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Impact of media coverage on epidemic spreading in complex networks

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HIGHLIGHTS

- A new SIS network model obtained by introducing an information variable is proposed.
- The diseases can be controlled through high efficiency of implementation.
- The introduced parameters have significant impact on the final prevalence density.
- The results may suggest effective control strategies incorporating media coverage.

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ABSTRACT

An SIS network model incorporating the influence of media coverage on transmission rate is formulated and analyzed. We calculate the basic reproduction number $R_0$ by utilizing the local stability of the disease-free equilibrium. Our results show that the disease-free equilibrium is globally asymptotically stable and that the disease dies out if $R_0$ is below 1; otherwise, the disease will persist and converge to a unique positive stationary state. This result may suggest effective control strategies to prevent disease through media coverage and education activities in finite-size scale-free networks. Numerical simulations are also performed to illustrate our results and to give more insights into the dynamical process.

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1. Introduction

Disease-induced death has accounted for a quarter of all deaths in the world annually, most of which happens in low- and middle-income countries [1]. Diseases such as severe acute respiratory syndrome (SARS) and flu present some distinct characteristics, including rapid spatial spread and visible symptoms. Once an infectious disease appears and spreads in a region, the disease control and prevention center will do its best to stop the propagation of the disease. One of the measures is to tell people the appropriate preventive knowledge of the disease as soon as possible through media (broadcasts, television, news, and so on) and education [2]. In practice, if residents have more preventive knowledge, the better they are able to protect themselves by adopting necessary measures. For example, during outbreaks of serious infectious diseases such as the SARS outbreak in 2003 and the H1N1 influenza pandemic in 2009, public media had massive reports on the number of infections and deaths per day, the locations where they happened, the symptoms of the disease, proper protection to reduce...
the possibility of being infected, etc. [3]. Therefore, it is important to refine classical mathematical models to cover these features by adding the dimensions of massive news coverage that may have an impact not only on individual behavior but also on the formation and implementation of public intervention and control policies [4,5].

There is much evidence that media coverage can play an appreciable role in the spread and control of infectious diseases [4,6–9,5]. Liu et al. [4] illustrated a possible mechanism for multiple outbreaks or even sustained periodic oscillations of emerging infectious diseases due to the psychological impact of the reported numbers of infectious and hospitalized individuals. Cui et al. [6] found that the model might exhibit multiple positive equilibria due to stronger media impact, which posed challenges to the prediction and control of the outbreaks of infectious diseases. Misra et al. [9] proposed and analyzed a nonlinear mathematical model, in which susceptible individuals formed another separate class and avoided contacts with the infective class.

Indeed, media coverage is a key factor in the transmission process of infectious disease. For example, a survey found that three tabloids and two broadsheets ran a total of 1153 news stories involving severe acute respiratory syndrome (SARS) in Britain from March to July 2003 [10], while the New Zealand Herald ran 261 articles from 13 March 2003 to 11 June 2003 [11] during the spread of SARS. On the one hand, as the number of infected cases increases, the media coverage may give more reports (maybe the number of infections and deaths per day, the locations where these happen, the symptoms of the disease) about them, while on the other hand, it can certainly cut down the opportunity and probability of contact transmission among the alerted susceptible populations, which is beneficial to the control and prevention of disease for further spreading. Many compartmental models [2,6–8,5,12] have assumed that media coverage could reduce the contact rate between susceptibles and infectives.

However, most of the previous models involving media coverage have assumed homogeneous mixing populations, i.e., the contact transmission between susceptible and infective individuals occurs randomly, without any geographical or social contact structure [2,6,7], although heterogeneity in the network structure significantly influences the transmission dynamics [13–15]. A few models have used network-based or individual-based approaches by adding the dimensions of massive news coverage. In fact, the spread of an infectious diseases in a population depends not only on the pathology of the disease itself, but also on the structure and mix patterns of the population. There are extensive investigations on overcoming this shortcoming, and one important effort along this direction is the use of complex network models, since the spread mechanism of diseases on networks has been widely studied by many researchers [16–24]. Researchers have given different interpretations for the mechanism of the propagation of diseases on complex networks [16,19–22].

One of the basic topological properties of the networks is the degree distribution $p(k)$, which is defined as the fraction of vertices in the network that have degree $k$. Statistically speaking, $p(k)$ is the probability that a vertex chosen randomly has degree $k$ [25]. Various studies [26,25,27] have revealed that the degree distribution $p(k)$ of most real-world networks is highly skewed, and that it usually follows a power law $p(k) \sim k^{-\gamma}$ [28]. Pastor-Satorras et al. [29] found that there was a nonzero epidemic threshold $\lambda_0$ in a homogeneous network as the classical prediction [30], and a surprising absence of any epidemic threshold or $\lambda_0 = 0$ in the heterogeneous counterpart if $2 < \gamma \leq 3$ and if the network size approaches infinity. This surprising result gave a good explanation to the behavior of a long-standing computer virus in computer networks [16]. For finite size scale-free networks, Pastor-Satorras et al. [19] introduced a maximum degree $k_c$ that came to a nonzero threshold, but when $k_c$ increased or the network size approached infinity, $\lambda_0$ would still approach zero. However, Zhou et al. [22] considered the fact that in reality the hub nodes might be only able to contact a limited population at one period of time despite their wide acquaintance, and proposed an epidemic model with identical infectivity for each infected individual which came to a constant threshold $\lambda_0$ independent of the degree distributions and the network size.

