Stochastic epidemic models revisited: analysis of some continuous performance measures

J.R. Artalejo\textsuperscript{a}, A. Economou\textsuperscript{b} and M.J. Lopez-Herrero\textsuperscript{c}\textsuperscript{*}

\textsuperscript{a}Faculty of Mathematics, Complutense University of Madrid, 28040 Madrid, Spain; \\
\textsuperscript{b}Department of Mathematics, University of Athens, Panepistemiopolis, 15784 Athens, Greece; \\
\textsuperscript{c}School of Statistics, Complutense University of Madrid, 28040 Madrid, Spain

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We deal with stochastic epidemic models having a set of absorbing states. The aim of the paper is to study some continuous characteristics of the epidemic. In this sense, we first extend the classical study of the length of an outbreak by investigating the whole probability distribution of the extinction time via Laplace transforms. Moreover, we also study two almost new epidemic descriptors, namely, the time until a non-infected individual becomes infected and the time until the individual is removed from the infective group. The obtained results are illustrated by numerical examples including an application to a stochastic SIS model for head lice infections.

\textbf{Keywords:} extinction time; head lice; recovery time; stochastic SIS model; stochastic SIR model; time to infection

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1. Introduction

Understanding the mechanism that underlies the spread of an infectious disease can give important insights to help in the fight against the disease itself. Epidemic models are widely used for increasing the understanding of infectious disease dynamics and for determining preventive measures to control infection spread.

In the present work, we describe the dynamics of the epidemic in terms of a general birth–death model (including epidemic SIS models) and the stochastic SIR model. We suppose that a closed population is divided into susceptible, infective and, for SIR models, also removed individuals. The associated process describes the composition of the population and terminates when the number of infectives becomes zero, which almost surely happens within finite time. As a result, the stationary distribution is degenerate on the set of absorbing states. We refer the reader to the textbooks by Allen [2], Andersson and Britton [3] and Daley and Gani [12] in order to find the
mathematical background of these models as well as the main results and applications within a biological framework.

In this paper, we concentrate mainly on three continuous characteristics of this spread. The first characteristic, the extinction time, quantifies the spread of the epidemic on the whole population and describes the time till the end of the epidemic process. The other two characteristics concern the individuals’ behaviour. More concretely, we deal with the time till the infection and the recovery/removal time of a selected infected individual.

The extinction time has been the subject matter of many papers. First, we focus on the determination of the moments in finite birth–death processes. Norden [32] first obtained an explicit expression for the mean time to extinction, given an initial state. Then, he used the backward Kolmogorov equations to get an expression for higher moments involving the moments of one order less. In addition, the density function of the extinction time was approximated in terms of a gamma function. For an alternative proof of the moment formulae based on the use of the Laplace transform method, we refer to Goel and Richter-Dyn [16]. For the stochastic logistic epidemic, Kryscio and Lefèvre [20] obtained an approximation for the mean time to extinction based on the combination of several previous results. More recently, Newman et al. [31] derived explicit expressions for the mean and the variance of the extinction time for the special case of a single initial infective. Stone et al. [35], for an SIS model with external source of infection, dealt with the time to reach 0 infectives starting from a certain number of infected individuals and determined expressions for higher-order moments using the moment-generating function. The study of the moments can be extended to the infinite case; that is, the case where birth–death process takes values on \( \mathbb{N} \) [2,33].

The problem of determining the whole distribution of the extinction time (i.e. distribution function or density function) can be investigated using various methodologies including, for instance, spectral decompositions [19] and generating function methods [32]. At this point, we remark that this problem is much more involved than the calculation of the moments. There is no doubt about the theoretical value of the existing results, but their suitable computation is an intricate matter. Since the extinction time can be reduced to the transient analysis of the birth–death process, we next give a brief overview of some existing methods.

In the more general framework of a continuous-time Markov chain on a finite state space with rate matrix \( Q \), the unconditional version of the absorption time, \( L \), satisfies the following results [21,22]:

(R1) \[ P\{L \leq x\} = 1 - \alpha \exp\{Mx\}e, \text{ for } x \geq 0. \]

(R2) If \( M \) is invertible, \( L \) is finite with probability 1. Then, we have that \( \varphi(s) = E[e^{-sL}] = -\alpha(sI - M)^{-1}Me \) and \( E[L^k] = k!\alpha(-M^{-1})^k e, \text{ for } k \geq 1, \)

where \( M \) is the submatrix of \( Q \) corresponding to the set of the \( N \) transient states, \( I \) is the identity matrix of order \( N \), \( \alpha \) is a row vector of dimension \( N \) containing the initial probabilities and \( e \) is a column vector of dimension \( N \) with all entries equal to one.

Coming back to the context of the finite birth–death process, it is clear that the matrix \( M \) is an irreducible diagonally dominant matrix. Therefore, \( M \) is invertible and it is possible to analyse the behaviour of transforms and moments of the extinction time, given an initial state, in terms of the results for the unconditional absorption time, \( L \). Thus, Equation (R1) gives a closed-form solution for the problem under study. However, we readily notice that the computation of the previous formulae in Equations (R1) and (R2) requires to deal with powers and inverses of matrices having positive and negative entries, which is numerically unstable.

Finding accurate methods to compute the matrix exponential is a non-trivial matter which still attracts the interest of many investigators in numerical analysis. Following Moler and Van Loan [25], we remark that ‘the exponential of a matrix can be computed in many ways. In practice,
consideration of computational stability and efficiency indicates that some of the methods are preferable to others, but that none are completely satisfactory. One of the best methods is the scaling and squaring method implemented in MATLAB, which computes a Padé approximation to the matrix exponential [17]. Kulkarni [21] presented four methods for dealing with the transient analysis of a finite Markov chain. In addition to the exponential matrix, those methods also include differential equations, Laplace transforms and uniformization techniques. Some discussion is given, but it does not lead to a definite evidence in favour of one of the methods.

In this paper, we develop recursive schemes for Laplace transforms and appeal to numerical inversion methods. The numerical inversion of Laplace transforms is based on Fourier series methods. More concretely, our numerical results are obtained by employing the Post-Widder method [1,10]. A BASIC code implementing the algorithm can be found in Abate and Whitt [1]. Other methods for numerically inverting Laplace transforms are available in the literature. In fact, it is recommended to perform two different numerical inversion algorithms (e.g. Post-Widder method and Euler method) so that they can be used in parallel to determine the desired accuracy by agreement of the two methods. The possibility of performing the inversion using various alternative methods, as a checking mechanism, gives an initial motivation to use the Laplace transform method.

At this point, we mention that the Laplace transform provides a methodological approach not only for the computation of the distribution of the extinction time, but also for the study of the time to infection and recovery of a selected individual, as we will show in the sequel. However, the study of these two descriptors could also be reduced to an absorption time problem by introducing an auxiliary absorbing state. Transition into this state would mean that the marked individual gets infected (or he/she has recovered).

A detailed comparison among different methods of numerical computation is not our objective here, but a few general considerations are presented in what follows. The variety of numerical methods for computing the matrix exponential makes it difficult to summarize how they should be compared. Moreover, it is interesting to notice that computing only \( \exp(A) \) requires a different approach from computing \( \exp(Ax) \), for several values of \( x \). We also observe that when dealing with Markov chains, the computation of the matrix exponential is subject to probability constraints. As a result, a first obvious remark is that there is no unique conclusion about which method is the best and it probably depends on each concrete application.

Concerning the stochastic epidemic models, the possibility of developing an exhaustive comparative study is a promising research topic for forthcoming studies. At present, we may comment that the Padé approach used by MATLAB is primarily concerned with small dense matrices and large sparse matrices (as the tridiagonal matrix of the SIS model). In these cases, the MATLAB routines are perhaps more straightforward than the numerical inversion methods. However, the numerical inversion methods require neither the computation of the whole matrix nor its eigenvalues. Moreover, the recursive schemes developed in this paper exploit the special transition structure of the epidemic models under study and avoid subtractions, which greatly improves the stability. Thus, we recommend our methods especially when the primary attention is put on accuracy rather than in computer time required, and as far as the population size increases and the underlying matrix is not sufficiently sparse (e.g. the SIR model and other more sophisticated epidemic models).

On the other hand, we observe that the differentiation of the Laplace transform equations provides the quickest way to obtain recursive schemes for computing the moments. In this way, we avoid the computation of the powers of matrices involved in (R2).

The above discussion on the extinction time needs to be completed with a few comments regarding approximations and asymptotic results. A remarkable result establishes that the extinction time, when the initial distribution equals the quasi-stationary distribution, follows a simple exponential distribution. For a proof in the context of the finite birth–death process, we refer to
Norden [32]. This approximation has been extensively used. For the generalization to bivariate epidemic models, see, e.g. [28,30]. The literature for asymptotic expansions is very rich; as an example, we mention the papers by Doering et al. [14] and Nåsell [29].

