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How heterogeneous susceptibility and recovery rates affect the spread of epidemics on networks

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ABSTRACT

In this paper, an extended heterogeneous SIR model is proposed, which generalizes the heterogeneous mean-field theory. Different from the traditional heterogeneous mean-field model only taking into account the heterogeneity of degree, our model considers not only the heterogeneity of degree but also the heterogeneity of susceptibility and recovery rates. Then, we analytically study the basic reproductive number and the final epidemic size. Combining with numerical simulations, it is found that the basic reproductive number depends on the mean of distributions of susceptibility and disease course when both of them are independent. If the mean of these two distributions is identical, increasing the variance of susceptibility may block the spread of epidemics, while the corresponding increase in the variance of disease course has little effect on the final epidemic size. It is also shown that positive correlations between individual susceptibility, course of disease and the square of degree make the population more vulnerable to epidemic and avail to the epidemic prevalence, whereas the negative correlations make the population less vulnerable and impede the epidemic prevalence.

1. Introduction

The method of establishing the network-based models, which incorporates the contact patterns among people as static or dynamic networks into epidemiology modeling, has become an essentially important research method to study the role of nodes standing for individuals or edges denoting the interaction between individuals in the spreading process (Dorogovtsev, Goltsev, & Mendes, 2008; Jin, Sun, & Zhu, 2014; Luo, Chang, & Jin, 2017; Pastor-Satorras, Castellano, Van Mieghem, & Vespignani, 2015). The frequently investigated and most classical epidemiological models as the basic conceptual tools in understanding the epidemic spreading and the related effective strategies for epidemic controlling are the SIS model and the SIR model. As is well known that the most valuable result in the standard SIS model (Boguná & Pastor-Satorras, 2002; Boguná, Pastor-Satorras, & Vespignani, 2003; Luo, Zhang, Sun, & Jin, 2014; Pastor-Satorras & Vespignani, 2001) or SIR (May & Lloyd, 2001; Moreno, Pastor-Satorras, & Vespignani, 2002; Newman, 2002) model is that the basic reproductive numbers of an

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infection R_0 is always larger than 1 for the scale-free networks in the thermodynamic limit, which implies that the heterogeneity of node's degree can reduce or even eliminate the existence of an epidemic threshold.

In most epidemiological models (Anderson, May, & Anderson, 1992; Jin et al., 2011; Keeling & Rohani, 2008; Li, Sun, Wu, Zhang, & Jin, 2014, 2017; Peng, Xu, Small, Fu, & Jin, 2016), all individuals are assumed to be homogeneous, e.g., all susceptible individuals acquire the disease with the same probability whenever in contact with an infected individual, and all infected individuals recover, or go back to being susceptible, with the same rate. Such consideration is, however, far from the actual situation. The individual variations of infectivity (Lloyd Smith et al., 2005; Riley et al., 2003), susceptibility (Boon et al., 2011; Hardie et al., 2008; Li, Cao, & Cao, 2010; Zhang, Xie, Tang, & Lai, 2014; Zhang, Xie, Chen, Liu, & Small, 2016) and recovery rates or infectious period are as widespread as network heterogeneity. These internal properties of individuals varying across a population result from genetic (Segal & Hill, 2003) and immunogenetic (Fryer et al., 2010) factors, differences in age, previous disease history, history of drug abuse, or differences in healthcare quality and can exert a non-negligible influence on the epidemic spreading process.

Recently, several authors have applied the percolation theory to explore the effects of individual heterogeneity in the context of network epidemics (Kenah & Robins, 2007; Miller, 2007, 2009; Neri, Pérez Reche, Taraskin, & Gilligan, 2010; Neri et al., 2011). For instance, Kenah and Robins (Kenah & Robins, 2007) showed that the bond percolation model failed to predict the correct outbreak size distribution and probability of an epidemic when there was a nondegenerate infectious period distribution. Miller (Miller, 2007) showed that an epidemic was most likely if infectivity was homogeneous and least likely if the variance of infectivity was maximized. Similarly, the attach rate was largest if susceptibility was homogeneous and smallest if the variance was maximized. Later, Miller (Miller, 2009) showed that heterogeneity infectiousness was the dominant factor controlling the probability of an epidemic and heterogeneity in susceptibility was the dominant factor controlling the size of an epidemic.