During the disease transmission process, the spread of an infectious disease depends mainly on the interactions between susceptible and infected individuals. However, there are many other factors, such as media coverage, vaccinations, and migration of population, which also influence the spread of an infectious disease [31–33]. In particular, media coverage has a great impact on individual behavior towards the disease, and further affects the government health care interventions to control the spread of such disease. It is media coverage that may provide people with more information about the disease to take precautions such as distancing, wearing protective masks, vaccinations, to reduce the chances of being infected.

The rest of this paper is organized as follows. In Section 2, we introduce the main idea and formulate an SIS network model incorporating media coverage. We derive the basic reproduction number $R_0$ in Section 3 and prove the local and global stability of the disease-free equilibrium. In addition, the existence and uniqueness of the endemic equilibrium is given. Extensive numerical simulations are performed to give more insights to the system in Section 4. Finally, Section 5 concludes the paper.

2. An SIS network model with media coverage

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 $N_k$ ($k = 1, 2, \ldots, n$) 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 $N (N = N_1 + N_2 + \cdots + N_n)$, then the degree distribution is $p(k) = N_k/N$ according to the previous section. The value $\langle k \rangle = \sum_k kp(k)$ is the average number of contacts each node (individual) can make. In epidemiology, there are two important and fundamental epidemic models: SIS and SIR [30]. For the SIS model, each individual can be in two states: $S$, susceptible to the disease, and $I$, infectious individuals who can transmit the disease to the susceptible one. A susceptible
individual may get infected at rate $\beta$ if contacted by one infected individual; at the same time, the infected individuals may recover and return to being susceptible individuals at rate $\mu$. For the SIR model, the individuals can exist in another state: R, recovered/removed; they cannot be infected again.

Denote by $S_k(t)$ and $I_k(t)$ the number of susceptible and infected individuals within the group $k$ at time $t$, respectively; then $N_k(t) = S_k(t) + I_k(t)$. 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 $t$. d’Onofrio et al. [34] considered two distinct possibilities: (a) $M$ only summarizes information about the current state of the disease, i.e. $M$ only depends on current values of state variables, and (b) $M$ also summarizes information about past values of state variables. In the present study, we adopt a linear form of (a), i.e. $M$ is a linear function of the current prevalence of the disease (see Ref. [34] and the references cited therein). This preliminary work will focus on a static network, i.e. ignoring the population demographics (recruitment or death), and it considers the situation where the media coverage only reduces the transmission rate between susceptible individuals and infected individuals.

Based on the above assumptions, one has the following system of $2n + 1$ ordinary differential equations (ODEs):

$$
\begin{align*}
\frac{dS_k(t)}{dt} &= -\beta(1 - P(M,k))kS_k(t)\Theta(t) + \mu I_k(t), \\
\frac{dI_k(t)}{dt} &= \beta(1 - P(M,k))kS_k(t)\Theta(t) - \mu I_k(t), \quad k = 1, 2, \ldots, n, \\
\frac{dM(t)}{dt} &= m_0 + \omega \sum_{k=1}^{n} I_k(t) - \lambda M(t).
\end{align*}
$$

Here, we consider the media coverage function $P(M,k) = \frac{\frac{\omega}{\lambda} M}{1 + \frac{\omega}{\lambda} M}$ as being similar to the Michaelis–Menten function $p_1(M) = \frac{CM}{1 + DM}$ (see [34,35] and the references cited therein). $P(M,k)$ 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 $\beta P(M,k)$ 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. For uncorrelated networks [16], $\Theta(t) = \frac{\sum_{k=1}^{n} I_k(t)}{\sum_{k=1}^{n} I_k(t) + \sum_{k=1}^{n} k N_k}$ represents the expectation that any given edge points to an infected individual. The parameter $\omega$ is a proportional constant with which media coverage is being implemented based on the current state of infectious cases, and $\lambda$ represents the depletion rate of media coverage due to ineffective implementation. In addition, we use another parameter $m_0$ to reflect the density level of media coverage on the disease even though there is no infectious case in the region under consideration. This can be explained as follows: due to the increasing trend of globalization and the development of information technology, information about infectious cases found in one region may spread rapidly to another region without this disease through media coverage, for example, in the case of the media coverage of the outbreak of SARS in 2003.

The parameters are also summarized in Table 1; all the parameters in the model are assumed to be positive except that $m_0$ is nonnegative.

Table 1: Parameters of the model.

| Parameter | Description |
|-----------|-------------|
| $\beta$ | Transmission rate per contact between one susceptible and one infectious individual |
| $a$ | Half-saturation constant, reflecting the impact of media coverage on the transmission |
| $\mu$ | Recovery rate of infected individuals |
| $m_0$ | Density level of media coverage on the disease from another region with the disease |
| $\omega$ | Rate of media coverage being implemented |
| $\lambda$ | Depletion rate of media coverage due to ineffective implementation |

