Applied Mathematics and Computation 263 (2015) 342–352

Contents lists available at ScienceDirect

Applied Mathematics and Computation

journal homepage: www.elsevier.com/locate/amc

Dynamic analysis of a delayed model for vector-borne diseases on bipartite networks

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ARTICLE INFO

Keywords:
Vector-borne diseases
Bipartite networks
Dynamic model
Time delay
Basic reproduction number
Global asymptotic stability

ABSTRACT

In this paper, to study the spread of vector-borne diseases in human population, we build two coupled models for human population and vector population respectively on bipartite networks. By taking approximate expression for the density of infective vectors, we reduce the coupled models to a delayed SIS model describing the spread of diseases in human population. For the delayed dynamic model, we analyze its dynamic behavior. The basic reproduction number R_0 is given. And based on the Lyapunov–LaSalle invariance principle, we prove the global asymptotic stability of the disease-free equilibrium and the endemic equilibrium. Finally we carry out simulations to verify the conclusions and reveal the effect of the topology structure of networks and the time delay on the transmission process. Our results show that the basic reproduction number depends on the topology structure of bipartite networks and the time delay. It is also pointed out that the time delay can reduce the basic reproduction number. Furthermore, when the disease will disappear, the delay speeds up the disappearing process; when disease will become endemic, the delay slows the disease spreading down and reduces the density of infective humans.

1. Introduction

Time delays are related to several periods in mathematical epidemiology, such as incubation period [1–3], maturation period and infectious period [4], etc. Particularly, the incubation period is common in the spread of vector-borne diseases. Vector-borne diseases of humans, for instance, malaria, yellow fever, and dengue fever, are transmitted via blood-suck arthropods called vectors, such as mosquitoes, biting flies and bugs. Vectors typically become infected by disease agents while feeding on infective humans, and then pass on the disease agents to susceptible humans. In the transmission process, there always is an incubation period during which disease agents develop in vectors, and only after that time, the infected vectors become themselves infective. For example, in malaria spread, mosquitoes become infected by malaria agents while biting infective humans, after 10 to 14 days [5,6], they become infective and pass on malaria agents to susceptible humans, so that malaria is widespread.

In order to discover the effect of time delays on the spread of vector-borne diseases, Cooke [7] proposed a Susceptible-Infected-Susceptible (SIS) model with an incubation period

$$y'(t) = by(t-T)[1-y(t)] - cy(t),$$

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http://dx.doi.org/10.1016/j.amc.2015.04.074
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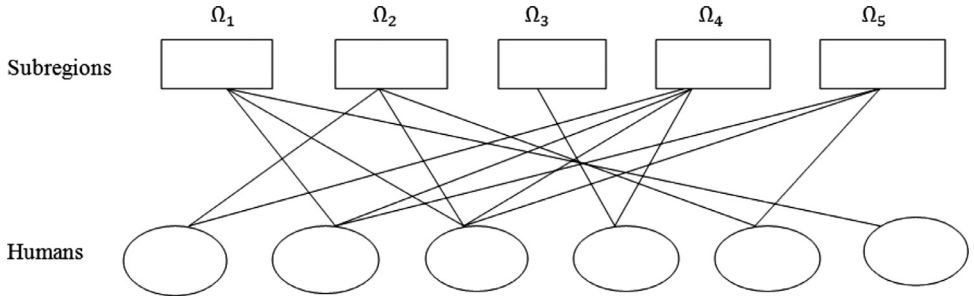

Fig. 1. A bipartite network with two kinds of nodes, subregions and humans, where $n = 5, N = 6$.

where $y(t)$ is the density of infective human, the time delay T is the incubation period, $b > 0$ and $c \geq 0$. Subsequently, the model was extended by Marcati and Pozio [8], Volz [9], Beretta et al. [10] , Jin and Ma [11], etc. By introducing incubation period, Aron and May [12], Dye and Williams [13], Ruan et al. [14] and Martcheva and Prosper [15] modified Ross–Macdonald model [16] which describes malaria transmission. Among these researches, Ruan et al. [14] have found that the incubation period could reduce the basic reproduction number and the prevalence of infection. Martcheva and Prosper [15] have shown the delay can lead to oscillations and chaos. These results are obtained based on the assumption that vectors and humans are fully mixed. While in the spreading process of infectious diseases, real contact patterns take the form of networks. In networks, a node represents individual and an edge is placed between two individuals if there is the possibility of transmission between them. In particular, the contact patterns between humans and vectors caused by vector-borne diseases form bipartite networks where the nodes belong to two mutually classes (human and vector) and the edges can only occur between two nodes in different classes.

Thus, in recent ten years, increasing attention and interest is paid to infectious diseases spread on complex networks which display complex topological properties of networks [17,18], such as small-world phenomenon [19] and scale-free degree distributions [20,21], etc. All kinds of epidemic models, Susceptible-Infected-Susceptible (SIS) [22–24], Susceptible-Infected (SI) [25,26] and Susceptible-Infected-Removed (SIR) [27–30], were proposed on homogeneous networks and heterogeneous networks and focused on the effect of topology characters of networks on diseases spread. In these models, no matter SIS, SI or SIR, an interesting result is that: there exists a critical epidemic threshold $\lambda_c > 0$ for homogeneous networks and when the spreading rate below λ_c, the epidemic will disappear; while the critical epidemic threshold may tend to zero for infinite heterogeneous networks such as scale-free networks. This means that, for infinite heterogenous networks, an epidemic disease will outbreak for any spreading rate. Even the size of a heterogeneous network is finite, the critical value is rather small compared with the one in a homogeneous network with the same size. The great change in the behavior of the processes indicates that we should take into account the topology character of networks in the propagation of epidemics.

The same is true for vector-borne diseases spread. Masuda and Konno [31] have analyzed a model describing malaria spreading between humans and mosquitos on bipartite networks. The study has revealed that the epidemic threshold [23] depends on the degree distributions of humans and mosquitoes and it will disappear if either of the second-order moments of the two degree distributions diverges. However, capturing the degree of each mosquito is impracticable. Considering human–human and human–mosquito infection, SIS models have been proposed [32–34]. In [33,34], the heterogeneity is induced by the heterogeneous connectivity among different persons, human and mosquitos are fully mixed.

The time delay and the structure of contact networks are important to the spread of vector-borne diseases. It is, therefore, necessary to study the impacts of the time delay and the structure of networks on the transmission of the vector-borne diseases. But, so far, there is no work focusing on vector-borne diseases spreading with a time delay on bipartite networks.

