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Research Article

SEIR Epidemic Dynamics in Random Networks

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Predicting disease transmission on complex networks has attracted considerable recent attention in the epidemiology community. In this paper, we develop a low-dimensional system of nonlinear ordinary differential equations to model the susceptible-exposed-infectious-recovered (SEIR) epidemics on random network with arbitrary degree distributions. Both the final size of epidemics and the time-dependent behaviors are derived within our simple framework. The underlying network is represented by the configuration model, which appropriately accounts for the heterogeneity and finiteness of the degree observed in a variety of real contact networks. Moreover, a generalized model where the infectious state of individual can be skipped is treated in brief.

1. Introduction

Infectious diseases spread over networks of contacts between susceptible and infectious individuals. Typical mathematical representation of an epidemic assumes that the host populations are fully mixed (mass-action approximation) [1, 2], that is, every individual has an equal opportunity to infect others and the underlying network topology is modelled as a fully connected graph. However, in the real world, the contact patterns are characterized by high levels of heterogeneity and each individual only has contact with a small fraction of the population [3–5].

In recent years, a number of researches have addressed the contact patterns among individuals as random networks [6–14], which allow for more realistic and accurate capture of heterogeneities in the number of contacts compared with classical fully mixed models. Network epidemic models make use of network topology of potential contacts instead of assuming that contact is possible with the total population. Some quantities of interest such as epidemic probability and mean final size of epidemics have been precisely solved in random networks with specified degree distributions (configuration models) using ideas drawn from percolation theory [9, 10, 15].

The heterogeneity introduced in the network framework, nevertheless, makes it rather difficult to analytically describe the time-dependent properties and the dynamical course of an epidemic. Some researchers made it by using high-dimensional pair-approximation methods (or moment closure methods) [4, 16, 17], which typically neglect the correlations between the states of nodes some steps away from each other, while others adopted approximate approaches that assume all nodes of the same degree having the same infection probability at any given time [3, 18, 19]. In addition, a good deal of effort has been devoted to simulation-based studies of epidemic dynamics [20–22].

Recently, Volz [23] and Miller [24] manage to introduce a low-dimensional system of nonlinear ordinary differential equations to model susceptible-infected-recovered (SIR) epidemics on random networks assuming infection and recovery occur at constant rates. A variant SIR model is also developed in [25]. Their calculations account for the effects induced by heterogeneous connectivity and finiteness of degree that are missed in standard well-mixed SIR equations. In contrast to the prior moment closure methods, the number of equations in the resulting system does not grow with the number of different degrees.

In the present paper, we move a further step beyond this framework by considering more complex susceptible-exposed-infectious-recovered (SEIR) epidemics in random networks, where an exposed period exists during which the individual has been infected but cannot transmit infection. We show that it is possible to analyze the dynamics of SEIR epidemics spread on configuration models [11] using a

coupled system of only three ordinary differential equations. The epidemic growth at any given time as well as its final size are investigated in this relatively simple framework, which is less computationally demanding and amenable to the analytical derivations. We also consider a situation where a host can be recovered directly after it is exposed. If a disease, for example, is detected and treated in the exposed status, no secondary infection will occur. Still, we will see that three differential equations suffice in this scenario.

The rest of the paper is organized as follows. In Section 2, we develop the theoretical framework and present some preliminaries. The network SEIR dynamics and its generation are then developed in Sections 3 and 4, respectively. Finally, we discuss the applicability and limitations in Section 5 with several open problems.

2. Definitions and Notations

Let the population of interest consist of n individuals represented by a network with n nodes. The population is modeled by the configuration model [11], in which the degree distribution is specified, but the graph is in other respects random. To define a configuration model network, one specifies the degree distribution by giving the properly normalized probability p_k that a randomly chosen node has degree k. To each node v assign an i.i.d. degree d_v drawn from the distribution p_k. If the sum of degrees is odd, all degrees are reassigned until the sum is even. Then generate a set X of half-edges with d_v copies of node v for all nodes. A pair of these stubs v_1, v_2 is then chosen uniformly at random and connected together to form a complete edge while X is not empty. This procedure generates a uniform choice from the set of all networks with the specified degree distribution. The resulting network has negligible loops and multiple edges in the limit of large network size n for degree distributions with finite mean [11].

The probability generating function [26] of the degree distribution p_k is defined as

$$G(x) = \sum_{k=0}^{\infty} p_k x^k, \tag{1}$$

where the dummy variable x serves as a placeholder. The mean degree of the network is then given by $\langle k \rangle = G'(1)$.

Nodes in the network fall into one of four exclusive states: susceptible, exposed, infectious, or recovered. In many infectious diseases, there is a period of time after the transmission of infection from susceptibles to potentially infective members but before these potential infectives develop symptoms and become infectious. This latent period is usually called exposed one [27]. We denote the fraction of the population in each state at time t by $S = S(t)$, $E = E(t)$, $I = I(t)$, and $R = R(t)$, respectively.

