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On the time to reach a critical number of infections in epidemic models with infective and susceptible immigrants

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ABSTRACT

In this paper we examine the time T to reach a critical number K_0 of infections during an outbreak in an epidemic model with infective and susceptible immigrants. The underlying process $\mathcal{X}$, which was first introduced by Ridler-Rowe (1967), is related to recurrent diseases and it appears to be analytically intractable. We present an approximating model inspired from the use of extreme values, and we derive formulae for the Laplace–Stieltjes transform of T and its moments, which are evaluated by using an iterative procedure. Numerical examples are presented to illustrate the effects of the contact and removal rates on the expected values of T and the threshold K_0, when the initial time instant corresponds to an invasion time. We also study the exact reproduction number $R_{exact,0}$ and the population transmission number R_p, which are random versions of the basic reproduction number $\mathcal{R}_0$.

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1. Introduction

The stochastic epidemic model with infective and susceptible immigrants, as introduced by Ridler-Rowe (1967), describes the dynamics of a homogeneously mixed population of individuals by means of a time-homogeneous continuous-time Markov chain (CTMC) $\mathcal{X} = \{X(t) = (M(t), N(t)) : t \geq 0\}$ defined on the state space $\mathcal{S} = \mathbb{N}_0 \times \mathbb{N}_0$ with $\mathbb{N}_0 = \{0, 1, 2, \ldots\}$, where $M(t)$ and $N(t)$ denote the numbers of susceptible individuals and infectives, respectively, at time t. The model accounts for four basic events: infection of a susceptible individual, immigration of a susceptible individual, immigration of an infective, and removal (or death) of an infective. Four strictly positive parameters are used at the outset, namely the *per capita* contact rate λ, the immigration rates ε and ν of infectives and susceptible individuals, and the removal rate per infective μ. For initial numbers of $m \in \mathbb{N}_0$ susceptible individuals and $n \in \mathbb{N}_0$ infectives, the hypotheses for the model (Fig. 1) are set out by specifying the non-null transition rates of $\mathcal{X}$ as follows:

$$q_{(m,n),(m',n')} = \begin{cases} \lambda mn, & \text{if } (m', n') = (m-1, n+1), \\ \varepsilon, & \text{if } (m', n') = (m, n+1), \\ \nu, & \text{if } (m', n') = (m+1, n), \\ \mu n, & \text{if } (m', n') = (m, n-1), \end{cases} \tag{1}$$

and $q_{(m,n)} = -q_{(m,n),(m,n)} = \lambda mn + \varepsilon + \nu + \mu n$. Note that, by Anderson (1991, Lemma 3.1 and 3.2), the process $\mathcal{X}$ is regular and recurrent positive since $\varepsilon + \nu > 0$.

Similarly to the Bartlett model (Bartlett, 1956; Reuter, 1961, Example 2), the model defined by (1) appears to be totally intractable, but various special cases have been extensively studied. The *general stochastic epidemic* (Bailey, 1975) is obtained by selecting $\varepsilon = \nu = 0$, which yields a finite state space since no immigration can occur. States of the form $(m, 0)$ with $m \in \mathbb{N}_0$ become absorbing states and, once the process $\mathcal{X}$ hits the n-axis, it executes a simple death process down it until it is absorbed at $(0, 0)$. One quantity which is analytically tractable and does supply some information about the general stochastic epidemic is the total size W of the epidemic. The exact distribution of W has been determined in several ways and by as many authors; for instance, a simple proof based on

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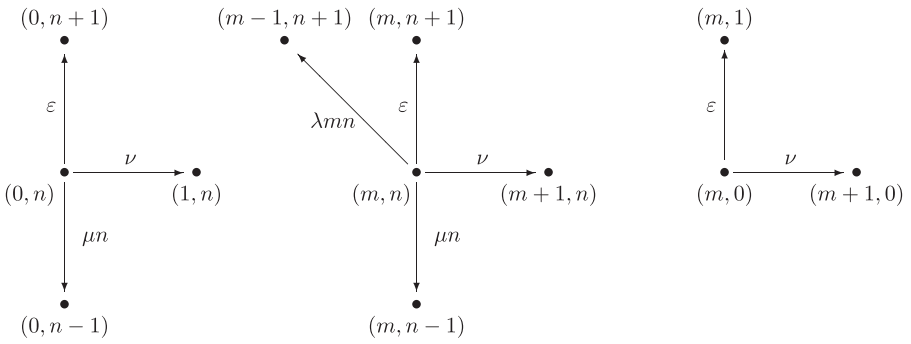

Fig. 1. Transitions among states in the epidemic model $\mathcal{X}$ with infective and susceptible immigrants.

the embedded jump chain can be found in Anderson (1991, Section 9.4), where the reader may also find two versions (Rajarshi, 1981) of the threshold theorem. In a recent work, Black and Ross (2015) develop a new method for computing the distribution of W, which is based on the replacement of the original numbers $X(t) = (M(t), N(t))$ of susceptible and infectives by a bivariate representation $Z(t) = (Z_1(t), Z_2(t))$ in terms of the numbers of infection and recovery events at time t. By using a co-lexicographic labeling of states for the resulting process $\{Z(t) : t \geq 0\}$, Black and Ross (2015) derive an iterative method in a variety of Markovian models, including the general stochastic epidemic, a variant with a phase-type infectious period distribution and a model with waning immunity.

