

Comparison of deterministic and stochastic SIS and SIR models in discrete time

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Abstract

The dynamics of deterministic and stochastic discrete-time epidemic models are analyzed and compared. The discrete-time stochastic models are Markov chains, approximations to the continuous-time models. Models of SIS and SIR type with constant population size and general force of infection are analyzed, then a more general SIS model with variable population size is analyzed. In the deterministic models, the value of the basic reproductive number \mathcal{R}_0 determines persistence or extinction of the disease. If $\mathcal{R}_0 < 1$, the disease is eliminated, whereas if $\mathcal{R}_0 > 1$, the disease persists in the population. Since all stochastic models considered in this paper have finite state spaces with at least one absorbing state, ultimate disease extinction is certain regardless of the value of \mathcal{R}_0 . However, in some cases, the time until disease extinction may be very long. In these cases, if the probability distribution is conditioned on non-extinction, then when $\mathcal{R}_0 > 1$, there exists a quasi-stationary probability distribution whose mean agrees with deterministic endemic equilibrium. The expected duration of the epidemic is investigated numerically. © 2000 Elsevier Science Inc. All rights reserved.

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1. Introduction

The extinction behavior exhibited by stochastic population models is frequently not characteristic of their deterministic analogs. Even the simple stochastic exponential growth model has a finite probability of extinction (see e.g., [1]). It is the goal of this investigation to examine the relationship between some stochastic and deterministic epidemic models. In particular,

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deterministic and stochastic SIS and SIR models with constant population size and general force of infection and SIS models with variable population size are analyzed and compared. The models are formulated in terms of discrete-time approximations to the continuous-time models. In the deterministic case, the model is formulated as a system of difference equations and in the stochastic case, the model is a Markov chain.

Some of the first analyses of stochastic and deterministic continuous-time epidemic models are due to Bailey [2] and Bartlett [3]. The Reed-Frost and Greenwood models are probably the most well-known discrete-time stochastic epidemic models [2]. In these Markov chain models, it is assumed that the discrete-time interval corresponds to the length of the incubation period and the infectious period is assumed to have length zero. The contact process depends on the binomial distribution and hence, these models are referred to as chain-binomial models. Zero, one, or more than one infection may take place during the fixed time interval. Some extensions of the Reed-Frost model are discussed in [4] and applied to diseases such as polio and influenza. Ackerman et al. [4] performed extensive computer simulations of the epidemic models and studied the impact of various vaccination strategies.

Continuous and discrete-time stochastic SI models were analyzed by West and Thompson [5] and their behavior compared to the analogous deterministic models. In the SI model, the susceptible proportion eventually converges to zero; the entire population becomes infected. West and Thompson [5] showed that deterministic and stochastic SI models have much different convergence behavior when the size of the susceptible population is varied and that the behavior of the continuous and discrete-time stochastic models agree when the time steps are small.

Jacquez and O'Neill [6] and Jacquez and Simon [7] compared the behavior of a continuous-time stochastic SI epidemic model with recruitment and deaths to the analogous deterministic model. The extinction behavior of the stochastic model was demonstrated, a behavior not possible in the analogous deterministic models. However, when the probabilities in the stochastic model were conditioned on non-extinction, the deterministic and stochastic models were more closely related; a quasi-stationary state exists in the stochastic model whose mean is given by the deterministic endemic equilibrium.

Quasi-stationary distributions in discrete-time Markov chains were first studied by Seneta and Vere-Jones [8] and in continuous-time by Darroch and Seneta [9]. In an epidemic setting, quasi-stationary distributions in continuous time were first studied by Kryscio and Lefèvre [10]. Recently, Nåsell analyzed the quasi-stationary distribution for a continuous-time stochastic SIS model with no births and deaths [11,12] and continuous-time stochastic SIR models with births and deaths [13]. He showed that the quasi-stationary distribution has different forms depending on the value of \mathcal{R}_0 and its relationship to N , the total population size. Three different parameter regions determine the form of the quasi-stationary distribution. When \mathcal{R}_0 is less than 1, the distribution is approximately geometric and when \mathcal{R}_0 is greater than 1, the distribution is approximately normal. However, there exists a transition region when \mathcal{R}_0 is near 1, where the form of the distribution is more complex. The time to disease extinction is also determined by these three regions [13]. We investigate the form of the probability distribution for the number of infectives for our discrete-time models and note that the quasi-stationary distributions are approximately normal when $\mathcal{R}_0 > 1$.

Due to the generality of the force of infection, the discrete-time deterministic and stochastic models in this investigation are new formulations. The stochastic formulations are more general

than the models of West and Thompson [5] and Nåsell [11–13] and are more closely tied to the deterministic models than are the Reed-Frost models. If the time step is chosen sufficiently small, then the discrete-time deterministic and stochastic models approximate the behavior of the continuous-time models. In particular, there is agreement between the behavior shown in special cases of our models and in the continuous-time stochastic models of Jacquez and O’Neill [6], Jacquez and Simon [7], and Nåsell [11–13].

It is assumed that the time step is sufficiently small so that only one change in state is possible during the time step. A change may be either a birth or death of a susceptible or infected individual, recovery of an infected individual, or an infection of a susceptible individual. The transition probabilities in our models approximate the Poisson process generally applied in continuous-time models [14]. The probability of each event depends only on their state at the current time interval; thus, making the stochastic processes Markovian. The discrete-time stochastic models are formulated as Markov chains and in the simple case of an SIS model with constant population size, the entire transition matrix is given.

In the following sections, three models are discussed: SIS model with constant population size, SIR model with constant population size, and SIS model with variable population size. In each section, first, a summary and analysis of the dynamics of the deterministic model are given. Second, the corresponding stochastic model is formulated, analyzed, and compared to the deterministic model. The probability an epidemic occurs, the quasi-stationary distribution, the mean, the quasi-stationary mean, and the mean duration of the epidemic are discussed. Third, numerical results from the deterministic and stochastic simulations are presented and discussed.

2. SIS model with constant population size

2.1. Deterministic SIS

The discrete-time deterministic SIS model has the form

$$\begin{aligned} S(t + \Delta t) &= S(t)(1 - \lambda(t)\Delta t) + (\beta\Delta t + \gamma\Delta t)I(t), \\ I(t + \Delta t) &= I(t)(1 - \beta\Delta t - \gamma\Delta t) + \lambda(t)\Delta tS(t), \end{aligned} \quad (1)$$

where $t = n\Delta t$, $n = 0, 1, 2, \dots$, Δt is a fixed time interval (e.g., 1 h, one day), $S(0) > 0$, $I(0) > 0$ and $S(0) + I(0) = N$. It is assumed that the parameters are positive, $\alpha > 0$, $\beta > 0$ and $\gamma > 0$. It follows that $S(t) + I(t) = N$ for all time; the total population size remains constant. The function $\lambda(t)$ is the force of infection (number of contacts that result in infection per susceptible individual per unit time), $\beta\Delta t$ is the number of births or deaths per individual during the time interval Δt (number of births = number of deaths), and $\gamma\Delta t$ is the removal number (number of individuals that recover in the time interval Δt). Individuals recover but do not develop immunity, they are immediately susceptible. In addition, it is assumed that there are no deaths due to the disease, no recruitment, and no vertical transmission of the disease (all newborns are susceptible). Since births can be combined with recoveries, $\gamma' = \beta + \gamma$, model (1) is equivalent to an SIS model without any births or deaths.

Model (1) generalizes epidemic models considered in [15] through the form of the force of infection. In [15], the force of infection was assumed to have the form

$$\lambda(t) = \frac{\alpha}{N}I(t), \quad (2)$$

where α is the contact rate, the number of successful contacts made by one infectious individual during a unit time interval. In this case, the incidence rate (number of new cases per unit time) $\lambda(t)S(t)$ is referred to as the standard incidence rate [16]. When the population size is constant, the standard incidence has the same form as the mass action incidence rate: constant $I(t)S(t)$. Another form for the force of infection arises from the Poisson distribution. The ratio $\mu = \alpha I \Delta t / N$ is the average number of infections per susceptible individual in time Δt . The probability of k successful encounters resulting in a susceptible individual becoming infective is assumed to follow a Poisson distribution:

$$p(k) = \frac{\exp(-\mu)\mu^k}{k!}.$$

Only one successful encounter is necessary for an infection to occur; therefore, when there are no successful encounters, the expression

$$p(0) = \exp(-\mu) = \exp\left(-\frac{\alpha \Delta t}{N}I\right)$$

represents the probability that a susceptible individual does not become infective. The number of susceptibles that do not become infective in time Δt is $S(t) \exp(-\alpha \Delta t I / N)$ and the number of susceptibles that do become infective is $S(t)(1 - \exp(-\alpha \Delta t I / N))$. Thus, another form for the force of infection $\lambda(t)\Delta t$ is

$$1 - \exp\left(-\frac{\alpha \Delta t}{N}I(t)\right). \quad (3)$$

The force of infection in (3) was applied to discrete-time epidemic models studied by Cooke et al. [17]. The force of infection in (2) can be seen to be a linear approximation to the one given in (3). Other forms for the force of infection are discussed by Hethcote [16].

Several general assumptions are made regarding the force of infection which includes the particular forms considered above:

- (i) $0 < \lambda(t) \equiv \lambda(I(t)) \leq \alpha I(t) / N$ for $I \in (0, N]$.
- (ii) $\lambda(I) \in C^2[0, N]$, $d\lambda(I)/dI > 0$, and $d^2\lambda(I)/dI^2 \leq 0$ for $I \in [0, N]$.
- (iii) $\lambda(I) |_{I=0} = 0$ and $\lambda'(I) |_{I=0} = \alpha/N$.
- (iv) $0 < (\beta + \gamma)\Delta t \leq 1$ and $0 < \alpha \Delta t \leq 1$.

The above assumptions imply that the force of infection increases with the number of infectives at a decreasing rate and is bounded above by a linear function of the number of infectives. The incidence rate is bounded above by the standard incidence rate of infection. Conditions (i)–(iv) are sufficient to guarantee non-negative solutions and asymptotic convergence to an equilibrium. However, they are not necessary conditions, for example, less restrictive assumptions on $\alpha \Delta t$ guarantee convergence to an equilibrium in the case of (2) [15]. In the analogous stochastic model, even more restrictive conditions will be put on the parameters to guarantee that the transition probabilities are true probabilities.

Model (1) with the force of infection given by (2) was analyzed by Allen [15] and the one with force of infection (2) by Cooke et al. [17] and Sumpter [18]. For the more general model (1), it is straightforward to show that conditions (i) and (iv) imply solutions are non-negative.

The basic reproductive number \mathcal{R}_0 determines asymptotic behavior of (1) and is expressed in terms of the model parameters as

$$\mathcal{R}_0 = \frac{\alpha}{\beta + \gamma}.$$

The basic reproductive number is defined as the average number of secondary infections caused by one infective individual during his/her infective period in an entirely susceptible population [19].

Theorem 1. (i) If $\mathcal{R}_0 \leq 1$, then solutions to (1) approach the disease-free equilibrium

$$\lim_{t \rightarrow \infty} I(t) = 0, \quad \lim_{t \rightarrow \infty} S(t) = N.$$

(ii) If $\mathcal{R}_0 > 1$, then solutions to (1) approach a unique positive endemic equilibrium

$$\lim_{t \rightarrow \infty} I(t) = \bar{I} > 0, \quad \lim_{t \rightarrow \infty} S(t) = \bar{S} > 0.$$

Proof. Denote the right side of $I(t + \Delta t)$ in (1) by $g(I)$

$$g(I) = I(1 - \beta\Delta t - \gamma\Delta t) + \lambda(I)\Delta t(N - I).$$

Note that

$$g'(I) = 1 - \beta\Delta t - \gamma\Delta t + \lambda'(I)\Delta t(N - I) - \lambda(I)\Delta t,$$

$$g''(I) = \lambda''(I)\Delta t(N - I) - 2\lambda'(I)\Delta t.$$

Since $\lambda''(I) \leq 0$ and $\lambda'(I) > 0$ for $I \in [0, N]$, it follows that $g''(I) < 0$ for $I \in [0, N]$.

For case (i), where $\mathcal{R}_0 \leq 1$, $g(0) = 0$ and $g'(0) \leq 1$. Since $g''(I) < 0$, $g'(I) < 1$ or $g(I) < I$ for $I \in (0, N]$. It follows that $\{I(t)\}$ is a strictly decreasing sequence bounded below by zero and must approach a fixed point of g on $[0, N]$. The only fixed point of g on $[0, N]$ is 0; hence, $\lim_{t \rightarrow \infty} I(t) = 0$.

For case (ii), where $\mathcal{R}_0 > 1$, it shown that there exists a unique $\bar{I} > 0$ such that $g(\bar{I}) = \bar{I}$, $g(I) > I$ for $I \in (0, \bar{I})$ and $g(I) < I$ for $I \in (\bar{I}, N]$. In this case, $g(0) = 0$, $g(N) < N$, and $g'(0) > 1$. Thus, there exists at least one fixed point $\bar{I} > 0$, $g(\bar{I}) = \bar{I}$. Let \bar{I} be the smallest positive fixed point, then $g(I) > I$ for $I \in (0, \bar{I})$. It follows that $g'(\bar{I}) \leq 1$. Since $g''(I) < 0$, $g'(I) < g'(\bar{I}) \leq 1$ for $I \in (\bar{I}, N]$. Integration of the last inequality over the interval $[\bar{I}, I]$ shows that $g(I) < I$ for $I > \bar{I}$. Thus, g has a unique positive fixed point \bar{I} .

