

Dynamic Population Epidemic Models

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ABSTRACT

Most multipopulation epidemic models are of the contact distribution type, in which the locations of successive contacts are chosen independently from appropriate contact distributions. This paper is concerned with an alternative class of models, termed dynamic population epidemic models, in which infectives move among the populations and can infect only within their current population. Both the stochastic and deterministic versions of such models are considered. Their threshold behavior is analyzed in some depth, as are their final outcomes. Velocities of spread of infection are considered when the populations have a spatial structure. A criterion for finding the equivalent contact distribution epidemic for any given dynamic population epidemic is provided, enabling comparisons to be made for the velocities and final outcomes displayed by the two classes of models. The relationship between deterministic and stochastic epidemic models is also discussed briefly.

1. INTRODUCTION

Research into the behavior of spatial epidemic models has generally concentrated on contact distribution models (e.g., [24]), in which the population remains static and the locations of successive contacts of an infectious individual are chosen independently from a contact distribution. Although such models are clearly appropriate for certain plant diseases spread by infected spores (see, e.g., [32]–[36]), for certain human and animal diseases it seems sensible to consider models in which the spatial spread of infection is caused by the mobility of the population. Consider, for example, fox rabies. In many habitats foxes live in well-defined social groups with little overlap between neighboring territories [22]. Thus a model in which susceptible foxes remain fixed while rabid foxes move among the groups infecting only within their current group seems plausible. For other epidemics, such as the spread of influenza among Russian cities (see the work of Baroyan and his coworkers reported in Bailey [2, chapter

19)), a model incorporating movement of both susceptibles and infectives is preferred. Models in which both infectives and susceptibles move tend to be rather difficult to treat theoretically. More progress can be made with models in which only the infectives move. These latter models, which we term *dynamic population epidemic models*, are the subject of this paper.

Dynamic population epidemic models have been studied by Ball [3, 6], Faddy [15, 16], and Faddy and Slorack [17]. Ball considered a stochastic model for the spread of fox rabies, for which he proved some threshold results and described some simulations. Faddy and Slorack [17] and Faddy [15] considered a one-dimensional model in which infectives can move only forward. For the simple epidemic (no removal of infectives) they obtained bounds on the asymptotic velocity of spread of infection; for the general epidemic (with removal of infectives) Faddy [15] considered threshold and (spatial) equilibrium behavior. In [16] Faddy considered a more general deterministic spatial epidemic model, incorporating both movement of infectives and contact between neighboring locations, and derived a set of equations that determine the final outcome of the epidemic. My aim in this paper is to develop a general theory for dynamic population epidemic models, with particular emphasis on comparing their properties with those of equivalent contact distribution models and on the relationship between the deterministic and stochastic formulations.

The basic models are described in Section 2, and their threshold behavior is discussed in Section 3. A criterion for matching dynamic population and contact distribution epidemics is given in Section 4, where the equivalent contact distribution epidemic for any given dynamic population epidemic is derived. Final outcomes and velocities of spread of infection for the various models are considered in Sections 5 and 6, respectively. Finally, some comments on the connection between deterministic and stochastic models are made in Section 7.

2. BASIC MODELS

Consider a population partitioned into groups, each consisting of N susceptibles. The groups are labeled $1, 2, \dots, m$, where m may be ∞ . Let $\{Z(t); t \geq 0\}$ be a time-homogeneous continuous-time Markov chain, with state space $\mathbb{N}$ and transition-rate matrix Q . That is, $Q = (q_{ij})$, where

$$q_{ij} = \begin{cases} \lim_{h \downarrow 0} h^{-1} \Pr[Z(h) = j \mid Z(0) = i] & \text{if } i \neq j, \\ -\sum_{j \neq i} q_{ij} & \text{if } i = j. \end{cases}$$

A given infective moves around the groups according to the law of $\{Z(t); t \geq 0\}$, starting from their initial group, for a time that is exponentially distributed with mean γ^{-1} and is then removed. Throughout that period it

makes contacts at the points of a homogeneous Poisson process at rate β . Each contact is with an individual within our infective's current group; the individual contacted is chosen independently and uniformly from the N initial susceptibles in that group. If the individual contacted is still susceptible, then it is infected and immediately starts moving around the groups contacting other individuals; that is, there is no latent period. If it is not still susceptible, then nothing happens. The behaviors of different infective individuals are probabilistically mutually independent. The epidemic is initiated by one of the susceptibles in group 1 becoming infectious and terminates as soon as there are no infectives remaining in the population.

