



Research article

The impact of media converge on complex networks on disease transmission

Maoxing Liu^{1,*}, Shushu He¹ and Yongzheng Sun²

¹ School of Science, North University of China, Taiyuan, Shanxi 030051, P. R. China

² School of Science, China University of Mining and Technology, Xuzhou, Jiangsu, 221008, P. R. China

* **Correspondence:** Email: liumaoxing@126.com; Tel: +86-351-394-2729.

Abstract: In this paper, we propose an epidemic disease model about the effect of media coverage on complex networks, where the contacts between nodes are treated as a social network. We calculate the basic reproduction number R_0 and get that the disease-free equilibrium is locally and globally asymptotically stable if $R_0 < 1$, otherwise disease-free equilibrium is unstable and there exists a unique endemic equilibrium, and the disease is permanent. And two immunization strategies are considered: proportional and target immunization. By comparing two immunization strategies, it is found that the target immunization is better than the proportional immunization. Finally, numerical simulations verify our results and some discussions of vaccination strategies are done in the control of infectious diseases.

Keywords: infectious diseases; complex network; basic reproduction number; stability; immunization

1. Introduction

In the real world many systems can be described by complex networks, ranging from nature to society. Recently the study of epidemic systems on complex networks has attracted a lot of attention and interest [1, 2, 3]. Within these studies, individuals are modeled as nodes, and possible contacts between individuals are linked by edges. The susceptible-infected-susceptible (SIS)[4], the susceptible-infected (SI) [5], the susceptible-infected-recovered (SIRS) [6], and the susceptible-exposed-infected-recovered (SEIRS) models on complex networks [7] have been extensively studied recently. According to reference[8], successful immunization strategies can be developed only by taking into account the inhomogeneous connectivity properties of scale-free networks. Alberto d’Onofrio description of the global behavior of the web-based SIS epidemic model is given in reference [9]. Jie Lou et al. studied the transmission dynamics and immunization strategies of sexually transmitted diseases on scale-free networks [10]. One of reasons for studying networks is to understand the mechanisms by which dis-

eases and other things, such as disease information spread over. For instance, the study of networks of sexual contact [11, 12] is helpful for us to understand and perhaps control the spread of sexually transmitted diseases. It also has been shown that there is an epidemic threshold on the homogeneous Watts Strogatz small-world network model, while the epidemic model on SF networks has no epidemic threshold; infections can be proliferated, whatever small infection rate they have. This result disproves the threshold theory in traditional epidemiology.

As we all know, human behavior in an epidemic include the contacts between human, media coverage, vaccination, migration of population etc. In fact, these factors are important for infectious disease spread. Particularly, it is media coverage which make people knowledgeable about the diseases to take precautions such as social distancing, wearing protective masks, vaccination etc. At present, some researchers take the media converge into considering on mathematical models and there is a lot of evidence that media coverage can play an appreciable role in the spread and control of infectious diseases [13, 14, 15, 16]. Zuo et al. proposed a compartment epidemic model with delay, and discussed the influence of consciousness planning on the spread and control of infectious diseases in a specific area [17]. In reference [18, 19], the influence of media during influenza epidemics is simulated. In recent paper [20], the authors review the literature on the effect of media on infectious disease outbreaks, and they considered a deterministic differential equation mean field SIS epidemic model for the spread of an infection in the presence of awareness programs, and analyzed two types of time delays and then perform simulations based on real parameter values to verify their theoretical results. However, the spread of an infectious disease depends not only on the characters of the disease, but also on the structure of the population. Thus many epidemic models with media coverage on network are proposed [21, 22]. Wang et al. [21] studied SIS models with media coverage in complex networks. They discussed the relationship between the parameters of the model and the basic reproduction number, and analyzed the stability of equilibria. Yuan et al. [22] proposed an epidemic disease model about the effect of awareness programs on complex networks, where the contacts between nodes were treated as a social network, and two forms on increasing rate of awareness programs were analyzed. These results can suggest effective control strategies, such as proportional immunization, targeted immunization etc., to prevent disease through media coverage and education activities on networks.

Based on predecessors' work, we propose a complex network model with media converge for predicting the future course of any epidemic such as smallpox, measles and flu, and so on. We consider that due to media converge, some susceptible will avoid their contacts with the infective. In the dynamic analysis, we use the method of next generation matrix to obtain the expression of the reproduction number and the condition for existence of the endemic equilibria. Furthermore, we analyze the eigenvalues of Jacobian matrix to prove the stability of the disease-free equilibrium and the permanence of infection. Two immunization strategies were considered: proportional and target immunization, and by comparing the two immunization strategies, it was found that the target immunization was better than proportional immunization in controlling the spread of the disease. Finally, we take some numerical simulations to illustrate the effect of media coverage on the disease.

This paper is organized as follows. We first propose the model in Section 2. Section 3 is devoted to the existence of equilibria and basic reproduction number, followed by the stability of the disease-free equilibrium and the permanence of infection, and then two immunization strategies are given. Numerical simulations and analysis are given in Section 4. Section 5 concludes the paper.

2. A model with media coverage

In this paper we consider the whole population and their contacts in networks. When disease transmission is modeled on networks, individuals are treated as nodes, and potential contacts among individuals as edges. To address the heterogeneity in the contacts among individuals, the population is divided into n distinct groups of size $N_k (k = 1, 2, \dots, n)$ such that each individual in group k has exactly k contacts; Here n denotes the maximum degree of all nodes. If the whole population size is N , then $N = N_1 + N_2 + \dots + N_n$ and the degree distribution is $p(k) = N_k/N$. The value $\langle k \rangle = \sum_{k=1}^n kp(k)$ is the average number of contacts each node.

We consider the fundamental epidemic model: SIS model. In this model each individual can be in two states: S , susceptible to the disease, and I , infectious individuals. Denote by $S_{1k}(t)$, $S_{2k}(t)$ and $I_k(t)$ the number of unaware susceptible, the aware susceptible and infected individuals within the group k at time t , respectively; then $N_k(t) = S_{1k}(t) + S_{2k}(t) + I_k(t)$. It is assumed that the disease spreads due to direct contacts between susceptible and infected individuals only. In order to incorporate the dimensions of massive news coverage, we introduce the information variable $M(t)$ to describe the cumulative density of media coverage in that region at time t .

