Physica A 515 (2019) 587–599

Contents lists available at ScienceDirect

Physica A

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

Global stability of a network-based SIRS epidemic model with nonmonotone incidence rate

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HIGHLIGHTS

- A network-based SIRS epidemic model with vaccination and a nonmonotone incidence rate is proposed.
- Epidemic threshold is obtained.
- Criteria of global stability of the equilibrium are established.
- Numerical experiments for a finite scale-free network are presented.

ARTICLE INFO

Article history:
Received 26 February 2018
Received in revised form 28 April 2018
Available online xxxx

Keywords:
SIRS epidemic model
Complex networks
Global stability
Nonmonotone incidence

ABSTRACT

This paper studies the dynamics of a network-based SIRS epidemic model with vaccination and a nonmonotone incidence rate. This type of nonlinear incidence can be used to describe the psychological or inhibitory effect from the behavioral change of the susceptible individuals when the number of infective individuals on heterogeneous networks is getting larger. Using the analytical method, epidemic threshold R_0 is obtained. When R_0 is less than one, we prove the disease-free equilibrium is globally asymptotically stable and the disease dies out, while R_0 is greater than one, there exists a unique endemic equilibrium. By constructing a suitable Lyapunov function, we also prove the endemic equilibrium is globally asymptotically stable if the inhibitory factor α is sufficiently large. Numerical experiments are also given to support the theoretical results. It is shown both theoretically and numerically a larger α can accelerate the extinction of the disease and reduce the level of disease.

1. Introduction

Mathematical models which describe the dynamics of infectious diseases have played a crucial role in the disease control in epidemiological aspect. In the traditional epidemiology, it is commonly assumed that individuals mix uniformly and all hosts have identical rates of disease-causing contacts. This over-simplified assumption makes the analysis tractable but not realistic [1]. Virtually, the interpersonal contact underlying disease transmission can be thought of expanding a complex network, where relations (edges) join individuals (nodes) who interact with each other [2,3]. Therefore, the disease transmission should be modeled over complex networks. Related studies indicated that networks structures have profound impacts on the spreading dynamics [4–7].

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https://doi.org/10.1016/j.physa.2018.09.152
0378-4371/

One of the pioneer works in this area was done by Pastor-Satorras and Vespignani [8,2], where they first succeeded in studying susceptible–infectious–susceptible (SIS) epidemic model on scale-free networks by large-scale simulations. The most striking result is that they found the absence of the epidemic threshold in these networks. That is, the threshold approaches zero in the limit of a large number of edges and nodes, and even a quite small infectious rate can produce a major epidemic outbreak, for which the rigorous proof was given by Wang and Dai [9] by a monotone iterative technique. Moreno et al. [10] studied susceptible–infectious–recovered (SIR) epidemic model on scale-free complex population networks. They show that the large connectivity fluctuations usually found in these networks strengthen considerably the incidence of epidemic outbreaks. These outstanding results have inspired a great number of related works (see Refs. [11–16] and the references therein).

It is well-known that the spread of many human diseases can be prevented or reduced by vaccination of the susceptible individuals. Chen and Sun [17] firstly succeeded in studying optimal control of an susceptible–infectious–recovered–susceptible (SIRS) epidemic model with vaccination on heterogeneous networks. They showed that if the percentage of vaccination of the susceptible is smaller than the recovered rate, the diseases may persist on heterogeneous networks.