The dynamics of the disease propagation can be described as follows. An infectious node transmit infection to each of its neighbors independently at a constant rate β. A susceptible node becomes infected and hence assigned to the exposed state, at rate $k\beta$ where k is the number of infectious neighbors it has. Exposed nodes become infectious at a constant rate α. Once infectious, a node recovers (becomes immune) at a constant rate γ, whereupon it will never infect any neighbors. By definition, we have $S + E + I + R = 1$. A flow chart is shown in Figure 1.

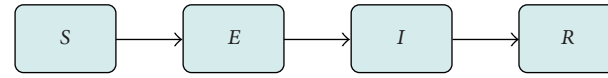

Figure 1: Flow chart for the SEIR model.

Similarly, as in [23–25], we define an "infectious contact" from an infected node u to its neighbor v to be a contact that would cause infection of v if v were susceptible. Now, we choose a node v in the network uniformly at random and modify the spread of the disease by disallowing infectious contacts from v to its neighbors. Denote a neighbor of v by u. Let $\theta = \theta(t)$ be the probability that there has not been infectious contact from u to v at time t. It is noteworthy that, by doing so, disease transmission along different edges to node v is independent. Moreover, disallowing infection originated from v *does not* modify the probability that v has become infected (more precisely, exposed), although it *does* influence the dynamics after v is infected (more precisely, infectious). Hence, if v has k neighbors (i.e., $d_v = k$), then the probability that v is still susceptible at time t is $\theta(t)^k$ [24]. In what follows, we refer to u as a *base* node while v as a *target* node. θ will be a critical quantity in our latter derivation.

3. Network SEIR Dynamics

In the limit as population size n goes to infinity, the epidemic spread can be viewed effectively as a deterministic behavior in terms of expected fractions $(S, I, \ldots)$ of the entire population size [10]. In this section, we aim to derive a low-dimensional system of ordinary differential equations to characterize exactly the epidemic dynamics.

The fraction of the population that has not yet been exposed (i.e., still susceptible) at time t is $S(t)$, which can be calculated as

$$S(t) = \sum_{k=0}^{\infty} p_k \theta(t)^k = G(\theta(t)), \tag{2}$$

by using (1) and the comments in the Section 2.

To derive the dynamics of θ, we need to introduce two augmented variables. Let $\phi = \phi(t)$ be the probability that the base node u of an edge from u to v is exposed, and the edge has not transmitted an infectious contact at time t. Similarly, let $\psi = \psi(t)$ be the probability that the base node of an edge is infectious, but the edge has not transmitted an infectious contact at time t. Note that those edges which satisfy the definitions for ϕ or ψ are subsets of those which satisfy the definition for θ.

Since the rate of change in the probability, a random edge that has not transmitted infection is equal to the rate at which infection crosses edges, we have

$$\dot{\theta} = -\beta\psi. \tag{3}$$

An edge from u to v begins to satisfy the definition of ϕ if the base node u becomes exposed. The rate at which neighbors of target node v become exposed matches the rate at which neighbors stop being susceptible. On the other hand, an edge no longer satisfies the definition for ϕ when infection crosses the edge or when the base node u becomes infectious. Set $f = f(t)$ to be the probability that a neighbor u is susceptible. Hence, we obtain

$$\dot{\phi} = -\alpha\phi - \beta\phi - \dot{f}. \tag{4}$$

Likewise, an edge from u to v begins to satisfy the definition of ψ if the base node u becomes infectious. An edge no longer satisfies the definition for ψ when infection crosses the edge or when the base node u recovers. Recall that only infectious state can transmit disease. Then we have

$$\dot{\psi} = -\gamma\psi - \beta\phi - \dot{f}. \tag{5}$$

Now we need to calculate f. The probability that a neighbor u reached following a randomly chosen edge has degree k is expressed by the *excess degree* distribution $kp_k/\langle k\rangle$ [15]. By our assumption, the neighbor u can only be infected by an edge other than the one starting from the target node v. Therefore,

$$f(t) = \frac{\sum_{k=0}^{\infty} kp_k\theta^{k-1}}{\langle k\rangle} = \frac{G'(\theta)}{G'(1)}, \tag{6}$$

by virtue of Definition (1). Thus, we obtain

$$\dot{f} = \frac{G''(\theta)\dot{\theta}}{G'(1)} = -\frac{G''(\theta)\beta\psi}{G'(1)}, \tag{7}$$

using (3). Substituting (7) into (4) and (5), we have

$$\dot{\phi} = -(\alpha+\beta)\phi + \frac{G''(\theta)\beta}{G'(1)}\psi, \tag{8}$$

$$\dot{\psi} = -\beta\phi + \left(\frac{G''(\theta)\beta}{G'(1)} - \gamma\right)\psi. \tag{9}$$

Finally, we can calculate the fraction of susceptible nodes $S(t) = G(\theta(t))$ directly by solving the coupled system of (3), (8), and (9). Furthermore, the values of E, I, and R can be derived in light of

$$\dot{R} = \gamma I, \tag{10}$$

$$\dot{E} = \beta SI - \alpha E, \tag{11}$$

and the appropriate normalization $I = 1 - S - R - E$. The complete system of equations is summarized in Table 1.