While many univariate epidemic models can be formulated as birth–death processes, the SIR models provide a natural framework to deal with the bivariate case. Daley and Gani [12] considered an SIR formulation to model stochastically a general epidemic. Then, the extinction time is investigated by using generating functions and Laplace transforms, but the authors conclude that the obtained solution is algebraically formidable. For the special case where the epidemic starts with one infective and the population size is very small, Gani [15] gave simpler results. Billard and Zhao [7] investigated the transient analysis of an SIR model, and consequently, the extinction time distribution can be derived from that study. However, the numerical implementation of their results is likely to be cumbersome and it seems to involve unstable elements (i.e. subtractions, alternating signs). Barbour [6] investigated the asymptotic behaviour of the extinction time as the population size tends to infinity. The computation of the expected extinction time is underlying in the numerical examples given in this paper.

Our contribution to the analysis of the extinction time is twofold. The first one is the computation of the whole distribution of the extinction time by applying numerical Laplace inversion algorithms. Although mean and variance summarize the main statistical properties of a random variable, knowledge of the whole probability distribution is of interest in its own right. In particular, the probability that the length of the outbreak exceeds a certain critical threshold may be helpful to take preventive actions. Moreover, the mere knowledge of the first moments is somewhat deceptive, when the distribution under study is not unimodal. This is the case of the final size distribution and the extinction time distribution of the SIR model, which are bimodal [5,6]. At this point, we remark that the methodology developed in this paper is also helpful to explore the shape of the density function by identifying its modes and the behaviour at the time origin 0. A second contribution concerns the moments of the conditional extinction times for the SIR epidemic model. By exploiting the special transition structure of this model, we get stable recursive schemes for computing transforms and moments. In this paper, we propose simple recursive schemes that avoid subtractions. In this way, we circumvent the above-mentioned drawbacks inherent to the computation of formulae in (R1) and (R2).

Finally, we now turn our attention to the time to infection and to the recovery time. As far as we know, these two epidemic descriptors have not been studied yet in the framework of stochastic biological models. Stone et al. [35] defined the probability that an individual becomes infected. In Section 2.2, we show how this probability is related to the time to infection. We perform an exhaustive analysis of these descriptors both for the birth–death model and the stochastic SIR model. In this paper, we focus on the birth–death model and the basic formulation of the stochastic SIR model. However, our results can be extended to other stochastic epidemic models. In any subsequent study, we would like to extend our analysis to models with killing and catastrophes (see, e.g. the papers by Coolen-Schrijner and van Doorn [11] and Artalejo et al. [4]) and also to more complicated variants of the SIS and SIR epidemic models (see, e.g. some recent publications in this journal such as the papers by McCormack and Allen [24], Clémençon et al. [9] and Martins et al. [23]).

The rest of the paper is organized as follows. In Section 2, we introduce the stochastic birth–death model and the random variables representing the extinction time, the time to infection and the recovery time. We develop algorithmic schemes for their analysis, first focusing on their Laplace transforms and then proceeding to the study of the moments. Section 3 presents a parallel analysis for the stochastic SIR model. In Section 4, we present some selected numerical results. In particular, in Section 4.1 we employ the descriptors under study in the context of the head lice infection reported by Stone et al. [35]. The behaviour characteristics of the SIR model are numerically...
investigated in Section 4.2. Finally, Section 4.3 contains some numerical results regarding the number of modes for the characteristics of the SIS model.

2. Birth–death process

We consider a closed population of $N$ individuals, where each individual is classified as either a susceptible or an infective. Individuals move from the susceptible to the infected group and then they recover, returning to the susceptible pool. The stochastic model describing the evolution of the epidemic can be seen as a birth–death process $\{I(t); t \geq 0\}$ with state space $S = \{0, \ldots, N\}$, where $I(t)$ gives the number of infectives at time $t$. The birth rates, corresponding to infections, are denoted by $\lambda_i$ and the death rates, corresponding to recuperations, are denoted by $\mu_i$, $i = 0, \ldots, N$. The infections are supposed to occur because of a contagious disease. Hence, when there are no infectives, the process stays there forever. The other states are assumed transient. More specifically, we assume that $\lambda_0 = \lambda_N = \mu_0 = 0$, while $\mu_1, \ldots, \mu_N$ and $\lambda_1, \ldots, \lambda_{N-1}$ are strictly positive.

A particular example of birth–death processes is, for instance, the classical SIS model, with $\lambda_i = \beta_i (N - i)/N$ for the birth rate and $\mu_i = \gamma_i$ for the death rate, where $\beta$ is the contact rate and $\gamma$ is the recovery rate per individual. A more general model is the Verhulst model (see [29] for details), with infection rates $\lambda_i = \beta_i (1 - (\alpha_1 i/N))$, for $0 \leq i \leq N - 1$, and recovery rates $\mu_i = \gamma_i (1 + (\alpha_2 i/N))$, for $0 \leq i \leq N$.

2.1. Extinction time

Let us assume that at the initial time $t = 0$ the population has $i$ infective individuals and define a continuous random variable $L_i$ to be the extinction time of the epidemic given the current population state. This variable can be seen as the absorption time by the state 0 given that $I(0) = i$.

Next we introduce some notation for absorption probabilities, Laplace transforms and moments of $L_i$, for $0 \leq i \leq N$. Let us define

$$u_i = P\{L_i < \infty\}, \quad 0 \leq i \leq N,$$

$$\varphi_i(s) = E[e^{-sL_i}], \quad 0 \leq i \leq N, \quad \text{Re}(s) \geq 0,$$

$$M_k^i = E[L_i^k], \quad 0 \leq i \leq N, \quad k \geq 0.$$

First we observe that $u_i = 1$, for $1 \leq i \leq N$, because the set $\{1, \ldots, N\}$ is a non-decomposable set of states.

Theorem 1 provides a computationally stable recursive scheme, from which the computation of Laplace transforms can be done at a low computational cost. More concretely, the proposed scheme only deals with algebraic operations involving positive terms, which guarantees stability even for large values of $N$. Once the Laplace transforms $\varphi_i(s)$ have been computed, the density functions $f_{L_i}(x)$ (or, alternatively, the probabilities $P\{L_i > x\}$) can be obtained numerically by using Fourier series methods.

**Theorem 1** The Laplace transforms $\varphi_i(s)$, for $1 \leq i \leq N$, are computed by the equations

$$\varphi_N(s) = \frac{\mu_N D_{N-1}}{s \lambda_{N-1} + (s + \mu_N)(s + g_{N-1})}, \quad (1)$$

$$\varphi_i(s) = \sum_{k=i}^{N-1} \frac{D_k}{\lambda_k} \prod_{n=i}^{k-1} \frac{\lambda_n}{s + g_n + \lambda_n} + \varphi_N(s) \prod_{k=i}^{N-1} \frac{\lambda_k}{s + g_k + \lambda_k}, \quad 1 \leq i \leq N - 1, \quad (2)$$
where the coefficients $g_i$ and $D_i$, for $1 \leq i \leq N - 1$, are given by the recursive scheme

\begin{align*}
g_1 &= \mu_1, \\
g_i &= \mu_i \frac{s + g_{i-1}}{s + g_{i-1} + \lambda_{i-1}}, \quad 2 \leq i \leq N - 1, \\
D_1 &= \mu_1, \\
D_i &= \frac{\mu_i D_{i-1}}{s + g_{i-1} + \lambda_{i-1}}, \quad 2 \leq i \leq N - 1.
\end{align*}

Proof
Conditioning on the exponentially distributed time to the first transition, we have

\begin{align*}
\varphi_0(s) &= 1, \\
\varphi_i(s) &= \frac{\mu_i}{s + \lambda_i + \mu_i} \varphi_{i-1}(s) + \frac{\lambda_i}{s + \lambda_i + \mu_i} \varphi_{i+1}(s), \quad 1 \leq i \leq N.
\end{align*}

Equation (7), for $1 \leq i \leq N - 1$, can be expressed as follows:

\begin{align*}
\beta_i \varphi_{i-1}(s) + \gamma_i \varphi_i(s) + \alpha_i \varphi_{i+1}(s) &= \delta_i,
\end{align*}

where

\begin{align*}
\beta_1 &= 0, \quad \beta_i = -\mu_i, \quad 2 \leq i \leq N - 1, \\
\gamma_i &= s + \lambda_i + \mu_i, \quad 1 \leq i \leq N - 1, \\
\alpha_i &= -\lambda_i, \quad 1 \leq i \leq N - 1, \\
\delta_1 &= \mu_1, \quad \delta_i = 0, \quad 2 \leq i \leq N - 1.
\end{align*}

Using a forward-elimination–backward-substitution procedure, the tridiagonal system (8) becomes

\begin{align*}
G_i \varphi_i(s) + \alpha_i \varphi_{i+1}(s) &= D_i, \quad 1 \leq i \leq N - 1,
\end{align*}

where

\begin{align*}
G_1 &= \gamma_1 = s + \lambda_1 + \mu_1, \\
G_i &= \gamma_i - \frac{\beta_i \alpha_{i-1}}{G_{i-1}} = s + \lambda_i + \mu_i - \frac{\mu_i \lambda_{i-1}}{G_{i-1}}, \quad 2 \leq i \leq N - 1, \\
D_1 &= \delta_1 = \mu_1, \\
D_i &= \delta_i - \frac{\beta_i D_{i-1}}{G_{i-1}} = \frac{\mu_i D_{i-1}}{G_{i-1}}, \quad 2 \leq i \leq N - 1.
\end{align*}

In order to avoid negative terms, we introduce a new set of coefficients $g_i = G_i - (s + \lambda_i)$, for $1 \leq i \leq N - 1$. Then, we easily find that the coefficients $g_i$ and $D_i$ are as claimed in Equations (3)–(6).