We shall emphasize that the incidence rate plays an important role in guaranteeing that the model can give a reasonable approximative description for the disease dynamics. However, in most existing epidemic models on complex networks, the incidence rate is usually assumed to be bilinear function based on the mass action law for infection. In fact, there are several reasons for using nonlinear incidence rates, even nonmonotone incidence function [18–22]. In practical situations, the number of effective contacts between infective individuals and susceptible individuals usually decreases at high infective levels due to the quarantine of infective individuals or due to the protection measures by the susceptible individuals. Very recently, for modeling such a psychological behavior on complex networks, Li [23] studied the dynamics of a network-based SIS epidemic model with nonmonotone incidence rate,

$$\begin{cases} \dfrac{dS_k(t)}{dt} = -\lambda k S_k(t) g(\Theta) + I_k(t), \\ \dfrac{dI_k(t)}{dt} = \lambda k S_k(t) g(\Theta) - I_k(t), \quad k = 1, 2, \ldots, n, \end{cases} \tag{1.1}$$

where $\lambda > 0$ is the transmission rate when susceptible individuals contact with infectious. $S_k(t)$ and $I_k(t)$ denote the relative densities of susceptible and infectious with degree k at time t on the complex networks with maximum degree n. The connectivity of nodes on the network is assumed to be uncorrelated, thus, we have $\Theta(t) = \frac{1}{\langle k \rangle} \sum_{k=1}^{n} kP(k)I_k(t)$ with $\langle k \rangle = \sum_{k=1}^{n} kP(k)$ is the average degree of the network and $P(i)$ is the connectivity distribution.

The function $g(\Theta)$ is introduced in SIS model (1.1) to reflect the psychological behavior mentioned above, which leads to nonlinear incidence rate defined by

$$\lambda k S_k(t) g(\Theta) := \lambda k S_k(t) \frac{\Theta}{1 + \alpha \Theta^2}, \tag{1.2}$$

where $\alpha \geq 0$ is a parameter.

When $\alpha = 0$, then the nonlinear incidence rate (1.2) becomes the bilinear one. Hence, the SIS model with (1.2) can be seen a generalization of the existing SIS model. Meanwhile, if α is large enough (e.g., $\alpha > 2$), the function g becomes a nonmonotone function. The biological meaning is that at high infective risk (i.e., when Θ is sufficiently large), the incidence rate may decrease as Θ increases because individuals become more careful and tend to reduce their contacts with other ones. In this sense, we call the parameter α inhibitory factor from the behavioral change of the susceptible individuals.

Inspired by the works of [17] and [23], in this paper we propose the following SIRS epidemic model with vaccination and the nonmonotone incidence rate as follows,

$$\begin{cases} \dfrac{dS_k(t)}{dt} = -\lambda k S_k(t) \dfrac{\Theta(t)}{1 + \alpha \Theta^2(t)} + \delta R_k(t) - \mu S_k(t), \\ \dfrac{dI_k(t)}{dt} = \lambda k S_k(t) \dfrac{\Theta(t)}{1 + \alpha \Theta^2(t)} - \gamma I_k(t), \\ \dfrac{dR_k(t)}{dt} = \gamma I_k(t) - \delta R_k(t) + \mu S_k(t), \quad k = 1, 2, \ldots, n. \end{cases} \tag{1.3}$$

where $S_k(t)$, $I_k(t)$, $R_k(t)$ denote the relative densities of susceptible nodes, infectious nodes and recovered nodes with degree k respectively. $\delta > 0$ represents the rate of immunization-lost for recovered nodes; $\gamma > 0$ represents the recovery rate of infected nodes; $\mu > 0$ represents the vaccination percentage for the susceptible nodes.

Among the existing epidemic models on complex networks, there has far been relatively little research into network epidemic models with nonlinear incidence rate. In [23], the author successfully proposed the SIS model with nonmonotone incidence rate and obtained the epidemic threshold. It was proved that if the transmission rate is below the threshold, the disease-free equilibrium is globally asymptotically stable, otherwise the endemic equilibrium is permanent. In our recent paper [24], the global stability and attractivity of the endemic equilibrium of system (1.1) is rigorously proved. When $\alpha = 0$, the proposed model (1.3) can be simplified to the model proposed by Chen and Sun [17]. In this paper, the global stability of disease-free equilibrium as well as the endemic equilibrium is rigorously proved without additional assumptions on the constants, which improves the existing results.