The possibility of two-cycles, $g(I_1) = I_2$ and $g(I_2) = I_1$ is ruled out by showing that $1 + g'(I) > 0$ for $I \in [0, N]$ [20]. If $I_1 < I_2$, $I_1, I_2 \in (0, N)$, then

$$0 < \int_{I_1}^{I_2} (1 + g'(I)) dI = I_2 - I_1 + g(I_2) - g(I_1) = 0,$$

a contradiction. Now, for $I \in [0, N)$, $1 + g'(I) > 1 - \lambda(I)\Delta t \geq 1 - \alpha\Delta t/N \geq 1 - \alpha\Delta t \geq 0$. In addition, McCluskey and Muldowney [20] proved that the condition $1 + g'(I) \neq 0$ implies non-existence of any m -cycle for $m > 1$. A result of Cull [21] can be applied. The difference equation $I(t + \Delta t) = g(I(t))$ is a population model as defined by Cull [21]: g has a unique positive fixed point $\bar{I} > 0$ such that $g(I) > I$ for $I \in (0, \bar{I})$ and $g(I) < I$ for $I \in (\bar{I}, N]$, g has a unique maximum, and is strictly increasing before the maximum and strictly decreasing after

the maximum. The non-existence of two-cycles for the population model $I(t + \Delta t) = g(I(t))$ implies global stability of \bar{I} (Theorem 1, p. 143, [21]). \square

The value of the endemic equilibrium depends on the form of the force of infection. With the force of infection given by (2), the endemic equilibrium is

$$\bar{I} = N(1 - 1/\mathcal{R}_0),$$

which agrees with the analogous continuous-time model. With the force of infection (3), the endemic equilibrium is the positive implicit solution of

$$\exp\left(\frac{-\alpha\Delta t}{N}\bar{I}\right) = \frac{N - (1 + \beta\Delta t + \gamma\Delta t)\bar{I}}{N - \bar{I}}.$$

2.2. Stochastic SIS

2.2.1. Model

The discrete-time stochastic SIS model is a Markov chain with finite state space. The corresponding continuous-time model is a Markov jump process with the jumps forming a Markov chain. In the discrete-time model, it is assumed that at most one event occurs in the time period Δt , either an infection, birth, death, or recovery which depends only on the values of the state variables at the current time. Since the population size remains constant, a birth and death must occur simultaneously.

Let \mathcal{I} and \mathcal{S} denote random variables for the number of infectives and susceptibles, respectively, in a population of size N . The random variable \mathcal{I} is integer-valued with state probabilities $p_i(t) = \text{Prob}\{\mathcal{I}(t) = i\}$ for $i \in \{0, 1, \dots, N\}$ and time $t \in \{0, \Delta t, 2\Delta t, \dots\}$. Let the probability of a new infective in time Δt be $\Pi_i\Delta t = \lambda(i)\Delta t(N - i)$, where $\lambda(i)$ denotes the force of infection, e.g., in case (2), $\lambda(i) = \alpha i/N$. If $i \notin [0, N]$, then $\Pi_i = 0$. Let the probability of recovery or death in time Δt be $(\beta + \gamma)i\Delta t$. Since \mathcal{I} has a finite state space, in Refs. [6,7], the N in case (2) is replaced by $s + i - 1$, then $s/(s + i - 1)$ is the proportion of susceptibles that can be infected by one infective. However, in the models considered here, it is assumed that $s/(s + i - 1) \approx s/N = (N - i)/N$. Thus, the transition probabilities for the SIS model are

$$\text{Prob}\{\mathcal{I}(t + \Delta t) = i + 1 \mid \mathcal{I}(t) = i\} = \Pi_i\Delta t,$$

$$\text{Prob}\{\mathcal{I}(t + \Delta t) = i - 1 \mid \mathcal{I}(t) = i\} = (\beta + \gamma)i\Delta t.$$

The probabilities $p_i(t)$ satisfy the following difference equations:

$$p_i(t + \Delta t) = p_{i-1}(t)\Pi_{i-1}\Delta t + p_{i+1}(t)(\beta + \gamma)(i + 1)\Delta t \\ + p_i(t)[1 - \Pi_i\Delta t - \beta i\Delta t - \gamma i\Delta t],$$

$$p_0(t + \Delta t) = p_0(t),$$

where $i = 1, \dots, N$ and $p_i(t) = 0$ for $i \notin \{0, 1, \dots, N\}$.

The above transition probabilities approximate the transition probabilities in a continuous-time Markov jump process, where the transition probabilities follow a Poisson process and the time between jumps is given by an exponential distribution with mean $1/[\Pi_i + (\beta + \gamma)i]$. The probability of recovery or death of an infective in the continuous-time model satisfies

$$\text{Prob}\{\mathcal{I}(t + \Delta t) = i - 1 \mid \mathcal{I}(t) = i\} = (\beta + \gamma)i\Delta t + o(\Delta t).$$

If $\Delta t \rightarrow 0$, the continuous-time model is obtained. For Δt sufficiently small, the discrete-time process assumes that the transition probability is given by $(\beta + \gamma)i\Delta t$.

The difference equations for the discrete-time model can be expressed in matrix form with the definition of the $N + 1 \times N + 1$ transition matrix. Let

$$T = \begin{pmatrix} 1 & (\beta + \gamma)\Delta t & 0 & 0 & \dots & 0 \\ 0 & 1 - \Pi_1\Delta t - (\beta + \gamma)\Delta t & 2(\beta + \gamma)\Delta t & 0 & \dots & 0 \\ 0 & \Pi_1\Delta t & 1 - \Pi_2\Delta t - 2(\beta + \gamma)\Delta t & 3(\beta + \gamma)\Delta t & \dots & 0 \\ \vdots & \vdots & \vdots & \vdots & \dots & \vdots \\ 0 & 0 & 0 & 0 & \dots & N(\beta + \gamma)\Delta t \\ 0 & 0 & 0 & 0 & \dots & 1 - N(\beta + \gamma)\Delta t \end{pmatrix}.$$

and $p(t)^\top = (p_0(t), p_1(t), \dots, p_N(t))$. The probability density for \mathcal{I} satisfies $p(t + \Delta t) = Tp(t)$.

To ensure that the elements of T are probabilities it is required that

$$\Pi_i\Delta t + (\beta + \gamma)i\Delta t \leq 1.$$

The following restrictions on the parameters are sufficient to guarantee that the elements of T are less than 1:

$$N(\alpha + \beta + \gamma)^2\Delta t \leq 4\alpha \text{ if } \mathcal{R}_0 > 1 \quad \text{and} \quad (\beta + \gamma)N\Delta t \leq 1 \text{ if } \mathcal{R}_0 \leq 1. \tag{4}$$

These restrictions are satisfied if Δt is sufficiently small. Note that they are stronger conditions than those imposed in the deterministic model.

Matrix T is a stochastic matrix with a single absorbing state, the zero state. From the theory of Markov chains, it follows that $\lim_{t \rightarrow \infty} p_0(t) = 1$ [14]. Eventually, there are no infectives in the population, regardless of the threshold value \mathcal{R}_0 . However, for $\mathcal{R}_0 > 1$, it may take a long time for the disease to be completely eliminated. For the continuous-time stochastic SIS model without births and standard incidence, it has been shown by Kryscio and Lefèvre [10] and Nåsell [11,12] that the time until absorption increases exponentially in N as N approaches infinity when $\mathcal{R}_0 > 1$.

2.2.2. Quasi-stationary distribution

A closer relationship between the stochastic and deterministic models can be seen through examination of another probability distribution referred to as the quasi-stationary probability distribution. Define

$$q_i(t) = \frac{p_i(t)}{1 - p_0(t)},$$

and $q(t)^\top = (q_1(t), q_2(t), \dots, q_N(t))$, where $i = 1, \dots, N$. The probability q is conditioned on non-extinction. The quasi-stationary distribution has been studied in continuous-time stochastic SI, SIS, and SIR models with standard incidence of infection [6,7,11–13].

The difference equations for q_i can be shown to satisfy

$$q_i(t + \Delta t)[1 - (\beta + \gamma)q_1(t)\Delta t] = q_{i-1}(t)\Pi_{i-1}\Delta t + q_{i+1}(t)(\beta + \gamma)\Delta t(i + 1) + q_i(t)[1 - \Pi_i\Delta t - (\beta + \gamma)i\Delta t], \tag{5}$$

where $i = 1, \dots, N$ and $q_i(t) = 0$ if $i \notin [1, N]$. This equation agrees with the difference equation for the probability function p with the exception of the factor $[1 - (\beta + \gamma)q_1(t)\Delta t]$.

A time-independent solution of (5) is a stationary or equilibrium solution, $q^{*T} = (q_1^*, q_2^*, \dots, q_N^*)$. It can be seen that a stationary solution satisfies the eigenvalue equation, $Tx^* = \delta x^*$, where $x^{*T} = (-1, q_1^*, \dots, q_N^*)$ and δ is the eigenvalue $[1 - (\beta + \gamma)q_1^*\Delta t]$. A similar relationship was shown by Jacquez and Simon [7] and Nåsell [11,12] for the continuous-time stochastic SIS model with standard incidence. After simplification, the matrix equation, $(T - I)x^*/\Delta t = -(\beta + \gamma)q_1^*x^*$, $\Delta t \neq 0$, is the same as the matrix equation satisfied by the continuous-time model. Nåsell describes an iterative procedure for calculating the quasi-stationary distribution.

Two approximations to the quasi-stationary distribution can be derived for q^* which also agree with the continuous-time approximations [7,10–12]. One approximation assumes there is no decrease in the number of infectives when $\mathcal{I}(t) = 1$. This first approximation \tilde{q} is the positive eigenvector associated with eigenvalue one of the reduced stochastic matrix \tilde{T} ; the first row and first column of T are deleted and $(\beta + \gamma)\Delta t$ is set to 0 in the second row and second column of T :

$$\tilde{T} = \begin{pmatrix} 1 - \Pi_1\Delta t & 2(\beta + \gamma)\Delta t & 0 & \dots & 0 \\ \Pi_1\Delta t & 1 - \Pi_2\Delta t - 2(\beta + \gamma)\Delta t & 3(\beta + \gamma)\Delta t & \dots & \cdot \\ \cdot & \cdot & \cdot & \dots & \cdot \\ 0 & 0 & 0 & \dots & N(\beta + \gamma)\Delta t \\ 0 & 0 & 0 & \dots & 1 - N(\beta + \gamma)\Delta t \end{pmatrix}.$$

Matrix \tilde{T} is a transition matrix of a regular Markov chain. The eigenvector \tilde{q} is the stationary distribution of \tilde{T} . The components of \tilde{q} can be shown to equal the following expressions:

$$\tilde{q}_n = \tilde{q}_1 \frac{\Pi_{n-1}\Pi_{n-2}\dots\Pi_1}{n!(\beta + \gamma)^{n-1}}, \quad n = 2, \dots, N, \quad 1 = \sum_{i=1}^N \tilde{q}_i.$$

In the special case of standard incidence (2), the formulas for the approximate quasi-stationary distribution simplify to those given by the continuous-time model [7,10–12]:

$$\begin{aligned} \tilde{q}_n &= \tilde{q}_1 \frac{(N - 1)!}{n(N - n)!} \left(\frac{\mathcal{R}_0}{N}\right)^{n-1}, \quad n = 2, \dots, N, \\ \tilde{q}_1 &= \left[\sum_{k=1}^N \frac{(N - 1)!}{k(N - k)!} \left(\frac{\mathcal{R}_0}{N}\right)^{k-1} \right]^{-1}. \end{aligned} \tag{6}$$

A second approximation to the quasi-stationary distribution assumes

$$\text{Prob}\{\mathcal{I}(t + \Delta t) = i - 1 \mid \mathcal{I}(t) = i\} = (\beta + \gamma)(i - 1)\Delta t.$$

In this second approximation, the approximate quasi-stationary distribution again satisfies $\tilde{T}\tilde{q} = \tilde{q}$, where \tilde{q} is the stationary distribution of the regular Markov chain and

$$\tilde{T} = \begin{pmatrix} 1 - \Pi_1\Delta t & (\beta + \gamma)\Delta t & 0 & \dots & 0 \\ \Pi_1\Delta t & 1 - \Pi_2\Delta t - (\beta + \gamma)\Delta t & 2(\beta + \gamma)\Delta t & \dots & 0 \\ \cdot & \cdot & \cdot & \dots & \cdot \\ 0 & 0 & 0 & \dots & (N - 1)(\beta + \gamma)\Delta t \\ 0 & 0 & 0 & \dots & 1 - (N - 1)(\beta + \gamma)\Delta t \end{pmatrix}.$$

In the case of standard incidence, the stationary distribution for this second approximation is given by

$$\tilde{q}_n = \tilde{q}_1 \frac{(N-1)!}{(N-n)!} \left(\frac{\mathcal{R}_0}{N}\right)^{n-1}, \quad n = 2, \dots, N,$$

$$\tilde{q}_1 = \left[\sum_{k=1}^N \frac{(N-1)!}{(N-k)!} \left(\frac{\mathcal{R}_0}{N}\right)^{k-1} \right]^{-1}. \quad (7)$$

The approximate quasi-stationary distributions, \tilde{q} , given by (6) and (7) with the quasi-stationary distribution q^* calculated via the implicit relation $(T - I)x^*/\Delta t = -(\beta + \gamma)q_1^*x^*$, are graphed in Figs. 1 and 2 for $N = 50$ and $\mathcal{R}_0 = 1.5, 2,$ and 3 . For $\mathcal{R}_0 = 2$ and 3 , the distributions agree

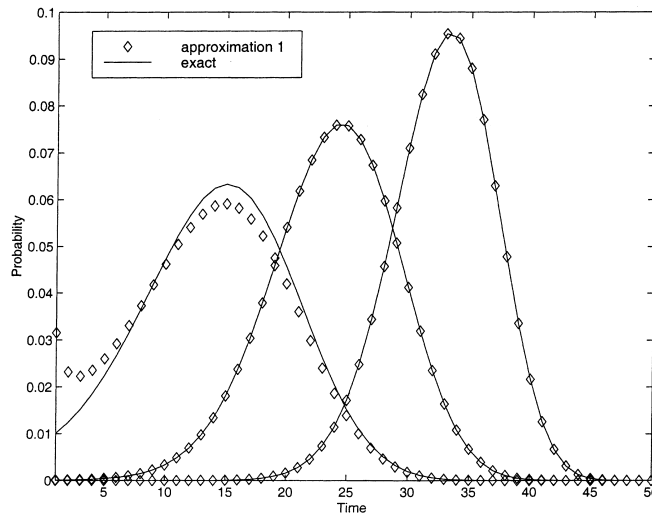


Fig. 1. The quasi-stationary probability distribution and the approximate distribution given by (6) when $N = 50$ and $\mathcal{R}_0 = 1.5, 2,$ and 3 .