In this paper, in view of the habit of vectors limited dispersal from their breeding sites, we divide the whole region where vector-borne disease occurs into several subregions. Then we construct bipartite networks (as illustrated in Fig. 1) where there are two class nodes, humans and subregions, and the edge represents the relation that a human enters into a subregion and is fully mixed with the vectors in the subregion. Two separate degree distributions may be reasonable to interpret the contact patterns between humans and subregions in reality. On bipartite networks, we derive a delayed SIS model for vector-borne diseases spread only caused by human–vector infection and following we analyze the dynamic behavior of the delayed model to discover the effect of topology character of networks and the time delay on the propagation process.

The rest of this paper is organized as follows: In Section 2, we derive the SIS model with a time delay on bipartite networks. The existence of equilibriums both of disease-free equilibrium and endemic equilibrium is discussed in Section 3. And we analyze the global asymptotic stability of the equilibriums in Section 4. In Section 5, we perform simulations to illustrate the results. Finally, we discuss in Section 6.

2. Model formulation

Assume Ω is the region where the transmission of vector-borne diseases occurs. It may be a city or a village, and so on. In the region Ω, most vectors remain near their breeding sites, for example, mosquitoes tend to travel limited distances nearby their

Table 1
Description of parameters.

Parameter	Description
a	Rate of biting on humans by a single vector
b	The probability of infected bites on human that produce an infection
c	The probability of infected bites on vector that produce an infection
γ	The probability that an infective human is cured and become susceptible
δ	The birth and death rate of vectors
τ	The incubation period in vectors
N	The total size of human population
V	The vector size in each subregion
n	The number of subregions

breeding sites [35], moist surfaces, rivers, forest and residential area. So it is reasonable to divide the region Ω into n domains which correspond to breeding sites of vectors and are denoted by $\Omega_1, \Omega_2, \ldots, \Omega_n$ respectively.

Human moves between these subregions daily. When humans enter a subregion, the humans and vectors in the subregion are homogeneous mixed. Thus, the contact patterns between humans and subregions form bipartite networks. Two separate degree distributions $q(k)$ and $p(k)$ can be defined for subregions and humans respectively. As done in the study [36], to measure the dependence of the epidemic threshold on the network size, the degree distribution is Poisson for the vectors. Here, we assume each subregion has the same degree. While the displacement distribution of human mobility, for both long-range travels and daily movements, approximately follows a power law distribution [37–40]. Then the degree distribution of humans $p(k)$ may be power law. Here it is treated as arbitrary degree distribution.

In order to describe the spread of vector-borne disease on networks, we make the following assumptions:

(H1) The total size N of human population is constant. Let N_k represent the number of humans who visit k subregions daily. In addition, we divide human population into two discrete states, susceptible and infective. Let us denote by $X_k(t)$ and $Y_k(t)$ the number of susceptible and infective humans with degree k at time t respectively. Thus $N_k = Np(k) = X_k(t) + Y_k(t)(k = 1, 2, \ldots, n)$ and $N = N_1 + N_2 + \cdots + N_n$.
(H2) The birth and death rate for vectors are equal and denoted by δ, then the number of vectors in each subregion nearly remains unchanged with time. We denote by V the size of vectors in each subregion. The vectors in each subregion are divided into susceptible (i.e. healthy) vectors V_s and infective vectors V_i. So $V = V_s(t) + V_i(t)$, where $V_s(t)(V_i(t))$ is the amount of susceptible (infective) vectors at time t.
(H3) A susceptible human becomes infective when he(she) is bitten by an infective vector and a infective human is cured and becomes a susceptible node with recovery rate γ. Let a be the rate of biting on humans by a single vector. Let b be the probability of infected bites on human that produce an infection and the probability of infected bites on vector that produce an infection is c.
(H4) A susceptible vector becomes infected upon biting an infective human and after its incubation period τ, it becomes infective.
(H5) The life span of vectors is much shorter than the duration of infectiousness of humans, i.e. $\gamma \ll \delta$.
(H6) The death and birth rate of vectors, δ, is assumed "large" and assure $ac/(\delta e^{\delta\tau}) \ll 1$.

For the sake of clarity, we present the description of parameters in Table 1.

According to the above assumptions, in each subregion, vectors contact with $N\langle k\rangle/n$ humans and the number of bits on humans per day per human is $a/\frac{N\langle k\rangle}{n}$. So we have the following SIS model for the human population

$$\begin{cases} \dfrac{dX_k(t)}{dt} = -\dfrac{ab}{N\langle k\rangle/n} kX_k(t)V_i(t) + \gamma Y_k(t), \\ \dfrac{dY_k(t)}{dt} = \dfrac{ab}{N\langle k\rangle/n} kX_k(t)V_i(t) - \gamma Y_k(t), \end{cases} \quad k = 1, 2, \ldots, n. \tag{2.1}$$

In each subregion, vectors contact with $N\langle k\rangle/n$ humans including $\sum_k kY_k(t)/n$ infective humans at time t. So for the vector population in each subregion, we have the following SI model

$$\begin{cases} \dfrac{dV_s(t)}{dt} = -\dfrac{ac}{N\langle k\rangle/n} e^{-\delta\tau} V_s(t-\tau) \sum\limits_k kY_k(t-\tau)/n + \delta V_i(t), \\ \dfrac{dV_i(t)}{dt} = \dfrac{ac}{N\langle k\rangle/n} e^{-\delta\tau} V_s(t-\tau) \sum\limits_k kY_k(t-\tau)/n - \delta V_i(t). \end{cases} \tag{2.2}$$

Let $S_k(t)$ and $I_k(t)$ be the relative density of the susceptible and infective nodes with degree k, then $S_k(t) = X_k(t)/N_k$ and $I_k(t) = Y_k(t)/N_k$ are the dimensionless human variables and satisfy $S_k(t) + I_k(t) = 1$. Let $v_s(t) = V_s(t)/V$ and $v_i(t) = V_i(t)/V$ be the dimensionless vector variables, then $v_s(t) + v_i(t) = 1$.