Now we use Equation (9) and express $\varphi_i(s)$ in terms of $\varphi_{i+1}(s)$

\begin{align*}
\varphi_i(s) &= \frac{D_i - \alpha_i \varphi_{i+1}(s)}{G_i} = \frac{D_i + \lambda_i \varphi_{i+1}(s)}{s + g_i + \lambda_i}, \quad 1 \leq i \leq N - 1.
\end{align*}

Iterating Equation (10) we obtain Equation (2).
Now using Equation (7) for $i = N$, we have

$$(s + \mu_N)\varphi_N(s) = \mu_N\varphi_{N-1}(s).$$

(11)

Finally, from Equations (2), for $i = N - 1$, and (11), we get Equation (1). □

On the other hand, the value of the density function at the point $x = 0$ follows by differentiating expression (R1), which yields

$$f_{L_i}(0) = \left\{ \begin{array}{ll}
\mu_1, & \text{if } i = 1, \\
0, & \text{otherwise.}
\end{array} \right.$$  

Next we focus on the calculation of the moments $\{M^k_i; 0 \leq i \leq N\}$, for any arbitrary non-negative integer $k$. Note that moments of order $k = 0$ are $M^0_i = u_i = 1$, for $0 \leq i \leq N$. For $k \geq 1$, by differentiating Equation (7) $k$ times with respect to $s$ and setting $s = 0$, we find that

$$M^k_0 = 0,$$

$$\lambda_i + \mu_i)M^{k}_i = \mu_iM^{k-1}_{i-1} + \lambda_iM^{k}_{i+1} + kM^{k-1}_i, \quad 1 \leq i \leq N.$$  

(13)

These equations were also derived by Norden [32] using the backward equations and also, for the particular case of an SIS epidemic with external source of infection, by Stone et al. [35], from the moment-generating function.

Moments of $L_i$, for $1 \leq i \leq N$, are determined recursively on $k$ by using the following formula, which agrees with Equation (6.8) in [32] and generalizes the corresponding one stated in Stone et al. [35]:

$$M^k_i = \sum_{j=0}^{i-1} \rho_j \sum_{n=j+1}^{N} \frac{kM^{k-1}_n}{\lambda_n\rho_n},$$

(14)

where $\rho_0 = 1$ and $\rho_j = \prod_{m=1}^{j}(\mu_m/\lambda_m)$, for $1 \leq j \leq N - 1$.

2.2. Time to infection and recovery time

Let us consider the population at an arbitrary time $t$ and suppose that there are $i$ infected individuals at this time, for $0 \leq i \leq N - 1$. We mark one of the $N - i$ non-infected individuals and denote by $S_i$ a random variable representing the time until the selected individual gets infected. Obviously, $S_0 = +\infty$ and the case $i = N$ has no sense. To study the variables $S_i$, for $0 \leq i \leq N - 1$, we define

$$v_i = P\{S_i < \infty\}, \quad 0 \leq i \leq N - 1,$$

$$\psi_i(s) = E[e^{-sS_i}\mathbf{1}_{[S_i<\infty]}], \quad 0 \leq i \leq N - 1, \quad Re(s) \geq 0,$$

$$\tilde{M}^k_i = E[S^k_i\mathbf{1}_{[S_i<\infty]}], \quad 0 \leq i \leq N - 1, \quad k \geq 0,$$

where, for an event $A$, $\mathbf{1}_A$ is the indicator random variable that takes the value 1 when the event $A$ occurs and is 0 otherwise.

Note that the probabilities $v_i$ are strictly between 0 and 1, for $1 \leq i \leq N - 1$. Indeed, $P\{S_i < \infty\} \geq (\lambda_1/(\lambda_1 + \mu_1)) \cdots (\lambda_{N-1}/(\lambda_{N-1} + \mu_{N-1})) > 0$ and $P\{S_i = \infty\} \geq (\mu_i/(\lambda_i + \mu_i)) \cdots (\mu_1/(\lambda_1 + \mu_1)) > 0$; hence, $P\{S_i < \infty\} < 1$.

A first-step argument, conditioning on the identity of the next infected individual (i.e. either
a recovery, an infection for a non-tagged individual or the infection of the marked individual), shows that the Laplace transforms, $\psi_i(s)$, satisfy the following set of equations:
\begin{align}
\psi_0(s) &= 0, \\
\psi_i(s) &= \frac{\mu_i}{s + \lambda_i + \mu_i} \psi_{i-1}(s) + \frac{\lambda_i}{s + \lambda_i + \mu_i} \frac{N - i - 1}{N - i} \psi_{i+1}(s) \\
&\quad + \frac{\lambda_i}{s + \lambda_i + \mu_i} \frac{1}{N - i}, \quad 1 \leq i \leq N - 1.
\end{align}

For every $s$, the system of equations (15) and (16) is tridiagonal and the coefficient matrix is strictly diagonally dominant. We again solve the system by using a forward-elimination—backward-substitution method. After some algebra, we obtain a stable recursive scheme which appears in the following theorem.

**Theorem 2**  
*The Laplace transforms $\psi_i(s)$, for $1 \leq i \leq N - 1$, are computed by the equations*
\begin{align}
\psi_{N-1}(s) &= \frac{2(\lambda_{N-1}(s + g_{N-2} + \lambda_{N-2}) + \mu_{N-1}D_{N-2})}{2(s + \lambda_{N-1})(s + g_{N-2} + \lambda_{N-2}) + \mu_{N-1}(2(s + g_{N-2} + \lambda_{N-2})}, \\
\psi_i(s) &= \sum_{k=i}^{N-2} \frac{D_k}{\lambda_k} \frac{N - k}{N - k - 1} \prod_{n=i}^{k} \frac{\lambda_n}{s + g_n + \lambda_n} \frac{N - n - 1}{N - n} \\
&\quad + \psi_{N-1}(s) \prod_{k=i}^{N-2} \frac{\lambda_k}{s + g_k + \lambda_k} \frac{N - k - 1}{N - k}, \quad 1 \leq i \leq N - 2,
\end{align}

*where the coefficients $g_i$ and $D_i$, for $1 \leq i \leq N - 2$, are given by the recursive scheme*
\begin{align}
g_1 &= \mu_1, \\
g_i &= \frac{\mu_i}{s + g_{i-1} + \frac{\lambda_{i-1}}{N-i+1}}, \quad 2 \leq i \leq N - 2, \\
D_1 &= \frac{\lambda_1}{N - 1}, \\
D_i &= \frac{\lambda_i}{N - i} + \frac{\mu_i D_{i-1}}{s + g_{i-1} + \lambda_{i-1}}, \quad 2 \leq i \leq N - 2.
\end{align}

**Proof**  
The proof follows along the lines described in the proof of Theorem 1, so it is omitted. ■

The starting value of the density function $f_S(x)$ follows from the Tauberian result $f_S(0) = \lim_{s \to \infty} s \psi_i(s)$. In light of Equation (16), it gives $f_S(0) = \lambda_i/(N - i)$, for $1 \leq i \leq N - 1$.

Observe that $v_i = \tilde{M}_i^0 = P[S_i < \infty] = \psi_i(0)$. Consequently, the probabilities $v_i$, $0 \leq i \leq N - 1$, can be determined by setting $s = 0$ in Equations (17)–(22). It should be pointed out that the resulting equations for the probabilities $v_i$ correspond to those given by Stone et al. [35] for the probability that an individual becomes infected.

We now concentrate on the calculation of the moments $\{\tilde{M}_i^k; 0 \leq i \leq N - 1\}$. By differentiating Equations (15) and (16) $k \geq 1$ times and evaluating at $s = 0$, we find that
\begin{align}
\tilde{M}_0^k &= 0, \\
(\lambda_i + \mu_i)\tilde{M}_i^k &= \mu_i \tilde{M}_{i-1}^k + \lambda_i \frac{N - i - 1}{N - i} \tilde{M}_{i+1}^k + k \tilde{M}_i^{k-1}, \quad 1 \leq i \leq N - 1.
\end{align}

This system of equations provides, after some algebra, a stable recursive scheme for the computation of $\tilde{M}_i^k$, for $1 \leq i \leq N - 1$ and $k \geq 1$, having a similar structure to the one appearing
in Theorem 2. In fact, for each fixed $k \geq 1$, the following simple modifications are needed: (i) replace $\psi_i(s)$ by $\tilde{M}_k^i$, for $1 \leq i \leq N - 1$, (ii) set $s = 0$ in Equations (17)–(20), and (iii) replace $D_i$ in Equations (21) and (22) by $D_1 = k\tilde{M}_k^{i-1}$ and $D_i = k\tilde{M}_k^{i-1} + \mu_iD_{i-1}(g_{i-1} + \lambda_{i-1})^{-1}$, for $2 \leq i \leq N - 2$. Now, each iteration allows us to compute the unknown moments of order $k \geq 1$ in terms of the moments of one order less. Note that the moments of order $k = 0$ are $v_i$.