Based on the above assumptions, we have the following dynamic model of $3n + 1$ ordinary differential equations:

$$\begin{cases} \frac{dS_{1k}(t)}{dt} = -\beta k S_{1k}(t)\Theta(t) + \gamma q I_k(t) - \xi S_{1k}(t), \\ \frac{dS_{2k}(t)}{dt} = -\beta(1 - B(M, k))k S_{2k}(t)\Theta(t) + \gamma(1 - q)I_k(t) + \xi S_{1k}(t), \\ \frac{dI_k(t)}{dt} = \beta k S_{1k}(t)\Theta(t) + \beta(1 - B(M, k))k S_{2k}(t)\Theta(t) - \gamma I_k(t), \\ \frac{dM(t)}{dt} = m_0 + \omega \sum_{k=1}^n I_k(t) - \mu_0 M(t). \end{cases} \quad (2.1)$$

In this model, $k = 1, 2, \dots, n$. The parameter β is transmission rate per contact between one susceptible and one infectious individual; parameter γ is recovery rate of infected individuals; since the information reported by the media is always available, parameter ξ represents the conversion rate of transformation from high susceptible to low susceptible; parameter q denotes the probability that the person transformed into a highly susceptible person after recovery; parameter μ_0 is depletion rate of media coverage due to ineffective implementation; parameter m_0 is density level of media coverage on the disease from another region with the disease; parameter ω is the media reporting rate being implemented.

Here in uncorrelated networks, $\Theta(t)$ represents the expectation that any given edge points to an infected individual, defined as:

$$\Theta(t) = \frac{\sum_{k=1}^n k I_k}{\sum_{k=1}^n k N_k}.$$

And we consider the media coverage function $B(M, k) = \frac{kM(t)}{a+kM(t)}$ as being similar to the Michaelis-Menten function $p_1(M) = \frac{cM}{c+dM} (c, d \geq 0)$ (see [15, 21] and the references cited therein). $B(M, k)$ measures the impact of media coverage on a individual in group k , which is related to the number of people he/she meets and the current density level of media coverage. The term $B(M, k)$ measures the

effect of the reduction of transmission rate between susceptible and infectious individuals due to the reporting of infectious cases in the media. The half-saturation constant $a > 0$ reflects the impact of media coverage on contact transmission. All parameters in the model are assumed to be positive except that m_0 is nonnegative.

Note that $\frac{dS_{1k}(t)}{dt} + \frac{dS_{2k}(t)}{dt} + \frac{dI_k(t)}{dt} = 0$, which means that $S_{1k}(t) + S_{2k}(t) + I_k(t) = N_k$ is constant. Denote the relative densities of the unaware susceptible nodes, the aware susceptible nodes and infected nodes of degree k at time t by $s_{1k}(t) = \frac{S_{1k}(t)}{N_k}$, $s_{2k}(t) = \frac{S_{2k}(t)}{N_k}$, $i_k(t) = \frac{I_k(t)}{N_k}$, respectively and $\Theta(t) = \frac{1}{\langle k \rangle} \sum_{k=1}^n kp(k)i_k(t)$. Then system (2.1) can be rewritten as:

$$\begin{cases} \frac{ds_{1k}(t)}{dt} = -\beta k s_{1k}(t)\Theta(t) + \gamma q i_k(t) - \xi s_{1k}(t), \\ \frac{ds_{2k}(t)}{dt} = -\beta(1 - B(M, k))k s_{2k}(t)\Theta(t) + \gamma(1 - q)i_k(t) + \xi s_{1k}(t), \\ \frac{di_k(t)}{dt} = \beta k s_{1k}(t)\Theta(t) + \beta(1 - B(M, k))k s_{2k}(t)\Theta(t) - \gamma i_k(t), \\ \frac{dM(t)}{dt} = m_0 + \omega N \sum_{k=1}^n p(k)i_k(t) - \mu_0 M(t), \end{cases} \quad (2.2)$$

with the normalization condition $s_{1k}(t) + s_{2k}(t) + i_k(t) = 1$, and $\Omega \doteq \prod_{k=1}^n [0, 1] \times [m_0/\mu_0, (m_0 + \omega N)/\mu_0]$ is the region of attraction, which attracts all solutions initiating in the interior of the positive orthant.

3. Analysis of the model

3.1. The stability of the disease-free equilibrium

It can be easily see system (2.2) always exists a unique disease-free equilibrium $E_0(\underbrace{0, 1, 0, \dots, 0, 1, 0}_{3n}, M_0)$, where $M_0 = m_0/\mu_0$. We notice that only compartments i_k are involved in

the calculation of the basic reproduction number R_0 , which is defined as the expected number of secondary infections produced by an index case[23]. For this disease-free equilibrium E_0 , the production of new infections \mathcal{F} and the rate of transfer of individuals \mathcal{V} are given respectively by

$$\mathcal{F} = \begin{pmatrix} \beta s_{11}(t)\Theta(t) + \beta(1 - B(M, 1))s_{21}(t)\Theta(t) \\ 2\beta s_{12}(t)\Theta(t) + 2\beta(1 - B(M, 2))s_{22}(t)\Theta(t) \\ \vdots \\ n\beta s_{1n}(t)\Theta(t) + n\beta(1 - B(M, n))s_{2n}(t)\Theta(t) \end{pmatrix}, \mathcal{V} = \begin{pmatrix} \gamma i_1(t) \\ \gamma i_2(t) \\ \vdots \\ \gamma i_n(t) \end{pmatrix}.$$

Then we have the derivatives at the disease-free equilibrium E_0 :

$$\bar{F} = \frac{\beta}{\langle k \rangle} \begin{pmatrix} a_1 p(1) & a_1 2p(2) \cdots & a_1 n p(n) \\ a_2 p(1) & a_2 2p(2) \cdots & a_2 n p(n) \\ \vdots & \vdots & \vdots \\ a_n p(1) & a_n 2p(2) \cdots & a_n n p(n) \end{pmatrix}, \bar{V} = \begin{pmatrix} \gamma & 0 & 0 \cdots & 0 \\ 0 & \gamma & 0 \cdots & 0 \\ 0 & 0 & \gamma \cdots & 0 \\ \vdots & \vdots & \ddots & \vdots \\ 0 & 0 & 0 \cdots & \gamma \end{pmatrix},$$

where $a_1 = \frac{a}{a+M_0}$, $a_2 = \frac{2a}{a+2M_0}$, \dots , $a_n = \frac{na}{a+nM_0}$. Thus, the reproduction number is given by $R_0 = \rho(\bar{F}\bar{V}^{-1})$, the spectral radius of the matrix $\bar{F}\bar{V}^{-1}$. Then we obtain the basic reproduction number as follows:

$$R_0 = \frac{a\beta\mu_0}{\gamma\langle k \rangle} \sum_{k=1}^n \frac{k^2 p(k)}{a\mu_0 + km_0}.$$

Remark 1. When $m_0 \rightarrow 0^+$, we obtain $R_0 = \frac{\beta \langle k^2 \rangle}{\gamma \langle k \rangle}$, which is consistent with the classical result obtained by Pastor et al.[4]. If $m_0 = 0$, the existence of media coverage will not change the epidemic threshold, but it may affect the prevalence of the disease.