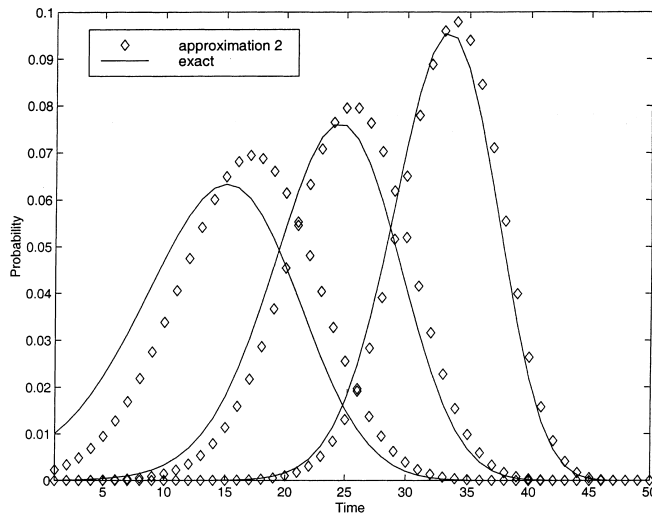


Fig. 2. The quasi-stationary probability distribution and the approximate distribution given by (7) when $N = 50$ and $\mathcal{R}_0 = 1.5, 2,$ and 3 .

reasonably well with the quasi-stationary distribution which assumes an approximate normal shape. However, for smaller values of N and \mathcal{R}_0 small, the approximations are not as good and the distribution may not have a normal shape [11,12]. Also, note that as \mathcal{R}_0 increases, the variance of the quasi-stationary distribution decreases.

2.2.3. *Mean and quasi-stationary mean*

The mean of the probability distributions of p and q satisfy

$$m(t) = \sum_{i=0}^N ip_i(t), \quad m^*(t) = \sum_{i=1}^N iq_i(t).$$

Applying the difference equations for p and q , another set of difference equations for the mean, $m(t)$, and quasi-stationary mean, $m^*(t)$, can be derived:

$$m(t + \Delta t) - m(t) = \sum_{i=0}^N \left[\frac{\Pi_i \Delta t}{i} - \beta \Delta t - \gamma \Delta t \right] ip_i(t) = (\beta + \gamma) \Delta t [\mathcal{R}_0 \xi(t) - 1] m(t), \tag{8}$$

$$m^*(t + \Delta t) [1 - (\beta + \gamma) q_1(t) \Delta t] - m^*(t) = (\beta + \gamma) \Delta t [\mathcal{R}_0 \xi^*(t) - 1] m^*(t),$$

where

$$\xi(t) = \frac{\sum_{i=0}^N p_i(t) \Pi_i}{\alpha \sum_{i=0}^N ip_i(t)} \quad \text{and} \quad \xi^*(t) = \frac{\sum_{i=1}^N q_i(t) \Pi_i}{\alpha \sum_{i=1}^N iq_i(t)}.$$

Note that $\Pi_i/i \leq \alpha s/N$; thus, $\xi(t)$ and $\xi^*(t)$ are strictly less than 1.

The asymptotic behavior of the mean, $m(t)$, depends on the basic reproductive number, \mathcal{R}_0 . When $\mathcal{R}_0 \leq 1$, it follows from (8) that the mean $m(t)$ of the stochastic process is strictly decreasing, bounded below by zero, and therefore, must approach an equilibrium. Since the only non-negative equilibrium for $m(t)$ is 0, $m(t)$ approaches 0. When $\mathcal{R}_0 > 1$, however, the mean $m(t)$ has an additional steady-state given by

$$\xi(t) = \frac{1}{\mathcal{R}_0}.$$

In the particular case of (2), where $\Pi_i = \alpha is/N$, the above expression can be written as

$$m(t) \left[N \left(1 - \frac{1}{\mathcal{R}_0} \right) - m(t) \right] = \sigma^2(t),$$

where $\sigma^2(t)$ is the variance. The deterministic endemic equilibrium is $\bar{I} = N(1 - 1/\mathcal{R}_0)$. Hence, the above inequality shows that when the mean of the stochastic process is approximately constant, it is less than the deterministic equilibrium.

The mean of the distribution of q^* is calculated for various values of N and \mathcal{R}_0 in Table 1 and is compared to the deterministic endemic equilibrium \bar{I} . There is closer agreement between the quasi-stationary mean of the stochastic process and the deterministic endemic equilibrium when \mathcal{R}_0 and N are large. The agreement is poorer when \mathcal{R}_0 is small. Also, note the quasi-stationary mean lies below the endemic equilibrium.

Table 1
The quasi-stationary mean m^* and the deterministic equilibrium \bar{I} , (m^*/\bar{I})

N	$\mathcal{R}_0 = 3$	$\mathcal{R}_0 = 2$	$\mathcal{R}_0 = 1.5$
25	16.09/16.67	11.27/12.5	7.26/8.33
50	32.80/33.33	23.83/25	14.48/16.67

2.2.4. *Relation to a random walk*

If the population size N is sufficiently large, initially, the nonlinear stochastic process can be approximated by a random walk on $[0, \infty)$ with an absorbing barrier at zero. If p denotes the probability of moving to the right and q the probability of moving to the left, then the probability of eventual absorption beginning from position $a > 0$ is

$$\left(\frac{q}{p}\right)^a \text{ if } q < p \text{ and } 1 \text{ if } q \geq p.$$

[14]. If p and q are interpreted in terms of the epidemic model, then p denotes the probability of becoming infected, $\Pi_i \Delta t$, and q the probability of recovery or death, $(\beta + \gamma) i \Delta t$. When there are a small number of infectives, $s \approx N$ and $\Pi_i \approx \alpha i$, so that the probability of no epidemic is $(q/p)^a \approx [(\beta + \gamma)/\alpha]^a$, or approximately

$$\left(\frac{1}{\mathcal{R}_0}\right)^a \text{ if } \mathcal{R}_0 > 1 \text{ and } 1 \text{ if } \mathcal{R}_0 \leq 1,$$

where a is the initial number of infectives. The numerical examples show that the value of $p_0(t)$ (probability of no epidemic) approaches $(1/\mathcal{R}_0)^a$ at the outset of the epidemic.

2.2.5. *Expected duration of the epidemic*

Let τ_j be the probability distribution for the time to extinction beginning with j infectives. A system of difference equations for the expected duration of the epidemic, $E(\tau_j)$, for the discrete-time model takes the form

$$\begin{aligned} E(\tau_j) &= \Delta t + j(\beta + \gamma)\Delta t E(\tau_{j-1}) + \Pi_j \Delta t E(\tau_{j+1}) \\ &\quad + [1 - \Pi_j \Delta t - (\beta + \gamma)j\Delta t]E(\tau_j), \\ E(\tau_1) &= \Delta t + \Pi_1 \Delta t E(\tau_2) + [1 - \Pi_1 \Delta t - (\beta + \gamma)\Delta t]E(\tau_1) \end{aligned}$$

for $j = 2, \dots, N$. Assuming that $\Delta t \neq 0$, the system above can be expressed in the same form as the continuous-time SIS model [12]:

$$\begin{aligned} E(\tau_j) &= \frac{1}{\Pi_j + j(\beta + \gamma)} + \frac{\Pi_j}{\Pi_j + j(\beta + \gamma)} E(\tau_{j+1}) + \frac{j(\beta + \gamma)}{\Pi_j + j(\beta + \gamma)} E(\tau_{j-1}), \\ E(\tau_1) &= \frac{1}{\Pi_1 + (\beta + \gamma)} + \frac{\Pi_1}{\Pi_1 + (\beta + \gamma)} E(\tau_2) \end{aligned} \tag{9}$$

for $j = 2, \dots, N$. This system of linear equations can be solved explicitly for $E(\tau_j)$ [1,22]. It was shown for the continuous-time SIS model with standard incidence that, when $\mathcal{R}_0 > 1$, the expected duration of the epidemic increases exponentially in N [10–12]. This exponential increase in the expected duration can be observed in the numerical examples in the next section.

2.3. Numerical examples

Several numerical examples are simulated with various values for the population size, N , the initial number of infectives, \mathcal{I} , and the basic reproductive number, \mathcal{R}_0 . In all the simulations, the incidence rate has the form of the standard incidence rate of infection, $\lambda(t)S(t) = \alpha I(t)S(t)/N$.

In the first example, the behavior of individual sample paths of the stochastic model are compared to the deterministic solution. Three sample paths of the stochastic model are graphed against the corresponding deterministic solution in Fig. 3. Initially, one infective is introduced into

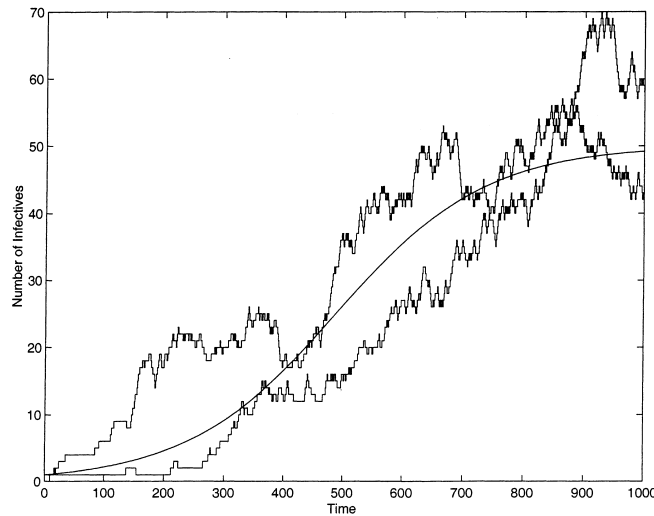


Fig. 3. Three sample paths of stochastic SIS model are graphed with the deterministic solution when $\mathcal{R}_0 = 2$, $\mathcal{I}(0) = 1$, $\mathcal{S}(0) = 99$, $\alpha = 1.6$, $\beta = 0.4 = \gamma$, and $\Delta t = 0.01$.

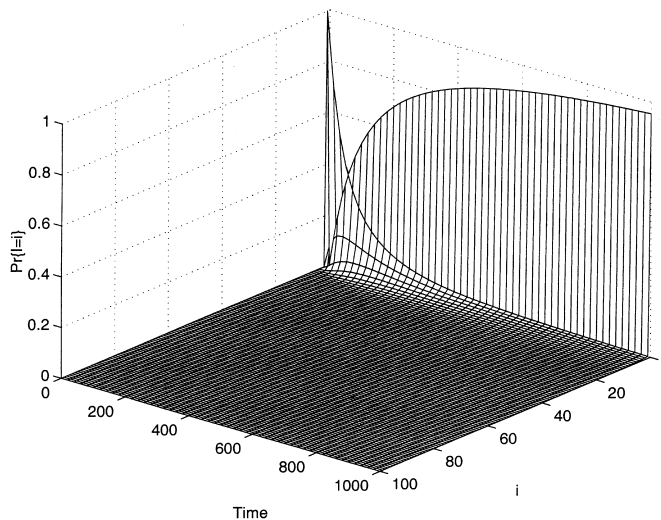


Fig. 4. The probability function, $p(t)$, for the stochastic SIS model is graphed when $\mathcal{R}_0 = 0.9$, $\mathcal{I}(0) = 1$, $\mathcal{S}(0) = 99$, $\alpha = 0.9$, $\beta = 0.5 = \gamma$, and $\Delta t = 0.01$.

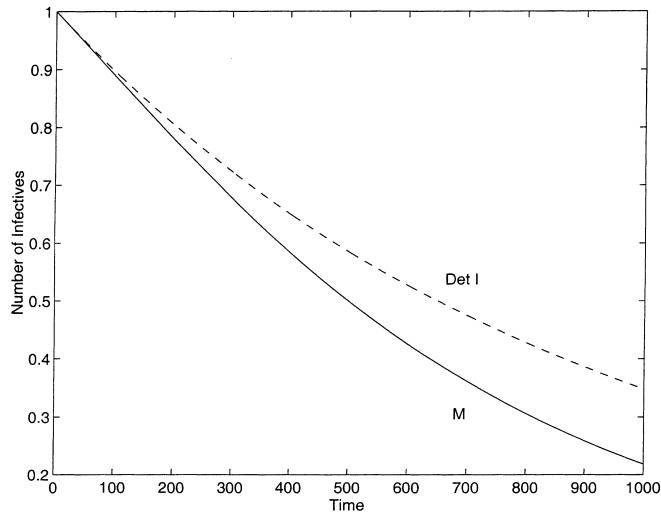


Fig. 5. The mean, M , of the stochastic SIS model and the deterministic solution, $\text{Det } I$, are graphed for the probability function and parameter values given in Fig. 4.