Note that $\frac{dS_k(t)}{dt} + \frac{dI_k(t)}{dt} = 0$, which means that $S_k(t) + I_k(t) = N_k$ is constant. Denote the relative densities of susceptible and infected nodes of degree $k$ at time $t$ by $s_k(t) = S_k(t)/N_k$ and $i_k(t) = I_k(t)/N_k$, respectively. Then, system (1) can be rewritten as

$$
\begin{align*}
\frac{ds_k(t)}{dt} &= \beta(1 - P(M,k))k(1 - i_k(t))\Theta(t) - \mu i_k(t), \quad k = 1, 2, \ldots, n, \\
\frac{di_k(t)}{dt} &= \beta(1 - P(M,k))kS_k(t)\Theta(t) - \mu i_k(t), \\
\frac{dM(t)}{dt} &= m_0 + \omega N \sum_{k=1}^{n} p(k)i_k(t) - \lambda M(t),
\end{align*}
$$

with the normalization condition $s_k + i_k = 1$, and also $\Theta(t) = \frac{1}{\lambda N} \sum_k k p(k)i_k$. It is not difficult to verify that $\Gamma = \prod_{k=1}^{n}[0, 1] \times [m_0/\lambda, (m_0+\omega N)/\lambda]$ 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 basic reproduction number and global stability of the disease-free equilibrium

We can easily see that system (2) has a unique disease-free equilibrium $E_0(0, \ldots, 0, m_0/\lambda)$. Following the recipe of van den Driessche and Watmough [36], 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. In the disease-free state $E_0$, the production of new infections $F$ and the rate of transfer of individuals $\gamma$ are given respectively by

$$ F = D^T E_0 = \begin{pmatrix} \frac{1}{\alpha + nM_0} & \frac{1}{\alpha + nM_0} & \cdots & \frac{1}{\alpha + nM_0} \\ \frac{1}{\alpha + 2M_0} & \frac{1}{\alpha + 2M_0} & \cdots & \frac{1}{\alpha + 2M_0} \\ \vdots & \vdots & \ddots & \vdots \\ \frac{n}{\alpha + nM_0} & \frac{n}{\alpha + nM_0} & \cdots & \frac{n}{\alpha + nM_0} \end{pmatrix}, $$

$$ \gamma = \begin{pmatrix} \mu_1 \\ \mu_2 \\ \vdots \\ \mu_n \end{pmatrix}. $$

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

$$ F = D^T E_0 = \begin{pmatrix} \frac{1}{\alpha + nM_0} & \frac{1}{\alpha + nM_0} & \cdots & \frac{1}{\alpha + nM_0} \\ \frac{1}{\alpha + 2M_0} & \frac{1}{\alpha + 2M_0} & \cdots & \frac{1}{\alpha + 2M_0} \\ \vdots & \vdots & \ddots & \vdots \\ \frac{n}{\alpha + nM_0} & \frac{n}{\alpha + nM_0} & \cdots & \frac{n}{\alpha + nM_0} \end{pmatrix}, $$

$$ \gamma = \begin{pmatrix} 1 \\ 1 \\ \vdots \\ 1 \end{pmatrix}. $$

where $M_0 = m_0/\lambda$.

Thus, the reproduction number is given by $R_0 = \rho(FV^{-1})$, the spectral radius of the matrix $FV^{-1}$. We obtain the basic reproduction number as follows:

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

**Remark 1.** When $m_0 \to 0^+$, we obtain $R_0 = \frac{\beta k^2 \rho(k^2)}{\mu(k^2)}$, which is consistent with the classical result (set $\mu = 1$; then $\beta_c = \frac{(k^2)}{\langle k^2 \rangle}$) obtained by Pastor et al. [16]. That is to say, if $m_0 = 0$, the existence of media coverage will not change the epidemic threshold, but it may affect the prevalence of the disease.

In summary, we have the following result.

**Theorem 1.** If $R_0 < 1$, the disease-free equilibrium $E_0$ of system (2) is locally asymptotically stable, whereas if $R_0 > 1$ the disease-free equilibrium $E_0$ is unstable.

Furthermore, we can obtain the global stability of $E_0$.

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

**Proof.** From system (2), we have

$$ \begin{cases} \frac{di_k(t)}{dt} \leq \beta k(1-i_k(t))\Theta(t) - \mu i_k(t), \\ \frac{dM(t)}{dt} = m_0 + \omega N \sum_{k=1}^{n} p(k)i_k(t) - \lambda M(t). \end{cases} \tag{3} $$
Let us consider the following auxiliary system:

\[
\begin{aligned}
\frac{di_k(t)}{dt} &= \beta k(1 - i_k(t))\Theta(t) - \mu i_k(t), \\
\frac{dM(t)}{dt} &= m_0 + \omega N \sum_{k=1}^{n} p(k)i_k(t) - \lambda M(t).
\end{aligned}
\]  

(4)

The basic reproduction number \(R_0\) for system (4) is

\[ R_0 = \frac{\beta \langle k^2 \rangle}{\mu \langle k \rangle}. \]

From \(R_0 < 1\), since \(R_0\) is a decreasing function of \(m_0\), we obtain

\[ R_0 = \lim_{m_0 \to 0^+} R_0 \leq 1. \]

Since the first equation of system (4) is independent of the second, according to the results obtained by d’Onofrio et al. [37], one obtains that the disease-free equilibrium is globally asymptotically stable if \(R_0 \leq 1\), i.e., \(\lim_{t \to +\infty} i_k(t) = 0\), \(k = 1, 2, \ldots, n\). The limit system of the information variable \(M(t)\) satisfies \(dM(t)/dt = m_0 - \lambda M(t)\), and it comes to \(\lim_{t \to +\infty} M(t) = M_0\). By comparison arguments, we know that the disease-free equilibrium \(E_0\) is globally attractive in \(\Gamma\) for system (2). This, combined with Theorem 1, implies that \(E_0\) is globally asymptotically stable, and hence the proof is complete. \(\square\)

**Remark 2.** From Theorem 2, the global asymptotic stability of the disease-free equilibrium \(E_0\) excludes any possibility of the phenomenon of bifurcation. In order to eradicate the disease, what we need to do is to reduce the basic reproduction number \(R_0\) below 1. To clarify this, the sensitivity analysis of \(R_0\) in terms of the model parameters will be performed in Section 4.