For the stability of the disease-free equilibrium we have the following result.

Theorem 1 *If $R_0 < 1$, the disease-free equilibrium E_0 of system (2.2) is locally asymptotically stable, whereas if $R_0 > 1$ the disease-free equilibrium E_0 is unstable.*

Proof The Jacobian matrix of system (2.2) at the disease-free equilibrium E_0 is

$$J|_{E_0} = \begin{pmatrix} F & O \\ H & -\mu_0 \end{pmatrix},$$

where

$$F = \begin{pmatrix} F_1 & F_2 \\ F_3 & F_4 \end{pmatrix}, O = (0, 0, \dots, 0)^T, H = (0, \dots, \omega NP(1), \dots, \omega NP(n)).$$

The matrix $J|_{E_0}$ has one eigenvalues which is equal to $-\mu_0$. Next we need to find the eigenvalues of the matrix F , where

$$F_1 = \begin{pmatrix} -\xi \cdots 0 & 0 & \cdots & 0 \\ \vdots & \vdots & \vdots & \vdots \\ 0 \cdots -\xi & 0 & \cdots & 0 \\ \xi \cdots 0 & 0 & \cdots & 0 \\ \vdots & \vdots & \vdots & \vdots \\ 0 \cdots \xi & 0 & \cdots & 0 \end{pmatrix}, F_3 = \begin{pmatrix} 0 \cdots 0 & 0 \cdots 0 \\ 0 \cdots 0 & 0 \cdots 0 \\ \vdots \cdots \vdots & \vdots \cdots \vdots \\ 0 \cdots 0 & 0 \cdots 0 \end{pmatrix}.$$

$$F_4 = \begin{pmatrix} \beta \frac{a}{a+1M} 1 \frac{1}{\langle k \rangle} 1P(1) - \gamma & \beta \frac{a}{a+1M} \frac{1}{\langle k \rangle} 2P(2) & \cdots & \beta \frac{a}{a+1M} \frac{1}{\langle k \rangle} nP(n) \\ \beta \frac{a}{a+2M} 2 \frac{1}{\langle k \rangle} 1P(1) & \beta \frac{a}{a+2M} 2 \frac{1}{\langle k \rangle} 2P(2) - \gamma & \cdots & \beta \frac{a}{a+2M} 2 \frac{1}{\langle k \rangle} np(n) \\ \vdots & \vdots & \ddots & \vdots \\ \beta \frac{a}{a+nM} n \frac{1}{\langle k \rangle} 1P(1) & \beta \frac{a}{a+nM} n \frac{1}{\langle k \rangle} 2P(2) & \cdots & \beta \frac{a}{a+nM} n \frac{1}{\langle k \rangle} np(n) - \gamma \end{pmatrix}.$$

By analysis, the eigenvalues of F are only related to F_1 and F_4 , the matrix F_1 has n eigenvalues which are equal to $-\xi$, and has n eigenvalues which are equal to 0. Through the determinant changes, the matrix F_4 has $(n - 1)$ eigenvalues which are equal to $-\gamma$ and one eigenvalue which is equal to $\frac{\beta a \mu_0}{\langle k \rangle} \sum_{k=1}^n \frac{k^2 p(k)}{a \mu_0 + k m_0} - \gamma$. Obviously, if $R_0 < 1$, we can get $\frac{\beta a \mu_0}{\langle k \rangle} \sum_{k=1}^n \frac{k^2 p(k)}{a \mu_0 + k m_0} - \gamma$ is negative. Thus the proof is completed.

For the globally stability of the disease-free equilibrium, we have

Theorem 2 *If $R_0 < 1$, the disease-free equilibrium E_0 of system (2.2) is globally asymptotically stable.*

Proof From system (2.2), we have

$$\begin{cases} \frac{di_k(t)}{dt} \leq \beta k(1 - i_k(t))\Theta(t) - \gamma i_k(t), \\ \frac{dM(t)}{dt} = m_0 + \omega N \sum_{k=1}^n p(k) i_k(t) - \mu_0 M(t). \end{cases} \quad (3.1)$$

Let us consider the following auxiliary system:

$$\begin{cases} \frac{di_k(t)}{dt} = \beta k(1 - i_k(t))\Theta(t) - \gamma i_k(t), \\ \frac{dM(t)}{dt} = m_0 + \omega N \sum_{k=1}^n p(k) i_k(t) - \mu_0 M(t). \end{cases} \quad (3.2)$$

The basic reproduction number for system (3.2) is $R'_0 = \frac{\beta \langle k^2 \rangle}{\gamma \langle k \rangle}$. When $R_0 < 1$, since R_0 is decreasing function of m_0 , we can get $R'_0 = \lim_{m_0 \rightarrow 0^+} R_0 \leq 1$. Since the first equation of system (3.2) is independent of the second, according to the results obtained by d'Onofrio et al.[9], we can obtain that the disease-free equilibrium is globally asymptotically stable if $R'_0 < 1$, i.e. $\lim_{t \rightarrow +\infty} i_k(t) = 0, k = 1, 2, \dots, n$. The limit system of the information variable $M(t)$ satisfies $\frac{dM(t)}{dt} = m_0 - \mu_0 M(t), M(t) = \frac{m_0 - ce^{-\mu_0 t}}{\mu_0}$, and it comes to $\lim_{t \rightarrow +\infty} M(t) = M_0$. By comparison arguments, we know that the disease-free equilibrium E_0 is globally attractive for system (2.2). Thus if $R_0 < 1$, the disease-free equilibrium E_0 of system (2.2) is globally asymptotically stable.

3.2. The existence of the endemic and permanent

As for the existence of the endemic equilibrium of system (2.2), we have the following theorem.

Theorem 3 If $\gamma \xi \omega N \langle k \rangle + a \mu_0 \xi \beta \geq \gamma n m_0 q \beta$, and $R_0 > 1$, system (2.2) exists one and only one positive equilibrium $E^*(s_{11}^*, s_{21}^*, i_1^*, \dots, s_{1n}^*, s_{2n}^*, i_n^*, M^*)$.