In many situations the partitioning of the population into groups and the continuous-time Markov chain $\{Z(t); t \geq 0\}$ will reflect an underlying spatial structure. In this paper we consider models in which the groups are located at the points of the d -dimensional integer lattice, $\mathbb{Z}^d$, where d will usually be 1 or 2. For such models it is convenient to index the groups by $\mathbb{Z}^d$ rather than by $\mathbb{N}$. The continuous-time Markov chain that we consider is the d -dimensional simple symmetric continuous-time random walk, in which jumps to each of the $2d$ nearest neighbors occur at rate $(2d)^{-1}\lambda$. I refer to these spatial models as *spatial dynamic population epidemics* and to our earlier model as the *general dynamic population epidemic*. Faddy [15] considered a model similar to our one-dimensional spatial dynamic population epidemic, except in his model infecteds stepped one to the right at rate λ .

We can derive deterministic models corresponding to the above stochastic models by viewing the infinitesimal transition probabilities in the latter as rates of change in the former. Thus if in our general model we let $x_i(t)$ and $y_i(t)$ be the numbers of susceptibles and infectives, respectively, in group i at time t , we obtain that

$$\frac{dx_i}{dt} = -\beta N^{-1} x_i y_i, \quad (2.1)$$

and

$$\frac{dy_i}{dt} = \beta N^{-1} x_i y_i + \sum_{j=1}^{\infty} q_{ji} y_j - \gamma y_i, \quad i = 1, 2, \dots, \quad (2.2)$$

with initial condition $x_i(0) = N$, $y_i(0) = 0$, $i \neq 1$, and $x_1(0) = N - 1$, $y_1(0) = 1$. It will sometimes be convenient to work in terms of densities (proportions) rather than absolute numbers, so let $\tilde{x}_i(t) = N^{-1} x_i(t)$ and $\tilde{y}_i(t) = N^{-1} y_i(t)$ be the densities of susceptibles and infectives, respectively, in

group i at time t . Then (2.1) and (2.2) become

$$\frac{d\tilde{x}_i}{dt} = -\beta \tilde{x}_i \tilde{y}_i, \quad (2.3)$$

$$\frac{d\tilde{y}_i}{dt} = \beta \tilde{x}_i \tilde{y}_i + \sum_{j=1}^{\infty} q_{ji} \tilde{y}_j - \gamma \tilde{y}_i, \quad i = 1, 2, \dots, \quad (2.4)$$

with initial condition $\tilde{x}_i(0) = 1$, $\tilde{y}_i(0) = 0$, $i \neq 1$, and $\tilde{x}_1(0) = 1 - N^{-1}$, $\tilde{y}_1(0) = N^{-1}$. The model described by (2.1) and (2.2) is a special case of a model studied by Faddy [16].

The deterministic version of our one-dimensional spatial model is, in obvious notation,

$$\frac{d\tilde{x}_i}{dt} = -\beta \tilde{x}_i \tilde{y}_i, \quad (2.5)$$

$$\frac{d\tilde{y}_i}{dt} = \beta \tilde{x}_i \tilde{y}_i + \frac{\lambda}{2} (\tilde{y}_{i-1} + \tilde{y}_{i+1}) - \lambda \tilde{y}_i - \gamma \tilde{y}_i, \quad i \in \mathbb{Z}, \quad (2.6)$$

with initial conditions $\tilde{x}_i(0) = 1$, $\tilde{y}_i(0) = 0$, $i \neq 0$, and $\tilde{x}_0(0) = 1 - N^{-1}$, $\tilde{y}_0(0) = N^{-1}$. Note that the density formulations of our deterministic models depend only on the group size N through the initial conditions.