### 3.2. The uniqueness of the endemic equilibrium

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

\[
\begin{aligned}
\beta(1 - P(M, k))k(1 - i_k^*)\Theta - \mu i_k^* &= 0, \\
m_0 + \omega Ni^* - \lambda M^* &= 0,
\end{aligned}
\]

(5)

where \(i^* = \sum_{k=1}^{n} p(k)i_k^*\). On the one hand, the first equation of (5) yields

\[ a\beta k(1 - i_k^*)\Theta - \mu i_k^*(a + km^*) = 0, \quad k = 1, \ldots, n. \]

(6)

The second equation of (5) yields

\[ M^* = \frac{m_0 + \omega Ni^*}{\lambda}. \]

(7)

Multiplying Eq. (6) by \(p(k)\) and summing over \(k\) and then substituting (7) into it, we obtain the result

\[ i^* = \frac{(a\beta\lambda - a\beta\lambda\Theta - \mu m_0)\langle k \rangle \Theta}{a\mu\lambda + \mu\omega N\langle k \rangle \Theta}. \]

(8)

which implies that \(\Theta < 1 - \frac{\mu m_0}{a\beta\lambda}\) (the inequality \(1 - \frac{\mu m_0}{a\beta\lambda} > 0\) holds naturally when \(R_0 > 1\)).

On the other hand, from Eq. (5) one obtains

\[ i_k^* = \frac{a\beta k\theta}{a\beta k\Theta + \mu(a\lambda + k m_0) + \mu k\omega N}. \]

(9)

Substituting (8) and (9) into the expression of \(\Theta = \frac{1}{\langle k \rangle} \sum_k kp(k)i_k^*\), then we obtain a self-consistency equation as follows:

\[
\Theta = \frac{1}{\langle k \rangle} \sum_k kp(k) \left( \frac{a^2\beta\mu\lambda^2 k\Theta + a\beta\lambda k\mu\omega N(k)\Theta^2}{(a\lambda + k m_0)a\mu^2\lambda + a^2\beta\mu\lambda^2 k\Theta + (\mu + \beta k) a\mu\lambda\omega N(k) \Theta} \right) \triangleq G(\Theta).
\]

(10)

Obviously, Eq. (10) has a trivial solution, \(\Theta = 0\). By straightforward computation, we have

\[
\frac{dG(\Theta)}{d\Theta} = \frac{1}{\langle k \rangle} \sum_k kp(k) \frac{A}{B^2},
\]

(11)