Proof To discuss the endemic equilibrium, by imposing the stationary condition we consider the following equations:

$$\begin{cases} -\beta k s_{1k}^* \Theta + \gamma q i_k^* - \xi s_{1k}^* = 0, \\ -\beta(1 - B(M^*, k)) k s_{2k}^* \Theta + \gamma(1 - q) i_k^* + \xi s_{1k}^* = 0, \\ \beta k s_{1k}^* \Theta + \beta(1 - B(M^*, k)) k s_{2k}^* \Theta - \gamma i_k^* = 0, \\ m_0 + \omega N \sum_{k=1}^n p(k) i_k^* - \mu_0 M^* = 0. \end{cases} \quad (3.3)$$

The fourth equation of (3.3) yield $M^* = \frac{m_0 + \omega N i^*}{\mu_0}$, where $i^* = \sum_{k=1}^n p(k) i_k^*$. The first equation of (3.3) yield

$$s_{1k}^* = \frac{\gamma q i_k^*}{\beta k \Theta + \xi}.$$

The second equation of (3.3) yield

$$s_{2k}^* = \frac{\gamma(1 - q)(a + kM^*)(\beta k \Theta + \xi) i_k^* + (a + kM^*) \xi \gamma q i_k^*}{\beta k a \Theta (\beta k \Theta + \xi)}.$$

Then we obtain

$$\begin{aligned} i_k^* &= 1 - s_{1k}^* - s_{2k}^* = 1 - \frac{\gamma q i_k^*}{\beta k \Theta + \xi} - \frac{\gamma(1 - q)(a + kM^*)(\beta k \Theta + \xi) i_k^* + (a + kM^*) \xi \gamma q i_k^*}{\beta k a \Theta (\beta k \Theta + \xi)} \\ &= \frac{\beta k a \Theta (\beta k \Theta + \xi) - \beta k a \Theta \gamma q i_k^* - \gamma(1 - q)(a + kM^*)(\beta k \Theta + \xi) i_k^* - (a + kM^*) \xi \gamma q i_k^*}{\beta k a \Theta (\beta k \Theta + \xi)}. \end{aligned}$$

Thus

$$i_k^* = \frac{\beta k a \Theta (\beta k \Theta + \xi)}{\beta k a \Theta (\beta k \Theta + \xi) + \beta k a \Theta \gamma q + \gamma(1 - q)(a + kM^*)(\beta k \Theta + \xi) + (a + kM^*) \xi \gamma q}.$$

Substituting M^* into the expression of i_k^* , then we get

$$i_k^* = \frac{\beta k a \Theta (\beta k \Theta + \xi)}{\beta k a \Theta (\beta k \Theta + \xi) + \beta k a \Theta \gamma q + \gamma(1 - q)(a + kM^*)(\beta k \Theta + \xi) + (a + kM^*) \xi \gamma q}$$

$$\begin{aligned}
&= \frac{\beta ka\Theta(\beta k\Theta + \xi)}{\beta ka\Theta(\beta k\Theta + \xi) + \beta ka\Theta\gamma q + \gamma(1-q)(a + k\frac{m_0 + \omega Ni^*}{\mu_0})(\beta k\Theta + \xi) + (a + k\frac{m_0 + \omega Ni^*}{\mu_0})\xi\gamma q} \\
&= \frac{\beta ka\Theta(\beta k\Theta + \xi)\mu_0}{\beta ka\Theta(\beta k\Theta + \xi)\mu_0 + \beta ka\Theta\gamma q\mu_0 + [\gamma(1-q)(\beta k\Theta + \xi) + \xi\gamma q](a\mu_0 + km_0 + \omega N\langle k\rangle\Theta)}.
\end{aligned}$$

Substituting i_k^* into the expression of $\Theta(t)$, then we obtain a self-consistency equation as follows:

$$\Theta = \frac{1}{\langle k \rangle} \sum_{k=1}^n kp(k)i_k^* = \frac{1}{\langle k \rangle} \sum_{k=1}^n kp(k)\frac{A}{B} \doteq G(\Theta).$$

Here

$$\begin{aligned}
A &= \beta^2 k^2 \Theta^2 a \mu_0 + \beta ka \Theta \xi \mu_0, \\
B &= \beta^2 k^2 \Theta^2 a \mu_0 + \beta ka \Theta \xi \mu_0 + \gamma \beta k \Theta a \mu_0 + \gamma \beta k^2 \Theta m_0 + \gamma \beta k \Theta^2 \omega N \langle k \rangle \\
&\quad + \gamma \xi a \mu_0 + \gamma \xi k m_0 + \gamma \xi \omega N \langle k \rangle \Theta - \gamma q \beta k^2 \Theta m_0 - \gamma q \beta k \Theta^2 \omega N \langle k \rangle.
\end{aligned}$$

So, we obtain

$$\left. \frac{dG(\Theta)}{d\Theta} \right|_{\Theta=0} = \frac{\beta a \mu_0}{\gamma \langle k \rangle} \sum_{k=1}^n \frac{k^2 p(k)}{a \mu_0 + k m_0} = R_0 > 1,$$

$$\frac{d^2 G(\Theta)}{d\Theta^2} = \frac{1}{\langle k \rangle} \sum_{k=1}^n kp(k) \frac{C}{D},$$

where

$$\begin{aligned}
C &= -2\gamma a \beta^3 k^3 \mu_0 (N \langle k \rangle \omega q \xi - \beta k^2 m_0 q + a \beta k \mu_0 + \beta k^2 m_0) (N \langle k \rangle \gamma \omega q - N \langle k \rangle \gamma \omega - a \beta k \mu_0) \Theta^3 \\
&\quad - 6\gamma a \beta^3 k^3 \mu_0 \xi (a \mu_0 + k m_0) (N \langle k \rangle \gamma \omega q - N \langle k \rangle \gamma \omega - a \beta k \mu_0) \Theta^2 \\
&\quad - 6\gamma a \beta^2 k^2 \mu_0 \xi^2 (a \mu_0 + k m_0) (N \langle k \rangle \gamma \omega q - N \langle k \rangle \gamma \omega - a \beta k \mu_0) \Theta \\
&\quad + 2\gamma a k \mu_0 \xi^2 (a \mu_0 + k m_0) \beta [\gamma \xi \omega N \langle k \rangle + a \mu_0 \xi \beta - \gamma k m_0 q \beta],
\end{aligned}$$

