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# Stochastic epidemic models: New behavioral indicators of the disease spreading

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ABSTRACT

The purpose of this paper is to propose new indicators of the dynamics of infectious disease spread in stochastic epidemic models, including both global system-oriented descriptors (e.g. the final size measured as the number of individuals infected on a least one occasion during an outbreak) and individual-oriented descriptors (e.g. the time to reach an individual run of infections). We focus on birth-and-death models and the basic *SIR* epidemic model but the methodology remains valid for other nonlinear stochastic epidemic models. The theory is illustrated by numerical experiments which demonstrate that the proposed behavioral indicators can be applied efficiently.

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## 1. Introduction

Epidemic models are widely used for understanding the mechanism that underlies the spread of an infectious disease. Roughly speaking, there are two main mathematical approaches: deterministic models based on differential equations and stochastic models that employ Markov chains, branching and diffusion processes. Early literature for studying epidemiological models was largely deterministic. However, when population sizes are small, it is now accepted that a stochastic model is more realistic [1]. This is the case of small communities sharing confined spaces such as intensive care units in hospitals, schools, prisons, small herds or local area networks of moderate size [2–5]. Even when the population size increases and the stochastic analysis becomes more difficult, the random nature of the disease dynamic requires to be taken into account.

One of the most important differences between deterministic and stochastic epidemic models is related to the role played by the reproduction ratio, $R_0$. The reproduction ratio, $R_0$, is probably the most important quantity in epidemiology. The classical biological interpretation presents $R_0$ as the expected number of secondary cases produced by one individual during the period of infectivity, when the individual is introduced into a completely susceptible population. From the deterministic perspective, it is well-known the threshold value of $R_0$ that establishes that an infection persists if $R_0 > 1$ (endemic equilibrium), while the model reaches the disease-free equilibrium (extinction) if $R_0 \leqslant 1$. In contrast, if we deal with a stochastic model with a finite population, the epidemic extinction is certain (with probability 1) regardless of the magnitude of $R_0$. However, the utility of $R_0$ to quantify the spread and severity of the stochastic epidemic still holds. For example, in a susceptible-infective-susceptible (SIS) stochastic epidemic the parameter region where the time to extinction is short can be identified by small values of $R_0$. In contrast, if $R_0$ is large, then the epidemic tends to persist for a very long time, so a state

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of quasi-stationary equilibrium may be reached before a random fluctuation leads to the extinction of the epidemic. For further comparison between deterministic and stochastic models, we refer to the literature [1,6–8].

There is no reason to see a conflict between the two approaches. The advantage of the deterministic model lies in its generally simpler analysis. However, Andersson and Britton [1] conclude that stochastic models are to be preferred when their analysis is possible. To this end, the stochastic model so often contains several simplifying assumptions (e.g. exponentiality, homogeneity, randomly-mixing), and thus it is not entirely realistic. A compromise between mathematical tractability and realistic assumptions is always a premise.

In the present work, we are concerned with stochastic epidemic models that employ continuous time Markov chains (*CTMC*). More concretely, we describe the dynamics of the epidemic in terms of birth-and-death processes and the susceptible-infective-removed (*SIR*) stochastic model. Despite of the limitations assumed, this framework allows a detailed mathematical elaboration. We compute not only the expected value of the epidemic descriptors, but also their whole probability distribution (i.e., density function or probability mass function). Moreover, our mathematical results are well suited for numerical implementation. We show how algebraic computations of the epidemic indicators can be reduced to deal with iterative stable schemes.

Among the classical indicators of the spread, severity and persistence of the infectious disease, we mention the final size of the epidemic, the time to extinction and the quasi-stationary distribution [1,6–9]. The goal of this paper is to make progress by investigating other indicators of interest related both to global system-oriented characteristics and to individual-oriented descriptors of the infectious disease. Most of the indicators studied in the sequel are completely new, except the number of infections per individual which was considered in [5].

More specifically, in the case of epidemic models of birth-and-death type, we consider the following descriptors: (i) the time to reach an individual run of infections, (ii) the final size, and (iii) the number of infections per individual; while in the case of the stochastic *SIR* model the attention is focused in: (iv) the time to reach a specific state, and (v) the time to reach a critical number of infections.

In order to complete the description of the literature, we now mention a selection of related epidemiological stochastic models based on *CTMCs* [4,5,10–15], some recent papers investigating indicators of the transmissibility in *SIS* and *SIR* stochastic models [16,17], as well as some works of the authors where the main epidemic indicators were revisited [2,3,18].

The dimensionality of the underlying *CTMC* determines the order of presentation of the results, so we start presenting our findings for the unidimensional birth-and-death processes (Section 2) and the *SIS* stochastic epidemic, which is subsumed under the birth-and-death formalism as a special case. However, we remark that the bidimensional *SIR* model (Section 3) has a simpler algebraic structure, which makes the technical calculations easier. Theoretical results in Sections 2 and 3 provide the paper contribution to the development of a methodology. Our numerical results and discussion on their implications are presented in Section 4. Along the numerical experiments we perform a sensitivity analysis to determine how the system parameters will impact the new indicators. Finally, concluding remarks are given in Section 5.

## 2. Birth-and-death processes

We consider a closed population of size $N$, where each individual is classified as either a susceptible or an infective. Let $S(t)$ and $I(t)$ be the number of susceptibles and infectives, respectively, at time $t$. Since $S(t)+I(t)=N$, the evolution of the epidemic is simply described by the process $\{I(t); t \geqslant 0\}$ with state space $S=\{0,\ldots,N\}$. The infection ends when $I(t)=0$. The birth rates, corresponding to infections, are denoted by $\lambda_i>0$, for $1 \leqslant i \leqslant N-1$, $(\lambda_0=\lambda_N=0)$ and the death rates, corresponding to recoveries, are denoted by $\mu_i>0$, for $1 \leqslant i \leqslant N$, $(\mu_0=0)$. The transitions among states are represented in Fig. 1. A selected sample of epidemiological stochastic models obtained as particular cases of the birth-and-death formalism includes the basic *SIS* epidemic model, the *SIS* model with external source of infection and the Verhulst model.

The *SIS* model assumes that a recovered individual does not acquire immunity but immediately becomes susceptible. Thus, the process $\{I(t); t \geqslant 0\}$ is usually modelled as a particular case of a birth-and-death process with an absorbing state 0. The birth and death rates are

$$\lambda_i = \frac{\beta}{N} i(N-i), \quad 0 \leqslant i \leqslant N,$$
$$\mu_i = \gamma i, \quad 0 \leqslant i \leqslant N,$$

where $\beta$ is the contact rate and $\gamma$ is the individual recovery rate.

In the model with a external source of infection the birth rate should be modified as $\lambda_i=(\xi+\beta i/N)(N-i)$, for $0 \leqslant i \leqslant N$, where $\xi>0$ denotes the rate associated with the external source of infection. We notice that $\lambda_0>0$. Another generalization

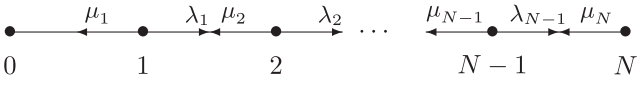


**Fig. 1.** States and transitions in the birth-and-death model.

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