where \(A = a^2\beta\mu\lambda^3 k(a\lambda + k m_0) + 2a^2\beta\mu^2\lambda^2 k\mu\omega N(k)(a\lambda + k m_0)\Theta + a^2\beta^2\mu^2\lambda^3 k^2\omega N(k)\Theta^2 + a^2\beta^2\mu^2\lambda^2 k\omega N(k)^2(\mu + \beta k)\Theta^2 > 0\), \(B = (a\lambda + k m_0)a\mu^2\lambda + a^2\beta\mu\lambda^2 k\Theta + (\mu + \beta k) a\mu\lambda\omega N(k) \Theta\). Hence, it follows that \(dG(\Theta)/d\Theta > 0\). Through a
similar derivation, we obtain that
\[ \frac{d^2G(\Theta)}{d\Theta^2} = \frac{1}{\langle k \rangle} \sum_k kp(k) \frac{B^2 \frac{dA}{d\Theta}}{B^4} - 2AB \frac{dB}{d\Theta}, \]
where
\[ B^2 \frac{dA}{d\Theta} - 2AB \frac{dB}{d\Theta} = -2a^2 \beta^2 \mu^6 \lambda^7 k^2 \omega N(k) - 2a^2 \beta^2 \mu^6 \lambda^7 k^2 - 4a^2 \beta^2 \mu^6 \lambda^7 k^3 m_0 - 4a^2 \beta^2 \mu^6 \lambda^7 k^3 m_0 \omega N(k) \\
- 2a^2 \beta \mu^8 \lambda^3 k^3 \omega N(k) - 2a^2 \beta \mu^8 \lambda^3 k^3 \omega (k^2) \omega N(k) \\
- 2a^2 \beta \mu^6 \lambda^5 k^4 m_0^2 - 2a^2 \beta \mu^6 \lambda^5 k^4 m_0^2 \omega N(k) - 2a^2 \beta \mu^5 \lambda^7 k^4 m_0 \omega N(k) \\
- 4a^2 \beta \mu^6 \lambda^5 k^4 m_0 \omega N(k) - 2a^2 \beta \mu^5 \lambda^7 k^4 m_0 \omega^2 N(k) (k^2) \omega N(k) + 2a^2 \beta \mu^6 \lambda^5 k^4 \omega N(k) \\
- 2a^2 \beta \mu^6 \lambda^5 k^4 \omega N(k) - 2a^2 \beta \mu^5 \lambda^7 k^4 \omega^2 N(k) (k^2) \omega N(k) + 4a^2 \beta \mu^6 \lambda^5 \omega^2 N(k) m_0^2 \\
+ 2a^2 \beta \lambda^2 k^2 \mu^7 \omega^2 N^2 (k^2) m_0 \Theta + 2a^2 \beta \lambda^2 k^2 \mu^7 \omega^2 N^2 (k^2) m_0 \Theta + 2a^2 \beta \lambda^2 k^2 \mu^7 \omega^2 N^2 (k^2) m_0 \Theta \\
+ 2a^2 \beta \lambda^2 k^2 \mu^7 \omega^2 N^2 (k^2) m_0 \Theta + 2a^2 \beta \lambda^2 k^2 \mu^7 \omega^2 N^2 (k^2) m_0 \Theta \\
= 2a^2 \beta \lambda^2 k^2 \mu^7 \omega^2 N^2 (k) (m_0 - \alpha \beta \lambda) + 4a^2 \beta \lambda^2 \mu^6 k^3 m_0 \omega N(k) (m_0 - \alpha \beta \lambda) \\
+ 2a^2 \beta \lambda^2 \mu^6 k^3 m_0 \omega N(k) \Theta (m_0 - \alpha \beta \lambda) + 2a^2 \beta \lambda^2 \mu^6 k^3 m_0 \omega N(k) \Theta (m_0 - \alpha \beta \lambda) \\
+ 2a^2 \beta \lambda^2 \mu^6 k^3 m_0 \omega N(k) \Theta (m_0 - \alpha \beta \lambda) + 2a^2 \beta \lambda^2 \mu^6 k^3 m_0 \omega N(k) \Theta (m_0 - \alpha \beta \lambda) \\
- 2a^2 \beta \lambda^2 \mu^6 k^3 \Theta - 2a^2 \beta \lambda^2 \mu^6 k^3 \Theta \omega N(k) - 2a^2 \beta \lambda^2 \mu^6 k^3 m_0 \Theta \\
- 2a^2 \beta \lambda^2 \mu^6 k^3 \Theta - 2a^2 \beta \lambda^2 \mu^6 k^3 \Theta \omega N(k) - 2a^2 \beta \lambda^2 \mu^6 k^3 m_0 \Theta.
\]
Notice that the first seven terms of above equality have the factor \((\mu m_0 - \alpha \beta \lambda)\) and \(\mu m_0 - \alpha \beta \lambda < 0\) if \(R_0 > 1\), and the other terms are all negative; hence we conclude that \(d^2G(\Theta)/d\Theta^2 < 0\) when the inequality \(\Theta > 1 - \mu m_0 / \alpha \beta \lambda (R_0 > 1)\) holds. Therefore, the necessary and sufficient condition for Eq. (10) to have a unique positive solution, \(\Theta > 0\), is that
\[ \frac{dG(\Theta)}{d\Theta} \bigg|_{\Theta=0} > 1. \]
Then, we compute that
\[ \frac{dG(\Theta)}{d\Theta} \bigg|_{\Theta=0} = \frac{a \beta \lambda}{\langle k \rangle \mu} \sum_{k=1}^{\infty} \frac{k^2 p(k)}{\alpha \lambda + km_0} = R_0 > 1. \]
That is to say, if \(R_0 > 1\), there exists a unique positive equilibrium \(E^*(i_1^*, \ldots, i_n^*, M^*)\) of system (2).

**Theorem 3.** If \(R_0 > 1\), system (2) admits a unique endemic equilibrium \(E^*(i_1^*, \ldots, i_n^*, M^*)\).

In the following, we will investigate the order parameter (prevalence) \(i(t)\) using the relation
\[ i(t) = \sum_k p(k)i_k(t). \]
To perform an explicit calculation for the Barabasi-Albert (BA) [26] scale-free model, we use the continuous \(k\) approximation that allows the practical substitution of sum series with integrals [38,39,29]. The full connectivity distribution of the BA scale-free network is embedded with \(p(k) = 2m^2 k^{-3}\), where \(m\) is the minimum number of links at each node. Noting that the average connectivity is \(\langle k \rangle = \int_0^\infty k p(k) dk = 2m\), from Eq. (10), we have
\[ \Theta = \frac{\mu (\alpha + \mu m_0) - \mu mm_0 e^{\alpha / \mu}}{\mu (\alpha + 2m \omega N \Theta) + (\mu m_0 + \alpha \beta \lambda \Theta + 2m \beta \omega N \Theta) \kappa}, \]
which yields the solution
\[ \Theta = \frac{\mu (\alpha + \mu m_0) - \mu mm_0 e^{\alpha / \mu}}{\mu (\alpha + 2m \omega N \Theta) + (\mu m_0 + \alpha \beta \lambda \Theta + 2m \beta \omega N \Theta) \kappa}. \]
Then, the order parameter is calculated as follows:
\[ i = \sum_k p(k)i_k \]
\[ = 2m^2 \beta \Theta (\alpha + \mu m_0 \Theta) \int_0^\infty \frac{1}{k^2} \frac{dk}{\mu (\alpha + \mu m_0 \Theta) + (\mu m_0 + \alpha \beta \lambda \Theta + 2m \beta \omega N \Theta) \kappa} \]
\[ = \frac{2m \Theta [\alpha \beta \lambda (1 - \Theta) - \mu m_0]}{\mu (\alpha + 2m \Theta)} \]
By substituting the expression for \(\Theta\) into the above equality, we can get the order parameter \(i\) analytically.
Fig. 1. The relationship between the basic reproduction number $R_0$ and the model parameters $a$, $\lambda$, $m_0$, and $\beta/\mu$ on BA scale-free networks.
Fig. 2. The influence of different values of $\lambda$ on the total density of infected nodes, with $a = 10$, $\beta = 0.02$, $\mu = 0.11$, $\omega = 0.0005$, $m_0 = 0.05$, $N = 1000$, $\lambda = 0.01, 0.03, 0.05, 0.07, 0.09, 0.1, 0.3, 0.5, 0.7, 0.9$ (from bottom to top), respectively (semilog plot). The inset shows the steady infected density scaling with $\lambda$.