Apply the dimensionless variables, the system (2.1) is reduced to the following equation

$$\frac{dI_k(t)}{dt} = ab\frac{nV}{N\langle k\rangle}k(1-I_k(t))v_i(t) - \gamma I_k(t), \quad k = 1, 2, \ldots, n. \tag{2.3}$$

Furthermore, the dimensionless vector system is

$$\frac{dv_i}{dt} = \mathrm{ace}^{-\delta\tau}(1-v_i(t-\tau))\frac{\sum_k kp(k)I_k(t-\tau)}{\langle k\rangle} - \delta v_i(t). \tag{2.4}$$

Associating (2.3) and (2.4), we have a coupled system

$$\begin{cases} \dfrac{dI_k(t)}{dt} = ab\dfrac{nV}{N\langle k\rangle}k(1-I_k(t))v_i(t) - \gamma I_k(t), \quad k = 1, 2, \ldots, n, \\ \dfrac{dv_i}{dt} = \mathrm{ace}^{-\delta\tau}(1-v_i(t-\tau))\dfrac{\sum_k kp(k)I_k(t-\tau)}{\langle k\rangle} - \delta v_i(t). \end{cases} \tag{2.5}$$

For the coupled system (2.5), by the theory and methods used in researches [12,13], we will reduce its dimension. As usual, we assume $\gamma \ll \delta$ in (H4), that means the life span of vectors is much shorter than the duration of infectiousness of humans. In fact, it is common for malaria transmitted by mosquitoes. In this case, the dynamics of the vector population reaches its equilibration much faster than that of human population (Aron andMay [12]). So disease among humans can be predicted without modeling the vector explicitly. Namely, we can study the system (2.5) with $v_i(t)$ frozen at the following case (Dye and Williams [13])

$$\frac{dv_i}{dt} = 0. \tag{2.6}$$

From (2.6), we have an approximate expression for $v_i(t)$, i.e.

$$v_i(t) \approx \frac{\mathrm{ace}^{-\delta\tau}\frac{\sum_k kp(k)I_k(t-\tau)}{\langle k\rangle}}{\delta + \mathrm{ace}^{-\delta\tau}\frac{\sum_k kp(k)I_k(t-\tau)}{\langle k\rangle}}.$$

By the assumption (H5), we know that $ace^{-\delta\tau} \ll \delta$. So

$$v_i(t) \approx \frac{\mathrm{ace}^{-\delta\tau}}{\delta}\frac{\sum_k kp(k)I_k(t-\tau)}{\langle k\rangle}. \tag{2.7}$$

Substituting (2.7) into the system (2.5), the coupled system can be reduced to the following system

$$\frac{dI_k}{dt} = \frac{a^2bc}{\delta e^{\delta\tau}}\frac{nV}{N\langle k\rangle}k(1-I_k(t))\frac{\sum_k kp(k)I_k(t-\tau)}{\langle k\rangle} - \gamma I_k(t), \quad k = 1, 2, \ldots, n. \tag{2.8}$$

For analyze conveniently, we set $s = \gamma t$, then the model (2.8) can be written as

$$\frac{dI_k}{ds} = \frac{a^2bcnV}{\gamma\delta e^{\delta\tau}N\langle k\rangle}k(1-I_k(s))\frac{\sum_k kp(k)I_k(s-\tau_s)}{\langle k\rangle} - I_k(s), \quad k = 1, 2, \ldots, n.$$

To simplify the notations, s will be replaced by t and τ_s by τ. So the above system becomes

$$\frac{dI_k}{dt} = \frac{a^2bcnV}{\gamma\delta e^{\delta\tau}N\langle k\rangle}k(1-I_k(t))\frac{\sum_k kp(k)I_k(t-\tau)}{\langle k\rangle} - I_k(t), \quad k = 1, 2, \ldots, n. \tag{2.9}$$

Let $\beta = \frac{a^2bcnV}{\gamma\delta e^{\delta\tau}N\langle k\rangle}$ and $\Theta(I(t)) = \frac{\sum_k kp(k)I_k(t)}{\langle k\rangle}$, we can rewrite the system (2.9) as

$$\frac{dI_k(t)}{dt} = \beta k[1-I_k(t)]\Theta(I(t-\tau)) - I_k(t), \quad k = 1, 2, \ldots, n. \tag{2.10}$$

In the following sections, we will analyze the model (2.10).

3. The equilibriums and invariants of (2.10)

In order to analyze the delayed model (2.10), we specify the notations used in the following sections.

Let $C \triangleq C([-\tau, 0], [0,1]^n)$ be the Banach space of continuous functions mapping the interval $[-\tau, 0]$ into $[0,1]^n$. By the fundamental theory of functional differential equations [41], it is easy to show that there exists a unique solution $I(t) = (I_1(t), I_2(t), \ldots, I_n(t))$ satisfying initial condition $I_0(\theta) = \phi(\theta) \in C$, where $I_0(\theta) = (I_1(\theta), I_2(\theta), \ldots, I_n(\theta))$ and $\phi(\theta) = (\phi_1(\theta), \phi_2(\theta), \ldots, \phi_n(\theta))$, $\theta \in [-\tau, 0]$.

In order to discuss clearly, we first make a note on notations. As usual, let $I_t(\theta) = I(t+\theta)(-\tau \le \theta \le 0)$ be a function in C. In the following, $I_t(\theta)$ and $I_0(\theta)$ represent functions in C, while $I_k(t)(k = 1, 2, \ldots, n)$ is the kth element of the vector $I(t) = (I_1(t), I_2(t), \ldots, I_n(t))$. Additionally for all $u, v \in R^n$, we write $u < v \Leftrightarrow u_k \le v_k$, $u \ne v$.

Next, we shall show that the results about the equilibriums and invariants of (2.10).

For obtaining the equilibriums of the system (2.10), we set $I_k(t) = I_k^*$ and $\Theta(t) = \Theta^*$. Then the equilibriums of the system (2.10) satisfy $I_k^* = \frac{\beta k\Theta^*}{1+\beta k\Theta^*}$, $k = 1, 2, \ldots, n$, where $\Theta^* = \frac{\sum_k kp(k)I_k^*}{\langle k\rangle}$. By substituting I_k^* into $\Theta^* = \frac{\sum_k kp(k)I_k^*}{\langle k\rangle}$, we have the self-consistency equation

$$\Theta^* = \langle k\rangle^{-1}\sum_k kp(k)\frac{\beta k\Theta^*}{1+\beta k\Theta^*} \triangleq G(\Theta^*). \tag{3.1}$$

The solution $\Theta^* = 0$ always satisfies the consistency equation. That means the system (2.10) has always disease-free equilibrium $E^0 = (0, 0, \ldots, 0)$. Upon further differentiation of $G(\Theta^*)$, we have

$$G'(\Theta^*) = \frac{\beta}{\langle k\rangle}\sum_k \frac{k^2p(k)}{(1+\beta k\Theta^*)^2} > 0,$$

$$G''(\Theta^*) = -2\frac{\beta^2}{\langle k\rangle}\sum_k \frac{k^3p(k)}{(1+\beta k\Theta^*)^3} < 0.$$

So $G(\Theta^*)$ is rigorously increasing concave function. In view of $G(0) = 0$ and $G(1) < 1$, the self-consistency equation (3.1) allows a nontrivial solution $\Theta^* \neq 0(\Theta^* \in (0, 1])$ only if

$$\frac{dG}{d\Theta^*}|_{\Theta^*=0} = \frac{\beta\langle k^2\rangle}{\langle k\rangle} > 1.$$

That means there exists a unique endemic equilibrium $E^* = (I_1^*, I_2^*, \ldots, I_n^*)$, where I_k^* satisfies $I_k^* = \frac{\beta k\Theta^*}{1+\beta k\Theta^*}$. So the basic reproduction number R_0 is given by $R_0 = \frac{\beta\langle k^2\rangle}{\langle k\rangle}$, where $\langle k^2\rangle = \frac{\sum k^2p(k)}{\langle k\rangle}$.