For the process $\mathcal{X}$ defined in (1), Ridler-Rowe (1967) obtains certain limiting probabilities of the population size when immigration of both susceptibles and infectives into the population takes place; more concretely, it is shown in Ridler-Rowe (1967) that the long-term distribution of the process $\mathcal{X}$, as $\nu \to 0+$, tends to its counterpart for $\nu = 0$. A more important result (Ridler-Rowe, 1967, Theorem 1) states that, for initial numbers of m_0 susceptibles and n_0 infectives, the infectives almost surely die out at some time, with the mean of the time $\tau(m_0, n_0)$ at which this event first occurs satisfying

$$\tau(m_0, n_0) \sim \mu^{-1} \log(m_0 + n_0), \tag{2}$$

uniformly as $m_0 + n_0 \to \infty$ with $n_0 > 0$. We aim to complement the treatment of Ridler-Rowe (1967) by focusing here on the time T to reach a certain number K_0 of infections during an outbreak. The value K_0 may in a sense be regarded as a critical threshold at which, when a typical outbreak is in progress, the population starts to be saturated with the disease. In analyzing the distribution of T, we shall replace the bivariate process $\mathcal{X}$ by an *augmented* version $(\mathcal{X}, \mathcal{Y})$ allowing us to record the number of infections that take place during the outbreak. Our approach is based on the distribution of the maximum number of susceptible individuals in the population, and it is related to the use of extreme values in two-species competition processes (Gómez-Corral and López García, 2012a,b, 2015), where the maximum number of individuals alive at an arbitrary time is seen as an important measure in studying the effects of overpopulation in the ecosystem.

For recurrent diseases, the use of extreme values is equally applicable to other probabilistic descriptors, such as the exact reproduction number $R_{exact,0}$ and the population transmission number R_p; for convenience, we recall here that $R_{exact,0}$ is a random variable that counts the exact number of secondary cases produced by a focal infective during its entire infectious period, whereas R_p is defined as the exact number of secondary cases produced by all currently infective individuals prior to the first recovery. The exact reproduction number is first introduced and evaluated by Ross (2011) in the setting of SIS and SIR epidemics for a homogeneously mixed population of N individuals, including the cases of exponentially distributed infectious periods and a two-phase gamma infectious period distribution, and the random variables $R_{exact,0}$ and R_p are then presented by Artalejo and López-Herrero (2013) as two alternative measures that do not count repeated contacts that the basic reproduction number $\mathcal{R}_0$ overestimates. The measures $R_{exact,0}$ and R_p are applied by Economou et al. (2015, Section 3, Appendix D) to the spread of a respiratory disease and infections caused by nosocomial pathogens in intensive care units, where heterogeneous contacts are appropriately translated into a directed graph $\mathcal{G}$ and a 2^N-state Markov chain model.

Models for recurrent diseases usually treat the epidemic events of infection and removal of an infective in identical ways to standard epidemics, but they differ in their treatment of demographic forces. For example, in the Martini model (Anderson and May, 1991; Nåsell, 1999, Section 2), individuals are all subject to an immigration-death process, with the death-rate independent of the state of infection, and all newly immigrated individuals are susceptibles, so that the infection of susceptible individuals and the removal of an infective are treated in the same way as in the standard SIR model. The Bartlett model (Nåsell, 1999, Section 3) differs from the Martini model only in the sense that no deaths of individuals are assumed to occur; further work on the resulting model has been published by Pollett and Stewart (1994), Ridler-Rowe (1967), Stewart and Bebbington (1996), and Stirzaker (1975). The paper by Nåsell (1999) is a good summary on the Martini and Bartlett models, and it presents a review of Bartlett's work; specifically, the interest of Nåsell (1999) is in the problem of determining the time to extinction in recurrent diseases, which is proved to be a surprisingly difficult matter. The Martini and Bartlett models (Nåsell, 1999, Sections 2 and 3) omit factors that are known to be of importance in certain recurrent diseases, such as age-dependent exposure and seasonality transmission (Schenzle, 1984) and non-exponential distributional assumptions on waiting times (Keeling and Grenfell, 1997).

The paper is organized as follows. To begin with, we define in Section 2 the underlying Markov chain model, which is formulated as a reducible CTMC $(\mathcal{X}, \mathcal{Y})$ over a state space consisting of a single class of communicating transient states, and two sets of absorbing states that allow us to reflect the end of an outbreak and how the critical threshold K_0 of infections might be eventually reached during the outbreak. In Section 3, we characterize the distribution of T in terms of its Laplace–Stieltjes transform, which is seen to satisfy a system of linear equations that is not amenable to numerical implementation. Therefore, we adopt an approximating procedure that, for large enough numbers of susceptibles, examines the dynamics of the process $(\mathcal{X}, \mathcal{Y})$ till absorption. Numerical examples in Section 4 are presented to illustrate the effects of the contact rate λ and the removal rate μ on the expected values of T, as the initial time instant is an invasion time. We close with a few concluding remarks in Section 5. We summarize in Appendices A and B the analytical treatment of the random measures $R_{exact,0}$ and R_p of disease spread, and we present in Appendix C the distribution of

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