Fig. 3. The influence of different values of $m_0$ on the total density of infected nodes, with $a = 10$, $\beta = 0.02$, $\mu = 0.11$, $\omega = 0.0005$, $N = 1000$, $\lambda = 0.05, m_0 = 0.003, 0.007, 0.01, 0.03, 0.05, 0.07, 0.09, 0.1, 0.3, 0.5$ (from top to bottom), respectively (semilog plot). The inset shows the steady infected density as a function of $m_0$.

Remark 3. If we let $m_0 = 0$, then $\omega = 0$; that is, there is no media coverage on disease transmission. After some algebraic operations, one has $i = \frac{2}{e^{\frac{m_0}{m}\beta} - 1} \left[ 1 - \frac{\mu}{m\beta} (e^{\frac{m_0}{m}\beta} - 1) \right]$, i.e. $i \propto 2e^{-\frac{m_0}{m}\beta}$, which is consistent with the result of Pastor-Satorras et al. [16] (set $\mu = 1$ therein).

4. Numerical simulations

In this section, we will perform a series of numerical simulations to give more insight into the dynamical process on BA random scale-free networks ($p(k) = 2m^2k^{-3}$, $m = 3$). First, we perform a sensitivity analysis of the basic reproduction number $R_0$ in terms of the model parameters.

From Fig. 1, we can see that the basic reproduction number $R_0$ increases with increasing the model parameters $a$, $\lambda$, and $\beta/\mu$, while it decreases with increasing the parameter $m_0$. The influence of parameter $\lambda$ (depletion rate of media coverage) on the basic reproduction number $R_0$ is greater than that of parameters $a$ and $\beta/\mu$, which shows that we should improve the effectiveness of the campaigning (i.e. lower $\lambda$) to reduce $R_0$.

In the following, we will perform a series of numerical simulations to gain more insight into system (2). We consider a BA network with $m = 3$ ($p(k) = 2m^2k^{-3}$); the other parameters are chosen as $a = 10$, $\beta = 0.02$, $\mu = 0.11$, $\lambda = 0.08$, $\omega = 0.0005$, and $N = 1000$. To see the effect of different model parameters on the transmission dynamics in greater depth, we illustrate the influence of these parameters on the prevalence (total density) of the disease in Figs. 2–4.
Fig. 4. The impact of different values of $\omega$ on the total density of infected nodes, with $a = 10$, $\beta = 0.02$, $\mu = 0.11$, $N = 1000$, $\lambda = 0.08$, $\omega = 0.0005, 0.001, 0.005, 0.01, 0.05, 0.1$ (from top to bottom), respectively. (a) $R_0 < 1$ ($m_0 = 0.05$); (b) $R_0 > 1$ ($m_0 = 0.04$). The inset shows the steady infected density as a function of $\omega$.

In Fig. 2 the influence of different values $\lambda$ on the total infected density is shown. We can see that the smaller the value of $\lambda$ is (the more effective the media and education that is implemented), the lower the total density of infected nodes is. The inset of Fig. 2 shows how the infected density in the steady state scales with $\lambda$. The same operation is done for the parameters $m_0$ and $\omega$. From Fig. 3, one obtains that the larger the value of $m_0$ is, the more easily the disease could be prevented. This means that the disease control and prevention center had better timely report the disease emerging in another neighboring region due to rapid spatial spread, although there is no infected case found in the present region. The inset of Fig. 3 also shows the infected density scaling with $m_0$.

Although the parameter $\omega$ is independent of $R_0$ (see Section 3), it has a great influence on the total density of infected nodes; see Fig. 4. On the one hand, when $R_0 < 1$, increasing the value of $\omega$ could accelerate the extinction of the disease (see Fig. 4(a)); on the other hand, when $R_0 > 1$, increasing the value of $\omega$ lead to a large reduction of the final (total) infected density (see Fig. 4(b)).

Next, we consider the case $R_0 = 0.9878 < 1$ by setting $m_0 = 0.05$. Fig. 5(a) shows the time distribution of disease extinction within each group $k$: the smaller $k$ is, the faster the disease vanishes in that group, since the smaller $k$ group has less chance to contact infected nodes compared to the larger $k$ group. Fig. 5(b) shows the time evolution of the total infected density nodes with different initial infections. Obviously, one can see that, if $R_0 < 1$, the disease will die out independent of different initial infections, which implies that the disease-free equilibrium is globally stable in $\Gamma$.

Fig. 6(a) presents the time when the spreading arrives at the endemic state within each group $k$, $R_0 = 1.0824 > 1$, by letting $m_0 = 0.04$. Fig. 6(b) indicates that the disease will persist and converge to a unique positive equilibrium state $\hat{I}$ whatever the initial total infected density is, which means that the endemic states are stable.