From above discussion, we have the following theorem:

Theorem 3.1. *The system (2.10) has always disease-free equilibrium $E^0 = (0, 0, \ldots, 0)$. If $R_0 > 1$, there exists a unique endemic equilibrium $E^* = (I_1^*, I_2^*, \ldots, I_n^*)$, where I_k^* satisfies $I_k^* = \frac{\beta k\Theta^*}{1+\beta k\Theta^*}$.*

Set $C_1 = \{\phi|\phi \in C, E^0 < \phi(\theta) < E^*, \theta \in [-\tau, 0]\}$ and $C_2 = \{\phi|\phi \in C, E^* < \phi(\theta) < (1, 1, \ldots, 1), \theta \in [-\tau, 0]\}$.

Theorem 3.2. *C, C_1 and C_2 are invariants with respect to (2.10).*

Proof. We prove only that C is invariant with respect to (2.10), C_1 and C_2 are invariants with respect to (2.10) can be proved by the similar way.

By the definition of invariant, we will prove that if $I(t) = (I_1(t), I_2(t), \ldots, I_n(t))$ is a solution of the system (2.10) with initial condition $I_0(\theta) = \phi(\theta) \in C$, then $0 \le I_k(t) \le 1$ for all $t > 0$, $k = 1, 2, \ldots, n$.

Suppose for the sake of contradiction that this is not true. Then by the continuity of $I(t)$, there exists $l \in \{1, 2, \ldots, n\}$ and $\xi > 0$, such that $0 \le I_k(t) \le 1$ for $0 \le t \le \xi$, $k = 1, 2, \ldots, n$, and either

(i) $I_l(\xi) = 0$ and $I_l(t) < 0$ on $(\xi, \xi + \varepsilon)$ for some ε, or
(ii) $I_l(\xi) = 1$ and $I_l(t) > 1$ on $(\xi, \xi + \varepsilon)$ for some ε.

If (i) holds, assume, as we may, that $\varepsilon < \tau$, from (2.10), we have $\frac{d(I_l(t)e^t)}{dt} = \beta e^t l[1 - I_l(t)]\Theta(I(t-\tau))$. It can be seen that $I_l(t)e^t$ is non-decreasing on $(\xi, \xi + \varepsilon)$, which is contrary to (i). If (ii) holds, from (2.10), we have $\frac{dI_l(t)}{dt} = \beta l[1 - I_l(t)]\Theta(I(t-\tau)) - I_l(t) \le -I_l(t) \le -1$ for $\xi < t < \xi + \varepsilon$. It shows that $I_l(t)$ is non-increasing, which contradicts (ii). Thus $0 \le I_k(t) \le 1$ for all $t > 0$, $k = 1, 2, \ldots, n$. □

4. Stability analysis of (2.10)

In this section, we first discuss the local stability of the disease free equilibrium E^0 and endemic equilibrium E^*, after that, we illustrate the global asymptotic stability of the equilibriums.

Theorem 4.1. *For the model (2.10), one has*

(i) *If $R_0 < 1$, the disease-free equilibrium E^0 is locally asymptotically stable;*
(ii) *If $R_0 = 1$, the disease-free equilibrium E^0 is neutrally stable;*
(iii) *If $R_0 > 1$, the disease-free equilibrium E^0 is unstable.*

Proof. The linearized system of (2.10) at the equilibrium E^0 is

$$\frac{dI_k(t)}{dt} = \beta k\frac{\sum_k kp(k)I_k(t-\tau)}{\langle k\rangle} - I_k(t), \quad k = 1, 2, \ldots, n.$$

The associated characteristic equation is

$$F(\lambda)=\begin{vmatrix} \lambda+1-\frac{\beta}{\langle k\rangle}p(1)e^{-\lambda\tau} & -\frac{\beta}{\langle k\rangle}2p(2)e^{-\lambda\tau} & \cdots & -\frac{\beta}{\langle k\rangle}np(n)e^{-\lambda\tau} \\ -\frac{2\beta}{\langle k\rangle}p(1)e^{-\lambda\tau} & \lambda+1-\frac{2\beta}{\langle k\rangle}2p(2)e^{-\lambda\tau} & \cdots & -\frac{2\beta}{\langle k\rangle}np(n)e^{-\lambda\tau} \\ \cdots & & & \\ \frac{n\beta}{\langle k\rangle}p(1)e^{-\lambda\tau} & -\frac{n\beta}{\langle k\rangle}2p(2)e^{-\lambda\tau} & \cdots & \lambda+1-\frac{n\beta}{\langle k\rangle}np(n)e^{-\lambda\tau} \end{vmatrix}$$
$$=(\lambda+1)^{n-1}(\lambda+1-R_0e^{-\lambda\tau}). \tag{4.1}$$

It is easy to see that the characteristic equation (4.1) has $n-1$ eigenvalues which are all -1 and the remain eigenvalues satisfy the equation

$$\lambda+1-R_0e^{-\lambda\tau}=0. \tag{4.2}$$

So the stability of the disease-free equilibrium is decided by the roots of Eq. (4.2). Assume $\lambda=x+iy$ satisfies Eq. (4.2). Then we have $x+1+iy-R_0e^{-\lambda\tau}e^{iy}=0$, i.e. $x+1+iy=R_0e^{-\lambda\tau}e^{iy}$. So $(x+1)^2+y^2=R_0^2e^{-2\lambda\tau}$.

(i) If $R_0<1$, we are only required to verify that the all roots of (4.2) have negative real part.

For the sake of contradiction, suppose $x\geq 0$, then $1\leq(x+1)^2+y^2\leq R_0^2<1$. That is contrary, so $x<0$. Therefore E^0 is locally asymptotically stable.

(ii) If $R_0=1$, we are only required to prove $x\leq 0$.

When $y\neq 0$, suppose $x\geq 0$, then $1<(x+1)^2+y^2<R_0^2<1$. That is contrary, so $x<0$.

When $y=0$, suppose $x>0$, then $1<(x+1)^2+y^2<R_0^2=1$. That is contrary, so $x\leq 0$. if $x=0$, then (4.2) holds. This means that $\lambda=0$ is a root of (4.2). Since $F'(0)\neq 0$, $\lambda=0$ is only a simple root of (4.2). Thus E^0 is linear neutral stable.