Note, however, that the system (23) cannot lead to a more explicit expression as in the case of Equations (13). The reason is that the recursion scheme (23) is of second order with no constant coefficients and, more importantly, the sum of the coefficients at each row of the system is not $0$, as in the case of Equations (13). Therefore, it is not possible to reduce the order of the scheme by considering the differences. The same holds as well for the other recursive schemes for the computation of the moments of various descriptors that we report in the rest of the paper (see, e.g. Equations (29) and (34)).

Next we study the recovery time, $R_i$, defined as the time until a tagged infected individual gets recovered, given that the population consists of $i$ infective individuals, for $1 \leq i \leq N$. Obviously, $R_0$ has no sense. To study these random variables, we define

$$w_i = P\{R_i < \infty\}, \quad 1 \leq i \leq N,$$

$$\phi_i(s) = E[e^{-sR_i}], \quad 1 \leq i \leq N, \quad Re(s) \geq 0,$$

$$m^k_i = E[R^k_i], \quad 1 \leq i \leq N, \quad k \geq 0.$$

It is clear that the recovery time of a marked individual is at most the time to extinction of the epidemic. Thus, we have that $R_i \leq L_i$, for all $1 \leq i \leq N$. Hence, as it was shown in Section 2.1, from $u_i = P\{L_i < \infty\} = 1$, we conclude that $w_i = m^0_i = P\{R_i < \infty\} = 1$, for $1 \leq i \leq N$.

Again, a first-step argument gives that the Laplace transforms, $\phi_i(s)$, satisfy

$$\phi_i(s) = \frac{\mu_i}{s + \lambda_i + \mu_i} \frac{i - 1}{i} \phi_{i-1}(s) + \frac{\lambda_i}{s + \lambda_i + \mu_i} \phi_{i+1}(s)
+ \frac{1}{s + \lambda_i + \mu_i} \frac{\mu_i}{i}, \quad 1 \leq i \leq N,$$

and the initial density value is $f_{R_i}(0) = \lim_{s \to \infty} s\phi_i(s) = \mu_i / i$, for $1 \leq i \leq N$.

For $k \geq 1$, by differentiating Equation (24) $k$ times with regard to $s$ and setting $s = 0$, we get that the moments of order $k$ satisfy the equations

$$(\lambda_i + \mu_i)m^k_i = \mu_i \frac{i - 1}{i} m^k_{i-1} + \lambda_i m^k_{i+1} + km^{k-1}_i, \quad 1 \leq i \leq N.$$

Once more, we notice that the coefficient matrix in Equation (24) is strictly diagonally dominant. This guarantees the existence of finite solutions both for transforms and moments. In addition, it is possible to develop algorithmic schemes for their computational analysis, whose structure is similar to the one appearing in Theorems 1 and 2.

Remark 3 When the rate at which recovery events occur is proportional to the number of infected individuals (i.e. the case $\mu_i = \gamma i$), we observe immediately that the recovery times, $R_i$, for $1 \leq i \leq N$, are exponentially distributed with rate $\gamma$, independently of the number of infective individuals in the population.

We may consider unconditional versions of the time to infection and the recovery time. Let us denote versions such as $S$ and $R$, respectively, and their corresponding Laplace transforms as
\[ \Psi(s) = \sum_{i=1}^{N-1} a_i \psi_i(s), \]
\[ \Phi(s) = \sum_{i=1}^{N} b_i \phi_i(s), \]

where the positive weights \( a_i \) and \( b_i \) satisfy that \( \sum_{i=1}^{N-1} a_i = \sum_{i=1}^{N} b_i = 1 \). There exist various ways to choose appropriate weights. A reasonable possibility is to employ the quasi-stationary probabilities, \( q_i \), of having \( i \) infective individuals given that the extinction has not occurred yet.

3. \textbf{Stochastic SIR model}

Let us consider again a closed population of \( N \) individuals, subdivided into susceptible, infective and removed or immune individuals. A susceptible individual can get infected and then can recover. Those recovered individuals are assumed permanently immune to further infections. As a result, we refer to them as removed individuals, while in fact they remain in the population. The denominator \( N \) in the transition rates (see next paragraph) shows that the contribution of the immunes is to waste potential infection capacity from the infectives.

The assumption that the total population is closed implies that, at any time \( t \), \( S(t) + I(t) + R(t) = N \), where \( S(t) \), \( I(t) \) and \( R(t) \) denote the number of susceptibles, infectives and removals, respectively, at time \( t \). The population dynamics is described in terms of a bidimensional continuous-time Markov chain \( \{(I(t), S(t)); t \geq 0\} \). Transitions from a state \((i, j)\) can be either to state \((i+1, j-1)\) at a rate \( \lambda_{ij} \), due to an infection, or to state \((i-1, j)\) at a rate \( \mu_i \), due to a removal. We consider that the epidemics consists of a single outbreak so the transition from \((0, j)\) to \((1, j-1)\) is not permitted. In addition, we have some trivial null rates, that is, \( \lambda_{i0} = \mu_0 = 0 \). Usually, the transition rates are assumed to have the form \( \lambda_{ij} = \beta ij/N \) and \( \mu_i = \gamma i \), where the parameters \( \beta > 0 \), \( \gamma > 0 \) are identified as the contact rate and removal rate, respectively. Various authors suggest that the model might be made more realistic by allowing contact and removal rates to depend upon the number of susceptibles and infectives present in the population. In that sense, Severo [34] and more recently Clancy and Green [8] suggest potential functions.

In what follows, let us assume that the population initially consists of \( m \) infectives and \( n \) susceptibles. The state space of the SIR epidemic model is \( S = \{(i, j); 0 \leq j \leq n, 0 \leq i \leq m + n - j\} \), with absorbing states \( \{(0, j); 0 \leq j \leq n\} \). The rest of states are transient and after leaving one of them, the chain never returns to this state. If we take \( j \) as the main level and arrange the states in lexicographic order, then we observe that the generator \( Q \) has a triangular structure which is really helpful to get stable recursive algorithms. Figure 1 shows the state space and the transitions rates when \((m, n) = (3, 3)\).

3.1. \textbf{Extinction time}

We introduce the random variable \( L_{ij} \) as the extinction time of the outbreak given that the current state is \((i, j)\). Let us define the absorption probabilities \( u_{ij} = P[L_{ij} < \infty] \). We notice that \( u_{ij} = 1 \) for all transient states. This is due to the fact that the submatrix of \( Q \) governing the motion in the set of transient states is invertible because it is a triangular matrix with non-null diagonal elements.
Next we define the Laplace transforms and moments of $L_{ij}$.

$$\phi_{ij}(s) = E[e^{-sL_{ij}}], \quad (i, j) \in S, \quad Re(s) \geq 0,$$

$$M^k_{ij} = E[L^k_{ij}], \quad (i, j) \in S, \quad k \geq 0.$$  

Using a first-step argument, we get that the functions $\phi_{ij}(s), (i, j) \in S,$ satisfy

$$\phi_{0j}(s) = 1, \quad 0 \leq j \leq n,$$

$$\phi_{ij}(s) = \frac{\mu_i}{s + \lambda_{ij} + \mu_i} \phi_{i-1,j}(s) + \frac{\lambda_{ij}}{s + \lambda_{ij} + \mu_i} \phi_{i+1,j-1}(s), \quad 0 \leq j \leq n, \quad 1 \leq i \leq m + n - j.$$  

Solving Equation (26) for $j = 0$, we have

$$\phi_{i0}(s) = \frac{\mu_i}{s + \mu_i} \phi_{i-1,0}(s) = \cdots = \prod_{k=1}^{i} \frac{\mu_k}{s + \mu_k}, \quad 1 \leq i \leq m + n.$$  

Note that Equations (25)–(27) can be combined, in the natural order $1 \leq j \leq n$ and $1 \leq i \leq m + n - j$, to determine recursively the values of $\phi_{ij}(s)$, at a fixed point $s$. In particular, we can get the Laplace transform of the extinction time from the initial state $(m, n)$. After that, the use of numerical inversion algorithms permits to determine numerically $P\{L_{mn} > x\}$, that is, the probability that an outbreak, starting with $m$ infectives and $n$ susceptibles, will last more than $x$ time units.

We observe that the density function at $x = 0$ is given by $f_{L_{ij}}(0) = \mu_1$, if $i = 1$, and it is 0 otherwise.

For determining the moments of $L_{ij}$ of any arbitrary order $k \geq 0$, we first notice that

$$M^k_{0j} = 0, \quad 0 \leq j \leq n, \quad k \geq 0,$$

and for the transient states

$$M^0_{ij} = u_{ij} = 1, \quad 0 \leq j \leq n, \quad 1 \leq i \leq m + n - j.$$
In order to get the moment of order \( k \geq 1 \), by differentiating Equations (26) and (27) \( k \) times with respect to \( s \) and setting \( s = 0 \), we find that

\[
M_{i0}^k = k \sum_{l=1}^{i} \frac{M_{l0}^{k-1}}{\mu_l}, \quad 1 \leq i \leq m + n, \quad k \geq 1,
\]

\[
M_{ij}^k = \frac{\mu_i}{\lambda_{ij} + \mu_i} M_{i-1,j}^k + \frac{\lambda_{ij}}{\lambda_{ij} + \mu_i} M_{i+1,j-1}^k + \frac{k}{\lambda_{ij} + \mu_i} M_{ij}^{k-1},
\]

\[1 \leq j \leq n, \quad 1 \leq i \leq m + n - j, \quad k \geq 1.\]

Equations (28) and (29) provide an efficient recursive scheme for the computation of \( M_{ij}^k \) in the natural order.