Finally, we explore the finite size dependence of the model process in Fig. 7. As the population size $N$ increases, the lower the steady state density of infected nodes. We can see this also from the order parameter $i(t)$ in Section 3.
Fig. 5. (a) The distribution of the times when the epidemic vanishes within each group $k$; (b) the time series for total density of infected nodes across networks with different initial infections. Parameter values: $a = 10$, $\beta = 0.02$, $\mu = 0.11$, $N = 1000$, $\lambda = 0.08$, $m_0 = 0.05$, $\omega = 0.0005$, $m = 3$, $n = 100$.

5. Conclusions and discussions

In this paper, we have proposed an SIS network model to investigate the influence of media coverage on disease transmission. Different from the classical network-based SIS models [14,16], which only consider contacts between susceptible and infectious individuals, the impact of media coverage on the reduction of the transmission rate is also taken into account. We derive the basic reproduction number $R_0$ by investigating the local stability of the disease-free equilibrium $E_0$ and perform a series of numerical simulations to gain more insight into the dynamical process.

Our results show that the basic reproduction number $R_0$ determines the dynamical behavior of the system. In particular, if $R_0 < 1$ the disease-free equilibrium $E_0$ is globally asymptotically stable (that is, the disease always dies out), so, in order to prevent the spread of the disease, what we need to do is to reduce $R_0$ below 1 (see Fig. 5(b)); if $R_0 > 1$, the disease will persist and converge to a unique positive stationary state (see Fig. 6(b)). There are some unanswered questions with our model. For example, we obtain the global asymptotic stability of the endemic equilibrium only through numerical simulations, and cannot provide an elaborate analysis.

The basic reproduction number $R_0$ is smaller than that of Ref. [16], since we introduce another parameter $m_0$. If $m_0 = 0$, although the media coverage does not change the basic reproduction number $R_0$, it can reduce the final prevalence (density); see Fig. 4(b). In order to prevent the disease, what we need to do is to reduce $R_0$ below 1. According to Figs. 2–4, we can do this by improving the efficiency of the media and education implemented, timely reporting new infectious cases emerging in another neighboring region as well as the local region, and teaching the residents to adopt necessary measures.

However, there are some limitations of our model: (i) we use mean-field theory to describe the interactions between the susceptibles and infected individuals without explicitly considering the network topology such as assortative or disassortative, clustering [26]; (ii) we use a maximum degree $n$ such that the ODE approximation has the same dynamics as
Fig. 6. (a) The time when the spreading arrives at the endemic state within each group $k$; (b) the time series for total density of infected nodes across networks with different initial infections. Parameter values: $a = 10$, $\beta = 0.02$, $\mu = 0.11$, $N = 1000$, $\lambda = 0.08$, $m_0 = 0.04$, $\omega = 0.0005$, $m = 3$, $n = 100$.

Fig. 7. The steady-state density of infected nodes as a function of population size $N$. Parameter values: $a = 10$, $\beta = 0.02$, $\mu = 0.11$, $\lambda = 0.08$, $m_0 = 0.04$, $\omega = 0.0005$, $m = 3$, $n = 100$. 
the stochastic process if \( N/n \) is large [40]. We emphasize that this preliminary work does not consider the factors of edges rewiring, but only focuses on the case in which the media coverage reduces the transmission rate between susceptible and infected individuals. We can generalize the current model in many aspects. For example, a susceptible individual may cut its links to an infected individual and rewire those links once the information he/she receives due to media coverage reaches a certain threshold [41]; or we could consider a multiplex network with two layers [42], in which one is the disease transmission network and the other is the information transmission network, but the two networks have different topologies. These and some other related issues are very good topics on networks, which deserves further exploration.