$$\begin{aligned}
D &= N^3 \gamma^3 \omega^3 \Theta^3 (\beta k q \Theta - \beta k \Theta - \xi)^3 \langle k \rangle^3 \\
&\quad - 3(a \beta^2 k^2 \mu_0 \Theta^2 - \beta \gamma k^2 m_0 q \Theta + a \beta \gamma k \mu_0 \Theta + a \beta k \mu_0 \Theta \xi + \beta \gamma k^2 m_0 \Theta + a \gamma \mu_0 \xi + \gamma k m_0 \xi) \\
&\quad \cdot N^2 \gamma^2 \omega^2 \Theta^2 (\beta k q \Theta - \beta k \Theta - \xi)^2 \langle k \rangle^2 \\
&\quad + 3(a \beta^2 k^2 \mu_0 \Theta^2 - \beta \gamma k^2 m_0 q \Theta + a \beta \gamma k \mu_0 \Theta + a \beta k \mu_0 \Theta \xi + \beta \gamma k^2 m_0 \Theta + a \gamma \mu_0 \xi + \gamma k m_0 \xi)^2 \\
&\quad \cdot N \gamma \omega \Theta (\beta k q \Theta - \beta k \Theta - \xi) \langle k \rangle \\
&\quad - (a \beta^2 k^2 \mu_0 \Theta^2 - \beta \gamma k^2 m_0 q \Theta + a \beta \gamma k \mu_0 \Theta + a \beta k \mu_0 \Theta \xi + \beta \gamma k^2 m_0 \Theta + a \gamma \mu_0 \xi + \gamma k m_0 \xi)^3.
\end{aligned}$$

Obviously, $N \langle k \rangle \omega q \xi - \beta k^2 m_0 q + a \beta k \mu_0 + \beta k^2 m_0 = N \langle k \rangle \omega q \xi + a \beta k \mu_0 + \beta k^2 m_0 (1 - q) > 0$, and $N \langle k \rangle \gamma \omega q - N \langle k \rangle \gamma \omega - a \beta k \mu_0 = N \langle k \rangle \gamma \omega (q - 1) - a \beta k \mu_0 < 0$, we have $\gamma \xi \omega N \langle k \rangle + a \mu_0 \xi \beta \geq \gamma n m_0 q \beta$, so $\gamma \xi \omega N \langle k \rangle + a \mu_0 \xi \beta \geq \gamma k m_0 q \beta$, for $k = 1, 2, \dots, n$, so $C > 0$. And we know, $\beta k q \Theta - \beta k \Theta - \xi = \beta k \Theta (q - 1) - \xi < 0$,

$a\beta^2 k^2 \mu_0 \Theta^2 - \beta \gamma k^2 m_0 q \Theta + a\beta \gamma k \mu_0 \Theta + a\beta k \mu_0 \Theta \xi + \beta \gamma k^2 m_0 \Theta + a\gamma \mu_0 \xi + \gamma k m_0 \xi = a\beta^2 k^2 \mu_0 \Theta^2 + k\Theta(a\mu_0 \gamma + a\mu_0 \xi + \gamma k m_0(1 - q))\beta + \gamma \xi(a\mu_0 + k m_0) > 0$, so $D < 0$. Thus

$$\frac{d^2 G(\Theta)}{d\Theta^2} = \frac{1}{\langle k \rangle} \sum_{k=1}^n k p(k) \frac{C}{D} < 0.$$

That it is to say, if $R_0 > 1$, there exists a unique positive the endemic equilibrium $E^*(s_{11}^*, s_{21}^*, i_1^*, \dots, s_{1n}^*, s_{2n}^*, i_n^*, M^*)$ of system (2.2).

Theorem 4 Suppose $R_0 > 1$, then system (2.2) is permanent of infection, that is, there exists a $\epsilon > 0$, such that $\liminf_{t \rightarrow \infty} i_k(t) > \epsilon, k = 1, 2, \dots, n$, for any solution of (2.2) with $s_{1k}(0) > 0, s_{2k}(0) > 0, i_k(0) > 0$, and $M(0) > 0$.

Proof The argument is similar to that of Lemma 3.5 of De Leenheer and Smith[24], which is based on Theorem 4.6 of Thieme[25]. For simplicity, we denote

$$x = (s_{11}, s_{12}, i_1, \dots, s_{1k}, s_{2k}, i_k)$$

to be the state variable of (2.2) and $x(t)$ represents a solution of (2.2). Define

$$\mathcal{X} = \{x \in R_+^{3k} : s_{1k} + s_{2k} + i_k = 1, k = 1, 2, \dots, n\},$$

$$\mathcal{X}_0 = \{x \in \mathcal{X} : \sum_{k=1}^n k p(k) i_k > 0\},$$

and

$$\partial \mathcal{X}_0 = \mathcal{X} \setminus \mathcal{X}_0.$$

In the following, we show that system (2.2) is uniformly persistent with respect to $(\mathcal{X}_0, \partial \mathcal{X}_0)$.

Obviously, \mathcal{X} is positively invariant with respect to (2.2). We can also see that \mathcal{X}_0 is positively invariant with respect to (2.2) since

$$\frac{d}{dt} \left(\sum_{k=1}^n k p(k) i_k \right) \geq -\gamma \sum_{k=1}^n k p(k) i_k.$$

Furthermore, there exists a compact set \mathcal{B} in which all solutions of (2.2) initiating in \mathcal{X} will enter and remain there.

Denote

$$\mathcal{M}_0 = \{x_0 \in \partial \mathcal{X}_0 : x(t) \in \partial \mathcal{X}_0, t \geq 0\},$$

and

$$\Omega = \bigcup \{\omega(x(t)) : x(0) \in \partial \mathcal{M}_0\}.$$

Restricting (2.2) on \mathcal{M}_0 gives

$$\begin{cases} \frac{ds_{1k}(t)}{dt} = \gamma q i_k(t) - \xi s_{1k}(t), \\ \frac{ds_{2k}(t)}{dt} = \gamma(1 - q) i_k(t) + \xi s_{1k}(t), \\ \frac{di_k(t)}{dt} = -\gamma i_k(t). \end{cases} \quad (3.4)$$

Clearly, the system (3.4) has a unique equilibrium E_0 . It is easy to see from the first and third equations of (3.4) that all $s_{1k}(t)$ and $i_k(t)$ tend to zero as $t \rightarrow \infty$. Then, $s_{2k}(t) \rightarrow 1$ as $t \rightarrow \infty$ since $s_{1k}(t) + s_{2k}(t) + i_k(t) = 1$. So, $\Omega = E_0$.