3. THRESHOLD BEHAVIOR

3.1. DETERMINISTIC MODELS

The threshold behavior of deterministic epidemic models can be related to the basic reproduction ratio R_0 , defined as the expected number of secondary cases produced by a typical infected individual during its entire period of infectiousness (see, e.g., [13]). Note that in the above definition of R_0 we ignore depletion of the susceptible population owing to the infection process; that is, all contacts result in infection. For epidemics in heterogeneous populations, care is required in determining what is meant by a typical infective, and instead R_0 can be defined as the dominant eigenvalue of a positive linear operator [13]. However, for our general dynamic population epidemic model, all infectives make an average of $\beta\gamma^{-1}$ contacts during their infectious periods, so $R_0 = \beta\gamma^{-1}$. The interpretation of the threshold behavior in the present deterministic context is that if $R_0 \leq 1$ the total number of infectives in the populations decreases monotonically to zero, whereas if $R_0 > 1$ it first increases and then decreases to zero.

3.2. STOCHASTIC MODELS

For stochastic models, threshold behavior has a quite different interpretation. To obtain a sharp change in behavior at the threshold it is usual to let the initial susceptible population size tend to infinity and say that a major epidemic occurs if the total size of the epidemic also tends to infinity (see e.g., [42], [43]). However, for multipopulation epidemic models there are

several ways in which the initial susceptible population size can tend to infinity, which gives rise to quite different threshold values for R_0 , as I now illustrate for our dynamic population epidemic models.

First consider the situation when the group size N tends to infinity; the number of groups m may be finite or countably infinite. The only difference between the process of infectives in our stochastic general dynamic population epidemic model and an equivalent linear branching process (in which individuals live for an exponentially distributed time with mean γ^{-1} , move around the groups according to the law of $\{Z(t); t \geq 0\}$, and have offspring at the points of a homogeneous Poisson process with rate β) is that in the epidemic process not all contacts result in a new infective. Thus the branching process provides an upper bound for the process of infectives in the epidemic process (cf. [42]). However, as the group size N tends to infinity, the probability of contacting one of a finite set of infectives tends to zero, so we would expect the epidemic processes to converge in some sense to the branching process. This intuition can be made rigorous by defining both the sequence of epidemic processes and the branching process on a common probability space and using a coupling argument, as in [4], [7], and [23]. We obtain that, as $N \rightarrow \infty$ over any finite time interval $[0, t]$, the process of infectives in the epidemic process converges almost surely to the branching process and that the total size of the epidemic process (i.e., the total numbers of initial susceptibles ultimately infected in each of the groups) converges to that of the branching process. Thus in the limit as $N \rightarrow \infty$, a major epidemic occurs if and only if the branching process does not go extinct.

The total number of individuals alive in the branching process follows a linear birth-and-death process with birth rate β and death rate γ . The following theorem follows immediately from elementary facts concerning birth-and-death processes.

THEOREM 3.1

In the limit as $N \rightarrow \infty$:

(i) *A major epidemic occurs with nonzero probability if and only if $R_0 = \gamma^{-1}\beta > 1$.*

(ii) *The probability of a major epidemic is $1 - p^a$, where a is the initial number of infectives and $p = \min\{1, \gamma\beta^{-1}\}$.*

(iii) *The total number of initial susceptibles ultimately infected by the disease, T say, has the distribution*

$$P(T = k) = \left(\frac{(2k + a - 1)! a}{k! (k + a)!} \right) \left(\frac{\gamma^{k+a} \beta^k}{(\gamma + \beta)^{2k+a}} \right), \quad k = 0, 1, \dots \quad (3.1)$$

If $R_0 > 1$, the distribution (3.1) is defective, with total mass p^a . The remaining mass $1 - p^a$ corresponds to $T = \infty$.

Further information concerning the behavior of the limiting behavior of the epidemic process as $N \rightarrow \infty$, such as the total size of the epidemic in each of the groups, can be obtained from more detailed knowledge of the branching process. Note that the threshold value of R_0 is the same as in the deterministic epidemic.

We now consider the situation when the group size N is finite but the number of groups M is countably infinite.