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**References**

1. R. Laxminarayan, A. Mills, J. Breman, A. Measham, G. Alleyne, M. Claeson, P. Jha, P. Musgrove, J. Chow, S. Shahid-Salles, D. Jamison, Advancement of global health: key messages from the disease control priorities project, Lancet 367 (2006) 1193–1208.
2. Y. Liu, J. Cui, H. Zhu, The impact of media coverage on the dynamics of infectious diseases, Int. J. Biomath. 1 (2008) 65–74.
3. D. Gao, S. Ruan, An SIS patch model with variable transmission coefficients, Math. Biosci. 232 (2011) 110–115.
4. R. Liu, J. Wu, H. Zhu, Media/psychological impact on multiple outbreaks of emerging infectious diseases, Comput. Math. Methods Med. 8 (2007) 153–164.
5. J. Tchuenche, N. Dube, C. Bhunu, R. Smith, C. Bauch, The impact of media coverage on the transmission dynamics of human influenza, BMC Public Health 11 (2011) 55.
6. Y. Liu, X. Sun, H. Zhu, The impact of media on the control of infectious diseases, J. Dynam. Differential Equations 20 (2008) 31–53.
7. J. Cui, X. Tao, H. Zhu, An SIS infection model incorporating media coverage, Rocky Mountain J. Math. 38 (2008) 1323–1334.
8. Y. Li, J. Cui, The effect of constant and pulse vaccination on SIS epidemic models incorporating media coverage, Commun. Nonlinear Sci. Numer. Simul. 14 (2009) 2353–2365.
9. M. Misra, A. Sharma, J. Shukla, Modeling and analysis of effects of awareness programs by media on the spread of infectious diseases, Math. Comput. Modelling 53 (2011) 1221–1228.
10. P. Wallis, B. Nerlich, Disease metaphors in new epemics: the UK media framing of the 2003 SARS epidemic, Soc. Sci. Med. 60 (2005) 2629–2639.
11. N. Wilson, G. Thomson, O. Mansoor, Print media response to SARS in New Zealand, Emerg. Infectious Dis. 10 (2004) 1461–1464.
12. S. Funk, E. Gilad, C. Watkins, V. Jansen, The spread of awareness and its impact on epidemic outbreaks, Proc. Natl. Acad. Sci. USA 106 (2009) 6872–6877.
13. M. Newman, Tks, The spread of epidemic disease on networks, Phys. Rev. E 66 (2002) 016128.
14. R. May, A. Lloyd, Infection dynamics on scale-free networks, Phys. Rev. E 64 (2001) 066122.
15. M. Keeling, The effects of local spatial structure on epidemiology of invasions, Proc. R. Soc. Lond. B 266 (1999) 859–867.
16. R. Pastor-Satorras, A. Vespignani, Epidemic spreading in scale-free networks, Phys. Rev. Lett. 86 (2001) 3200–3203.
17. R. Pastor-Satorras, A. Vespignani, Absence of epidemic threshold in scale-free networks with degree correlations, Phys. Rev. E 90 (2009) 028701.
18. M. Boguna, R. Pastor-Satorras, A. Vespignani, Epidemic spreading in complex networks with degree correlations, Lecture Notes in Phys. 625 (2003) 127–147.
19. R. Pastor-Satorras, A. Vespignani, Epidemic dynamics in finite size scale-free networks, Phys. Rev. E 65 (2002) 035108.
20. X.C. Fu, M. Small, D.M. Walker, H.F. Zhang, Epidemic dynamics on scale-free networks with piecewise linear infectivity and immunization, Phys. Rev. E 77 (2008) 036113.
21. J.Z. Wang, Z.R. Liu, J.H. Xu, Epidemic spreading on uncorrelated heterogeneous networks with non-uniform transmission, Physica A 382 (2007) 715–721.
22. T. Zhou, J.G. Liu, W.J. Bai, G.R. Chen, B.H. Wang, Behaviors of susceptible–infected epidemics on scale-free networks with identical infectivity, Phys. Rev. E 74 (2006) 056109.
23. J. Joo, J.L. Lebowitz, Behavior of susceptible–infected–susceptible epidemics on heterogeneous networks with saturation, Phys. Rev. E 69 (2004) 066105.
24. P.F. Zhang, X.C. Fu, Spreading of epidemics on scale-free networks with nonlinear infectivity, Nonlinear Anal. 70 (2009) 3273–3278.
25. M.E.J. Newman, The structure and function of complex networks, SIAM Rev. 45 (2003) 167–256.
26. R. Albert, A. Barabasi, Statistical mechanics of complex networks, Rev. Modern Phys. 74 (2002) 47–97.
27. S. Boccaletti, V. Latora, Y. Moreno, M. Chavez, D. Hwang, Complex networks: structure and dynamics, Phys. Rep. 424 (2006) 175–308.
28. A. Clauset, C. Shalizi, M.E.J. Newman, Powerlaw distributions in empirical data, SIAM Rev. 51 (2009) 661–703.
29. R. Pastor-Satorras, A. Vespignani, Epidemic dynamics and endemic states in complex networks, Phys. Rev. E 63 (2001) 066117.
30. N.T.J. Bailey, The Mathematical Theory of Infectious Diseases, second ed., Griffin, London, 1975.
31. J.D. Murray, Mathematical Biology, Springer Verlag, Berlin, 1993.
32. J. Li, Effects of behaviour change on the spread of AIDS epidemic, Math. Comput. Modelling 16 (1992) 103–111.
33. R. Naresh, S. Pandey, A.K. Misra, Analysis of a vaccination model for carrier dependent infectious disease with environmental effects, Nonlinear Anal. Model. Control 13 (2008) 313305.
34. R. Naresh, A. Tripathi, D. Sharma, Modeling and analysis of the spread of AIDS epidemic with immigration of HIV infectives, Math. Comput. Modelling 49 (2009) 880–892.
35. A. d’Onofrio, P. Manfredi, E. Salinelli, Vaccinating behaviour, information, and the spread of SIR vaccine preventable diseases, Theor. Popul. Biol. 71 (2007) 301–317.
36. J.D. Murray, Mathematical Biology, Springer, New York, Tokyo, 1989.
37. P. van den Driesche, J. Watmough, Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission, Math. Biosci. 180 (2002) 29–48.
38. A. d’Onofrio, A note on the behaviour of the network-based SIS epidemic model, Nonlinear Anal. 9 (2008) 1567–1572.
39. A. Barabasi, R. Albert, Emergence of scaling in random networks, Science 286 (1999) 509–512.
40. A. Barabasi, R. Albert, H. Jeong, Mean-field theory for scale-free random networks, Physica A 272 (1999) 173–187.
41. J. Lindquist, J.L. Ma, P. van den Driesche, F.H. Willeboorde, Network evolution by different rewiring schemes, Physica D 238 (2009) 370–378.
42. T. Gross, Carlos J. Dommar D’Lima, B. Blasius, Epidemic dynamics in an adaptive network, Phys. Rev. Lett. (2006) 208701.
43. M. Newman, A. Daz-Guilera, J. Gomez-Gardences, C.J. Perez-Vicente, Y. Moreno, A. Arenas, Diffusion dynamics on multiplex networks, Phys. Rev. Lett. (2013) 028701.