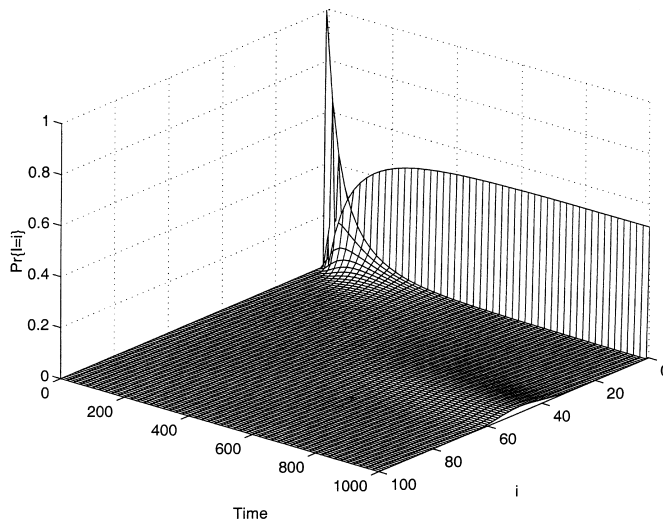


Fig. 6. The probability function, $p(t)$, for the stochastic SIS model is graphed when $\mathcal{R}_0 = 2$, $\mathcal{I}(0) = 1$, $\mathcal{S}(0) = 99$, $\alpha = 1.6$, $\beta = 0.4 = \gamma$, and $\Delta t = 0.01$.

a population of size $N = 100$ with $\mathcal{R}_0 = 2$. The time step is $\Delta t = 0.01$ and the Time axis is the number of time steps, e.g., Time = 1000 means 1000 time steps and thus, an actual total time of $1000\Delta t = 10$. One of the sample paths reaches 0 very quickly and the other two sample paths vary about the deterministic solution.

The probabilities for the number of infectives at each time step Δt can be obtained directly from the equation $p(t + \Delta t) = Tp(t)$. In Figs. 4–7, one infective is introduced into the population of size

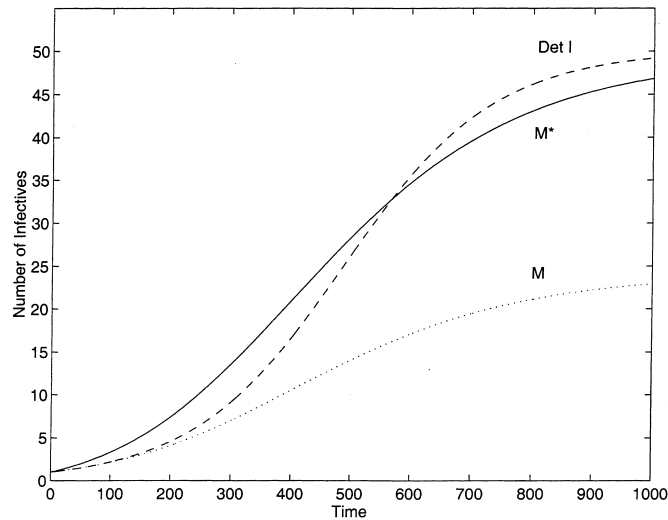


Fig. 7. The mean, M , the mean conditioned on non-extinction, M^* , of the stochastic SIS model and the deterministic solution, $\text{Det } I$, are graphed for the probability function and parameter values given in Fig. 6.

$N = 100$. When the basic reproductive number $\mathcal{R}_0 = 0.9$, the probability of no epidemic, $p_0(t)$, quickly approaches 1 (see Fig. 4). The mean of the stochastic process and the deterministic solution both approach 0 (see Fig. 5). When the basic reproductive number $\mathcal{R}_0 = 2$, the probability distribution is bimodal (Fig. 6); one mode is at zero and the second mode is approximated by the endemic equilibrium; the endemic equilibrium is $\bar{I} = 50$, whereas the mean of the distribution is $i \approx 46.8$ at $t = 1000\Delta t = 10$. If the probability at zero is neglected, the remaining probabilities represent the quasi-stationary distribution, $q(t)$, which appears to be approximately normal. The mean of the probability density $p(t)$, M , and the mean of $q(t)$, M^* , are graphed in Fig. 7 and compared to the deterministic solution, $\text{Det } I$. It can be seen that the mean, M , lies below the deterministic solution and the mean of the quasi-stationary distribution, M^* , is less than but much closer than M to the deterministic solution.

The value of $p_0(t)$ initially approaches the value estimated from the random walk. In Fig. 6, the probability that the disease is eliminated, $p_0(t)$, is approximately $1/\mathcal{R}_0 = 0.5$ for the time frame shown ($p_0(1000\Delta t) = p_0(10) \approx 0.51$). In Fig. 8, the initial number of infectives is increased to five individuals, then according to the estimate given by the random walk, $p_0(t)$ is approximately $(1/\mathcal{R}_0)^5 = 0.03125$, and $p_0(1000\Delta t) = p_0(10) \approx 0.039$.

The mean and mean conditioned on non-extinction show closer agreement to the deterministic solution as the population size and the initial number of infectives are increased. The mean and the quasi-stationary mean for the state probabilities (with five initial infectives), given in Fig. 8, are graphed in Fig. 9. Note that there is closer agreement between the means and the deterministic solution; however, the means are always less than the deterministic solution.

If the time were continued for a sufficiently long period, then it would be possible to observe $\lim_{t \rightarrow \infty} p_0(t) = 1$ and $\lim_{t \rightarrow \infty} m(t) = 0$. For a population size of $N = 100$, absorption or complete disease extinction did not occur in the time frame shown in Figs. 4–9. However, if the size of the

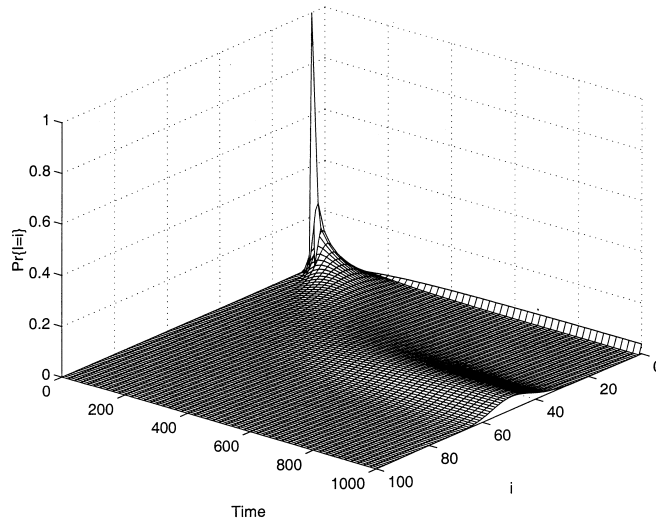


Fig. 8. The probability function, $p(t)$, for the stochastic SIS model is graphed when $\mathcal{R}_0 = 2$, $\mathcal{I}(0) = 5$, $\mathcal{S}(0) = 95$, $\alpha = 1.6$, $\beta = 0.4 = \gamma$, and $\Delta t = 0.01$.

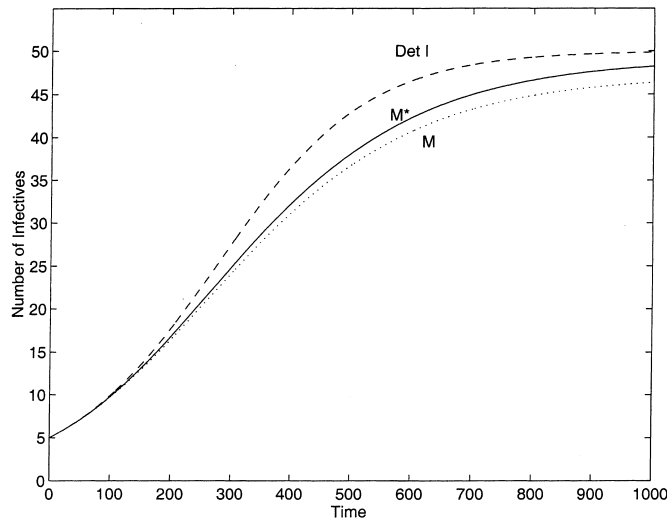


Fig. 9. The mean, M , the mean conditioned on non-extinction, M^* , of the stochastic SIS model and the deterministic solution, $\text{Det } I$, are graphed for the probability function and parameter values given in Fig. 8.

population is reduced and the time frame is extended, then ultimate extinction can be observed. In Fig. 10, $N = 15$ and $p_0(10000\Delta t) = p_0(100) \approx 1$. In addition, the expected duration of the epidemic is computed from the difference equations in (9) and compared to simulations, where the time to extinction is averaged over 10 000 sample paths with $\Delta t = 0.01$. In Fig. 11, the expected duration is graphed as a function of the initial number of infectives when $N = 20$ and $\mathcal{R}_0 = 2$. It can be seen that the discrete-time approximation for the expected duration of the epidemic agrees

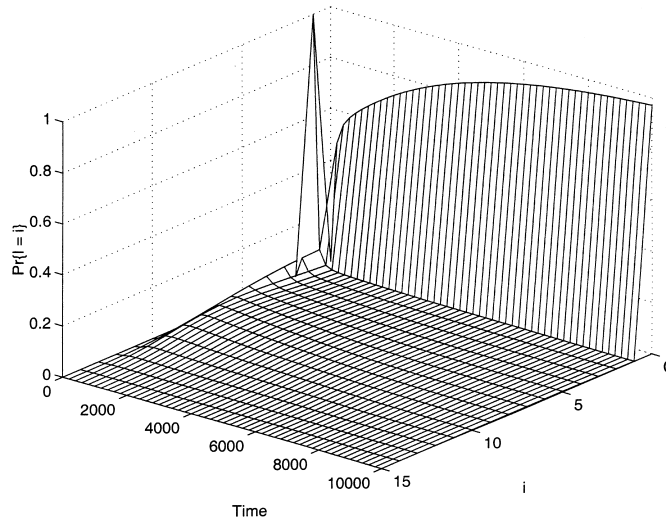


Fig. 10. The probability function, $p(t)$, for the stochastic SIS model is graphed when $\mathcal{R}_0 = 2$, $\mathcal{I}(0) = 1$, $\mathcal{S}(0) = 14$, $\alpha = 1.6$, $\beta = 0.4 = \gamma$, and $\Delta t = 0.01$.

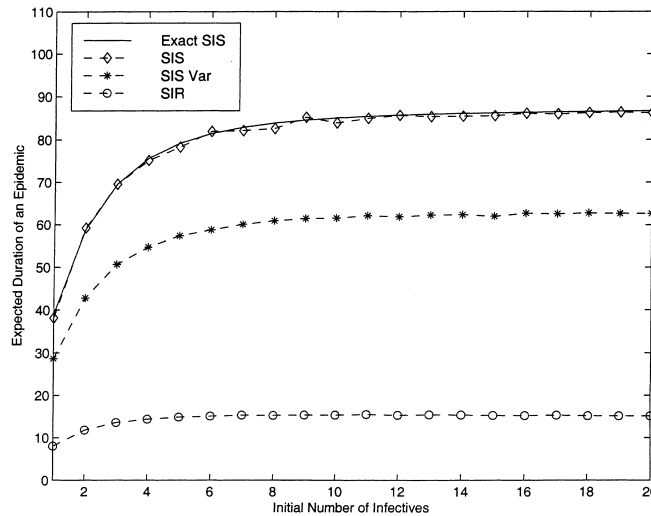


Fig. 11. The expected duration of an epidemic when $N = 20$, $\mathcal{R}_0 = 2$, $\alpha = 1.6$, and $\beta = 0.4 = \gamma$, as a function of the initial number of infectives, $\mathcal{I}(0)$. The exact values for the expected duration (formula (9)) are graphed for the SIS model and the approximate values calculated from an average of 10 000 sample paths when $\Delta t = 0.01$. The approximate duration is calculated for the SIR model and the SIS model with variable population size when $r = 0.25$, $K = 20$, and initial total population size $\mathcal{N}(0) = 20$.

with that obtained from the continuous-time model. Also, note that as the initial number of infectives increases, the expected duration approaches a constant. In Table 2, the expected duration when $\mathcal{I}(0) = 1$ is calculated for various values of N when using formula (9) and compared to the

Table 2

The expected duration of an epidemic for the SIS model as a function of N , $\mathcal{R}_0 = 2$, $\mathcal{I}(0) = 1$, $\alpha = 1.6$, $\beta = 0.4 = \gamma$

N	5	10	15	20	25
Exact	3.68	8.26	17.73	39.01	88.68
Approximate	3.69	8.20	17.83	39.44	90.93

approximate duration calculated from an average of 10 000 sample paths. There is close agreement between the exact and approximate duration and it is evident that there is a large increase in the expected duration with N .

In the next section, the SIR deterministic and stochastic models with constant population size are analyzed.

