Physica A 392 (2013) 1577–1585

Contents lists available at SciVerse ScienceDirect

Physica A

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

Effects of delayed recovery and nonuniform transmission on the spreading of diseases in complex networks

Cheng-yi Xia [a,b,*], Zhen Wang [c,d,e], Joaquin Sanz [c,f], Sandro Meloni [c], Yamir Moreno [c,f]

[a] Key Laboratory of Computer Vision and System (Ministry of Education), Tianjin University of Technology, Tianjin 300384, PR China

[b] Tianjin Key Laboratory of Intelligence Computing and Novel Software Technology, Tianjin University of Technology, Tianjin 300384, PR China

[c] Institute for Biocomputation and Physics of Complex Systems (BIFI), University of Zaragoza, 50018 Zaragoza, Spain

[d] Department of Physics, Hong Kong Baptist University, Kowloon Tong, Hong Kong

[e] Center for Nonlinear Studies and the Beijing-Hong Kong-Singapore Joint Center for Nonlinear and Complex Systems (Hong Kong) Baptist University, Kowloon Tong, Hong Kong

[f] Department of Theoretical Physics, University of Zaragoza, 50009 Zaragoza, Spain

ARTICLE INFO

Article history:
Received 31 July 2012
Received in revised form 29 October 2012
Available online 27 November 2012

Keywords:
Disease spreading
Complex networks
SIS model
Heterogeneous mean-field approach

ABSTRACT

We investigate the effects of delaying the time to recovery (delayed recovery) and of nonuniform transmission on the propagation of diseases on structured populations. Through a mean-field approximation and large-scale numerical simulations, we find that postponing the transition from the infectious to the recovered states can largely reduce the epidemic threshold, therefore promoting the outbreak of epidemics. On the other hand, if we consider nonuniform transmission among individuals, the epidemic threshold increases, thus inhibiting the spreading process. When both mechanisms are at work, the latter might prevail, hence resulting in an increase of the epidemic threshold with respect to the standard case, in which both ingredients are absent. Our findings are of interest for a better understanding of how diseases propagate on structured populations and to a further design of efficient immunization strategies.

1. Introduction

Infectious diseases have been a great threat to human beings for a long time [1]. Especially in recent years, some emerging infectious diseases, such as severe acute respiratory syndrome(SARS) [2], avian influenza [3,4], and swine influenza [5], have resulted in huge life and economical losses. Thus, 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. Traditionally, there are two typical epidemic models: the Susceptible–Infected–Susceptible (SIS) and the Susceptible–Infected–Removed (SIR) models. Both kinds of models have been intensively studied during the last few years, adding to the traditional well-mixed hypotheses usually invoked by the models [6] an ever increasing dose of realism.

As a matter of fact, real systems are neither regular (and/or well-mixed) nor random, but their topology is usually different to these limits [7,8]. Actually, the more abundant are those called scale-free (SF) networks, in which the probability $P(k)$ that an individual has k neighbors is a power-law distribution [8,9]. Today, the modeling of infectious diseases and their prevention and control has become an interdisciplinary issue which has attracted the attention of scientists from epidemiology, biology, mathematics, physics and computational sciences [10–13]. In particular, Pastor-Satorras and

* Corresponding author at: Key Laboratory of Computer Vision and System (Ministry of Education), Tianjin University of Technology, Tianjin 300384, PR China.
E-mail address: xialooking@163.com (C.-y. Xia).

0378-4371/$ – see front matter © 2012 Elsevier B.V. All rights reserved.
doi:10.1016/j.physa.2012.11.043

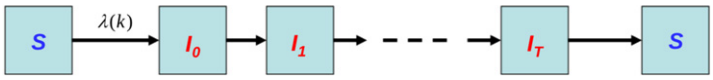

Fig. 1. The figure shows the cycle of infection of a susceptible individual. We assume that after the initial infection, the newly infected node will remain infectious during a time window of $T + 1$ time steps, after which the node recovers and gets back to the susceptible state.