Note that E_0 is a covering of Ω , which is isolated and is acyclic. To finish the proof, it suffices to show that E_0 is a weak repeller for X_0 , that is

$$\limsup_{t \rightarrow \infty} \text{dist}(x(t), E_0) > 0,$$

where $x(t)$ is any arbitrary solution of (2.2) with $x(0) \in X_0$. We only need to prove $W^s(E_0) \cap X_0 = \emptyset$, where $W^s(E_0)$ is the stable manifold of E_0 . We take the contradiction and assume that it's not true. Then there exists a solution $x(t) \in X_0$ such that $s_{1k}(t) \rightarrow 0$, $s_{2k}(t) \rightarrow 1$ and $i_k(t) \rightarrow 0$ as $t \rightarrow \infty$. Since $R_0 = \frac{\beta a \mu_0}{\gamma \langle k \rangle} \sum_{k=1}^n \frac{k^2 p(k)}{a \mu_0 + k m_0} > 1$, we can choose $\nu > 0$ such that $(1 - \nu)R_0 > 1$. For such $\nu > 0$, there exists a $T > 0$ such that, for $t \geq T$, $0 \leq s_{1k}(t) < \nu$, $1 - \nu < s_{2k}(t) \leq 1$, and $0 \leq i_k(t) < \nu$. Let

$$V(t) = \sum_{k=1}^n k p(k) i_k(t).$$

In fact $V(t) = \langle k \rangle \Theta(t)$. Then, for $t \geq T$, we have

$$\begin{aligned} \frac{dV(t)}{dt} &= \sum_{k=1}^n k p(k) \frac{di_k(t)}{dt} \\ &= \sum_{k=1}^n k p(k) [\beta k s_{1k}(t) \Theta(t) + \beta(1 - B(M, k)) k s_{2k}(t) \Theta(t) - \gamma i_k(t)] \\ &= \sum_{k=1}^n k^2 p(k) \beta \frac{V(t)}{\langle k \rangle} (s_{1k}(t) + \frac{a \mu_0}{a \mu_0 + k m_0} s_{2k}(t)) - \gamma V(t) \\ &\geq \left[\frac{a \mu_0 \beta}{\langle k \rangle} \sum_{k=1}^n \frac{k^2 p(k)}{a \mu_0 + k m_0} (1 - \nu) - \gamma \right] V(t) \\ &= \gamma [R_0(1 - \nu) - 1] V(t). \end{aligned}$$

Since $(1 - \nu)R_0 > 1$, it follows immediately that $V(t) \rightarrow \infty$ as $t \rightarrow \infty$, which contradicts with the fact that $V(t)$ is bounded. This completes the proof.

3.3. Immunization strategies

Vaccination is very helpful in controlling vaccine preventable diseases [8, 12]. In this section we discuss system (2.2) with two immunization schemes: the proportional immunization and the targeted immunization.

3.3.1. Proportional immunization

Let f be the immunization rate, $0 < f < 1$. Then system (2.2) becomes

$$\begin{cases} \frac{ds_{1k}(t)}{dt} = -\beta k(1 - f) s_{1k}(t) \Theta(t) + \gamma q i_k(t) - \xi s_{1k}(t), \\ \frac{ds_{2k}(t)}{dt} = -\beta(1 - B(M, k)) k(1 - f) s_{2k}(t) \Theta(t) + \gamma(1 - q) i_k(t) + \xi s_{1k}(t), \\ \frac{di_k(t)}{dt} = \beta k(1 - f) s_{1k}(t) \Theta(t) + \beta(1 - B(M, k)) k(1 - f) s_{2k}(t) \Theta(t) - \gamma i_k(t), \\ \frac{dM(t)}{dt} = m_0 + \omega N \sum_{k=1}^n p(k) i_k(t) - \mu_0 M(t). \end{cases} \quad (3.5)$$

Similar arguments as those in Section 3 give us the basic reproduction number

$$\hat{R}_0 = \frac{\beta a \mu_0 (1-f)}{\gamma \langle k \rangle} \sum_{k=1}^n \frac{k^2 p(k)}{a \mu_0 + k m_0} = (1-f) R_0.$$

Compare the expressions of \hat{R}_0 and R_0 , when $f = 0$, there is no immunity, $\hat{R}_0 = R_0$, otherwise $\hat{R}_0 < R_0$, indicating that the immune strategy is effective. When $f \rightarrow 0$, i.e. $\hat{R}_0 \rightarrow 0$, it means that under the strategy of total immunity, the disease cannot be transmitted on complex networks.

3.3.2. Targeted immunization

In addition to proportional immunization, target immunization is also an effective immunization strategy. Define the threshold of two network nodes are k_1 and k_2 respectively, $k_1 < k_2$ and the immunization rate f_k depends on the degree k . Then f_k is as follows:

$$f_k = \begin{cases} 1, & k > k_2; \\ p_k, & k_1 < k < k_2; \\ 0, & k < k_1. \end{cases} \quad (3.6)$$

Here $\bar{f}_k = \sum_k f_k p(k)$ is the average immunological rate, then we can re-write system (2.2) as

$$\begin{cases} \frac{ds_{1k}(t)}{dt} = -\beta k(1-f_k)s_{1k}(t)\Theta(t) + \gamma q i_k(t) - \xi s_{1k}(t), \\ \frac{ds_{2k}(t)}{dt} = -\beta(1-B(M,k))k(1-f_k)s_{2k}(t)\Theta(t) + \gamma(1-q)i_k(t) + \xi s_{1k}(t), \\ \frac{di_k(t)}{dt} = \beta k(1-f_k)s_{1k}(t)\Theta(t) + \beta(1-B(M,k))k(1-f_k)s_{2k}(t)\Theta(t) - \gamma i_k(t), \\ \frac{dM(t)}{dt} = m_0 + \omega N \sum_{k=1}^n p(k)i_k(t) - \mu_0 M(t). \end{cases} \quad (3.7)$$

By recalculating the basic number of regeneration, we get

$$\tilde{R}_0 = \frac{\beta a \mu_0}{\gamma \langle k \rangle} \sum_{k=1}^n \frac{\langle k^2 \rangle - \langle k^2 f_k \rangle}{a \mu_0 + k m_0}.$$

Since $\langle k^2 f_k \rangle = \bar{f} \langle k^2 \rangle + \sigma$, where σ is the covariance of f_k and k^2 , and

$$\sigma = Cov(f_k, k^2) = \langle (f_k - \bar{f})(k^2 - \langle k^2 \rangle) \rangle.$$

When appropriately small k is taken, σ can be guaranteed to be greater than 0, therefore:

$$\langle k^2 \rangle - \langle k^2 f_k \rangle = \langle k^2 \rangle (1 - \bar{f}) - \sigma < \langle k^2 \rangle (1 - \bar{f}).$$

Obviously, we can get $\tilde{R}_0 \leq R_0$, which implies that targeted immunization can be effective in controlling the spread of disease. If note $f = \bar{f}_k$, we can get $\tilde{R}_0 \leq \hat{R}_0$, it means that for the same average immune rate, target immunity is more effective than proportional immunity.

