Physica A 433 (2015) 42–50

Contents lists available at ScienceDirect

Physica A

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

The virus variation model by considering the degree-dependent spreading rate

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HIGHLIGHTS

- We present the virus variation model by considering the degree-dependent spreading rate.
- The epidemic threshold with degree-dependent spreading rate can be theoretical drawn.
- The average infected virus version in the BA network is less than in the ER network.
- The greater the initial infected degree, the smaller the average virus version of the whole infected node.

ARTICLE INFO

Article history:
Received 23 October 2014
Received in revised form 22 February 2015
Available online 2 April 2015

Keywords:
Virus mutation
Epidemic threshold
Degree-dependent spreading rate
Infected virus version

ABSTRACT

Considering the difference of different individuals' physical quality and antibody, this paper investigates the epidemic spreading model with the virus mutation. By using the mean-field theory, the epidemic threshold with degree-dependent spreading rate can be theoretical drawn. According to the numerical simulations, we can obtain that the average infected virus version in the BA network is less than the ER network. In addition, if the effective spreading rate is either small or large enough, the average virus version of the whole infected individuals will reduce. However, when the spreading rate takes some proper values, the average infected virus version can greatly increase. Finally, we study how the different initial infected nodes influence the average virus version of the whole infected individuals. The numerical results show that the greater of the initial infected degree, the smaller of the average virus version of the whole infected individuals.

1. Introduction

Human history has been related to epidemics, and various types of diseases have ravaged a lot of civilizations. For example the SARS outbreak in 2003 and the 2009 new flu strain H1N1 that hit the world leading to a pandemic with a large amount of infections and panic [1,2]. The outbreak of infectious disease has cost large amounts of money in health care and disease control. The whole world is devoted to stop spreading of such diseases. However, analyzing and understanding the propagation of infectious diseases is of great significance to efficiently control potentially devastating epidemic outbreaks as well as to deploy tailored immunization strategies [3,4].

Over the past decades, there has been a great deal of effort in understanding the dynamical process of epidemic spreading [5–12]. Scholars have used various mathematical models to analyze the spread of disease [13–15]. Zhang et al.

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http://dx.doi.org/10.1016/j.physa.2015.03.077
0378-4371/

studied the effects of behavioral response and vaccination policy on epidemic spreading. The results illustrated that under the partial-subsidy policy, the vaccination coverage depends monotonically on the sensitivity of individuals to payoff difference, but the dependence is non-monotonous for the free-subsidy policy [16]. Chen et al. investigated the global stability and optimal control of an SIRS epidemic model on heterogeneous networks. The obtained results showed that an optimal control exists for the control problem [17]. Yang et al. investigated the traffic-driven epidemic outbreak on complex networks. Their study indicated that increasing the average degree or inducing traffic congestion could slow down the spreading process significantly [18]. Cao et al. researched the impact of media coverage on epidemic spreading in complex networks, their results showed that the disease-free equilibrium is globally asymptotically stable and that the disease dies out if the basic reproduction number is below 1 [19].

One of the most easily mutation virus is the influenza, for example the influenza A virus subtype H1N1 which is a novel strain of influenza [20]. Existing vaccines against seasonal flu provide no protection. Meanwhile, a study at the US Centers for Disease Control and Prevention published in May 2009 found that children have no preexisting immunity to the new strain but that adults, particularly those over 60 have some degree of immunity. Children show no cross-reactive antibody reaction to the new strain, adults' aged 18 to 64 had 6%–9%, and older adults 33% [21–25]. Since the difference of individuals' physical quality and antibody, therefore, it is reasonable to assume that the virus can mutate in a part of infected people. On the other hand, the infected individuals spread the virus to their neighbors not only depending on the spreading rate but also the number of connections they have. This kind of spreading rate can be called the degree-dependent spreading rate (i.e., the spreading capability of an individual depends on the number of his/her neighbors). In general, if an individual i has lots of friends, the probability which one of his friends contacts him will decrease with the increase of his connections. Given an example, if an individual i has 5 friends and an individual j has 10 friends, and each individual could contact one of his friends with the same probability at each time step. Then i would contact one of his friends with probability 0.2 per unit time, however, j would contact one of his friends with probability 0.1. The virus variation model by considering the degree-dependent spreading rate has not been well considered [26–29].

Motivated by the above discussion, in this paper we propose the dynamics of the virus variation model. According to theoretical analysis and numerical simulations, the impact of the degree-dependent spreading rate on the epidemic spreading is investigated. Firstly, a constant θ is introduced to describe the different nodes spreading rate. Then, based on the mean-field theory, the epidemic thresholds are theoretical drawn both in the BA network and in the ER network. Finally, some meaningful results are obtained. (1) The average infected virus version in the BA network is less than in the ER network. (2) If the effective spreading rate is very small or large, the average virus version of the whole infected individuals will reduce. However, if the effective spreading rate takes some proper values, the average infected virus version can greatly increase. (3) The greater the initial infected degree, the smaller the average virus version of the whole infected individuals.

The remainder of this paper is organized as follows. In Section 2, the model of virus variation is introduced in detail, and the epidemic threshold with degree-dependent spreading rate is theoretical drawn both in the BA network and in the ER network. Section 3 presents the simulation results and the relevant discussions. Finally, we conclude the paper in Section 4.

2. The model of SIR virus variation

In this paper, we consider two types of connection networks models: BA network and ER network. With respect to the epidemic model, our model is based on the evolution of a Susceptible–Infected–Recovered (SIR) epidemic dynamics [30,31]. Considering the actual situation, since the difference of different individuals' physical quality and antibody, the whole people are divided into two types: the mutation-individual and the normal-individual. Some assumes and instructions are given as follows:

(1) $s_1, s_2, \ldots, s_n, \ldots$ denote the virus version.
(2) The original virus version of each infected is version 1.
(3) Due to the physical and physiological differences, the virus can mutate in a certain proportion of individuals. Typically, when a mutation-individual is infected by the version s_i, $(i = 1, 2, \ldots)$, then he will spread virus with the version s_{i+1}. However, when a normal-individual is infected by the version s_i, $(i = 1, 2, \ldots)$, then he will spread virus with the version s_i.
(4) If a susceptible i gets infected from more than one neighbor, the individual i will carry the newest virus version. For example, a susceptible i is infected by two neighbors a and b, however, the nodes a and b carry the virus version s_a and s_b, respectively. Then, the individual i will carry the virus version $s_i = \max\{s_a, s_b\}$.

2.1. Epidemic spreads in the heterogeneous networks

Let $\rho_k(t), S_k(t), R_k(t)$ denote the density of infected, susceptible, and recovered with connectivity k at time t, respectively. These variables are connected by means of the normalization condition:

$$\rho_k(t) + S_k(t) + R_k(t) = 1 \tag{1}$$