Note that we can reproduce the network SIR dynamics by letting α go to infinity. In fact, by definition $|\phi - \psi|$ tends to 0 as α approaches infinity. Hence, (8) breaks down while (9) asymptotically becomes

$$\dot{\psi} = -\beta\psi + \left(\frac{G''(\theta)\beta}{G'(1)} - \gamma\right)\psi, \tag{12}$$

which is equivalent to the dynamics describe by [24, equation (3)] for the SIR model.

Table 1: System of equations for network SEIR epidemics.

$\dot{\theta} = -\beta\psi$
$\dot{\phi} = -(\alpha+\beta)\phi + (G''(\theta)\beta/G'(1))\psi$
$\dot{\psi} = -\beta\phi - \gamma\psi + (G''(\theta)\beta/G'(1))\psi$
$S = G(\theta)$
$\dot{R} = \gamma I$
$\dot{E} = \beta SI - \alpha E$

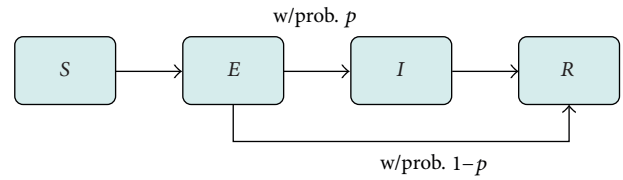

Figure 2: Flow chart for the generalized SEIR model.

3.1. Final Epidemic Size. The expected final size of network SEIR epidemics in the limit of infinite networks can be derived easily within our framework. By letting $\dot{R} = 0$, or equivalently $I = 0$, we arrive at $1 = S + R + E$. It follows from (11) that $\dot{E} = -\alpha E$, which yields $E(\infty) = 0$. Consequently, the final size of an epidemic is simply given by

$$R(\infty) = 1 - S(\infty) = 1 - G(\theta(\infty)). \tag{13}$$

3.2. Initial Conditions. In order to solve our equations, we need to find initial conditions. The initial conditions for the model can be chosen in many ways, but the most typical is to assume that a single node or a small fraction ε_1 of nodes in the network are selected at random and initially infected.

The quantity θ can be viewed as the fraction of nodes remaining susceptible. We then have $\theta(0) = 1 - \varepsilon_1$ with $\varepsilon_1 \ll 1$. The initial values of ϕ and ψ can be set to $\phi(0) = \varepsilon_2$ and $\psi(0) = \varepsilon_3$, respectively, with $\varepsilon_2, \varepsilon_3 \ll 1$ in the limit of large population size $n \to \infty$.

4. A Generalization

In this section, we consider a generalization of the SEIR model discussed above. A disease may not become infectious if treated in time once an individual is exposed. Hence, we denote by p a probability that an exposed node will become infectious (at a rate α). With probability $1 - p$, an exposed node will recover (at a rate γ). The corresponding flow chart can be shown as Figure 2.

Although most of the derivation in Section 3 still applies, we need to make some modifications to incorporate the new situation. Equation (4) should be replaced by

$$\dot{\phi} = -\alpha p\phi - \beta\phi - \dot{f}, \tag{14}$$

since only a fraction p of exposed nodes will develop to infectious period. Similarly, (10) and (11) will be reformulated as

$$\begin{aligned} \dot{R} &= \gamma I + (1-p)E\gamma, \\ \dot{E} &= \beta SI - \alpha pE - (1-p)\gamma R, \end{aligned} \tag{15}$$

respectively. It is clear that the system reproduces the SEIR model when $p = 1$.

5. Discussion

In this paper, we proposed a low-dimensional system of nonlinear ordinary equations to model SEIR epidemics in random networks. The calculations for the dynamic time-dependent behavior as well as the final size of the epidemic are placed in a common framework extending the prior work [23–25] on SIR epidemic models. A modification of the SEIR model where the state I may be skipped is also addressed.

The network used in the present study is a static configuration model. It would be highly desirable to extend the static random networks to dynamic ones [28]. Future research could be enhanced by invoking time-varying rates of infection and recovery, and more elaborated (realistic) models of epidemics may be considered. For example, epidemic spreading on random clustered networks are explored in [29]. Optimal strategies for various applications such as cyber security [30] and vaccination [31] are also valuable. Validation in a real-world setting is needed to establish the statistical models so that it can be in fact used to predict disease transmission.

Immunization strategies may also be taken into account. In [32], the authors proposed a distributive immunization where a recovered node can create an immunization agent with some given probabilities. The agent then spreads to all neighbors and immunizes the susceptible ones among them. Therefore, its dynamics can be viewed as a competition between two types of diffusion processes on a network: one transmits disease while the other transmits immune. It is hoped that the methodology described in the paper can be helpful in capturing this distributive immunization mechanism.

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