \tilde{\gamma}}{1 + \tilde{\gamma} + \tilde{\nu}}, 0 \right) \right\}_k$ of system (4.7) is globally asymptotically stable.

(ii) If $\tilde{R}_0 > 1$, system (4.7) is permanent, i.e. there exists an $\varepsilon > 0$, such that

$$
\liminf_{t \to \infty} \{X_k(t), Y_k(t)\}^N_{k=1} \geq \varepsilon,
$$

where $(X_k(t), Y_k(t))$ is any solution of system (4.7), satisfying $\mathcal{H}$ and $Y_k(0) > 0$.

**Proof** Firstly, it is easy to get that $E_0$ is the disease-free equilibrium solution of system (4.7).

Clearly, $\mathcal{H}$ defined in (4.8) is a compact convex set. In the following, we discuss the dynamic of system (4.7) in $\mathcal{H}$. The Jacobian matrix of the disease-free equilibrium of system (4.7), which is a $2N \times 2N$ matrix, can be written as follows:

$$
J = \begin{pmatrix}
    a & -b_1 - \tilde{\gamma}(1 + \tilde{\alpha}) & \cdots & 0 & -b_1j & \cdots & 0 & -b_{1N} \\
    0 & b_1(1 + \tilde{\alpha} + \tilde{\kappa}) & \cdots & 0 & b_{1j} & \cdots & 0 & b_{1N} \\
    \vdots & \vdots & \ddots & \vdots & \vdots & \ddots & \vdots & \vdots \\
    0 & -b_{i1} & \cdots & a & -b_j - \tilde{\gamma}(1 + \tilde{\alpha}) & \cdots & 0 & -b_{iN} \\
    0 & b_{i1} & \cdots & 0 & b_j(1 + \tilde{\alpha} + \tilde{\kappa}) & \cdots & 0 & b_{iN} \\
    \vdots & \vdots & \ddots & \vdots & \vdots & \ddots & \vdots & \vdots \\
    0 & -b_{N1} & \cdots & 0 & -b_{Nj} & \cdots & a & -b_N - \tilde{\gamma}(1 + \tilde{\alpha}) \\
    0 & b_{N1} & \cdots & 0 & b_{Nj} & \cdots & 0 & b_N(1 + \tilde{\alpha} + \tilde{\kappa})
\end{pmatrix},
$$

where $a = -1 + \tilde{\gamma} + \tilde{\nu}$, $b_j = -j \tilde{\nu} (1 + \tilde{\gamma}) / (k)$, $b_{ij} = \beta_i \frac{1 + \tilde{\gamma}}{1 + \tilde{\gamma} + \tilde{\nu}} (k)$, $i \neq j$, $i, j = 1, 2, \ldots, N$. 
The polynomial equation of the disease-free equilibrium is

\[(\lambda - a)^N(\lambda + 1 + \bar{\alpha} + \bar{\kappa})^{N-1}\left(\lambda - \sum_{j=1}^{N} b_j + 1 + \bar{\alpha} + \bar{\kappa}\right) = 0.\]

Hence, if \(\tilde{R}_0 > 1\), there exists a unique positive eigenvalue \(\lambda\) of \(J\). If \(\tilde{R}_0 < 1\), all real-valued eigenvalues \(\lambda\) of \(J\) are negative.

Perron–Feobenius theorem gives that the maximal real part of all eigenvalues of \(J\) is positive if and only if \(\tilde{R}_0 > 1\). Then, from Lemma 4.2, we complete the proof of this Theorem. □

4.3. Numerical simulations

This subsection will give some results of numerical simulations to investigate the dynamics of nodes with different degrees \(k\) in system (4.1).

Select the following initial conditions:

\[X_k(0) = 0.8, \ Y_k(0) = 0.2, \ Z_k(0) = 0.\]

We investigate the dynamics of nodes with different degrees \(k\). The results of numerical simulation are shown in Figures 6 and 7. In Figure 6:

\[\nu = 0.3, \ \beta = 0.1, \ \kappa = 0.5, \ \gamma = 0.1, \ \mu = 0.3, \ \alpha = 0.261, \ m = 3, \ M = 100, \ (4.10)\]

and hence \(\tilde{R}_0 = 0.7056 < 1\). In Figure 7:

\[\nu = 0.1, \ \beta = 0.25, \ \kappa = 0.5, \ \gamma = 0.1, \ \mu = 0.3, \ \alpha = 0.261, \ m = 3, \ M = 100, \ (4.11)\]

and hence \(\tilde{R}_0 = 2.4695 > 1\). Both simulations, given in Figures 6 and 7, have selected the same initial values: \(X_k(0) = 0.8, \ Y_k(0) = 0.2, \ Z_k(0) = 0.\)