### 3.2. Time to infection and removal time

Let us assume that the initial state of the population is \((I(0), S(0)) = (m, n)\), with \( m \geq 1 \). We choose one of the susceptible individuals, we mark it and investigate the random variable \( S_{mn} \) defined as the sojourn time until the marked individual becomes infected. We note that it is possible that the outbreak ends before the infection of the marked individual and consequently \( P\{S_{mn} = \infty\} > 0 \).

In order to investigate the probabilistic behaviour of \( S_{mn} \) we introduce, for the rest of possible states, analogous random variables, \( S_{ij} \), defined as the time to infection of the marked susceptible individual conditioned to the current state \((i, j) \in S\). Obviously, \( S_{0j} = +\infty \), for \( 1 \leq j \leq n \), and for \( 0 \leq i \leq m + n \), \( S_{i0} \) have no sense because there are no susceptible individuals. The rest of the probabilities \( v_{ij} = P\{S_{ij} < \infty\} \), associated with the transient states, are strictly between 0 and 1. Indeed, for \( 1 \leq j \leq n \) and \( 1 \leq i \leq m + n - j \), we notice that \( P\{S_{ij} < \infty\} \geq \frac{\lambda_{ij}}{(\lambda_{ij} + \mu_i) \cdots \lambda_{i+j-1,1}} / (\lambda_{i+j-1,1} + \mu_{i+j-1}) > 0 \) and \( P\{S_{ij} = \infty\} \geq \frac{\mu_i}{(\lambda_{ij} + \mu_i) \cdots \mu_1} / (\lambda_1 + \mu_1) > 0 \).

Next we investigate the Laplace transforms and moments of \( S_{ij} \), for \( 1 \leq j \leq n \) and \( 1 \leq i \leq m + n - j \), so we define

\[
\psi_{ij}(s) = E\{e^{-sS_{ij}} 1_{[S_{ij} < \infty]}\}, \quad 1 \leq j \leq n, \quad 0 \leq i \leq m + n - j, \quad Re(s) \geq 0,
\]

\[
\tilde{M}_{ij}^k = E\{S_{ij}^k 1_{[S_{ij} < \infty]}\}, \quad 1 \leq j \leq n, \quad 0 \leq i \leq m + n - j, \quad k \geq 0.
\]

By a first-step argument, conditioning on the next event (i.e. either a removal, an infection for a non-tagged individual or the infection of the marked individual), we find that the Laplace transforms satisfy

\[
\psi_{0j}(s) = 0, \quad 1 \leq j \leq n,
\]

\[
\psi_{ij}(s) = \frac{\mu_i}{s + \lambda_{ij} + \mu_i} \psi_{i-1,j}(s) + \frac{\lambda_{ij}}{s + \lambda_{ij} + \mu_i} \frac{j - 1}{j} \psi_{i+1,j-1}(s)
\]

\[+ \frac{\lambda_{ij}}{s + \lambda_{ij} + \mu_i} \frac{1}{j}, \quad 1 \leq j \leq n, \quad 1 \leq i \leq m + n - j. \quad (31)
\]

We can solve the system of linear equations (30) and (31) without evaluating its inverse matrix. Notice first that Equation (31), for \( j = 1 \), yields

\[
\psi_{11}(s) = \frac{\lambda_{11}}{s + \lambda_{11} + \mu_1},
\]
and this explicit expression provides the starting point to get the rest of the Laplace transforms. Via a stable procedure, we compute recursively $\psi_{ij}(s)$ in the natural order $1 \leq j \leq n$ and $1 \leq i \leq m + n - j$.

Moreover, we notice that $f_{S_{ij}}(0) = \lambda_{ij}/j$, for $1 \leq j \leq n$ and $1 \leq i \leq m + n - j$.

Now we observe that $v_{ij} = \tilde{M}_{ij}^0 = P\{S_{ij} < \infty\} = \psi_{ij}(0)$. Thus, it follows that the probabilities $v_{ij}$ satisfy Equations (30) and (31), with $s = 0$.

We can also get the moments of order $k \geq 1$, $\tilde{M}_{ij}^k$, as the solution of the following system of linear equations:

$$
\tilde{M}_{0j}^k = 0, \quad 1 \leq j \leq n, \quad k \geq 1,
$$

$$
\tilde{M}_{ij}^k = \frac{\mu_i}{\lambda_{ij} + \mu_i} \tilde{M}_{i-1,j}^k + \frac{\lambda_{ij}}{\lambda_{ij} + \mu_i} \frac{j - 1}{j} \tilde{M}_{i+1,j-1}^k
$$

$$
+ \frac{k}{\lambda_{ij} + \mu_i} \tilde{M}_{ij}^{k-1}, \quad 1 \leq j \leq n, \quad 1 \leq i \leq m + n - j, \quad k \geq 1.
$$

Finally, we study the removal time, $R_{ij}$, defined as the time until a tagged infected individual gets immunity, given that the current population has $i$ infective and $j$ susceptible individuals, for $0 \leq j \leq n$ and $1 \leq i \leq m + n - j$. In fact, we want to compute the characteristics of the random variable $R_{mn}$, but this computation involves all the variables $R_{ij}$.

First we notice that for any state $(i, j)$, the removal time is always shorter than the extinction time of the outbreak, that is $R_{ij} \leq L_{ij}$. Hence, if we define the probabilities $w_{ij} = P\{R_{ij} < \infty\}$, we can conclude that $w_{ij} = 1$, for $0 \leq j \leq n$ and $1 \leq i \leq m + n - j$.

Let us introduce some notation for the Laplace transforms and moments

$$
\phi_{ij}(s) = E[e^{-sR_{ij}}], \quad 0 \leq j \leq n, \quad 1 \leq i \leq m + n - j, \quad \text{Re}(s) \geq 0,
$$

$$
m_{ij}^k = E[R_{ij}^k], \quad 0 \leq j \leq n, \quad 1 \leq i \leq m + n - j, \quad k \geq 0.
$$

We now observe that the Laplace transforms satisfy

$$
\phi_{ij}(s) = \frac{\mu_i}{s + \lambda_{ij} + \mu_i} \frac{i - 1}{i} \phi_{i-1,j}(s) + \frac{\lambda_{ij}}{s + \lambda_{ij} + \mu_i} \phi_{i+1,j-1}(s)
$$

$$
+ \frac{\mu_i}{s + \lambda_{ij} + \mu_i} \frac{1}{i}, \quad 0 \leq j \leq n, \quad 1 \leq i \leq m + n - j.
$$

(32)

For any fixed $j$, formula (32) provides a recursion for computing the transforms $\phi_{ij}(s)$, for $i = 1, \ldots, m + n - j$, in terms of $\phi_{i+1,j-1}(s)$, which has been computed in the previous step.

The starting density value is now given by $f_{R_{ij}}(0) = \mu_i/i$, for $0 \leq j \leq n$, $1 \leq i \leq m + n - j$.

Differentiating Equation (32) with respect to $s$ and setting $s = 0$, yields

$$
m_{ij}^0 = w_{ij} = 1, \quad 0 \leq j \leq n, \quad 1 \leq i \leq m + n - j,
$$

(33)

$$
m_{ij}^k = \frac{\mu_i}{\lambda_{ij} + \mu_i} \frac{i - 1}{i} m_{i-1,j}^k + \frac{\lambda_{ij}}{\lambda_{ij} + \mu_i} m_{i+1,j-1}^k
$$

$$
+ \frac{k}{\lambda_{ij} + \mu_i} m_{ij}^{k-1}, \quad 0 \leq j \leq n, \quad 1 \leq i \leq m + n - j, \quad k \geq 1.
$$

(34)

Then, we can compute $m_{ij}^k$ recursively from Equations (33) and (34), in the order $k \geq 0$, $0 \leq j \leq n$ and $1 \leq i \leq m + n - j$.

**Remark 4** In agreement with Remark 3, in the case $\mu_i = \gamma i$, we observe that the removal times $R_{ij}$, for $0 \leq j \leq n$ and $1 \leq i \leq m + n - j$, are exponentially distributed with rate $\gamma$, independently of the number of susceptible and infective individuals in the population.
4. Numerical examples

In this section, we present some numerical illustrations of the theoretical results. A first set of numerical experiments concerns the application to outbreaks of head lice, *Pediculus humanis capitis*. To this end, in Section 4.1, we employ the data set provided by Stone *et al.* [35] corresponding to a study carried out in the UK schools. The underlying epidemic model is a stochastic SIS model with an external source of infection. In Section 4.2, we investigate the influence of various system parameters in the behaviour characteristics of the SIR epidemic model. Some special attention is paid to the identification of the modes. In fact, the analysis of the modes is extended in Section 4.3 to the SIS model.