(iii) If $R_0>1$, then $F(0)<0$ and $F(\infty)>0$. Therefore $F(\lambda)$ has at least one positive root. It indicates that E^0 is unstable. □

Theorem 4.2. *If $R_0>1$, the endemic equilibrium E^* is locally asymptotically stable.*

Proof. We take transform $u(t)=(u_1(t),u_2(t),\ldots,u_n(t))=I(t)-E^*$, i.e. $u_k(t)=I_k(t)-I_k^*$, $k=1,2,\ldots,n$, then

$$\frac{du_k(t)}{dt}=\beta k(1-I_k^*)\Theta(u(t-\tau))-(1+\beta k\Theta^*)u_k-\beta ku_k\Theta(u(t-\tau)),\ k=1,2,\ldots,n. \tag{4.3}$$

The linearized system of (2.10) at equilibrium E^* is

$$\frac{du_k(t)}{dt}=\beta k(1-I_k^*)\Theta(u(t-\tau))-(1+\beta k\Theta^*)u_k,\quad k=1,2,\ldots,n.$$

Denote $a_{ij}=\frac{\beta}{\langle k\rangle}(1-I_i^*)jp(j)e^{-\lambda\tau}$, $a_{ii}=-1-i\beta\Theta^*+\frac{\beta}{\langle k\rangle}(1-I_i^*)ip(i)e^{-\lambda\tau}$, $i,j=1,2,\ldots,n$, $i\neq j$.Then the associated characteristic equation is

$$F(\lambda)=\begin{vmatrix} \lambda-a_{11} & -a_{12} & \cdots & -a_{1n} \\ -a_{21} & \lambda-a_{22} & \cdots & -a_{2n} \\ \cdots & & & \\ -a_{n1} & -a_{n2} & \cdots & \lambda-a_{nn} \end{vmatrix}$$
$$=\prod_{k=1}^{n}(\lambda+1+k\beta\Theta^*)-\frac{\beta}{n}\sum_{k=1}^{n}k^2p(k)(1-I_k^*)\prod_{l\neq k,l=1}^{n}(\lambda+1+l\beta\Theta^*)e^{-\lambda\tau}. \tag{4.4}$$

Let

$$P(\lambda)=\prod_{k=1}^{n}(\lambda+1+k\beta\Theta^*),$$
$$Q(\lambda)=\frac{\beta}{n}\sum_{k=1}^{n}k^2p(k)(1-I_k^*)\prod_{l\neq k,l=1}^{n}(\lambda+1+l\beta\Theta^*).$$

Then Eq. (4.4) can be written as

$$F(\lambda)=P(\lambda)-Q(\lambda)e^{-\lambda\tau}. \tag{4.5}$$

Because $Q(-1-l\beta\Theta^*)Q(-1-(l+1)\beta\Theta^*)<0$, $l=1,2,\ldots,n-1$, $Q(\lambda)$ has at least one root in $(-1-(l+1)\beta\Theta^*,-1-l\beta\Theta^*)$, $l=1,2,\ldots,n-1$. Additionally $Q(\lambda)$ is $n-1$ order polynomial. So there exists only one root of $Q(\lambda)$ in $(-1-(l+1)\beta\Theta^*,-1-l\beta\Theta^*)$, $l=1,2,\ldots,n-1$. Denote it by $-\alpha_l,\alpha_l\in(1+l\beta\Theta^*,1+(l+1)\beta\Theta^*)$, $l=1,2,\ldots,n-1$. So $Q(\lambda)=\frac{\beta}{\langle k\rangle}\sum_{k=1}^{n}k^2p(k)(1-I_k^*)\prod_{l=1}^{n-1}(\lambda+\alpha_l)$. Notice that $\frac{\beta}{\langle k\rangle}\sum_{k=1}^{n}k^2p(k)(1-I_k^*)=1$, then $Q(\lambda)=\prod_{l=1}^{n-1}(\lambda+\alpha_l)$.

Next, we will prove at first $P(\lambda)$, $Q(\lambda)$ satisfy the conditions as follows:

(*I*) $P(\lambda)$ and $Q(\lambda)$ are analytic in the set $\Re\lambda\geq 0$ and $P(\lambda)\neq 0$, $\Re\lambda>0$,

(*II*) $\overline{P(-iy)} = P(iy)$, $\overline{Q(-iy)} = Q(iy)$, $0 \le y < +\infty$,
(*III*) $|Q(iy)| < |P(iy)|$, $0 \le y < +\infty$,
(*IV*) $\lim_{|\lambda|\to\infty, \Re\lambda > 0} \left|\frac{Q(\lambda)}{P(\lambda)}\right| = 0$.

Following, we will prove that there is no root of (4.5) in the set $\Re\lambda \ge 0$, i.e. all roots of (4.5) are in $\Re\lambda < 0$ for the given $\tau > 0$. It is obvious that the conditions (*I*) and (*II*) hold. Since

$$|P(iy)| = \prod_{k=1}^{n} \sqrt{(1 + k\beta\Theta^*)^2 + y^2},$$

$$|Q(iy)| = \prod_{l=1}^{n-1} \sqrt{\alpha_l^2 + y^2}, \quad 1 + l\beta\Theta^* < \alpha_l < 1 + (l+1)\beta\Theta^*, \quad l = 1, 2, \ldots, n-1,$$

we have

$$\left|\frac{P(iy)}{Q(iy)}\right| = \frac{\sqrt{(1+\beta\Theta^*)^2 + y^2}\sqrt{(1+2\beta\Theta^*)^2 + y^2} \ldots \sqrt{(1+n\beta\Theta^*)^2 + y^2}}{\sqrt{\alpha_1^2 + y^2} \ldots \sqrt{\alpha_{n-1}^2 + y^2}}$$
$$> \sqrt{(1+\beta\Theta^*)^2 + y^2} > 1.$$

This indicates that the condition (*III*) holds. Set $\lambda = x + iy$, then

$$\lim_{|\lambda|\to\infty, \Re\lambda > 0} \left|\frac{Q(iy)}{P(iy)}\right|$$
$$= \lim_{|\lambda|\to\infty, \Re\lambda > 0} \frac{\sqrt{(\alpha_1 + x)^2 + y^2} \ldots \sqrt{(\alpha_{n-1} + x)^2 + y^2}}{\sqrt{(1+\beta\Theta^* + x)^2 + y^2}\sqrt{(1+2\beta\Theta^* + x)^2 + y^2} \ldots \sqrt{(1+n\beta\Theta^* + x)^2 + y^2}}$$
$$< \lim_{|\lambda|\to\infty, \Re\lambda > 0} \frac{1}{\sqrt{(1+\beta\Theta^* + x)^2 + y^2}} = 0.$$

So $\lim_{|\lambda|\to\infty, \Re\lambda > 0} \left|\frac{Q(iy)}{P(iy)}\right| = 0$. The condition (*IV*) holds.