THEOREM 3.2

The one-dimensional spatial dynamic population epidemic process of Section 2 goes extinct with probability 1 if the initial number of infectives is finite.

Proof. The proof parallels the argument of [18] for the one-dimensional contact distribution epidemic. Consider the case $N = 1$ and suppose without loss of generality that the rightmost infective at time $t = 0$ is located at the origin. Let q be the probability that no susceptibles to the right of the origin are infected during the epidemic. Then q is not less than the corresponding probability if all individuals to the left of the origin are infectious at time $t = 0$. That is,

$$q \geq \prod_{i=1}^{\infty} q_i = \tilde{q},$$

where q_i is the probability that the infective at the origin fails to infect any susceptible located in group j for some $j \geq i$. Let q'_i be the corresponding probability when the underlying random walk $\{Z(t); t \geq 0\}$ makes unit steps to the right at rate λ . Then clearly $q_i \geq q'_i$ and

$$q'_i = 1 - \left(\frac{\lambda}{\lambda + \gamma} \right)^i \left(\frac{\beta}{\beta + \gamma} \right), \quad i = 1, 2, \dots,$$

because the probability that an infective steps to the right before being removed is $\lambda/(\lambda + \gamma)$, once reaching i the probability that it infects a susceptible before dying is $\beta/(\beta + \gamma)$, and we have exploited the lack-of-memory property of exponential random variables. It follows that $\sum_{i=1}^{\infty} (1 - q'_i) < \infty$, so $\prod_{i=1}^{\infty} q'_i > 0$ and hence $\tilde{q} > 0$. A similar argument shows that the expected number of susceptibles to the right of the origin infected by the initial infectives is finite. Thus even if susceptibles to the right of the origin are infected by the initial infectives, only finitely many will be so, so we can fix attention on the rightmost such individual and repeat the above

argument to deduce that the probability that the epidemic infects susceptibles to the right of this individual is at most $1 - \tilde{q}$. Thus the epidemic eventually stops spreading to the right with probability 1, since $\tilde{q} > 0$. Similarly, the epidemic eventually stops spreading to the left and hence goes extinct almost surely. The argument can be extended to the case $N > 1$ by replacing $\tilde{q}$ by $\tilde{q}^N$.

THEOREM 3.3

Let $\pi = \pi(\gamma, \lambda, \beta, N)$ be the probability that the two-dimensional spatial dynamic population epidemic, initiated by one infective at the origin, ultimately goes extinct. Then

- (i) There exists $\gamma_0 = \gamma_0(\lambda, \beta, N) > 0$ such that $\pi = 1$ for $\gamma > \gamma_0$ and $\pi < 1$ for $\gamma < \gamma_0$, and
- (ii) If $N = 1$, $\gamma_0 \leq \min[\beta, (\sqrt{3}/2)\lambda]$.

Proof. First note that, for fixed β , λ , and N , epidemics for different values of γ can be coupled so that their total size is stochastically decreasing with γ , and hence π increases with γ . Also, since the branching process both provides an upper bound for the epidemic process and goes extinct almost surely if $\gamma \geq \beta$, $\pi = 1$ if $\gamma \geq \beta$. Thus to prove (i) we have to show that $\pi < 1$ for γ sufficiently small. This can be done by comparing the epidemic with an appropriate percolation model (cf. [21], [24]) as follows.

A simple coupling argument shows that the total size of the epidemic is stochastically increasing with N , so we need only consider the case $N = 1$. Consider the initial infective at the origin and let p be the probability that it infects all of its four nearest neighbors. The random walk $\{Z(t); t \geq 0\}$ is recurrent so $p \uparrow 1$ as $\gamma \downarrow 0$. Consider the site percolation process in which each point independently is either open (i.e., has arcs to all four of its nearest neighbors) or closed (i.e., has no arcs emanating from it) with probabilities p and $1 - p$, respectively. Construct an epidemic from this site percolation process by saying that an individual is ultimately infected if there is a chain of open points (i.e., set of nearest-neighboring open points) joining the initial infective at the origin to that individual. It is well known that there is a nonzero probability that infinitely many points are so connected to the origin if p is greater than some critical value p_0 (see, e.g., [14]). Further, the total size of the dynamic population epidemic is clearly stochastically greater than that of the epidemic constructed from the percolation process. Thus $\pi < 1$ if γ is less than some critical value γ_0 .