4.1. An application to head lice infections

Head lice, or *pediculosis capitis*, is an important health problem among children worldwide. Although head lice rarely cause direct harm, it can cause distress to many patients. Healthcare professionals, school administrations and parents frequently seek solutions. Since head lice are extremely contagious, it is important to observe promptly the outbreak and to reduce the transmission rate. Some steps that can be followed to prevent the spread include regular checks in schools, existence of detection campaigns, as well as the engagement of parents. Once the outbreak starts, it is important to avoid head-to-head contact, not to share combs, towels and other personal hygiene objects. The common treatment options are environmental decontamination, mechanical removal and use of topical insecticides (lindane, malathion). For further details, we refer the reader to the recent studies by Diamantis *et al.* [13] and Ibarra *et al.* [18], and also to the references therein.

In what follows, we deal with the SIS epidemic model with an external source of infection studied by Stone *et al.* [35]. This means, that we have a closed population of $N$ individuals and assume that infection and removal rates are, respectively, $\lambda_i = (N - i)(\xi + \beta i / N)$ and $\mu_i = \gamma i$, for $0 \leq i \leq N$, where $\xi$ denotes the external rate of infection. Since $\xi > 0$, we observe that $\lambda_0 > 0$ and the resulting birth–death process has no absorbing states. At this point, we stress that all our results in Section 2 remain valid, when we focus on the dynamics of the infection during an individual outbreak. In other words, in this model, extinction means the first moment of no infectives. On the other hand, we notice that an outbreak starts when there is an external infection; that is, $i = 1$. However, the outbreak can be detected later on (or its control could start after some time), so in our numerical experiments we consider several choices for $i$.

To illustrate how our results can be used to investigate properties of the infection spread, we next assume the model parameters considered by Stone *et al.* [35]. They refer to the data from 31 Welsh (UK) schools. For a population size of $N = 100$ and $i = 1$ (i.e. one initial infective), they set $\gamma$ at 1.0 (i.e. the unit time is the expected time of the infectious period) and estimate the rates $\beta = 1.02$ and $\xi = 0.01$ using the maximum-likelihood method. These system parameters provide our basic scenario along this section.

First, we focus on the cumulative distribution function of the extinction time, $F_{L_i}(x) = P\{L_i \leq x\}$. The computation of $F_{L_i}(x)$ has been done by using numerical inversion methods (Section 1), involving the Laplace transforms equations shown in Theorem 1. In Figure 2, we display three curves corresponding to $I(0) = i = 1, 25$ and 50.

The distribution of $L_i$ presents heavy tails when there are larger number of initial infectives in the population. It seems intuitively plausible that the duration of an outbreak is stochastically larger for increasing $i$, having fixed $\beta$. In other words, for $i < j$, one expects that $L_i \leq_{st} L_j$, where the symbol $\leq_{st}$ denotes the usual stochastic order relation with respect to the distribution function. This result can be rigorously established using the following coupling argument.
For $i < j$, let $I_i(t)$ and $I_j(t)$ be independent realizations of the number of infectives starting from $i$ and $j$ infectives, respectively. Then, we define

$$
\tilde{I}_j(t) = \begin{cases} 
I_j(t), & \text{if } t \leq T, \\
I_i(t), & \text{if } t > T,
\end{cases}
$$

where $T = \inf\{t \geq 0 | I_i(t) = I_j(t)\}$.

We notice that $I_j(t)$ could be equal to $I_i(t) + 1$ at some time $t < T$, but the probability that $I_i(t)$ has a positive jump and $I_j(t)$ has a simultaneous negative jump is zero. This eliminates the possibility that $I_j(t') < I_i(t')$, for any $t' \in (t, T)$. Moreover, the strong Markov property guarantees that the constructed process $\tilde{I}_j(t)$ is a Markov process, stochastically equivalent to $I_j(t)$, and by construction satisfies that $\tilde{I}_j(t) \geq I_i(t)$. This proves the desired result.

The interested reader can find a detailed description of the coupling method in Thorisson [36]. For nice applications to epidemics, see the book by Andersson and Britton [3].

In addition, Figure 2 shows the importance of detecting the epidemic process at an early stage (i.e. when it involves a few infective individuals). Note that 78% of the outbreaks starting with only one infective ends before 20 units time. While, when the infectious disease affects initially 25 individuals, the time $L_{25}$ is longer than 20 time units in more than 67% of the infections. This proportion increases to 71% when $i = 50$.

Now we show how the distribution function can be used to halve the mean length of an individual outbreak with a given accuracy. We observe that $E[L_1] = 13.741$, so $E[L_1]/2 = 6.8705$. The objective is to see $P\{L_1 \leq 6.8705\}$ as a function of $\beta$, let us say $f(\beta)$, in order to determine the threshold $\beta^*$ at which we get that $f(\beta) > 0.8$. The numerical evaluation shows a monotone behaviour on $\beta$ and it readily yields $f(0.70) = 0.8099$ and $f(0.71) = 0.7968$, so we have $\beta^* = 0.70$. Thus, the conclusion is that the adoption of policies allowing the contact rate to drop down in $\beta - \beta^* = 1.02 - 0.70 = 0.32$ units would imply that the length of the outbreak is halved with a probability greater than 0.8.

The numerical results suggest that the duration of an outbreak is stochastically increasing with respect to $\beta$. Once more, a rigorous proof of this fact requires an appeal to coupling methods. Given the initial state $i$, we observe that the next transition of the process $\{I(t); t \geq 0\}$ is determined by a competition between two exponentially distributed random variables, let us say $\exp(\lambda_i)$ and $\exp(\mu_i)$, with rates $\lambda_i$ and $\mu_i$. For $\beta' > \beta$, let $I_i(t)$ and $I_i'(t)$ be two independent realizations associated with the contact rates $\beta$ and $\beta'$, respectively. Both realizations start with $i$ infectives.
associated with the SIS model with contact rate $\beta^\prime$. We now observe that the exponential variable $\exp(\lambda_i)$ can be expressed as the minimum between two independent exponential variables, let us say $\exp(\lambda_i)$ and $\exp(\omega_i)$, with $\omega_i = (\beta^\prime - \beta)i(N - i)/N$.

To construct $I_i(t)$ and an equivalent realization $\tilde{I}_i(t)$ of $I_i(t)$, we use common variables $\exp(\lambda_i)$ and $\exp(\mu_i)$. Let $N_i$ be the number of incidents of infection during an outbreak with $i$ initial infectives. For $1 \leq j \leq N_i$, define $T_j$ to be the time at which the $j$th infection occurs in the SIS model with contact rate $\beta$. Note that $T_1 = \infty$, if $N_i = 0$. Similarly, define $T_i^\prime$ for the SIS model with $\beta^\prime$. The coupling ensures that $T_i^\prime \leq T_1$. In fact, it follows that the updated number of infectives at time $T_i^\prime$ is given by

\[
I_i(T_i^\prime) = \begin{cases} 
  i^*, & \text{if } T_i^\prime < T_1, \\
  i^* + 1, & \text{if } T_i^\prime = T_1,
\end{cases}
\]

where $i^* \leq i$.

If $T_i^\prime = T_1$, then the realizations of the coupled processes continue their course. In contrast, if $T_i^\prime < T_1$, then we stop the coupling mechanism, update the initial number of infectives to their current values $i^*$ and $i^* + 1$ and define $\hat{T} = \inf\{t > T_i^\prime | I_i^\prime(t) = I_i^{\prime+1}(t)\}$. It may occur that the outbreak of the SIS model with rate $\beta$ ends before $\hat{T}$ (i.e., $L_i^\prime < \hat{T}$), showing that $L_i^* < L_i^{\prime+1}$; otherwise, it follows that $I_i^\prime(\hat{T}) = I_i^{\prime+1}(\hat{T})$ and the above construction must be restarted. The desired result follows, since the outbreak ends in a finite time with probability one.

Next we present results for $S_i$, the time till the infection of a selected non-infected individual, given that there are $i$ infectives in the population. In Table 1, we display results for the probability that the selected individual becomes infected before the end of the outbreak. We still keep $N = 100$ and $\xi = 0.01$ but we allow the contact and the recovery rates to vary as $\beta \in \{0.05, 0.5, 1.0, 5.0, 10.0\}$ and $\gamma \in \{0.5, 1.0, 2.0, 5.0\}$. For any fixed pair ($\beta, \gamma$), the cell gives from top to bottom these probabilities, when the initial number of infectives varies as $i = 1, 40$ and 90, respectively. We observe that $P\{S_i < \infty\}$ increases as a function of $i$. According to the intuition, the probability of being infected shows an increasing behaviour for increasing contact rates and decreases for increasing recovery rates.