Now, we prove that there is no root of (4.5) in the set $\Re\lambda \ge 0$ by the way similar to [42].

Because $P(\lambda)$ and $Q(\lambda)$ are analytic in the set $\Re\lambda \ge 0$ and $P(\lambda) \ne 0$, for $\Re\lambda \ge 0$. The function $P(\lambda)/Q(\lambda)$ is analytic in $\Re\lambda \ge 0$. On a sufficiently large semi-circle $|\lambda| = K$ in $\Re\lambda \ge 0$, $|Q(\lambda)/P(\lambda)| \le \rho < 1$ because of (*IV*) and $|Q(\lambda)/P(\lambda)| \le \rho < 1$ on the line $\lambda = iy$, $-K \le y \le K$ because of (*III*). So by the maximum modulus principle, in every large semi-circle in the right half-plane, $|Q(\lambda)/P(\lambda)| < 1$. But in the right half-plane, $|e^{\lambda\tau}| > 1$. So $-Q(\lambda)/P(\lambda) = e^{\lambda\tau}$ cannot have root in the right half-plane. Namely, there is no root of (4.5) in the set $\Re\lambda \ge 0$. Thus, the endemic equilibrium E^* is locally asymptotically stable.

Following, we will discuss the global asymptotical stability of the disease-free equilibrium E^0 and the endemic equilibrium E^*. □

Theorem 4.3. *For the model (2.10), one has*

(i) *If $R_0 \le 1$, the disease-free equilibrium E^0 is globally asymptotically stable;*
(ii) *If $R_0 > 1$, the endemic equilibrium E^* is globally asymptotically stable.*

Proof. (i) Since E^0 is stable when $R_0 \le 1$, we need only to prove E^0 is globally attractive.

Let $\phi(\theta) = (\phi_1(\theta), \phi_2(\theta), \ldots, \phi_n(\theta)) \in C$, we construct a functional

$$V(\phi) = \frac{1}{2}\left[\sum_k kp(k)\phi_k(0)\right]^2 + \frac{1}{2}\int_{-\tau}^{0}\left[\sum_k kp(k)\phi_k(\theta)\right]^2 d\theta.$$

Set $\Phi(\theta) = \Sigma_k kp(k)\phi_k(\theta)$, then

$$V'_{(2.10)}(\phi) = \lim_{t\to 0^+}\frac{1}{t}(V(I_t) - V(\phi))$$
$$= \Phi(0)\Phi'(0) + \int_{-\tau}^{0}\Phi(\theta)\Phi'(\theta)d\theta$$
$$= \Phi(0)\left[\frac{\sum_k \beta k^2 p(k)}{\langle k\rangle}\Phi(-\tau) - \Phi(0)\right] + \frac{1}{2}\Phi^2(0) - \frac{1}{2}\Phi^2(-\tau)$$
$$\le \Phi(0)\Phi(-\tau) - \frac{1}{2}\Phi^2(0) - \frac{1}{2}\Phi^2(-\tau) \le 0$$

So $V(\phi)$ is a Lyapunov functional on C. Further, let $E = \{\phi \in \overline{C} | V'(\phi) = 0\}$. From above, $E = \{\phi \in \overline{C} | \Phi(0) = \Phi(-\tau) = 0\}$. Let M denotes the largest subset of E that is invariant with respect to the system (2.10). Then M consists of E^0 only. By Lyapunov–LaSalle invariance principle 3.1 (see Chapter 5 in [41]), the disease-free equilibrium E^0 is globally attractive. So E^0 is globally asymptotically stable.

(ii) We need only to prove E^* is global attractive. It can be completed by two steps. Firstly we prove C_1 and C_2 are attractive domains respectively. Secondly, we prove $C\backslash(C_1 \cup C_2)$ is attractive domain.

Step 1: Let $\psi(\theta) = (\psi_1(\theta), \psi_2(\theta), \ldots, \psi_n(\theta))$ be such that $I_t(\theta) = \psi(\theta) + E^* \in C_1$, then $\psi_k(\theta) \le 0$, $k = 1, 2, \ldots, n$, $\theta \in [-\tau, 0]$. We construct a functional

$$V(\psi) = \frac{1}{2}\left[\sum_k kp(k)\psi_k(0)\right]^2 + \frac{1}{2}\int_{-\tau}^{0}\left[\sum_k kp(k)\psi_k(\theta)\right]^2 d\theta.$$

Set $U(\theta) = \Sigma_k kp(k)\psi_k(\theta)$, then $U(\theta) \le 0$, $\theta \in [-\tau, 0]$. So

$$\begin{aligned} V'_{(4.3)}(\psi) &= U(0)\left[\frac{\sum_k \beta k^2 p(k)(1-I_k^*)}{\langle k\rangle}U(-\tau) - U(0) - \sum_k \beta k^2 p(k)\psi_k(0)\Theta(I(t))\right] + \frac{1}{2}U^2(0) - \frac{1}{2}U^2(-\tau) \\ &= U(0)U(-\tau) - \frac{1}{2}U^2(0) - \frac{1}{2}U^2(-\tau) - U(0)\sum_k \beta k^2 p(k)\psi_k(0)\Theta(I(t)). \end{aligned}$$

Since $U(0)\Sigma_k \beta k^2 p(k)\psi_k(0) \ge 0$ and $\Theta(I(t)) \ge 0$. So $V'(\psi) \le 0$. Further, $V'(\psi) = 0$ only if $U(0) = U(-\tau) = 0$. It corresponds $\psi_k(\theta) = 0$, $k = 1, 2, \ldots, n$, $\theta \in [-\tau, 0]$. By Lyapunov–LaSalle invariance principle 3.1 (see Chapter 5 in [41]), $(u_1(t), u_2(t), \ldots, u_n(t)) \to (0, 0, \ldots, 0)$. So $\forall \phi \in C_1$, $I(t, \phi) \to E^*$.

By the same way, we can obtain that for $\forall \phi \in C_2$, $I(t, \phi) \to E^*$.