3. SIR model with constant population size

3.1. Deterministic SIR

In the SIR model, individuals develop an immunity to the disease, births and deaths are included but such that the population size remains constant. The discrete-time deterministic SIR model has the following form:

$$\begin{aligned}
 S(t + \Delta t) &= S(t)(1 - \lambda(t)\Delta t) + (N - S(t))\beta\Delta t, \\
 I(t + \Delta t) &= I(t)(1 - \beta\Delta t - \gamma\Delta t) + \lambda(t)\Delta tS(t), \\
 R(t + \Delta t) &= R(t)(1 - \beta\Delta t) + \gamma\Delta tI(t),
 \end{aligned}
 \tag{10}$$

where $t = n\Delta t$, $n = 0, 1, 2, \dots$, $S(0), I(0) > 0, R(0) \geq 0$, $S(0) + I(0) + R(0) = N$, $\alpha > 0$, $\beta > 0$, and $\gamma > 0$. The conditions (i)–(iv) on the force of infection are assumed to hold. In this model, recovery leads to immunity. Newborns are susceptible.

It is clear that conditions (i) and (iv) imply solutions are non-negative and $S(t) + I(t) + R(t) = N$ for all time. It can also be shown that the basic reproductive number determines the asymptotic behavior in some cases.

Theorem 2. (i) *If $\mathcal{R}_0 \leq 1$, then solutions to (10) approach the disease-free state*

$$\lim_{t \rightarrow \infty} I(t) = 0, \quad \lim_{t \rightarrow \infty} R(t) = 0, \quad \lim_{t \rightarrow \infty} S(t) = N.$$

(ii) *If $\mathcal{R}_0 > 1$, then there exists a unique positive endemic equilibrium and no cycles of period two exist. For the force of infection given by (2), the positive equilibrium is locally asymptotically stable.*

Proof. Let the right-hand side of $I(t + \Delta t)$ in (10) be denoted as g :

$$g(I, R) = I(1 - \beta\Delta t - \gamma\Delta t) + \lambda(t)\Delta t(N - I - R).$$

For case (i), $\mathcal{R}_0 \leq 1$, then

$$g(I, R) \leq I(1 - \beta\Delta t - \gamma\Delta t) + \alpha\Delta t I(N - I - R)/N \leq I + I(\alpha - \beta - \gamma)\Delta t \leq I.$$

Thus, $\{I(t)\}$ is a decreasing sequence bounded below by zero and has a limit \bar{I} . Also, it follows that $\{R(t)\}$ has a limit given by $\gamma\bar{I}/\beta$. The limit is a fixed point of g and must satisfy, $g(\bar{I}, \gamma\bar{I}/\beta) = \bar{I}$ or \bar{I} is a solution to

$$(\beta\Delta t + \gamma\Delta t)I = \lambda(I)\Delta t(N - I - \gamma I/\beta) = h(I). \tag{11}$$

Now, $h(0) = 0$, $h'(0) = \alpha\Delta t$, and $h''(I) < 0$ which implies $h(I) < \alpha\Delta t I$ for $I > 0$. Thus, $h(I) < \alpha\Delta t I \leq (\beta\Delta t + \gamma\Delta t)I$ for $I > 0$. The only fixed point satisfying (11) is the origin; solutions converge to the disease-free state.

For case (ii), where $\mathcal{R}_0 > 1$, the endemic equilibrium also satisfies (11). In this case, $h'(0) = \alpha\Delta t > (\beta\Delta t + \gamma\Delta t)$. In addition, $h(0) = 0$ and $h(N) < 0$. Thus, $h(I)$ must cross the line $(\beta\Delta t + \gamma\Delta t)I$ at least once. Uniqueness follows from the properties of $\lambda(I)$ as in the proof of Theorem 1.

Let $f(S, I)$ and $g(S, I)$ denote the right sides of $S(t + \Delta t)$ and $I(t + \Delta t)$ in (10), respectively. The results of McCluskey and Muldowney [20] can be applied to show there do not exist any cycles of period 2. Cycles of period 2 do not exist if the matrix, $\mathbf{I} + J$ is strictly diagonally dominant for $S + I \in [0, N]$ excluding the equilibrium $I = 0$ and $S = N$ [20]. Matrix \mathbf{I} is the identity matrix and matrix J is the 2×2 Jacobian matrix of $F = (f, g)$

$$\mathbf{I} + J = \begin{pmatrix} 2 - \lambda(I)\Delta t - \beta\Delta t & -\lambda'(I)\Delta t S \\ \lambda(I)\Delta t & 2 - \beta\Delta t - \gamma\Delta t + \lambda'(I)\Delta t S \end{pmatrix}.$$

Note from conditions (i)–(iv) that $\lambda(I)\Delta t \leq \alpha\Delta t I/N \leq 1$ and $\lambda'(I)\Delta t S \leq \alpha\Delta t S/N < 1$. Diagonal dominance in the first row of $\mathbf{I} + J$ follows from

$$2 - \lambda(I)\Delta t - \beta\Delta t - \lambda'(I)\Delta t S \geq 2 - \beta\Delta t - \alpha\Delta t(S + I)/N > 2 - \beta\Delta t - \alpha\Delta t \geq 0.$$

Diagonal dominance in the second row follows from

$$2 - \beta\Delta t - \gamma\Delta t + \lambda'(I)\Delta t S - \lambda(I)\Delta t \geq 2 - \beta\Delta t - \gamma\Delta t - \alpha\Delta t S/N > 0.$$

The matrix $\mathbf{I} + J$ is strictly row diagonally dominant for all $S + I \in [0, N]$, $S \neq N$ and hence, there do not exist any cycles of period 2.

Next, local stability of the positive equilibrium for the force of infection given by (2) is shown. The positive equilibrium is given by

$$\bar{S} = \frac{N}{\mathcal{R}_0}, \quad \bar{I} = \frac{\beta}{\alpha} N(\mathcal{R}_0 - 1).$$

The Jacobian matrix evaluated at the positive equilibrium is

$$J_e = \begin{pmatrix} 1 - \beta\Delta t \mathcal{R}_0 & -\alpha\Delta t/\mathcal{R}_0 \\ \beta\Delta t(\mathcal{R}_0 - 1) & 1 - \beta\Delta t - \gamma\Delta t + \alpha\Delta t/\mathcal{R}_0 \end{pmatrix}.$$

For local stability, the Jury conditions must be satisfied; the trace and determinant must satisfy the three conditions:

$$\text{trace}(J_e) < 1 + \det(J_e), \quad \det(J_e) < 1, \quad -\text{trace}(J_e) < 1 + \det(J_e)$$

[23]. It is tedious but straightforward to show that the trace and determinant of J_e satisfy the first two inequalities when $\mathcal{R}_0 > 1$. The third inequality is satisfied if

$$\alpha\Delta t < [4 - \beta\Delta t(\beta\Delta t + \gamma\Delta t)] \frac{\beta\Delta t + \gamma\Delta t}{\beta\Delta t(2 - \beta\Delta t - \gamma\Delta t)}. \quad (12)$$

The right side of the above inequality is greater than $3/2$. Hence, it follows from condition (iv) that the third inequality is satisfied. \square

Weaker conditions on the force of infection may result in periodic solutions. When $\mathcal{R}_0 > 1$ and inequality (12) is not satisfied, the model with the force of infection (2) exhibits periodic solutions [15].

The assumption $\beta > 0$ is important to the existence of the endemic state. If $\beta = 0$ and $\lambda(I) = \alpha I/N$ or $\lambda(I) = \alpha I$, then $\lim_{t \rightarrow \infty} I(t) = 0$ regardless of the magnitude of \mathcal{R}_0 [15]. The continuous-time version of this model with mass action incidence ($\lambda(I)S = \alpha IS$) and $\beta = 0$ is the classical SIR model studied by Kermack and McKendrick [24].

3.2. Stochastic SIR

The discrete-time stochastic SIR model is a Markov chain with finite state space. The analogous continuous-time model is a Markov jump process. The stochastic SIR model is a bivariate process dependent on the random variables \mathcal{I} and \mathcal{R} , the number of infected and immune individuals, respectively. The stochastic SIR has a joint probability function, $p_{ir}(t) = \text{Prob}\{\mathcal{I}(t) = i, \mathcal{R}(t) = r\}$, where $i, r = 0, 1, 2, \dots, N$ and $0 \leq i + r \leq N$.

Let $\Pi_{ir}\Delta t = \lambda(i)(N - i - r)\Delta t$ denote the probability of a new infective in time Δt :

$$\text{Prob}\{\mathcal{I}(t + \Delta t) = i + 1, \mathcal{R}(t + \Delta t) = r \mid \mathcal{I}(t) = i, \mathcal{R}(t) = r\} = \Pi_{ir}\Delta t,$$

where $\lambda(i)$ satisfies conditions (i)–(iv). Let $\gamma i\Delta t$, $\beta i\Delta t$, and $\beta r\Delta t$ denote the probability of recovery of an infective and the probability of death of an infective or of an immune individual, respectively:

$$\text{Prob}\{\mathcal{I}(t + \Delta t) = i - 1, \mathcal{R}(t + \Delta t) = r + 1 \mid \mathcal{I}(t) = i, \mathcal{R}(t) = r\} = \gamma i\Delta t,$$

$$\text{Prob}\{\mathcal{I}(t + \Delta t) = i - 1, \mathcal{R}(t + \Delta t) = r \mid \mathcal{I}(t) = i, \mathcal{R}(t) = r\} = \beta i\Delta t,$$

$$\text{Prob}\{\mathcal{I}(t + \Delta t) = i, \mathcal{R}(t + \Delta t) = r - 1 \mid \mathcal{I}(t) = i, \mathcal{R}(t) = r\} = \beta r\Delta t.$$

Each death is accompanied by a birth so that the population size remains constant. For example, a death of an immune individual is accompanied by a birth of a susceptible.

The difference equations satisfied by the joint probability $p_{ir}(t)$ are

$$\begin{aligned} p_{ir}(t + \Delta t) &= p_{i-1,r}(t)\Pi_{i-1,r}\Delta t + p_{i+1,r-1}(t)\gamma\Delta t(i + 1) + p_{i+1,r}(t)\beta\Delta t(i + 1) \\ &\quad + p_{i,r+1}(t)\beta\Delta t(r + 1) + p_{ir}(t)[1 - \Pi_{ir}\Delta t - \gamma i\Delta t - \beta(i + r)\Delta t], \end{aligned}$$

$$p_{0r}(t + \Delta t) = p_{0r}(t),$$

where $i, r = 0, 1, \dots, N$, $i + r \leq N$ and $p_{ir}(t) = 0$ if $i, r \notin [0, N]$. To ensure that the transition probabilities are positive and bounded by one, it is required that $\Pi_{ir}\Delta t + \gamma i\Delta t + \beta(i + r)\Delta t \leq 1$ for $i + r = 0, 1, \dots, N$ and $i + r \leq N$. The inequality is satisfied if Δt is chosen sufficiently small. The

discrete-time stochastic model is a Markov chain, but in this case, the transition matrix cannot be expressed in a simple form. There is a single absorbing state at the origin, $\mathcal{I} = 0$ and $\mathcal{R} = 0$.

3.2.1. Mean and quasi-stationary mean

The quasi-stationary distribution is defined by

$$q_{ir}(t) = \frac{p_{ir}(t)}{1 - \sum_{r=0}^N p_{0r}(t)}$$

for $i = 1, \dots, N$, $r = 0, \dots, N$ and $i + r \leq N$. The corresponding difference equations for $q_{ir}(t)$ are given by

$$q_{ir}(t + \Delta t)[1 - (\beta + \gamma)q_1(t)\Delta t] = q_{i-1,r}(t)\Pi_{i-1,r}\Delta t + q_{i+1,r-1}(t)\gamma\Delta t(i + 1) + q_{i+1,r}(t)\beta\Delta t(i + 1) + q_{i,r+1}(t)\beta\Delta t(r + 1) + q_{ir}(t)[1 - \Pi_{ir}\Delta t - \gamma i\Delta t - \beta(i + r)\Delta t]$$

for $i = 1, \dots, N$, $r = 1, \dots, N$, and $i + r \leq N$, where $q_i(t) = \sum_{r=0}^{N-i} p_{ir}(t)/(1 - \sum_{r=0}^{N-i} p_{ir}(t))$.

Note that the difference equations for the quasi-stationary distribution are similar to those for the SIS model. In addition, the mean and the mean conditioned on non-extinction for \mathcal{I} satisfy difference equations similar to those given for the SIS model.

Let $m_{\mathcal{I}}(t) = \sum_{i,r=0}^N ip_{ir}(t)$ and $m_{\mathcal{I}}^*(t) = \sum_{i=1,r=0}^N iq_{ir}(t)$ denote the mean number of infectives and the mean conditioned on non-extinction, respectively. In each case, the sum over i and r is understood to mean $i + r \leq N$. Then

$$m_{\mathcal{I}}(t + \Delta t) - m_{\mathcal{I}}(t) = \sum_{i,r=0}^N \left[\frac{\Pi_{ir}\Delta t}{i} - \gamma\Delta t - \beta\Delta t \right] ip_{ir}(t) = (\beta + \gamma)\Delta t[\mathcal{R}_0\xi_{\mathcal{I}}(t) - 1]m_{\mathcal{I}}(t), \tag{13}$$

$$m_{\mathcal{I}}^*(t + \Delta t)[1 - (\beta + \gamma)q_1(t)\Delta t] - m_{\mathcal{I}}^*(t) = (\beta + \gamma)\Delta t[\mathcal{R}_0\xi_{\mathcal{I}}^*(t) - 1]m_{\mathcal{I}}^*(t),$$

where

$$\xi_{\mathcal{I}}(t) = \frac{\sum_{i,r=0}^N p_{ir}(t)\Pi_{ir}}{\alpha \sum_{i,r=0}^N ip_{ir}(t)} \quad \text{and} \quad \xi_{\mathcal{I}}^*(t) = \frac{\sum_{i,r=0}^N q_{ir}(t)\Pi_{ir}}{\alpha \sum_{i,r=0}^N iq_{ir}(t)}.$$

Note that $\Pi_{ir}(t)/i \leq \alpha s/N$, where $s = N - i - r$ so that $\xi_{\mathcal{I}}(t) < 1$. When $\mathcal{R}_0 \leq 1$, it follows from (13) that $\{m_{\mathcal{I}}\}$ is a monotone decreasing sequence which converges to 0. It can be seen from (13) that there are two steady-state solutions for the mean, the zero solution and the solution of

$$\xi_{\mathcal{I}}(t) = \frac{1}{\mathcal{R}_0}.$$

There is a similar relation to a random walk as in the stochastic SIS model when the initial number of infectives is small and the population size is large. Initially, the epidemic fades out quickly with probability $(1/\mathcal{R}_0)^a$ when $\mathcal{R}_0 > 1$. However, it persists with probability, $1 - (1/\mathcal{R}_0)^a$, where a is the initial number of infectives.