To prove (ii), first note that our earlier comparison with the branching process shows that $\gamma_0 \leq \beta$. Let R be the number of susceptibles contacted by the initial infective. Then R is not greater than S , the number of groups (not including the origin) visited by the initial infective. The probability that the initial infective leaves the origin before being removed is $\lambda/(\lambda + \gamma)$,

and given it has left the origin it enters a new group with rate $3\lambda/4$ (it moves back to its previous group at rate $\lambda/4$) and is removed at rate γ . Thus, by the lack-of-memory property of the exponential distribution,

$$P(S \geq k) \leq \frac{\lambda}{\lambda + \gamma} \left(\frac{3\lambda}{3\lambda + 4\gamma} \right)^{k-1}, \quad k = 1, 2, \dots,$$

so

$$E[S] \leq \frac{\lambda}{\lambda + \gamma} \sum_{k=1}^{\infty} \left(\frac{3\lambda}{3\lambda + 4\gamma} \right)^{k-1} = \frac{\lambda(3\lambda + 4\gamma)}{4\gamma(\gamma + \lambda)}.$$

It follows that $E[R] \leq 1$ unless $\gamma < \sqrt{3}\lambda/2$, so again by comparison with the branching process $\gamma_0 \leq \sqrt{3}\lambda/2$, and (ii) follows.

Remarks

(1) The inequality (ii) can be extended easily to incorporate the case $N > 1$.

(2) One could probably prove threshold results similar to (i) but with varying β , λ , or N rather than γ . However, the situation is not so clear, and there will be constraints on the nonvarying parameters because of (ii).

(3) The threshold behavior is not as simple for these stochastic models as it was for the corresponding deterministic models and can no longer be related to the single quantity R_0 .

(4) Note that the proof of Theorem 3.3 breaks down in $d \geq 3$ dimensions because the random walk $\{Z(t); t \geq 0\}$ is no longer recurrent. However, the threshold phenomenon is still present as the following argument shows. For $(i, j) \in \mathbb{Z}^2$, let $S_{i,j} = \{(i, j, i_3, i_4, \dots, i_d): i_k \in \mathbb{Z}, k = 3, 4, \dots, d\}$. The random walk obtained by projecting $\{Z(t); t \geq 0\}$ onto its first two coordinates is recurrent, so if p is the probability that the initial infective at the origin infects susceptibles in each of the sets $S_{1,0}$, $S_{-1,0}$, $S_{0,1}$, and $S_{0,-1}$, then $p \uparrow 1$ as $\gamma \downarrow 0$. Thus if we treat the sets $S_{i,j}$ as our infectious units, rather than the individual groups, then by comparison with a two-dimensional site percolation process, as before, there is a nonzero probability of infinitely many $S_{i,j}$ being infected, and hence infinitely many susceptibles being infected, provided γ is sufficiently small.

4. MATCHING OF DYNAMIC POPULATION AND CONTACT DISTRIBUTION MODELS

We first make clear what we mean by a contact distribution model (see, e.g., [24]). The group structure is the same as for our general model of Section 2. Infectives are still infectious for a time that is exponentially distributed with mean γ^{-1} and make contacts at the points of a homoge-

neous Poisson process with rate β . Infectives no longer move, but the group with which each contact is made is chosen independently from an appropriate contact distribution. Specifically, there is a matrix $P = (p_{ij})$, where p_{ij} is the probability that a contact of an infective from group i is with an individual in group j . Once the contacted group is chosen, the individual contacted is chosen uniformly from the N initial susceptibles in that group and if susceptible becomes infected. Again the behaviors of different infectives are mutually independent.

In order to compare the properties of dynamic population and contact distribution epidemic models, we need some criterion for matching the two models. Specifically, given a dynamic population model we require some method of determining the contact matrix P for an equivalent contact distribution model. The criterion we adopt is that, for any i and j , the mean numbers of contacts in group j made by an infective from group i are the same in the two models.