![Figure 6](image_url1)

![Figure 7](image_url2)

Figure 6. Dynamics of nodes with degree \(k = m = 3, \ k = 22, \ k = 66\) and degree \(k = 98\): \(\tilde{R}_0 = 0.7056 < 1\). (a) Fraction of susceptible individuals \(X_k\) as a function of time. (b) Fraction of infected individuals \(Y_k\) as a function of time.
Figure 7. Dynamics of nodes with degree $k = m = 3$, $k = 22$, $k = 66$ and degree $k = 98$: $\bar{R}_0 = 2.4695 > 1$. (a) Fraction of susceptible individuals $X_k$ as a function of time. (b) Fraction of infected individuals $Y_k$ as a function of time.

Figures 6 and 7 show that if $\bar{R}_0 > 1$, then the disease will converge to a positive stationary level, which means the endemic states are stable. And if $\bar{R}_0 < 1$, then the disease will ultimately disappear. From these figures, it follows that the outcomes of $k = 66$ and $k = 98$ have no significant difference.

5. Conclusions

This paper introduces and studies two SIRS epidemic models on both homogenous networks and heterogeneous networks, respectively. The two models describe the variations of the total size of population which were not considered in [4]. Because vaccination has been an important strategy of disease control, we propose the vaccinated individuals $\nu S$, which was not considered in both [4,33]. The disease-related death is also considered in our models, which was not considered in [4]. The new results obtained in this paper are listed below:

1) On homogenous networks, we introduce the recruitment $\lambda$ of susceptible individuals corresponding to nature births and immigration. A formula of the basic reproduction number $R_0$ (see (3.5)) of the model on homogenous networks is obtained,

$$R_0 = \frac{\beta \langle k \rangle}{(\alpha + \kappa + \mu)(1 + \nu(\gamma + \mu))}.$$  

Theorem 3.2 suggests that high percentage (large $\nu$) of vaccinated population, and low connectivity (small $\langle k \rangle$) between the susceptible and infectious are both important factors to make epidemic diseases disappear. As we can see in the formula of $R_0$, both $\gamma$ and $\mu$ are much less than one in practice, therefore increasing the percentage ($\nu$) of the vaccinated population can be a very effective way to let $R_0$ decrease.

2) One distinguishing feature of our two models is that the threshold value or the basic reproduction number is independent of the total size of population, and is dependent of parameters which have significant biological meanings.
However, in [4], there is a connotation that the threshold value is dependent on the total size of population. Reference [4] assumed that the total number is constant, which was expressed as unit one. But the threshold value varies with the changes in the total number, which means that the more the total number of the population is, the easier the disease outbreaks. That is not supported by observations.

Compared with some developed countries whose populations are large, infectious diseases outbreak easier in developing countries, where the populations may be small, but the medical conditions and individuals’ physical health are poor. Consequently, the persistence or disappearance of infectious diseases should be independent of the total amount of population.

(3) Our SIRS epidemic model on heterogeneous networks has features similar to those of our model on homogeneous networks. Perron–Feobenius theorem and Lajmanovich–Yorke theorem [32] are used to study the globally asymptotical stabilities of the equilibria of the model. It is proved that if the threshold value \( \tilde{R}_0 \) is less than one, then the disease-free equilibrium is globally asymptotically stable, which means that the disease will disappear. Otherwise, if \( \tilde{R}_0 \) is more than one, system (4.1) is permanent. High percentage of vaccinated population and low connectivity between the susceptible and infectious are also important factors to make epidemic diseases disappear.

(4) Numerical experiments on the two models are given to illustrate theoretical results. In addition to presenting simulation figures illustrating the results of the theoretical expectations, we also numerically predict the effect of vaccination ratio on the size of HBV-infected mainland Chinese population.

With million years biological evolutions, the basic reproduction number \( R_0 \) of wild animals is less than one or slightly larger than one, which results in that wild animals hardly become extinct after various infectious diseases’ attacks. However, a large amount of domestic animals die during the spreading of epidemics. That is because dense living circumstances and no natural living ways may make domestic animals have larger \( \langle k \rangle \) (homogenous networks) and \( \beta \), or \( \langle k^2 \rangle / \langle k \rangle \) (heterogeneous networks) and \( \beta \) than wild animals.

For human beings, when a new infectious disease outbreaks, decreasing contact opportunities (reduce \( \langle k \rangle \) for homogenous networks or \( \langle k^2 \rangle / \langle k \rangle \) for heterogeneous networks) and protecting children and old people (they may have large \( \beta \)) may be effective control measures against epidemics. For a persistent infectious disease, improving the vaccination rate (\( \nu \)) and the cure rate (\( \kappa \)) of infected persons may be effective in disease prevention and control strategies.

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