Approximations are given for the expected time to extinction from quasi-stationarity for the continuous-time SIR model with standard incidence by Näsell [13]. In the numerical examples, we approximate the expected time to extinction for the corresponding discrete-time model.

For non-endemic SIR models, an important problem is to estimate the total size of the epidemic or the final size distribution. Since our models are endemic ($\beta > 0$), we do not investigate this

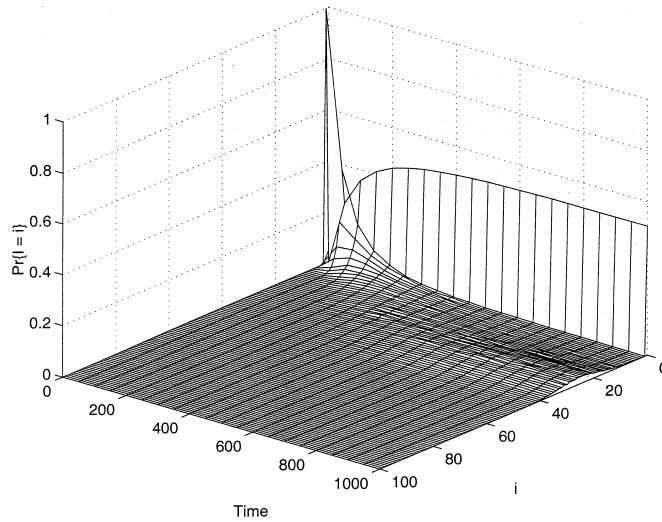


Fig. 12. The probability function for the number of infectives $\mathcal{I}(t)$ in the stochastic SIR model is graphed when $\mathcal{R}_0 = 2$, $\mathcal{I}(0) = 1$, $S(0) = 99$, $\mathcal{R}(0) = 0$, $\alpha = 1.6$, $\beta = 0.4 = \gamma$, and $\Delta t = 0.01$.

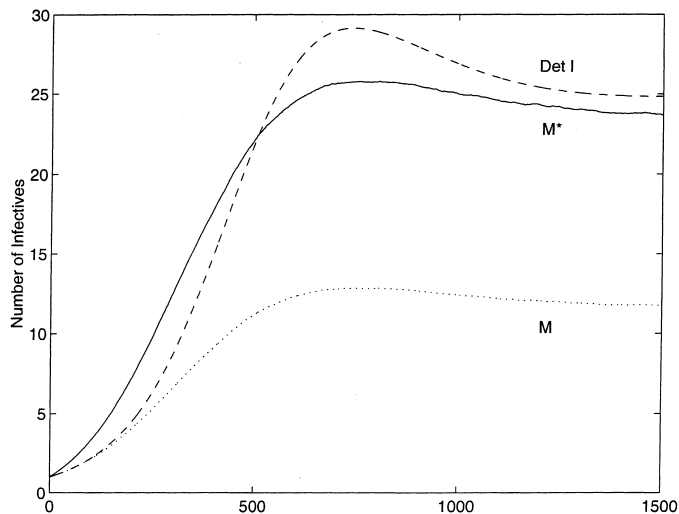


Fig. 13. The mean, M , the mean conditioned on non-extinction, M^* , of the stochastic SIR model and the deterministic solution, $\text{Det } I$, are graphed for the probability function and parameter values given in Fig. 12.

problem. Some references for the final size distribution in non-endemic, discrete and continuous-time stochastic SIR models are given in the list of Refs. [25–28].

3.3. Numerical examples

In the numerical examples, it is assumed that the incidence rate has the form of the standard incidence, $\lambda(t)S(t) = \alpha I(t)S(t)/N$. One initial infective is introduced into a population of size

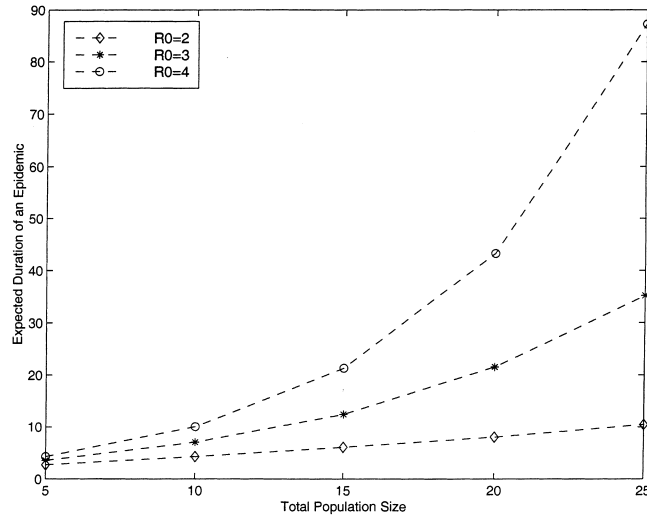


Fig. 14. The approximate expected duration of an SIR epidemic when $\mathcal{I}(0) = 1$, $\beta = 0.4 = \gamma$ and $\mathcal{R}_0 = 2, 3$, and 4 as a function of the total population size N . The duration is calculated from an average of 10 000 sample paths when $\Delta t = 0.01$.

$N = 100$, $\mathcal{R}_0 = 2$, and $\Delta t = 0.01$. The probability function for the number of infectives is graphed in Fig. 12, $p_i(t) = \sum_{r=0}^{N-i} p_{ir}(t)$. The probability function and its mean are calculated from 10 000 individual sample paths. The shape of the probability functions of the SIS and SIR models are similar; both are bimodal, with one mode at zero and one close to the endemic equilibrium of the deterministic model, $\bar{I} = 25$. Neglecting the probability at zero, the quasi-stationary distribution appears approximately normal. Initially, the disease is eliminated with probability close to $1/\mathcal{R}_0 = 0.5$. The deterministic solution of the SIR model is graphed with the mean and the mean conditioned on non-extinction in Fig. 13. Both means lie below the deterministic solution, although the mean conditioned on non-extinction is closer to the deterministic solution.

The expected duration of the epidemic is calculated numerically from an average of 10 000 sample paths. Fig. 14 is a graph of the expected duration as a function of N for \mathcal{R}_0 equal to 2, 3 and 4 with one initial infective. It can be seen that the expected duration appears to increase exponentially with N . The expected duration for a population of size $N = 20$ and $\mathcal{R}_0 = 2$ as a function of the initial number of infectives is graphed in Fig. 11 and compared to that of the SIS model. Since individuals recover in an SIR model and are not reinfected, the duration is much shorter for an SIR model than for an SIS model with the same parameter values.

4. SIS model with variable population size

In the SIS model with variable population size, it is assumed that the total population size is not constant but varies with time, $N \equiv N(t)$. Since the total population size satisfies $N(t) = S(t) + I(t)$, the model has two independent dynamic variables. The deterministic and stochastic SIS models are described in the next sections.

4.1. Deterministic SIS

The following difference equation models the growth of the population:

$$N(t + \Delta t) = N(t)[f(N(t)) + 1] = N(t)F(N(t)) = g(N(t)), \quad 0 < N(0) < K, \tag{14}$$

where $F(N) = f(N) + 1$ and $NF(N) = g(N)$. The functions f and g in (14) satisfy the following three conditions:

- (v) $f(N), g(N) \in C^1[0, K], f(N) > 0$ for $N \in [0, K)$,
- (vi) $g(0) = 0$ and $g(K) = K$, and
- (vii) $g'(0) > 1, g'(N) > 0$ for $N \in [0, K]$.

Thus, $F(N)N = g(N)$ implies $F(K) = 1$ and $f(K) = 0$. In addition, $g(N) > N$ for $N \in (0, K)$. Eq. (14) has two fixed points, $\bar{N} = 0$ and $\bar{N} = K$. Since the population is initially below K , conditions (v)–(vii) guarantee that solutions $N(t)$ increase monotonically to K , the carrying capacity. Such types of conditions were imposed in differential equation epidemic models with variable population size (e.g., [29–32]). Two examples satisfying the above restrictions are the difference equations for logistic growth:

$$N(t + \Delta t) = N(t) \frac{(r\Delta t + 1)K}{K + r\Delta t N(t)}, \quad r > 0 \tag{15}$$

or

$$N(t + \Delta t) = N(t) \left(1 + r\Delta t - \frac{N(t)r\Delta t}{K} \right), \quad 0 < r\Delta t < 1.$$

The deterministic SIS model with variable population size has the form:

$$\begin{aligned} S(t + \Delta t) &= S(t)[F(N(t)) - \lambda(t)\Delta t] + (\beta\Delta t + \gamma\Delta t)I(t), \\ I(t + \Delta t) &= I(t)[F(N(t)) - \beta\Delta t - \gamma\Delta t] + \lambda(t)\Delta t S(t), \\ N(t + \Delta t) &= N(t)F(N(t)), \end{aligned} \tag{16}$$

where $F(N(t)) = 1 + f(N(t))$, $\beta\Delta t$ is the per capita number of births, $\beta\Delta t - f(N(t))$ is the per capita number of deaths in time Δt , $S(0) > 0$, $I(0) > 0$, and $N(0) = S(0) + I(0) < K$.

Conditions (i)–(iv) are assumed to hold for the force of infection, but conditions (i)–(iii) are modified to account for the changing population size. Let $i(t) = I(t)/N(t)$. It is assumed that the force of infection is a function of the proportion of infectives, $i(t)$, rather than the number of infectives, $I(t)$. The three modified conditions are stated in terms of the proportion, $i(t)$:

- (i)' $\lambda(t) \equiv \lambda(i(t)) \leq \alpha i(t)$, where $i(t) = I(t)/N(t)$.
- (ii)' $\lambda(i) \in C^2[0, 1], d\lambda(i)/di > 0$, and $d^2\lambda(i)/di^2 \leq 0$ for $i \in [0, 1]$.
- (iii)' $\lambda(i)|_{i=0} = 0$ and $\lambda'(i)|_{i=0} = \alpha$.

With these restrictions, the incidence rate may take the form of the standard incidence given in (2) or the form in (3). However, the mass action incidence rate is not possible, $\lambda(t)S(t) \neq \text{constant}I(t)S(t)$. Also, note that the restrictions (i)'–(iii)' and (iv)–(vii) imply that solutions to (16) are non-negative.

The following theorem gives sufficient conditions that show the basic reproductive number determines asymptotic behavior.

Theorem 3. (i) If $\mathcal{R}_0 < 1$, then solutions to (16) satisfy

$$\lim_{t \rightarrow \infty} I(t) = 0, \quad \lim_{t \rightarrow \infty} S(t) = K.$$

(ii) If $\mathcal{R}_0 > 1$ and if the function $h(x) = (1 - \beta\Delta t - \gamma\Delta t)x + (1 - x)\lambda(x)\Delta t$ defined on $[0, 1]$ has a unique positive fixed point x^* , $0 < x^* < 1$, and $0 < x^* < x_M$, where $h(x_M) = \max_{0 \leq x \leq 1} h(x)$, then solutions to (16) satisfy

$$\lim_{t \rightarrow \infty} I(t) = \bar{I} > 0, \quad \lim_{t \rightarrow \infty} S(t) = \bar{S} > 0,$$

where $\bar{I} = x^*K$ and $\bar{S} + \bar{I} = K$.

The conditions imposed on h in part (ii) of Theorem 3 seem restrictive but it can be easily shown that they are satisfied by the force of infection $\lambda(i(t)) = \alpha i(t)$. In this case, $x^* = (\alpha - \beta - \gamma)/\alpha < \min\{1, (1 + [\alpha - \beta - \gamma]\Delta t)/(2\alpha\Delta t)\} = x_M$.

Proof. The difference equation for $I(t)$ can be expressed in terms of proportions, $i(t) = I(t)/N(t)$ and $s(t) = S(t)/N(t) = 1 - i(t)$

$$i(t + \Delta t) = \frac{N(t)}{N(t + \Delta t)} [i(t)(f(N(t)) + 1 - \beta\Delta t - \gamma\Delta t) + \lambda(i(t))\Delta t(1 - i(t))].$$

For case (i), let $\epsilon > 0$ be chosen such that $0 < \alpha\Delta t/(\gamma\Delta t + \beta\Delta t - \epsilon) < 1$. Since $N(t)$ approaches K and $f(N(t))$ approaches $f(K) = 0$ as $t \rightarrow \infty$, choose t sufficiently large such that $t \geq T$ implies $0 < f(N(t)) < \epsilon$. Then for $t \geq T$,

$$\begin{aligned} i(t + \Delta t) &\leq \frac{N(t)}{N(t + \Delta t)} [i(t)(1 + \epsilon - \beta\Delta t - \gamma\Delta t) + \alpha\Delta t(1 - i(t))] \\ &\leq i(t)(1 + \alpha\Delta t - \beta\Delta t - \gamma\Delta t + \epsilon) - \alpha\Delta t[i(t)]^2 \leq i(t). \end{aligned}$$

The sequence $\{i(t)\}$ is monotone decreasing, bounded below by zero and must converge to a fixed point of

$$h(i) = i(1 - \beta\Delta t - \gamma\Delta t) + \lambda(i)\Delta t(1 - i). \tag{17}$$

Note that

$$\begin{aligned} h'(i) &= 1 - \beta\Delta t - \gamma\Delta t + \lambda'(i)\Delta t(1 - i) - \lambda(i)\Delta t, \\ h''(i) &= \lambda''(i)\Delta t(1 - i) - 2\lambda'(i)\Delta t, \end{aligned}$$

$h''(i) < 0$ for $i \in [0, 1]$, and $h'(0) = 1 + \alpha\Delta t - \beta\Delta t - \gamma\Delta t < 1$. It follows that $h(i) < i$ for $i \in (0, 1]$. The only fixed point of h for $\mathcal{R}_0 < 1$ is 0. Hence, $i(t)$ converges to 0.