THEOREM 4.1

For a given general dynamic population epidemic model, the equivalent contact distribution model has

$$P = \gamma(\gamma I - Q)^{-1}. \quad (4.1)$$

Proof. Consider the dynamic population epidemic model. For $i, j = 1, 2, \dots$, let m_{ij} be the mean number of contacts in group j made by an infective from group i . Consider a given infective from group i . Let T be its infectious period, $R = \inf\{t > 0: Z(t) \neq i \mid Z(0) = i\}$, and $S = \min(T, R)$. Then S has an exponential distribution with mean $(\gamma - q_{ii})^{-1}$, and conditioning on S we obtain

$$\begin{aligned} m_{ii} &= \int_0^\infty (\gamma - q_{ii}) e^{-(\gamma - q_{ii})t} \left\{ \frac{\gamma}{\gamma - q_{ii}} \beta t + \sum_{k \neq i} \frac{q_{ik}}{\gamma - q_{ii}} (\beta t + m_{ki}) \right\} dt \\ &= \beta(\gamma - q_{ii})^{-1} + \sum_{k \neq i} q_{ik}(\gamma - q_{ii})^{-1} m_{ki}, \end{aligned} \quad (4.2)$$

using the Markov property and the lack-of-memory property of the exponential distribution. A similar conditioning argument shows that

$$m_{ij} = \sum_{k \neq i} q_{ik}(\gamma - q_{ii})^{-1} m_{kj}, \quad j \neq i. \quad (4.3)$$

It follows from (4.2) and (4.3) that

$$\gamma m_{ii} - \sum_{k=1}^{\infty} q_{ik} m_{ki} = \beta$$

and

$$\gamma m_{ij} - \sum_{k=1}^{\infty} q_{ik} m_{kj} = 0, \quad j \neq i.$$

Thus, if we let $M = (m_{ij})$,

$$\gamma M - QM = \beta I,$$

so $M = \beta(\gamma I - Q)^{-1}$.

Turning now to the contact distribution model with contact matrix P , let m_{ij}^c be the mean number of contacts in group j made by an infective from group i and $M^c = (m_{ij}^c)$. By a standard result concerning Poisson processes, the points in time at which a given infective from group i makes contacts within group j form a homogeneous Poisson process with rate $p_{ij}\beta$. Thus, since the infectious period has mean γ^{-1} , $m_{ij}^c = \gamma^{-1}\beta p_{ij}$, so $M^c = \gamma^{-1}\beta P$. Equation (4.1) follows by equating M and M^c .

We turn now to the one-dimensional spatial dynamic population epidemic model. Since that model is spatially homogeneous, the contact matrix of its equivalent contact distribution model takes the form

$$p_{ij} = p_{j-i}, \quad i, j \in \mathbb{Z},$$

where p_i is the probability that a contact of an infective from the origin is with an individual from group i .

THEOREM 4.2

For a one-dimensional spatial dynamic population epidemic model, the equivalent contact distribution model has a double-geometric contact distribution. Specifically,

$$p_i = \frac{(1-\theta)\theta^{|i|}}{1+\theta}, \quad i \in \mathbb{Z}, \quad (4.2)$$

where

$$\theta = \left\{ \gamma + \lambda - (\gamma^2 + 2\lambda\gamma)^{1/2} \right\} / \lambda. \quad (4.3)$$

Proof. Theorem 4.2 can be proved by inverting the appropriate infinite matrix $\gamma I - Q$ in (4.1). However, it is probably simpler to argue directly as follows. For $i \in \mathbb{Z}$, let m_i be the mean number of contacts in group i made by an infective from the group at the origin in the dynamic population model. The conditioning arguments used in the proof of Theorem 4.1 yield

$$m_0 = \frac{\beta}{\gamma + \lambda} + \frac{\lambda}{2(\gamma + \lambda)} (m_{-1} + m_1) \quad (4.4)$$

and

$$m_i = \frac{\lambda}{2(\gamma + \lambda)} (m_{i-1} + m_{i+1}), \quad i \neq 0. \quad (4.5)$$