| $P\{S_i < \infty\}$ | $\beta = 0.05$ | $\beta = 0.5$ | $\beta = 1.0$ | $\beta = 5.0$ | $\beta = 10.0$ |
|----------------------|----------------|----------------|----------------|----------------|----------------|
| $\gamma = 0.5$       | 0.07009        | 0.50948        | 0.71798        | 0.91515        | 0.95398        |
|                      | 0.20173        | 0.97877        | 0.99999        | 0.99999        | 0.99999        |
|                      | 0.25516        | 0.99056        | 0.99999        | 0.99999        | 0.99999        |
| $\gamma = 1.0$       | 0.01860        | 0.05750        | 0.27738        | 0.82882        | 0.90775        |
|                      | 0.07737        | 0.38662        | 0.92070        | 0.99999        | 0.99999        |
|                      | 0.10805        | 0.55150        | 0.96395        | 0.99999        | 0.99999        |
| $\gamma = 2.0$       | 0.00691        | 0.01320        | 0.02701        | 0.65068        | 0.81459        |
|                      | 0.03460        | 0.14955        | 0.32626        | 0.99999        | 0.99999        |
|                      | 0.05060        | 0.26159        | 0.50433        | 0.99999        | 0.99999        |
| $\gamma = 5.0$       | 0.00233        | 0.00368        | 0.00553        | 0.10302        | 0.52805        |
|                      | 0.01303        | 0.05250        | 0.10185        | 0.83154        | 0.99999        |
|                      | 0.01956        | 0.10182        | 0.19460        | 0.92210        | 0.99999        |

In Figure 3, we come back to the basic scenario (i.e. we set $(\gamma, \beta, \xi) = (1.0, 1.02, 0.01)$). Then, we draw the distribution function of the time to infection, restricted to individuals who become
infected before the outbreak ends; that is $P\{S_{i} \leq x|S_i < \infty\}$. The graphs correspond to 1, 25 and 50 initial infectives. If we start with 1 infective, the selected individual becomes infected in at most 5 units time with probability 0.39, but this probability is around 0.70 when the infectious disease involves at least 25 individuals. In general, for a fixed time $x$, we can see that the probability $P\{S_{i} \leq x|S_i < \infty\}$ increases as a function of the initial number of infectives. This shows again the importance of an early detection of the outbreak.

Since the probability $g(\beta) = P\{S_1 < \infty\}$ is an increasing function of $\beta$, we may find the value $\bar{\beta}$ such that the probability of becoming infected is sufficiently small; that is $g(\beta) < \epsilon$. For example, for $\epsilon = 0.03$ we observe that $g(0.25) = 0.0297$ and $g(0.26) = 0.0304$, so $\bar{\beta} = 0.25$. This illustrates that the computation of $P\{S_1 < \infty\}$ helps us to understand how much the contact rate should be reduced in order to control the epidemic spread at a desired level.

The results from Welsh schools data were summarized by Stone et al. [35] with a school of size 100 and one initial infective. However, the data collected in Table 3 of that paper show that the number of pupils checked in each school varies in a range from 24 (minimum size) to 201 (maximum size). Moreover, in their discussion, Stone et al. comment that it is impossible to know when an outbreak starts and ends, unless frequent checks of children are performed. These practical difficulties provide some motivation to study the epidemic characteristics for various choices of $N$ and $i$. This is done in the following.

Table 2 illustrates the behaviour of the two first moments of $L_i$ (i.e. the expectation and the standard deviation) and the probability of becoming infected, when $N$ varies in a range closer to the range observed in Welsh schools. The initial number of infectives takes values $i = 1, \lceil 0.05N \rceil, \lceil 0.10N \rceil, \lceil 0.25N \rceil$ and $\lceil 0.40N \rceil$, where $\lceil x \rceil$ is the ceiling function that gives the smallest integer larger or equal than $x$. The entries in the table measure the increasing effect of the population size and the initial number of infectives on the three selected infection characteristics. Moreover, for a fixed $N$, we observe that the three characteristics exhibit a wide range of variation. For example, for $N = 100$, the probability of infection $P\{S_i < \infty\}$ varies from 0.29203 to 0.93398. This shows again the importance of detecting the epidemic soon in order to facilitate the effort required to control the infection spread. The noteworthy differences observed for different values of $N$ motivate the need for an accurate estimation of the population size of each school. If the objective is to gain insight for the whole Welsh area, then a mixture, with appropriate weights, of the results obtained for several choices of $N$ could be helpful.
Table 2. Performance measures varying $N$ and $i$.

| $E[L_i] \sigma(L_i) P(S_i < \infty)$ | $N = 30$ | $N = 65$ | $N = 100$ | $N = 135$ | $N = 170$ | $N = 205$ |
|--------------------------------------|----------|----------|----------|----------|----------|----------|
| $i = 1$                             | 3.36257  | 6.68753  | 13.74157 | 25.26831 | 67.52877 | 158.89244 |
| $i = 0.05N$                         | 5.41608  | 12.11988 | 25.45760 | 45.29607 | 115.60655 | 256.51550 |
| $i = 0.10N$                         | 0.18803  | 0.22409  | 0.29207  | 0.37342  | 0.45526  | 0.52865  |
| $i = 0.25N$                         | 5.21413  | 14.05786 | 28.85272 | 52.49315 | 130.59850 | 283.68977 |
| $i = 0.40N$                         | 6.32719  | 15.31777 | 31.49979 | 54.41859 | 133.76732 | 287.35184 |
| $i = 0.50N$                         | 0.31166  | 0.50454  | 0.63397  | 0.78206  | 0.88145  | 0.94034  |
| $i = 0.75N$                         | 6.45181  | 17.11156 | 34.41010 | 58.81423 | 138.70265 | 293.38534 |
| $i = 0.95N$                         | 6.72013  | 15.85217 | 32.18647 | 54.85546 | 134.07593 | 287.54075 |
| $i = 1$                             | 0.40358  | 0.63585  | 0.77044  | 0.87730  | 0.93590  | 0.97075  |
| $i = 0.05N$                         | 9.35080  | 20.86563 | 39.23009 | 63.60564 | 144.71420 | 299.58522 |
| $i = 0.10N$                         | 7.17646  | 16.10818 | 32.37023 | 54.94491 | 134.13173 | 287.56646 |
| $i = 0.25N$                         | 0.65281  | 0.81343  | 0.89587  | 0.94760  | 0.97596  | 0.98914  |
| $i = 0.40N$                         | 10.29052 | 22.04059 | 40.65045 | 64.99889 | 146.18170 | 301.06668 |
| $i = 0.50N$                         | 7.22294  | 16.12733 | 32.38007 | 54.94924 | 134.13346 | 287.56717 |
| $i = 0.75N$                         | 0.74531  | 0.87299  | 0.93398  | 0.96718  | 0.98493  | 0.99329  |

4.2. SIR epidemic model

In this section, we consider an SIR epidemic model with $N = 30$. In Tables 3 and 4, we give the mean and the standard deviation, respectively, of the extinction time $L_{mn}$ for different choices of $\beta$ and $\gamma$. From top to bottom, each cell contains the entries for the initial states $(m, n) \in \{(1, 29), (15, 15), (29, 1)\}$.

First of all, it should be pointed out that the classical SIR model can be non-dimensionalized [26]. Once the time $t$ has been rescaled as $\tau = \gamma t$, the only relevant parameter is the ratio $\beta/\gamma$. This explains why the expectations associated with the pair $(\beta, \gamma) = (1.0, 1.0)$ have the corresponding expectations for $(\beta, \gamma) = (0.5, 0.5)$, while the quantities for $(\beta, \gamma) = (5.0, 5.0)$ are reduced 10-fold.

If we fix $\gamma$ and $(m, n)$, then we conclude that $E[L_{mn}]$ seems to have a maximum as a function of $\beta$. We also point out that it is easy to prove that $E[L_{mn}] \to (1/\gamma) \sum_{k=0}^{m} 1/k$, as $\beta \to 0$, while $E[L_{mn}] \to (1/\gamma) \sum_{k=0}^{m+1} 1/k$, as $\beta \to \infty$. On the other hand, it is clear that $E[L_{mn}]$ decreases with increasing values of $\gamma$. In fact, for fixed choices of $\beta$ and $(m, n)$, it is easy to prove that $E[L_{mn}] \to \infty$, as $\gamma \to 0$, and $E[L_{mn}] \to 0$, as $\gamma \to \infty$. Finally, for a fixed pair $(\beta, \gamma)$, we observe that among the values of $(m, n) \in \{(1, 29), (15, 15), (29, 1)\}$, $E[L_{mn}]$ is maximized when

Table 3. The mean extinction time $E[L_{mn}]$.

| $E[L_{mn}]$ | $\beta = 0.05$ | $\beta = 0.5$ | $\beta = 1.0$ | $\beta = 5.0$ | $\beta = 10.0$ |
|-------------|----------------|----------------|----------------|----------------|----------------|
| $\gamma = 0.5$ | 2.10268 | 3.86043 | 6.68164 | 8.01329 | 7.98511 |
|             | 6.87455 | 8.57078 | 9.03623 | 8.17696 | 8.06953 |
|             | 7.94191 | 8.02615 | 8.04014 | 7.99931 | 7.99387 |
| $\gamma = 1.0$ | 1.02489 | 1.33568 | 1.93021 | 4.08341 | 4.00664 |
|             | 3.37778 | 3.88558 | 4.28539 | 4.24926 | 4.08848 |
|             | 3.96647 | 3.99705 | 4.01307 | 4.00776 | 3.99965 |
| $\gamma = 2.0$ | 0.50613 | 0.57066 | 0.66784 | 1.87695 | 2.04170 |
|             | 1.67400 | 1.80662 | 1.94279 | 2.24597 | 2.12413 |
|             | 1.98205 | 1.99130 | 1.99852 | 2.00937 | 2.00388 |
| $\gamma = 5.0$ | 0.20097 | 0.21026 | 0.22188 | 0.38604 | 0.66816 |
|             | 0.66602 | 0.68745 | 0.71104 | 0.85707 | 0.90362 |
|             | 0.79252 | 0.79419 | 0.79580 | 0.80261 | 0.80401 |
Table 4. The standard deviation $\sigma(L_{mn})$.