Let $0 < \epsilon_1 < 1$ and $\epsilon_2 > 0$. Consider the function

$$\begin{aligned} \underline{h}(x) &= (1 - \epsilon_1)x(1 - \beta\Delta t - \gamma\Delta t) + (1 - \epsilon_1)(1 - x - \epsilon_1)\lambda(x)\Delta t \\ &= h(x) - \epsilon_1[h(x) + (1 - \epsilon_1)\lambda(x)\Delta t] \leq h(x). \end{aligned}$$

Also, the function

$$\bar{h}(x) = x(1 - \beta\Delta t - \gamma\Delta t + \epsilon_1) + (1 - x)\lambda(x)\Delta t = h(x) + \epsilon_1x \geq h(x),$$

where h is defined in (17). Note that $\underline{h}(0) = 0 = \bar{h}(0) = 0$.

The function h has the property that $h'(i) = 0$ for at most a single value of $i \in [0, 1]$. Thus, h is strictly increasing for $x < x_M$ and strictly decreasing for $x > x_M$. When $\mathcal{R}_0 > 1$ so that $h'(0) > 1$, the solution $x(t)$ to $x(t + \Delta t) = h(x(t))$ converges monotonically to x^* for $t > 0$.

For case (ii), choose ϵ_1 sufficiently small such that \underline{h} and \bar{h} have unique positive fixed points \underline{x} and \bar{x} , respectively, $\underline{x} < \bar{x} < 1$ such that $|x^* - \underline{x}| < \epsilon_2$ and $|x^* - \bar{x}| < \epsilon_2$. In addition, choose ϵ_1 sufficiently small such that $\underline{h}(x)$ and $\bar{h}(x)$ are strictly increasing for $0 < x < \bar{x}$, where \bar{x} is some point $\bar{x} < \bar{x} < x_M$, $\underline{h}(x) > x$ for $0 < x < \underline{x}$, $\underline{h}(x) < x$ for $x > \underline{x}$, $\bar{h}(x) > x$ for $0 < x < \bar{x}$, $\bar{h}(x) < x$ for $x > \bar{x}$, and $\bar{h}(x) < x_M$ for $x \in [0, 1]$ (possible since h and λ are $C^1[0, 1]$ and $h(x) < x_M$). Thus, $\lim_{t \rightarrow \infty} \underline{h}(x(t)) = \underline{x}$ and $\lim_{t \rightarrow \infty} \bar{h}(x(t)) = \bar{x}$.

Choose t sufficiently large such that $t \geq T$ implies $0 < f(N(t)) < \epsilon_1$, $1 - i(t) - s(t) < \epsilon_1$, and $1 - \epsilon_1 < N(t)/N(t + \Delta t)$. Then for $t \geq T$

$$\underline{h}(i(t)) \leq i(t + \Delta t) \leq \bar{h}(i(t)).$$

If $x(T) = i(T) = y(T)$, then

$$x(T + \Delta t) = \underline{h}(x(T)) = \underline{h}(i(T)) \leq i(T + \Delta t) \leq \bar{h}(i(T)) = \bar{h}(y(T)) = y(T + \Delta t) < x_M.$$

Since $h'(x) > 0$ for $x < x_M$, then

$$\begin{aligned} x(T + 2\Delta t) &= \underline{h}(x(T + \Delta t)) \leq h(x(T + \Delta t)) \leq h(i(T + \Delta t)) = i(T + 2\Delta t) \\ &\leq h(y(T + \Delta t)) \leq \bar{h}(y(T + \Delta t)) = y(T + 2\Delta t) < x_M. \end{aligned}$$

Continuing in this manner,

$$x(T + n\Delta t) = \underline{h}(x(T + (n - 1)\Delta t)) \leq i(T + n\Delta t) \leq \bar{h}(y(T + (n - 1)\Delta t)) = y(T + n\Delta t)$$

for $n = 2, \dots$. Since $x(t)$ converge to \underline{x} and $y(t)$ converges to \bar{x} , it follows that

$$x^* - \epsilon_2 \leq \underline{x} \leq \liminf_{t \rightarrow \infty} i(t) \leq \limsup_{t \rightarrow \infty} i(t) \leq \bar{x} \leq x^* + \epsilon_2.$$

Since ϵ_2 can be made arbitrarily small, it follows that $\lim_{t \rightarrow \infty} i(t) = x^*$. \square

4.2. Stochastic SIS

4.2.1. Model

The discrete-time stochastic SIS model with variable population size is formulated as a Markov chain. The stochastic process is bivariate. Let \mathcal{I} and \mathcal{N} denote the random variables for the number of infectives and total number of individuals. Let the joint probability function be denoted as

$$p_{in}(t) = \text{Prob}\{\mathcal{I}(t) = i, \mathcal{N}(t) = n\}.$$

There are five different transition probabilities in the discrete-time model:

$$\text{Prob}\{\mathcal{I}(t + \Delta t) = i + 1, \mathcal{N}(t + \Delta t) = n \mid \mathcal{I}(t) = i, \mathcal{N}(t) = n\} = \Pi_{in}\Delta t,$$

$$\text{Prob}\{\mathcal{I}(t + \Delta t) = i - 1, \mathcal{N}(t + \Delta t) = n \mid \mathcal{I}(t) = i, \mathcal{N}(t) = n\} = \gamma i \Delta t,$$

$$\text{Prob}\{\mathcal{I}(t + \Delta t) = i, \mathcal{N}(t + \Delta t) = n + 1 \mid \mathcal{I}(t) = i, \mathcal{N}(t) = n\} = \beta n \Delta t,$$

$$\text{Prob}\{\mathcal{I}(t + \Delta t) = i - 1, \mathcal{N}(t + \Delta t) = n - 1 \mid \mathcal{I}(t) = i, \mathcal{N}(t) = n\} = (\beta \Delta t - f(n))i,$$

$$\text{Prob}\{\mathcal{I}(t + \Delta t) = i, \mathcal{N}(t + \Delta t) = n - 1 \mid \mathcal{I}(t) = i, \mathcal{N}(t) = n\} = (\beta \Delta t - f(n))(n - i)$$

for $i \leq n$, $i, n = 0, 1, \dots, M$, where $\Pi_{in} = \lambda(i/n)(n - i)$, i/n is the proportion of infectives, and $f(n)$ is the function defined in the deterministic model (14). Assumptions (i)–(iii)', and (iv)–(vii) are assumed to hold. The population size may increase above carrying capacity, K ; thus, the definition of f needs to be extended to $n > K$ and in addition, the population size should be bounded. Two more conditions are made for the stochastic model:

(viii) There exists $M > K$ such that $f \in C^1[0, M]$, $f(n) < 0$ for $K < n \leq M$.

(ix) The probability of a birth, $\text{Prob}\{\mathcal{N}(t + \Delta t) = n + 1 \mid \mathcal{N}(t) = n\} = 0$ for $n \geq M$.

Conditions (viii) and (ix) assume that when $K < n \leq M$, the probability of a death is greater than the probability of a birth and that M is a bound on the population size.

The difference equations for the joint probability function $p_{in}(t)$ are given by

$$\begin{aligned} p_{in}(t + \Delta t) = & p_{i-1,n}(t)\Pi_{i-1,n}\Delta t + p_{i+1,n}(t)\gamma(i+1)\Delta t + p_{i,n-1}(t)\beta(n-1)\Delta t \\ & + p_{i+1,n+1}(t)(\beta\Delta t - f(n+1))(i+1) + p_{i,n+1}(t)(\beta\Delta t - f(n+1))(n+1-i) \\ & + p_{in}(t)[1 - \Pi_{in}\Delta t - \gamma i \Delta t - 2\beta n \Delta t + f(n)n], \end{aligned}$$

$$p_{00}(t + \Delta t) = p_{00}(t)$$

for $i \leq n \leq M$, $i, n = 0, 1, \dots, M$ and $p_{in}(t) = 0$ for $i, n \notin [0, M]$. The probabilities must satisfy $\Pi_{in}\Delta t + \gamma i \Delta t + 2\beta n \Delta t - f(n)n \leq 1$ for $0 \leq i \leq n$, $i, n = 0, 1, \dots, M$ which is possible if Δt is sufficiently small.

The only absorbing state is the state $\mathcal{I} = 0$ and $\mathcal{N} = 0$; eventually, the disease is eliminated and the population becomes extinct. However, it may take a long time for total population extinction to occur, especially for large initial values and large carrying capacity K .

4.2.2. Mean and quasi-stationary mean

The quasi-stationary probability distribution is defined by $q_{in}(t) = p_{in}(t) / (1 - \sum_{n=0}^M p_{0n}(t))$ for $i, n = 1, 2, \dots, M$, $i \leq n$. The probabilities $q_{in}(t)$ satisfy the difference equations

$$\begin{aligned} q_{in}(t + \Delta t) & \left[1 - \frac{\sum_{n=1}^M p_{1n}(t)(\beta\Delta t + \gamma\Delta t - f(n))}{1 - \sum_{n=0}^M p_{0n}(t)} \right] \\ & = q_{i-1,n}(t)\Pi_{i-1,n}\Delta t + q_{i+1,n}(t)\gamma(i+1)\Delta t + q_{i,n-1}(t)\beta(n-1)\Delta t \\ & \quad + q_{i+1,n+1}(t)(\beta\Delta t - f(n+1))(i+1) + q_{i,n+1}(t)(\beta\Delta t - f(n+1))(n+1-i) \\ & \quad + q_{in}(t)[1 - \Pi_{in}\Delta t - \gamma i \Delta t - 2\beta n \Delta t + f(n)n] \end{aligned}$$

for $i, n \in \{0, 1, \dots, M\}$, $i \leq n$. The difference equations for the quasi-stationary distribution differ from the SIS model with constant population size due to the presence of the term $f(n)$.

Difference equations for the mean and the mean conditioned on non-extinction also differ from those of the stochastic SIS model with constant population size. The mean and mean conditioned on non-extinction satisfy the difference equations:

$$m(t + \Delta t) - m(t) = \sum_{i,n=0}^M \left[\frac{\Pi_{in}\Delta t}{i} - \beta\Delta t - \gamma\Delta t + f(n) \right] ip_{in}(t) = (\beta + \gamma)\Delta t [\mathcal{R}_0 \xi_{\mathcal{S}}(t) - 1]m(t),$$

$$m^*(t + \Delta t) \left[1 - \frac{\sum_{n=1}^M p_{1n}(t)(\beta\Delta t + \gamma\Delta t - f(n))}{1 - \sum_{n=0}^M p_{0n}(t)} \right] - m^*(t) = (\beta + \gamma)\Delta t [\mathcal{R}_0 \xi_{\mathcal{S}}^*(t) - 1]m^*(t),$$

where

$$\xi_{\mathcal{S}}(t) = \frac{\sum_{i,n=0}^M (\Pi_{in}\Delta t + f(n)i)p_{in}(t)}{\alpha\Delta t \sum_{i,n=0}^M ip_{in}(t)},$$

$$\xi_{\mathcal{S}}^*(t) = \frac{\sum_{i,n=0}^M (\Pi_{in}\Delta t + f(n)i)q_{in}(t)}{\alpha\Delta t \sum_{i,n=0}^M iq_{in}(t)},$$

and the sum is over indices $i \leq n \leq M$. For this stochastic process, the threshold \mathcal{R}_0 may not determine the asymptotic behavior of the mean. Another threshold relates the random walk to the probability of disease elimination.

Initially, if there are a small number of infectives and a large population size, the probability of disease elimination can be approximated using the theory of random walks. Suppose $s \approx n_0$, where n_0 is the initial population size, $\text{Prob}\{\mathcal{N}(0) = n_0\} = 1$, then probability of infection is $\Pi_{in_0}\Delta t = \lambda(i/n_0)(n_0 - i)\Delta t \approx \alpha i\Delta t$ and the probability of recovery or death is $\gamma i\Delta t + (\beta\Delta t - f(n_0))i$. From the theory of random walks [14], the probability of ultimate absorption or disease elimination is approximately

$$\left(\frac{\gamma\Delta t + \beta\Delta t - f(n_0)}{\alpha\Delta t} \right)^a,$$

where a is the initial number of infectives. If $n_0 < K$, then $f(n_0) > 0$ and if $n_0 \leq K$, then $f(n_0) \geq 0$. If the initial population size is less than the carrying capacity, $n_0 < K$, then the probability that the disease is eliminated is less than in a population of constant size. In other words, a growing population is more likely to experience an epidemic than a population that has stabilized at a constant population size. However, in a population that is declining, $n_0 > K$, the probability that the disease is eliminated is greater than in a population that has stabilized.

