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Revisiting node-based SIR models in complex networks with degree correlations

Yi Wang [a,b], Jinde Cao [a,c,*], Abdulaziz Alofi [c], Abdullah AL-Mazrooei [c], Ahmed Elaiw [c]

[a] Department of Mathematics, Southeast University, Nanjing 210096, China

[b] Department of Mathematics and Statistics, University of Victoria, Victoria BC V8W 3R4, Canada

[c] Department of Mathematics, Faculty of Science, King Abdulaziz University, Jeddah 21589, Saudi Arabia

HIGHLIGHTS

- Two growing network models with the addition of new nodes or edges are introduced.
- Network growth may cause the presence of positive or negative degree correlations.
- Two node-based SIR models on correlated networks are revisited.
- The Miller model yields better estimation for disease properties on networks.
- The Miller model may have wider applicability than it should have been.

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ABSTRACT

In this paper, we consider two growing networks which will lead to the degree-degree correlations between two nearest neighbors in the network. When the network grows to some certain size, we introduce an SIR-like disease such as pandemic influenza H1N1/09 to the population. Due to its rapid spread, the population size changes slowly, and thus the disease spreads on correlated networks with approximately fixed size. To predict the disease evolution on correlated networks, we first review two node-based SIR models incorporating degree correlations and an edge-based SIR model without considering degree correlation, and then compare the predictions of these models with stochastic SIR simulations, respectively. We find that the edge-based model, even without considering degree correlations, agrees much better than the node-based models incorporating degree correlations with stochastic SIR simulations in many respects. Moreover, simulation results show that for networks with positive correlation, the edge-based model provides a better upper bound of the cumulative incidence than the node-based SIR models, whereas for networks with negative correlation, it provides a lower bound of the cumulative incidence.

1. Introduction

Mathematical modeling of infectious diseases, abbreviated for mathematical epidemiology (ME), can be dated back to Daniel Bernoulli [1], who studied the effect of variolation on smallpox to increase life expectancy, and initiated the idea of

* Corresponding author at: Department of Mathematics, Southeast University, Nanjing, 210096, China.
E-mail addresses: wangyi-mail@163.com (Y. Wang), jdcao@seu.edu.cn (J. Cao), aalofi1@kau.edu.sa (A. Alofi), aealmazrooei@kau.edu.sa (A. AL-Mazrooei), a_m_elaiw@yahoo.com (A. Elaiw).

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using differential mortality to estimate the rate of deaths attributable to a given disease. Mathematical epidemiology did not achieve much development in the late eighteenth and nineteenth centuries until the early twentieth century. In 1911, Sir Ross formulated a model to describe the spread of malaria through mosquitoes, and initiated one of the most important concepts in epidemiology "threshold effect", stating that the malaria in a region could be controlled by reducing the mosquito population size to a certain threshold [2]. This idea was further formalized and extended to general compartmental models in 1927 by Kermack and McKendrick [3,4], known as the basic reproduction number or basic reproductive ratio and often denoted as R_0, which is defined as the average number of secondary infections generated by a typical infected individual during its entire infectious period when introduced into a completely susceptible population [5]. Since then, a number of mathematical models have been formulated and proposed to analyze and estimate the spread and control of infectious diseases. Most of these models are variants of that of Kermack and McKendrick [3,4], see, for example, the book by Bailey [6] and a review paper by Hechcote [7].

To describe the spread of infectious diseases, Kermack and McKendrick formulated [3] the following basic SIR model

$$\begin{cases} S' = -\beta SI, \\ I' = \beta SI - \gamma I, \\ R' = \gamma I, \end{cases} \tag{1}$$

where S, I and R are all functions of time t and denote the number of the susceptible, infected and recovered individuals, respectively, β is the transmission coefficient between susceptible and infected individuals, and γ is the recovery rate of infected individuals. Here, the prime $'$ denotes derivative of a variable with respect to time t, and this notation is used throughout the paper without otherwise specified. There are two main features of this model: first, the number of susceptibles in the population decreases monotonically due to being infected but does not approach zero; and second, the number of infected eventually goes to zero as time t moves on. Denote by S_0 the initial number of susceptible individuals at the very beginning of a disease, then the basic reproduction number of model (1) is defined as $R_0 = \beta S_0/\gamma$, which completely determines whether there is a major epidemic or not. In particular, if $R_0 < 1$, the number of infected individuals goes down directly and there is no epidemic or minor outbreak; whereas if $R_0 > 1$, the number of infected individuals goes up at first due to the infection, then down again due to the recovery of infected individuals, and there is a major outbreak. The whole epidemic process ends with no infected individual left in the population. This kind of model assumes permanent immunity and is applicable to diseases such as flu, measles and chickenpox caused by a virus. Another fundamental model frequently used in mathematical epidemiology is of SIS type, which assumes no immunity against re-infection (once infected, the infected individuals can go back to the susceptible class), and is applicable to diseases such as cephalitis and gonorrhea caused by a bacterium. Both of these models yield the same basic reproduction number, i.e. $R_0 = \beta S_0/\gamma$. However, if $R_0 > 1$ the SIS type disease will persist at an endemic level. For more details about these basic epidemiological models, we refer the interested reader to, for example, Refs. [8,9]. In the present paper, however, we mainly concentrate on the SIR epidemic model.

Although model (1) is fitted well for some observed disease data, it is too oversimplified and ignores too many structures presented in the real population. For example, it is assumed that the sizes of the compartments are large enough such that the population is homogeneously mixed, that is, every individual has the equal probability of contacting any other individual in the population. This assumption, however, is not realistic. In fact, different individuals may have varying number of acquaintances [10,11]. One way to proceed would be to add contact heterogeneity to the population and see how much this alters the model behavior [12]. Contact network models are such attempts to represent the population. Under this framework, each individual is denoted by a node of the network, and possible contacts between two individuals (corresponding to two nodes in the network) are linked by an edge. These two nodes are called neighbors of each other, and thus a node can acquire infection only from one of its neighbors; in other words, the contact rate is proportional to the number of neighbors, i.e. the degree of a node.

In 2001, Pastor-Satorras and Vespignani [13] proposed a network SIS model to be account for long-lasting viruses in the computer networks. They found the absence of an epidemic threshold in the limit of large population size, which is in direct contrast to that of classical compartmental epidemic models. Later on, Moreno et al. [14] extended the network SIS framework in Ref. [13] and considered the following heterogeneous network SIR model

$$\begin{cases} S_k' = -\beta k S_k \Theta, \\ I_k' = \beta k S_k \Theta - \gamma I_k, \\ R_k' = \gamma I_k, \end{cases} \tag{2}$$

where S_k, I_k and R_k are the number of susceptible, infected and recovered individuals with degree k, respectively, and β is the per edge transmission rate between a susceptible node and an infected node. The term Θ denotes the probability that any randomly chosen edge points to an infected node. For uncorrelated networks, the probability that an edge points to a node of degree k is proportional to $kP(k)$ [13,14], where $P(k)$ is the degree distribution of the network. Thus, the probability that a randomly chosen edge connects to an infected node is

$$\Theta = \frac{\sum_k kI_k}{\sum_k kN_k} = \frac{\sum_k kP(k)i_k}{\langle k \rangle}, \tag{3}$$