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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 lowand 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

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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 [4,2,6–9,5]. 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 λ_c in a homogeneous network as the classical prediction [30], and a surprising absence of any epidemic threshold or $\lambda_c = 0$ in the heterogeneous counterpart if $2 < \gamma \le 3$ 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, λ_c 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 λ_c 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, ..., 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

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