Step 2: We define

$$F(t, \phi) = \begin{pmatrix} \beta[1-\phi_1(0)]\Theta(\phi(-\tau)) - \phi_1(0) \\ 2\beta[1-\phi_2(0)]\Theta(\phi(-\tau)) - \phi_2(0) \\ \cdots \\ k\beta[1-\phi_k(0)]\Theta(\phi(-\tau)) - \phi_k(0) \\ \cdots \\ n\beta[1-\phi_n(0)]\Theta(\phi(-\tau)) - \phi_n(0) \end{pmatrix}$$

for any $\phi \in C$. Thus, the system (2.10) can be reformulated as:

$$\frac{dI(t)}{dt} = F(t, I_t). \tag{4.6}$$

Define the solution semi-flow $\Psi(t)\phi = I_t(\phi) = I(t, \phi)$, $\phi \in C$, $t \ge 0$. Since f is cooperative in C, by Theorems 4.1 and 4.5 (see Hirsh and Smith [43]), semiflow $\Psi(t)$ defined by (4.5) is monotone. Thus, for all $\phi \in C\backslash(C_1 \cup C_2)$, there exist $\phi' \in C_1$, $\phi'' \in C_2$ such that $\phi'(\theta) < \phi(\theta) < \phi''(\theta)$, $\theta \in [-\tau, 0]$. By the monotony of $\Psi(t)$, we have $\Psi(t)\phi' < \Psi(t)\phi < \Psi(t)\phi''$, i.e. $I(t, \phi') < I(t, \phi) < I(t, \phi'')$. From the results obtained in step 1, it follows that $I(t, \phi) \to E^*$.

So the endemic equilibrium E^* is globally attractive. Furthermore, it is globally asymptotically stable. □

5. Simulations

In this section, we use model (2.9) to simulate the evolution behavior of the vector-borne diseases to support the results obtained in the previous sections. Meanwhile, we display the effect of topology structure of networks and the time delay τ on vector-borne diseases spread.

In [14], Ruan et al. have given a summary about the range of parameters in malaria transmission. So in our simulations, we choose some parameter values by the summary sheet in [14]. The recovery rate γ of an infective human varies from 0.01 to 0.05 per day, we take $\gamma = 0.01$. The biting rate on humans by a single mosquito is about 0.2 to 0.5 per day, here we take $a = 0.2$. The probability of infected bites on both human and mosquito that produces an infection is $b = c = 0.5$. And the death rate of mosquitoes δ varies from 0.05 to 0.5 per day, we choose $\delta = 0.5$ per day. We assume the size of human population is 1000, the size of vector population in each subregion is 1000, and the number of subregions $n = 100$. To study the effect the time delay τ on vector-borne diseases spread, we have chosen different values of the time delay τ which varies from 5 to 15 days [14]. The degree distribution for human population is assumed as $p(k) \sim k^{-\mu}$, where $\mu = 2.4$. Let $i(t) = \sum_k p(k)I_k(t)$ be the total density of infective humans.

As predicted by the analytic calculation, Fig. 2 shows that if $R_0 < 1$, the disease will disappear eventually. From Fig. 2(a), we can see, for different delay lengths, the relative density $I_{40}(t)$ reaches the same asymptotic value, 0. But the steady state is reached at different times. When the lengths of the delay are 13, 13.5 and 14 respectively, $I_{40}(t)$ takes 625, 230 and 140 time steps respectively to reach its steady state. Similar phenomenon can be found in Fig. 2(b). It indicates that when $R_0 < 1$, the time delay speeds up the disappearing process of disease.

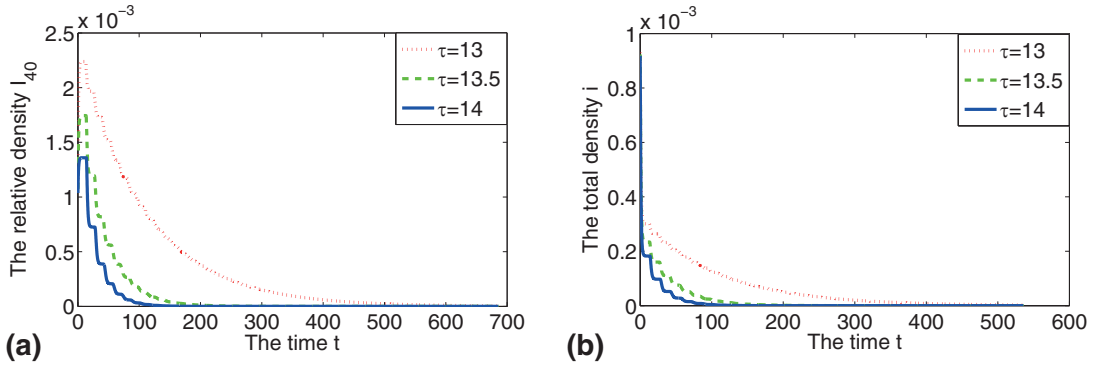

Fig. 2. The time evolutions under the condition of $R_0 < 1$ and $I_k(t) = 0.001$, $t \in [-\tau, 0]$, $k = 1, 2, \ldots, n$. (a) The time evolutions of the relative density I_{40} for different lengths of the delay τ; (b) The time evolutions of the total density i for different lengths of the delay τ.

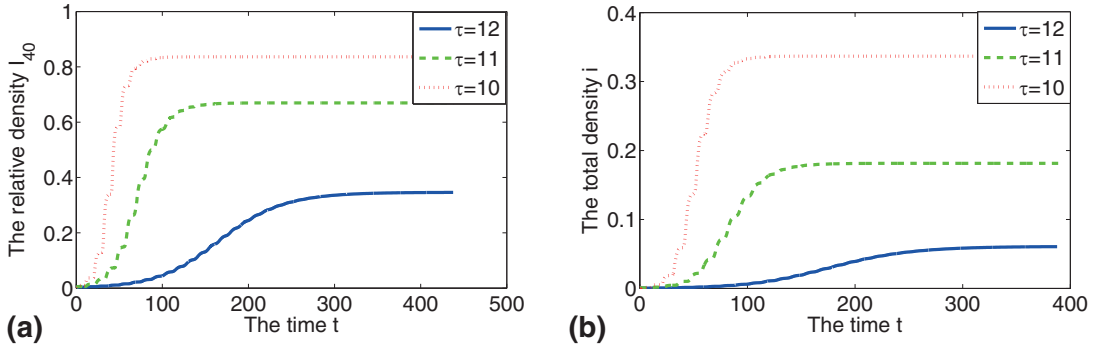

Fig. 3. The time evolutions under the condition of $R_0 > 1$ and $I_k(t) = 0.001$, $t \in [-\tau, 0]$, $k = 1, 2, \ldots, n$. (a) The time evolutions of the relative density I_{40} for different lengths of the delay τ; (b) The time evolutions of the total density i for different lengths of the delay τ.