| $\gamma$ | $\beta = 0.05$ | $\beta = 0.5$ | $\beta = 1.0$ | $\beta = 5.0$ | $\beta = 10.0$ |
|---------|----------------|----------------|----------------|----------------|----------------|
| 0.5     | 2.15571        | 4.62874        | 6.39547        | 3.59599        | 3.08677        |
|         | 2.63294        | 3.18315        | 2.99582        | 2.53983        | 2.53923        |
|         | 2.54595        | 2.56724        | 2.55992        | 2.53942        | 2.53939        |
| 1.0     | 1.03755        | 1.52032        | 2.31437        | 2.33676        | 1.79799        |
|         | 1.28715        | 1.50565        | 1.59157        | 1.29598        | 1.26991        |
|         | 1.27122        | 1.28131        | 1.28362        | 1.27118        | 1.26971        |
| 2.0     | 0.50922        | 0.60867        | 0.76016        | 1.57391        | 1.16838        |
|         | 0.63611        | 0.69920        | 0.75282        | 0.71802        | 0.64799        |
|         | 0.63513        | 0.63857        | 0.64065        | 0.63879        | 0.63559        |
| 3.0     | 0.20146        | 0.21557        | 0.23351        | 0.46287        | 0.63954        |
|         | 0.25263        | 0.26329        | 0.27445        | 0.31831        | 0.29958        |
|         | 0.25393        | 0.25459        | 0.25518        | 0.25672        | 0.25599        |

$(m, n) = (29, 1)$ for $\beta < \gamma$, while for $\beta \geq \gamma$ the maximum is reached at the balanced initial state $(15, 15)$.

The main conclusions inferred from Table 4 are that $\sigma(L_{mn})$ decreases as a function of $\gamma$, but it has a maximum as a function of $\beta$. A plausible explanation of this behaviour is as follows. If we view $\gamma$ as a scaling time parameter (see also Table 3), then we understand that increasing $\gamma$ reduces the mean and the variance of $L_{mn}$. When $\beta/\gamma < 1$, only small epidemics are expected, so the variance is small. In the case where $\beta/\gamma$ is slightly above one, we may find small and big epidemics and, as a result, the variance increases. Finally, for large values of $\beta/\gamma$, a rapid extinction is unlikely, so the variance becomes small again. For a fixed pair $(\beta, \gamma)$, several different patterns can be observed in the table.

In Figures 4 and 5, the epidemic is allowed to start with a single infective. Then, the density function of the extinction time $L_{1,29}$ is plotted for the parameter choices $(\beta, \gamma) \in \{(0.05, 0.5), (0.05, 5.0), (10.0, 0.5), (10.0, 5.0)\}$. Densities associated with pairs where $\beta < \gamma$ present a single mode at $x = 0$, while the case $\beta > \gamma$ leads to bimodal distributions. In this case, we observe a larger proper mode when the ratio $\beta/\gamma$ is larger (i.e. the case $(\beta, \gamma) = (10.0, 0.5)$). The initial value $f_{L_{1,29}}(0)$ is in agreement with the Tauberian result given in Section 3.1. This bimodal nature of the distribution agrees with the asymptotic approximation obtained by Barbour [6] (see Figure 2 in that paper). Barbour [6, pp. 478–479] gave an intuitive interpretation.
of the bimodal behaviour in terms of the trajectories associated with large epidemics. Those trajectories spend time twice near 0. The first time is due to the initial condition \( m = 1 \), while the second time is a consequence of taking \( \beta/\gamma \) sufficiently large, which make most realizations of the epidemic lead to a major outbreak, with relatively long extinction time. A similar discussion of the bimodal nature of the final size distribution is given on page 100 in [5] (see also [27]). If \( \beta \leq \gamma \), then one might expect only a minor epidemic, so the distribution is unimodal. In contrast, if \( \beta > \gamma \), for \( n \) sufficiently large, then either a minor epidemic occurs or a major epidemic occurs, so the distribution is bimodal.

For a better understanding of the bimodal nature of the extinction time density, we have performed some additional numerical experiments. For the initial state \((m, n) = (1, 99)\) and \( \gamma = 1.0 \), we have found that the density has two modes, one at the origin 0 plus a proper positive mode, while \( \beta \) remains greater than \( \hat{\beta} = 1.21731 \). For \( \beta \leq \hat{\beta} \), only the extreme mode at 0 is observed. The fact that the critical value of the contact rate for the distribution to be bimodal is larger than one (i.e. \( \hat{\beta} > 1 \)) is to be expected from the discussion given in [5,27] for the distribution of the total size of the epidemic. On the other hand, for fixed \( \gamma = 1.0 \) and \( \beta = 1.5 \), we have increased the initial number of infectives \( m \), but keeping \( m + n = 100 \). The conclusion was that \( f_{Lmn}(x) \) has two positive modes for \( 2 \leq m \leq 5 \). However, for the pair \((m, n) = (6, 94)\), we observed only one positive mode. These results corroborate that the existence of two modes is expected provided that \( m \) is small and the epidemic is well established.

Finally, in Table 5 we compute the first two moments of the removal time. In Remark 4, we have shown that the linear case \( \mu_i = \gamma i \) leads trivially to the exponential distribution. However, one way in which the model can be extended is to consider state dependent potential rates \( \lambda_{ij} = \beta i^a j^{1-b} \) and \( \mu_i = \gamma i^{1+c} \), as suggested by Severo [34] and Clancy and Green [8]. The powers \( a, b \) and \( c \) represent the infection power, the safety power and the removal power, respectively. In our numerical example, we assume that \((m, n) = (15, 15)\) and \( \lambda_{ij} = (\beta/30)\sqrt{ij} \) and \( \mu_i = \gamma \sqrt{i} \), for \( 0 \leq j \leq 15, 0 \leq i \leq 30 - j \). For this choice, as expected, we observe that \( R_{15,15} \) does not follow the exponential law. We notice that both the expectation, \( E[R_{15,15}] \), and the standard deviation, \( \sigma(R_{15,15}) \), are increasing functions of \( \beta \) but they are decreasing functions of \( \gamma \).

### 4.3. SIS model: analysis of the modes

The existence of bimodal distributions for the extinction time in the SIR model gives an initial motivation for a parallel study in the SIS model. To this end, we have carried out some numerical
Table 5. Mean and standard deviation of $R_{15,15}$.

| $\gamma$ | $E[R_{15,15}]$ | $\sigma(R_{15,15})$ |
|----------|----------------|-------------------|
| 0.5      | 5.41676        | 4.13972           |
|          | 5.60690        | 4.38859           |
| 1.0      | 2.70316        | 2.06306           |
|          | 2.75041        | 2.12473           |
| 2.0      | 1.35027        | 1.02983           |
|          | 1.36204        | 1.04516           |
| 5.0      | 0.53979        | 0.41152           |
|          | 0.54167        | 0.41397           |

Figure 6. Density function of $L_i$ when $N = 100$.

experiments, not reported here, for population sizes $N \in \{50, 100\}$ when the recovery rate is $\gamma = 1.0$ and the contact rate is chosen as $\beta \in \{1.5, 2.0, 2.5\}$. In all cases, we have obtained decreasing density functions, if $i = 1$, and unimodal densities if $i > 1$. To illustrate this situation, in Figure 6, we consider three densities for a population of $N = 100$ individuals. As is to be expected, the value of $f_{L_i}(0)$ obtained numerically is in agreement with the theoretical Tauberian result given in Section 2.1.

In order to explain why the density $f_{L_i}(x)$ is not bimodal, we recall that the extinction time of the SIS model, when the epidemic persists for a very long time, can be approximated by an exponential distribution with a very small rate [29,32]. The flat decreasing shape of such an exponential density helps to explain why the overall density of the extinction time is not bimodal.

In a second example, we deal with the density of the time to infection, $f_{S_i}(x)$, for the case $N = 100$. The three curves plotted in Figure 7 correspond to the contact and recovery rates and the initial number of infectives already used in Figure 6. We now observe that the initial value is $f_{S_i}(0) = \lambda_i/(N - i) = \beta i / N$. When $\beta > \gamma$, we see a proper mode which agrees with the fact that the epidemic is sufficiently large. In contrast, in the curve where $\beta < \gamma$, only the initial mode at 0 is observed.

As a general conclusion, we mention that it is not trivial to formulate intuitive explanations regarding the number of modes and the shape of the distribution. This problem depends on several facts including the number of system parameters, the nature of the rates (e.g. linear rates or logistic rates), the possible existence of interaction between different types of individuals (e.g. infectives
Figure 7. Density function of $S_i$ when $N = 100.$

and susceptibles in the SIR model) and the reproduction factor. The theoretical investigation seems complicated. Thus, the Laplace transform methodology used in this paper aims to provide a helpful tool.

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