There are some other works to study the effects of individual heterogeneity on the epidemic spreading process on networks. Karrer and Newman (Karrer & Newman, 2010) introduced the message passing approach to study a generalized SIR model that allowed for arbitrary distribution of transmission and recovery times. Using the message passing approach, Sherborne et al. (Sherborne et al., 1611) derived a new pairwise-like model for epidemics with Markovian transmission and arbitrary recovery period; and they also presented a novel extension of the edge-based compartmental model for epidemics with arbitrary distributions of transmission and recovery times. Li et al. (Li, Liu, Kim, Min, & Zhang, 2010) studied the network epidemic dynamics with both individual mobility and heterogeneity and showed that the heterogeneity of individual susceptibility and infectivity increased the epidemic threshold, and the positive correlation of individual susceptibility and infectivity availed to the epidemic prevalence. Wu et al. (Wu & Zhang, 2016) investigated the epidemic spreading on random and regular networks through a pairwise-type model to evaluate the influence of individual infectivity and susceptibility which were functions of individual activity. Smilkove et al. (Smilkov, Hidalgo, & Kocarev, 2014) found that heterogeneous susceptibility can make networks more vulnerable to the spread of epidemics if the correlation between a node's degree and susceptibility were positive. Yang et al., (Yang, Tang, & Gross, 2015) using the pairwise approximation method, showed that these correlations naturally arised in the adaptive network through considering a plausible scenario where people had intrinsic differences in susceptibility and adapted their social network structure to the presence of the disease. Abbas et al. (Abbas, Bhatia, Vorobeychik, & Koutsoukos, 2014) incorporated node properties into a node-based SIRS model for infection propagation and proposed new heuristics to curb the spread of infection in heterogeneous networks.

Although the studies mentioned above can explain how the individual heterogeneity influences the epidemic spreading process on networks to some extent, this complex phenomenon is still poorly understood and some questions remain open. Especially, there is less work to explore the effect of the heterogeneity of individual susceptibility and recovery rates at the same time on the dynamic behaviors of epidemics spreading on networks. And this invites us to follow the precious works and investigate how the heterogeneous susceptibility and recovery rates of individuals, combining with the network heterogeneity, influence the epidemic spreading process. We are interested in using or generalizing the heterogeneous mean-field theory to address this problem. Meantime, the incorporation of differential susceptibility and recovery rates into epidemic models does introduce a new dimension to epidemic modeling, since there are multiple ways for individuals with differences in susceptibility and recovery rates to be arranged in a network. Therefore, we should consider not only variations in the susceptibility and recovery rates of individuals, but also the correlations between individual susceptibility, recovery rates and connectivity to do a complete study.

In this paper, we establish an extended heterogeneous SIR epidemic model defined on networks with arbitrary network topology and analytically study the basic reproductive number R_0 and the final epidemic size $R(\infty)$ for this model. Our findings show that the spread of epidemics is closely correlated to the structure of population in terms of individual susceptibility and course of disease. When individual degree, susceptibility and recovery rate are independent, R_0 and $R(\infty)$ are increasing with the increment of the average susceptibility and the average course of disease of the entire networks. And if averages of susceptibility and disease course are identical, increasing the variance of susceptibility may block the spread of epidemics, while increasing the variance of disease course has little effect on the final epidemic size. Focusing on the individual level correlations between the two of the susceptibility, the course of disease, and the square of the degree or connectivity of individuals, we show that positive correlations between them make the network more vulnerable to epidemics (increasing R_0) and avail to the epidemic prevalence (increasing $R(\infty)$); whereas negative correlations make the network less vulnerable (decreasing R_0) and impede the epidemic prevalence (decreasing $R(\infty)$).