4. Numerical simulations

In this section, the stability of the disease-free equilibrium and endemic equilibrium are simulated respectively. We consider the dynamical process on BA random scale-free networks with $p(k) = 2m^2k^{-3}$ and $m = 3$.

Firstly, numerical simulations are presented to illustrate the results of system (2.2). In the simulations the parameters are given by the reference [6, 21], which are as follows: $N = 1000$, $\beta = 0.02$, $\gamma = 0.11$, $\xi = 0.05$, $q = 0.2$, $\omega = 0.0005$, $m_0 = 0.12$, $\mu_0 = 0.11$, $a = 10$, then $M = m_0/\mu_0 = 1.09$ and $R_0 < 1$, thus the disease-free equilibrium is globally asymptotically stable. As shown in Fig.1, when $R_0 < 1$, even for a large fraction of the infected at the initial time, the disease will disappear and all the individuals will become the aware susceptible. Moreover, the larger the cumulative density of media coverage is, the faster the disease dies out.

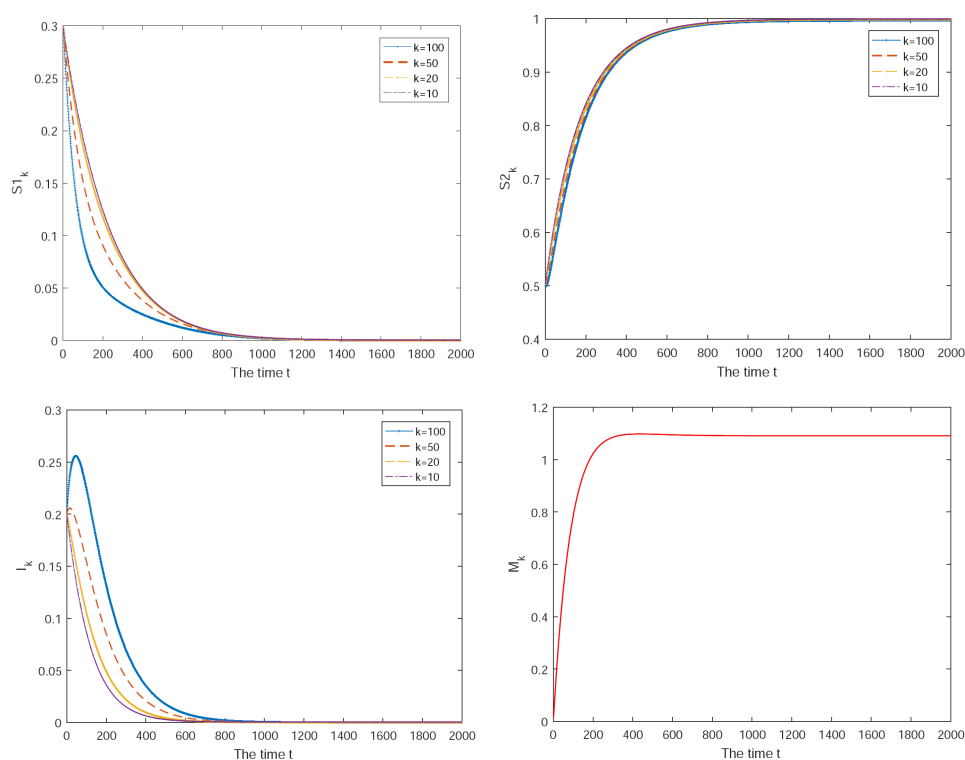


Figure 1. The time series of individuals for different degree and media coverage with $R_0 < 1$.

Secondly, the parameters are given by $N = 1000$, $\beta = 0.02$, $\gamma = 0.021$, $\xi = 0.05$, $q = 0.2$, $\omega = 0.0005$, $m_0 = 0.003$, $\mu_0 = 0.05$, $a = 10$, $M = m_0/\mu_0 = 0.06$ [6, 21], then $R_0 > 1$. One can see that, the disease will converge to a positive stationary level, even for a small fraction of the infected at the initial time, which means that the positive equilibrium is stable.

Finally, we consider the effects of immunization with the same values of parameters in the first case. In Fig. 3, we take $f = 0.2$ in the proportional immunization, and $f_k = 0.5$ for $5 < k < 10$ in the targeted

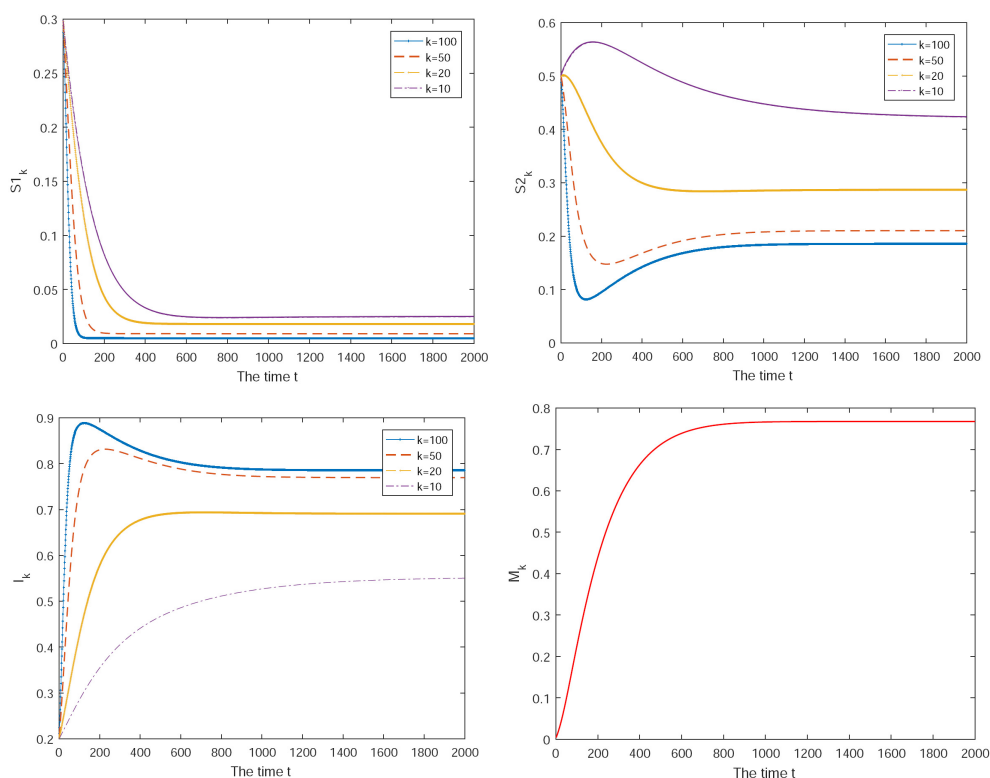


Figure 2. The time series of individuals for different degree and media coverage with $R_0 > 1$.

immunization

$$f_k = \begin{cases} 1, & k > k_2; \\ p_k, & k_1 < k < k_2; \\ 0, & k < k_1. \end{cases} \quad (4.1)$$

Numerical simulation verified the two immune strategy for disease control are effective, and the target immune strategy is more effective than proportion immunization strategy.