A possible reason for the difference between the constant and variable population size models is due to the death rate of infectives which changes with the population size. When the population size is below carrying capacity, births exceed deaths, and the length of infectivity increases from $1/(\gamma\Delta t + \beta\Delta t)$ to $1/(\gamma\Delta t + \beta\Delta t - f(n))$ ($f(n) > 0$). This longer period of infectivity due to a decreased death rate may result in more susceptibles becoming infective at the outset of an epidemic.

4.3. Numerical examples

Numerical examples are simulated for a growing population; the initial population size is below carrying capacity. In these examples, population growth follows the logistic equation (15):

$$f(n) = \frac{r\Delta t(K - n)}{K + r\Delta tn}$$

and the force of infection is $\lambda(i/n) = \alpha i/n$. The time step is $\Delta t = 0.002$, so that the actual time frame extends from 0 to time $(\Delta t) = 10\,000\Delta t = 20$. The probabilities and means are calculated from 10 000 individual sample paths.

In the first example, one infective is introduced into a population of size $n = 20$ with carrying capacity $K = 100$, maximum population size of $M = 200$, a basic reproductive number $\mathcal{R}_0 = 2$, and parameter values $\alpha = 1.5$, $\beta = 0.5$, $\gamma = 0.25$ and $r = 0.25$. In Figs. 15 and 16 the marginal probabilities for $\mathcal{I}(t)$ and $\mathcal{N}(t)$ are given. The probability function for infectives is bimodal, one mode is at zero and the second mode is the quasi-stationary mean which is near the endemic equilibrium of the deterministic model. The probability function of the total population size is unimodal for the time frame shown; there is one mode approaching carrying capacity, $K = 100$. The probability that the disease is eliminated approaches a value close to that predicted by the random walk in the time frame shown. The estimate for disease elimination in the random walk is given by

$$\frac{\beta\Delta t + \gamma\Delta t - f(n_0)}{\alpha\Delta t} \approx 0.367,$$

which is close to the value of $p_0(20) = \text{Prob}\{\mathcal{I}(20) = 0\}$ in Fig. 15. Initially, the probability of disease elimination is less than in the model with a constant population size due to the decreased

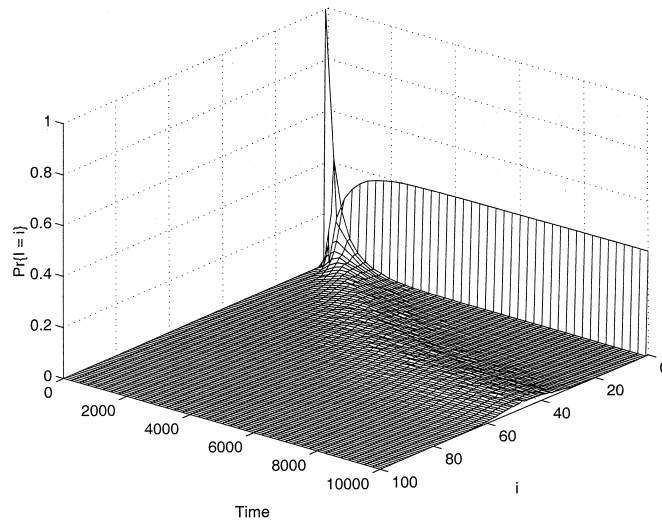


Fig. 15. The probability function for the number of infectives $\mathcal{I}(t)$ for the SIS model with variable population size is graphed when $\mathcal{R}_0 = 2$, $\mathcal{I}(0) = 1$, $\mathcal{S}(0) = 19$, $K = 100$, $\alpha = 1.5$, $\beta = 0.5$, $\gamma = 0.25$, $r = 0.25$, $M = 200$, and $\Delta t = 0.002$.

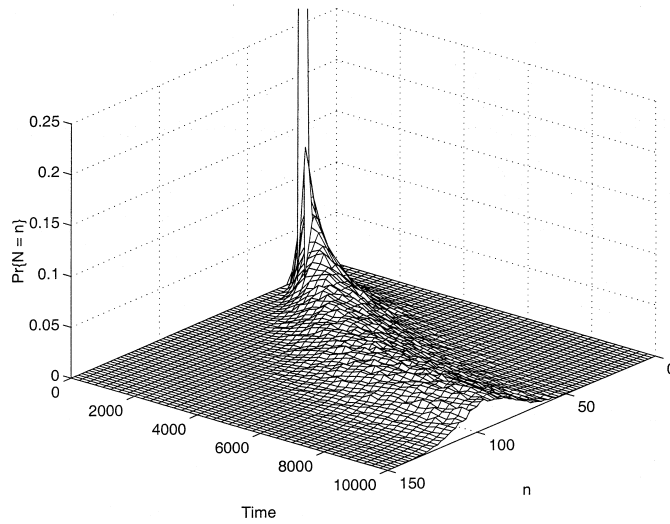


Fig. 16. The probability function for the total population size $\mathcal{N}(t)$ is graphed for the SIS model with variable population size of the parameter values given in Fig. 15.

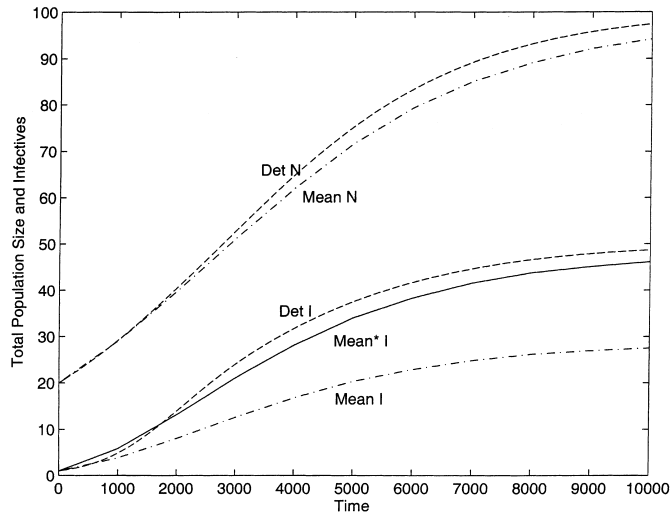


Fig. 17. The mean number of infectives, Mean I , the mean total population size, Mean N , the mean conditioned on non-extinction for infectives Mean *I , and the deterministic solutions, Det I and Det N , for the SIS model with variable population size are graphed for the probabilities and parameter values given in Figs. 15 and 16.

death rate of infectives. The mean and the mean conditioned on non-extinction for the probability distributions of $\mathcal{I}(t)$ and $\mathcal{N}(t)$ are graphed in Fig. 17.

In the next example, the carrying capacity is reduced to $K = 15$, the initial conditions are $I(0) = 1$ and $S(0) = 4$, and the parameter values are $r = 0.25$, $\alpha = 1.6$, $\beta = 0.4$, $\gamma = 0.4$, and $\Delta t = 0.002$. The graph in Fig. 18 can be compared to Fig. 10. For a small carrying capacity,

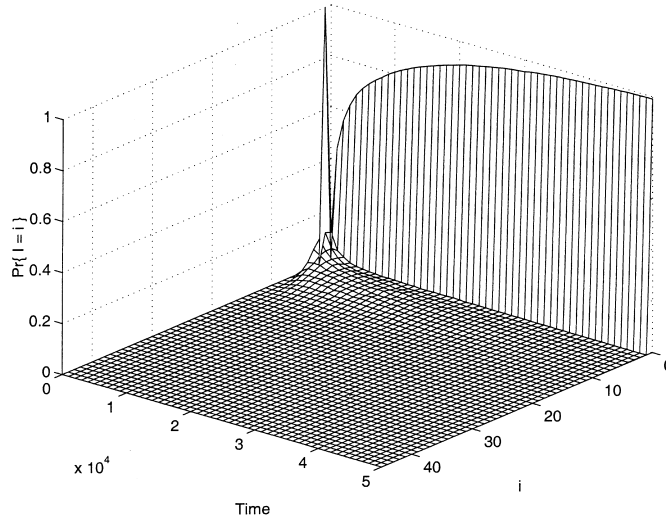


Fig. 18. The probability function for the number of infectives $\mathcal{I}(t)$ for the SIS model with variable population size is graphed when $\mathcal{R}_0 = 2$, $\mathcal{I}(0) = 1$, $\mathcal{S}(0) = 4$, $K = 15$, $\alpha = 1.6$, $\beta = 0.4 = \gamma$, $r = 0.25$, $M = 45$, and $\Delta t = 0.002$.

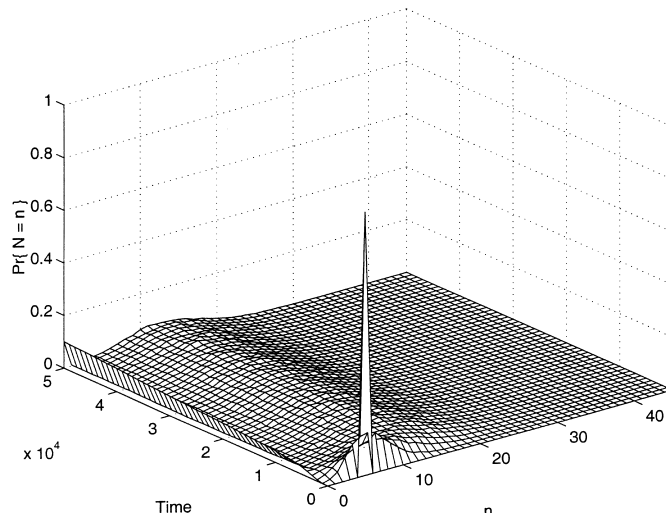


Fig. 19. The probability function for the total population size $\mathcal{N}(t)$ for the SIS model with variable population size is graphed for the parameter values given in Fig. 18.

$K = 15$, and for a longer time frame, time $(\Delta t) = 50000(0.002) = 100$, ultimate disease elimination can be observed. In Fig. 18, the disease is eliminated at a faster rate than in the SIS model with constant population size. This faster rate is probably due to the small population size and the small number of susceptibles, $S(0) = 4$ in Fig. 18 as opposed to $S(0) = 14$ in Fig. 10. In this example, it can also be observed that the total population size will eventually approach 0. In Fig. 19, the probability function for the total population size, $\mathcal{N}(t)$, shows that as time increases, the probability that the population size is 0 also increases.

In the last example, the expected duration of the epidemic is estimated by calculating the average duration from 10 000 sample paths when $K = 20$, $\mathcal{R}_0 = 2$, and $\mathcal{N}(0) = K$. In Fig. 11, the expected duration is compared to the SIS model with constant population size when the initial number of infectives is varied from 1 to 20. It can be seen that the expected duration is shorter for the variable population size model. This shortened duration may be due in part to the fact that the total size in the variable population size model is on the average less than K , $N < K$, but in the constant population size model $N = K$.

5. Summary

Discrete-time deterministic and stochastic models are formulated and analyzed for three different models: SIS model with constant population size, SIS model with variable population size, and SIR model with constant population size. These discrete-time models may be directly applicable to particular diseases (e.g., [4,33]) or may be considered as approximations to the more well-known continuous-time models. The discrete-time epidemic models are new formulations which generalize the form of the force of infection.

For the deterministic cases, Theorems 1–3 state asymptotic results for the three models. The basic reproductive number \mathcal{R}_0 determines whether the disease is eliminated or persists. In the case of persistence, the endemic equilibrium depends on the form of the force of infection.

The discrete-time stochastic epidemic models are formulated as Markov chains which may be considered approximations to the continuous-time Markov jump processes. Restrictions are put on the size of the time step to ensure that the models give true probability distributions.

In the stochastic models, the probability of disease elimination ultimately approaches 1, independent of the value of the basic reproductive number. However, as the population size increases, the time until absorption also increases. In these cases, the quasi-stationary distribution is significant. Difference equations for the mean and quasi-stationary mean are obtained. The numerical examples illustrate the form of the the probability distribution and the quasi-stationary distribution for the number of infectives. The probability distribution is bimodal when $\mathcal{R}_0 > 1$ and the initial number of infectives is small. One mode is at $\mathcal{I} = 0$. The second mode is the mean of the quasi-stationary distribution which is close to the deterministic endemic equilibrium. The shape of the quasi-stationary distribution appears to be approximately normal for $\mathcal{R}_0 > 1$ and N sufficiently large, which agrees with continuous-time stochastic SIS and SIR models with standard incidence studied by Nåsell [11–13]. In other examples, the behavior of the SIS Markov chain model agrees with that of the continuous-time SIS Markov jump process: the relation to a random walk and the expected duration of the epidemic. West and Thompson [5] also showed agreement between the behavior of the discrete and continuous-time stochastic SI models.

There is some distinction between the behavior of the SIS stochastic model with variable population size and the model with constant population size. The expected duration is less and the rate of convergence to extinction is faster in the variable population size model as illustrated in Figs. 10, 11 and 19. In the SIS model with variable population size, when the population size is less than the carrying capacity, births exceed deaths and the length of infectivity increases from $1/(\gamma\Delta t + \beta\Delta t)$ to $1/(\gamma\Delta t + \beta\Delta t - f(n))$ ($f(n) > 0$ when $n > K$). However, the reverse occurs when

the population size is greater than the carrying capacity, deaths exceed births and the length of infectivity decreases since $f(n) < 0$ when $n > K$.

There remain some open questions in regard to the behavior of the epidemic models. The global behavior of the deterministic SIR model in the case $\mathcal{R}_0 > 1$ needs to be verified. In addition, analytical approximations to the quasi-stationary distribution and the expected duration of the epidemic are needed for the stochastic SIR model and the SIS model with variable population size (see e.g., [11–13]).

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