When $R_0 > 1$, for different delay lengths, the time evolution of the relative density I_{40} and the total density i are displayed in Fig. 3. From the figure, it is clear that both of the relative density $I_{40}(t)$ and the total density $i(t)$ reach different asymptotic values for different lengths of the delay, and the asymptotic values of $I_{40}(t)$ and $i(t)$ decrease with increasing τ. Another phenomenon we can see, when we take 3 different values of the time delay, 10, 11 and 12, the relative density $I_{40}(t)$ takes 100, 200 and 420 time steps to reach its steady state, while for the total density $i(t)$, the time needed is 110, 180 and 370 respectively. That means the time it takes for the relative density $I_{40}(t)$ and the total density $i(t)$ to reach their steady states increases with increasing τ. From these phenomena, we can draw a conclusion: in the case of $R_0 > 1$, the larger the delay length τ is, the more slowly and less widely the disease spreads.

From Figs. 2 and 3, we know that the time delay plays important role in the propagation process and the larger the delay τ, the more the benefit to humans.

Fig. 4 shows the time evolutions of the relative densities I_{20}, I_{30} and I_{40} to demonstrate the heterogeneity induced by the presence of humans with different connectivity. Fig. 4(a) displays the case of $R_0 < 1$. From it, we can see that the relative densities $I_{20}(t)$, $I_{30}(t)$ and $I_{40}(t)$ with the same initial data take 655, 650 and 625 time steps respectively to reach the same steady state. As shown in Fig. 4(b), when $R_0 > 1$, $I_{20}(t)$, $I_{30}(t)$ and $I_{40}(t)$ take 110, 105 and 100 time steps respectively to reach their steady state. Particularly, the asymptotic values have the relation $I^*_{40} > I^*_{30} > I^*_{20}$. That is, in the steady state, the relative density I^*_k increases with the increasing connect degree k of humans. These phenomena can be explained easily by the fact: the larger the human's connect degree, the higher the probability to be infected.

6. Discussion

Various researches have shown that there are heterogeneities in host–vector contact (see the study [44]) for vector-borne diseases. As noted in [31], bipartite networks can provide the natural framework to investigate the spread of vector-borne diseases. However capturing the degree of each mosquito and each human is difficult.

In this paper, firstly, we provide a way to model the spread of vector-borne diseases on bipartite networks. In view of the habit of vectors limited dispersal from their breeding sites, the whole region where vector-borne disease occurs is divided into several subregions. Then we construct bipartite networks where there are two classes nodes, humans and subregions. An edge placed between a subregion and a human represents human enters the subregion and is fully mixed with the vectors in the subregion.

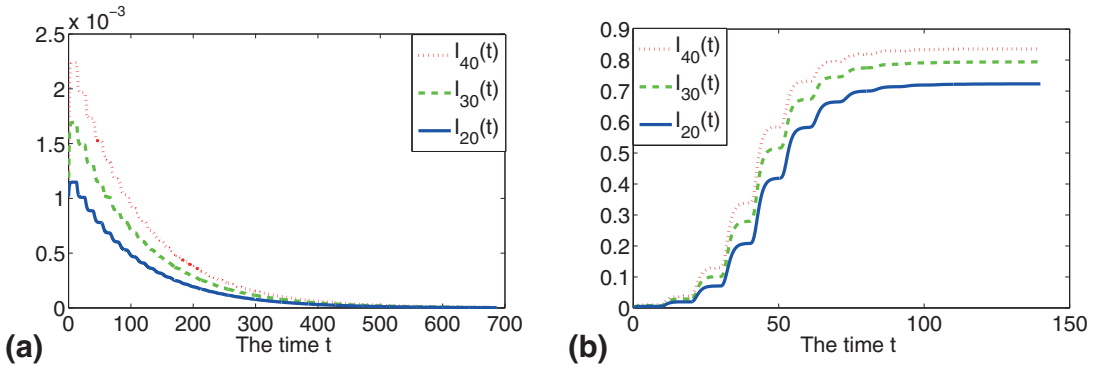

Fig. 4. (a) The time evolutions of the relative densities I_{20}, I_{30} and I_{40} with the initial condition $I_k(t) = 0.001$, $t \in [-13, 0]$, $k = 1, 2, \ldots, n$, when $R_0 = 0.8801$ and $\tau = 13$; (b) The time evolutions of the relative densities I_{20}, I_{30} and I_{40} with the initial condition $I_k(t) = 0.001$, $t \in [-10, 0]$, $k = 1, 2, \ldots, n$, when $R_0 = 3.9446$ and $\tau = 10$.

Thus heterogeneity of human behaviors can interpret the heterogeneity in human–vector contact. Moreover it can estimate the degree distribution of vectors.

Secondly, we derive a SIS model with a time delay on bipartite networks and analyze it to uncover the effect of the structure of networks and the time delay on vector-borne diseases spread. We have given the basic reproduction number R_0 that determines the epidemic spread and proved that if $R_0 \leq 1$, disease-free equilibrium E^0 is globally asymptotically stable; while $R_0 > 1$, the endemic equilibrium E^* is globally asymptotically stable. This result shows that the time delay doesn't destabilize the system. It is also confirmed by simulations.

From the expression for the basic reproduction number

$$R_0 = \frac{\beta \langle k^2 \rangle}{\langle k \rangle} = \frac{a^2 bcnV}{e^{\delta\tau} \gamma \delta N \langle k \rangle} \times \frac{\langle k^2 \rangle}{\langle k \rangle},$$

where $\frac{N\langle k \rangle}{n} = \frac{\sum_k k^2 q(k)}{\sum_k k q(k)}$ and $\frac{\langle k^2 \rangle}{\langle k \rangle} = \frac{\sum_k k^2 p(k)}{\sum_k k p(k)}$, we can obtain that the basic reproduction number depends on the structure of bipartite networks. The study [30] also demonstrated it. Furthermore, the basic reproduction number depends on the time delay and it decreases with the increasing delay length. By simulations, it is confirmed that when the disease will become endemic, the time delay can slow down the spreading of vector-borne diseases and the density of infective humans in the steady state decreases with the increasing delay length. The results suggest that the structure of the bipartite networks and the time delay play important roles in the propagation process.

There are some constraints on vector numbers in each subregion and the degree of each subregion in the present study. Even though our results will be greatly beneficial for us to understand the spreading behaviors and design effective epidemic-control strategies. In the near future, we will take into account various size of vector population in subregions and more realistic degree distribution of subregions to further study the vector-borne diseases spread on bipartite networks.

Acknowledgments

This work was supported by the National Natural Science Foundation (nos. 61272095, 61175067, 71031006, 11331009), Shanxi Scholarship Council of China (2013–014), Project Supported by National Science and Technology (no. 2012BAH33B01).

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