5. Conclusion

In this paper, we divided the susceptible into two parts (susceptible without aware, the aware susceptible) and added media information reports to study the impact of media reports on disease transmission. We obtained that when $R_0 < 1$, the disease-free equilibrium is globally asymptotically stable, while when $R_0 > 1$, the disease-free equilibrium instability, and the disease is permanent. In order to further control the spread of the disease, we studied two immunization strategies: proportional immunization and target immunization, both of which can control the spread of the disease well, but the target immunization is more effective. There will be more better strategies in the control the spread of the disease, and in the future we will continue to study them and get more better results.

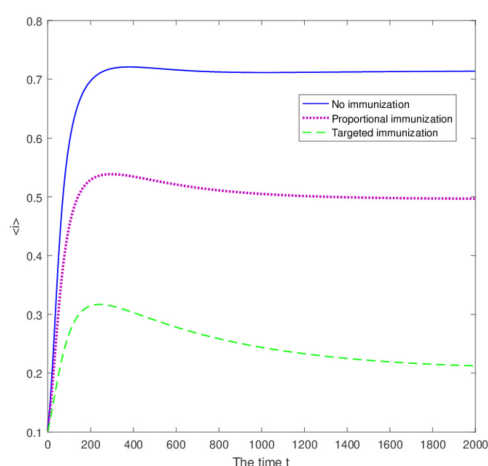


Figure 3. The time series of the total density of infected individuals with different immunization strategies.

Acknowledgments

The authors would like to thank the reviewers for their helpful comments and valuable suggestions, and the support of the National Sciences Foundation of China(11571324, 61403393), the Fund for Shanxi 1331KIRT, Shanxi Scholarship Council of China and the Scientific Activities of Selected Returned Overseas Professionals in Shanxi Province.

Conflict of interest

The authors declare there is no conflict of interest.

References

1. M. E. J. Newman, Spread of epidemic disease on networks, *Phys. Rev. E*, **66** (2002), 016128.
2. M. E. J. Newman, The structure and function of complex networks, *SIAM Rev.*, **45** (2003), 167–256.
3. M. J. Keeling, The implications of network structure for epidemic dynamics, *Theor. Popul. Biol.*, **67** (2005), 1–8.
4. R. Pastor-Satorras and A. Vespignani, Epidemic spreading in scale-free networks, *Phys. Rev. Lett.*, **86** (2001), 3200–3203.
5. T. Zhou, J. G. Liu, W. J. Bai, et al., Behaviors of susceptible-infected epidemics on scale-free networks with identical infectivity, *Phys. Rev. E*, **74** (2006), 056109.
6. M. Liu and Y. Chen, An SIRS model with differential susceptibility and infectivity on uncorrelated networks, *Math. Biosci. Eng.*, **12** (2015), 415–429.
7. J. Liu and T. Zhang, Epidemic spreading of an SEIRS model in scale-free networks, *Commun. Nonlinear Sci. Numer. Simul.*, **16** (2011), 3375–3384.

8. R. Pastor-Satorras and A. Vespignani, Immunization of complex networks, *Phys. Rev. E*, **65** (2002), 036104.
9. A. d’Onofrio, A note on the global behaviour of the network-based SIS epidemic model, *Nonlinear Anal.*, **9** (2008), 1567–1572.
10. J. Lou and T. Ruggeri, The dynamics of spreading and immune strategies of sexually transmitted diseases on scale-free network, *J. Math. Anal. Appl.*, **365** (2010), 210–219.
11. W. J. Reed, A stochastic model for the spread of a sexually transmitted disease which results in a scale-free network, *Math. Biosci.*, **201** (2006), 3–14.
12. M. Liu and J. Ruan, Modelling the spread of sexually transmitted diseases on scale-free networks, *Chinese Physics B*, **18** (2009), 2115–2120.
13. S. Funk, M. Salathé and V. A. A. Jansen, Modelling the influence of human behaviour on the spread of infectious diseases: a review, *J. R. Soc. Interf.*, **7** (2010), 1247–1256.
14. R. Liu, J. Wu and H. Zhu, Media/psychological impact on multiple outbreaks of emerging infectious diseases, *Comput. Math. Methods Med.*, **8** (2007), 153–164.
15. J. Tchuenche, N. Dube, C. Bhunu, et al., The impact of media coverage on the transmission dynamics of human influenza, *BMC Public Health*, **11** (2011), S5.
16. M. Liu, E. Liz and G. Röst, Endemic bubbles generated by delayed behavioral response—global stability and bifurcation switches in an SIS model, *SIAM J. Appl. Math.*, **75** (2015), 75–91.
17. L. Zuo, M. Liu and J. Wang, The impact of awareness programs with recruitment and delay on the spread of an epidemic, *Math. Prob. Eng.*, **2014** (2014), 235935.
18. S. Collinson and J. M. Heffernan, Modelling the effects of media during an influenza epidemic, *BMC Public Health*, **14** (2014), 1–10.
19. K. A. Pawelek, A. Oeldorf-Hirsch and L. Rong, Modeling the impact of twitter on influenza epidemics, *Math. Biosci. Eng.*, **11** (2014), 1337–1356.
20. D. Greenhalgh, S. Rana, S. Samanta, et al., Awareness programs control infectious disease—Multiple delay induced mathematical model, *Appl. Math. Comput.*, **251** (2015), 539–563.
21. Y. Wang, J. Cao, Z. Jin, et al., Impact of media coverage on epidemic spreading in complex networks, *Phys. A*, **392** (2013), 5824–5835.
22. X. Yuan, Y. Xue and M. Liu, Analysis of an epidemic model with awareness programs by media on complex networks, *Chaos Soli. Frac.*, **48** (2013), 1–11.
23. P. van den Driessche and J. Watmough, Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission, *Math. Biosci.*, **180** (2002), 29–48.
24. P. De Leenheer and H. L. Smith, Virus dynamics: A global analysis, *SIAM J. Appl. Math.*, **63** (2003), 1313–1327.
25. H. R. Thieme, Persistence under relaxed point-dissipativity (with application to an endemic model), *SIAM J. Math. Anal.*, **24** (1993), 407–435.



AIMS Press

©2019 the Author(s), licensee AIMS Press. This is an open access article distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>)