



## Research paper

## Edge-based SEIR dynamics with or without infectious force in latent period on random networks

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## ABSTRACT

In nature, most of the diseases have latent periods, and most of the networks look as if they were spun randomly at the first glance. Hence, we consider SEIR dynamics with or without infectious force in latent period on random networks with arbitrary degree distributions. Both of these models are governed by intrinsically three dimensional nonlinear systems of ordinary differential equations, which are the same as classical SEIR models. The basic reproduction numbers and the final size formulae are explicitly derived. Predictions of the models agree well with the large-scale stochastic SEIR simulations on contact networks. In particular, for SEIR model without infectious force in latent period, although the length of latent period has no effect on the basic reproduction number and the final epidemic size, it affects the arrival time of the peak and the peak size; while for SEIR model with infectious force in latent period it also affects the basic reproduction number and the final epidemic size. These accurate model predictions, may provide guidance for the control of network infectious diseases with latent periods.

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## 1. Introduction

The idea of mathematical methods to epidemiology was pioneered by Daniel Bernoulli [1], who focused on the effect of variolation against smallpox to prolong life expectancy. This topic, termed by mathematical epidemiology (ME), didn't have much achievement in the late eighteenth and nineteenth centuries until the early twentieth century. In 1911, Sir Ross developed a mathematical model to capture the spread of malaria, a disease which is transmitted via mosquitoes, and proposed a central concept "threshold effect" in epidemiology, highlighting that the malaria in a region could be eliminated through reducing the mosquito population [2]. This idea was further formalized and generalized to a large class of diseases by Kermack and McKendrick [3] in 1927, known as the basic reproduction number or basic reproductive ratio and usually denoted as  $R_0$  in epidemiology, which quantifies the expected number of secondary cases generated by a typical infected individual during its entire infectious period while introduced to a fully susceptible population [4]. Since then, numerous

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mathematical models have been proposed to study the spread and control of infectious diseases, using  $R_0$  as an important indicator, see [5] and [6], for example.

Two fundamental models commonly used in mathematical epidemiology are the so called susceptible-infected-recovered (SIR) and susceptible-infected-susceptible (SIS) models. For SIR models, susceptible individuals become infectious immediately once infected, and the infected individuals become fully immune once recovered (here, we use R to denote the recovered individuals; however, some authors also use R to denote the removal individuals that are either through the isolation or through death caused by the disease. In both cases, the recovered or removal individuals play no role in transmitting infection, and thus they are equivalent from a modeling point of view); while for SIS models, the infected individuals become fully susceptible that can be re-infected again. Usually, SIR models are appropriate for diseases such as influenza and measles caused by a virus, while SIS models are appropriate for diseases such as syphilis and gonorrhoea caused by a bacteria [7,8].

For the SIR model without demographic, if  $R_0 < 1$ , the infection decays directly and there is no epidemic prevalence; otherwise, the infection goes up at first as individuals get infected, then down again as they recover, and there is an outbreak. The whole process ends with no infection in the limit of large time  $t$  [7,8]. In addition, the final epidemic size, i.e. the total people affected by the disease in the end, is given by an implicit equation [3].

It should be mentioned that the classical SIR/SIS model implicitly assumes that the population is homogeneously mixed, i.e. every individual in the population has the same probability of contacting any other individual. This is obvious not the case in the real world. In fact, different individual may have varying number of contacts. This idea was initiated by Diekmann et al.[9]. Although Diekmann et al.[9] mainly considered the homogeneous population in which each individual had exactly the same number of  $k$  acquaintances, they pointed out in the concluding remarks that individuals might have different number of acquaintances, the situation pioneered by Pastor-Satorras and Vespignani [10] on epidemics in heterogeneous networks.

Indeed, contact network models are more realistic to describe the population mixing pattern. Under such framework, each individual of the population is denoted by a node, and possible contacts between two individuals are linked by an edge between the corresponding two nodes of the network; these two nodes are neighbors of each other. In [11], Moreno et al. considered the SIR model in complex heterogeneous networks, and obtained the following basic reproduction number

$$R_0 = \frac{\beta \langle k^2 \rangle}{\gamma \langle k \rangle}, \quad (1)$$

where  $\beta$  is the transmission rate along per edge,  $\gamma$  is the constant recovery rate of infected individuals,  $\langle k \rangle = \sum_k kP(k)$  is the average degree of the network, and  $\langle k^2 \rangle = \sum_k k^2P(k)$  is the second moment of the degree distribution  $P(k)$ . Moreover, they obtained the final size equations on various networks.

Network SIR models have been further studied by several authors in the literature. For example, by using a combination of bond percolation and generating function methods, Newman [12] derived the epidemic threshold and the final epidemic size. The basic reproduction number in [12] is defined as

$$R_0 = \frac{\beta}{\beta + \gamma} \frac{\langle k(k-1) \rangle}{\langle k \rangle} = \frac{\beta}{\beta + \gamma} \left( \langle k \rangle - 1 + \frac{\text{Var}[k]}{\langle k \rangle} \right), \quad (2)$$

where  $\text{Var}[k]$  denotes the variance of network degree distribution. The first factor of (2) describes the fact that an infected node spreads out the infection before it recovers, and the second factor is the average excess degree of the network. However, different from a dynamical model, one limitation of this mapping method is that it can not give us the dynamical evolution of a disease. Lindquist et al. [13] introduced effective degree SIR and SIS models on a contact network, and obtained the same basic reproduction number  $R_0$  for network SIR model as (2).

Considering infection across the edge, Keeling [14] proposed an edge-based SIR model on homogeneous networks, which leads to a basic reproduction number different from (2) since it takes the multiplicative correlation between connected nodes of various types into account. This formalism is further extended to SIR model on heterogeneous networks by adopting all combinations of degree pairs as variables [15], but it is very difficult to analyze (in fact, the dimension for the differential equations is of quadratic order of maximum degree of the network). Recently, using the probability generating function (PGF) method, Volz [16] considered SIR epidemics on random networks, and derived a system of three nonlinear ordinary differential equations (ODEs), the predictions of which agrees excellent well with the stochastic SIR simulations. By slightly modifying the definition of parameters, Miller [17] further showed that an SIR model on random networks was intrinsically two dimensional, which is consistent with the classical homogeneously mixed model. Both the models of Volz [16] and Miller [17] yield the same basic reproduction number as (2). There are lots of extensions using this edge-based compartmental modeling approach, see, e.g., [18–22].

However, in reality, many diseases have latent or incubation periods, i.e. individuals first entering an exposed class E and having no apparent symptoms, the length of which varies from disease to disease. For example, measles has a 8–13-day latent period, influenza has only a 1–3-day latent period, and Acquired Immune Deficiency Syndrome (AIDS) has the incubation time ranging from a few months to years after the patient has been shown to get antibodies to the human immunodeficiency virus (HIV) [23]. One can incorporate this effect by adding a new class E to the classical SIR model. During the latent period, the susceptible once infected remains no symptom for a certain length of time before entering the infective class I, and the individuals may or may not have infectious force depending on the particular disease under

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