Vespignani [14,15] showed that the epidemic threshold λ_c in an SIS model is absent for SF networks in the thermodynamic limit, that is, the threshold λ_c approaches zero and even a vanishingly small infection rate can produce an outbreak. Similar conclusions were also found for the SIR model on SF networks by Moreno et al. [16]. These alarming results have inspired a great number of related works, and most results point out that the topology of interactions dominates the spreading dynamics in complex networks [17–29].

Other works have also explored the effects of different infection mechanisms. For instance, in Ref. [30], the authors proposed a kind of connectivity-dependent infection scheme, which can yield threshold effects even in scale-free networks where they would otherwise be unexpected. Additional ingredients include saturation effects [31], constant infectivity [32], nonuniform transmission [33], finite populations [34], traffic-driven mechanisms [35], and piece-wise infection probability [36], which have been integrated into the SIS or SIR models.

On the other hand, there are other realistic elements that have been partially addressed in recent studies. For instance, the issue of delayed recovery can be thought of as the time elapsed since an individual becomes infected and the moment he/she starts the treatment that could lead to recovery [37,38]. This is especially relevant when studying spreading dynamics of diseases for which spontaneous recovery not due to medical treatment is unlikely. In what follows, we study the effects of such delayed recovery on the epidemic thresholds of an SIS dynamics that takes place on top of homogeneous and heterogeneous networks. Moreover, we also consider the case of nonuniform transmission (i.e., the fact that the spreading capabilities of an individual depend on his/her number of contacts) and the situation in which both mechanisms are concurrently active. To this end, we make use of the heterogeneous mean-field theory and perform large-scale numerical simulations, which we show are in agreement with the analytical predictions.

The rest of this paper is organized as follows. Section 2 describes in detail our model. In Section 3, the mean-field theory is used to derive the epidemic thresholds for homogeneous and heterogeneous networks. Large-scale numerical Monte Carlo simulations are also carried out to validate the mean-field approximation in Section 3. Finally, in Section 4, we round off the paper by presenting our concluding remarks.

2. The model

In the standard SIS model, individuals are divided into two categories: Susceptible (S) and Infected (I). Susceptible individuals are healthy ones which can be infected with the probability β through contacts with infectious subjects. Infective individuals in their turn are recovered with the probability γ, which we henceforth set to 1. Hence, individuals go through the cycle $S \longrightarrow I \longrightarrow S$, their dynamics being described by,

$$\begin{cases} \dfrac{\mathrm{d}s(t)}{\mathrm{d}t} = -\gamma\rho(t) + \beta s(t)\rho(t) \\ \dfrac{\mathrm{d}\rho(t)}{\mathrm{d}t} = \gamma\rho(t) - \beta s(t)\rho(t) \end{cases} \tag{1}$$

where $s(t)$ and $\rho(t)$ stand for the fraction of susceptible and infective individuals. Generally, we neglect the details of disease infection and fix the size of the total population, and thus $s(t)$ and $\rho(t)$ need to satisfy the normalization condition: $s(t) + \rho(t) = 1$.

In our modified SIS model with nonuniform spreading (transmission) probabilities and delayed recovery, we still assume that individuals can be susceptible or infectious. However, we introduce two new ingredients:

- If an individual is infected by his/her infected neighbors at any time step t, it will be infectious during a time window $T + 1$. Once this time has elapsed, the infective agent goes back to the susceptible state, S, with probability $\gamma = 1$, which can be assumed without loss of generality.
- At each time step t, infected individuals spread the disease to susceptible nodes with a probability that depends on the number of connections it has. Therefore, we assume that the effective spreading rate $\lambda = \beta/\gamma$ is a degree-dependent function $\lambda(k) = \frac{\lambda_0 k^\alpha}{k}$ (i.e., so-called nonuniform transmission).

The flow diagram of the disease spreading process for our modified model can be seen in Fig. 1, in which $I_0, I_1, \ldots, I_T$ denote the infective individuals at different stages and S represents the susceptible agents.

3. Epidemic thresholds

In this section, we investigate the critical thresholds of the model in both homogeneous and heterogeneous networks.