By symmetry, $m_i = m_{-i}$, so (4.4) yields

$$m_0 = (\beta + \lambda m_1) / (\gamma + \lambda). \quad (4.6)$$

The difference equation (4.5) has the general solution

$$m_i = A\theta_0^i + \beta\theta_1^i, \quad i = 0, 1, \dots,$$

where θ_0, θ_1 are the roots of $\lambda x^2 - 2(\gamma + \lambda)x + \lambda = 0$ and hence are given by

$$\theta_0, \theta_1 = \left\{ \gamma + \lambda \pm (\gamma + 2\gamma\lambda)^{1/2} \right\} / \lambda.$$

Now $\sum_{i=-\infty}^{\infty} m_i = \beta/\gamma < \infty$, so $A = 0$ because $\theta_0 > 1$. Thus m_i takes the form

$$m_i = B\theta^i, \quad i = 0, 1, \dots$$

Substitution into (4.6) determines B , and (4.2) follows upon noting that $p_i = (\gamma/\beta)m_i$.

5. TOTAL SIZE

5.1. DETERMINISTIC MODELS

An important epidemic characteristic is its final outcome, the numbers of initial susceptibles in each group that are ultimately infected by the epidemic. Consider the deterministic general dynamic population epidemic governed by Equations (2.3) and (2.4). Its final outcome can be determined by the following argument, due to Faddy [16], which generalizes one given

by Kendall [19, 20] for the single population. Note that it follows from (2.3) that

$$\tilde{y}_i = -\frac{1}{\beta} \frac{d}{dt} \ln \tilde{x}_i,$$

which upon substitution into (2.4) yields

$$\frac{d}{dt} \left(\tilde{y}_i + \tilde{x}_i + \frac{1}{\beta} \sum_{j=1}^{\infty} q_{ji} \ln \tilde{x}_j - \frac{\gamma}{\beta} \ln \tilde{x}_i \right) = 0. \quad (5.1)$$

For $i = 1, 2, \dots$, let n_i and $\hat{x}_i = x_i(\infty)$, respectively, be the initial and final density of susceptibles in group i . Now $\tilde{x}_i(0) + \tilde{y}_i(0) = 1$ and $\tilde{y}_i(\infty) = 0$, $i = 1, 2, \dots$. It then follows from (5.1) that

$$\hat{x}_i - 1 + \beta^{-1} \sum_{j=1}^{\infty} q_{ji} \ln \left(\frac{\hat{x}_j}{n_j} \right) - \gamma \beta^{-1} \ln \left(\frac{\hat{x}_i}{n_i} \right) = 0, \quad i = 1, 2, \dots \quad (5.2)$$

The set of equations (5.2) determines the final outcome of the deterministic general dynamic population epidemic.

Turning to the contact distribution epidemic, the equations governing the spread of disease are

$$\frac{d\tilde{x}_i}{dt} = -\beta \tilde{x}_i \sum_{j=1}^{\infty} \tilde{y}_j p_{ji}, \quad (5.3)$$

$$\frac{d\tilde{y}_i}{dt} = \beta \tilde{x}_i \sum_{j=1}^{\infty} \tilde{y}_j p_{ji} - \gamma \tilde{y}_i, \quad (5.4)$$

$$\frac{d\tilde{z}_i}{dt} = \gamma \tilde{y}_i, \quad (5.5)$$

where $\tilde{z}_i(t)$ denotes the density of removed individuals in group i at time t . The final outcome of this epidemic can be determined by the following argument due to Watson [38], which again is a generalization of Kendall [19, 20].

First note that it follows from (5.5) that

$$\sum_{j=1}^{\infty} \tilde{y}_j p_{ji} = \gamma^{-1} \sum_{j=1}^{\infty} \frac{d\tilde{z}_j}{dt} p_{ji},$$

which upon substitution into (5.3) yields, after integration,

$$\ln\left(\frac{\tilde{x}_i}{n_i}\right) = -\beta\gamma^{-1} \sum_{j=1}^{\infty} \tilde{z}_j p_{ji}, \quad i = 1, 2, \dots \quad (5.6)$$

If, as before, we let $\hat{x}_i = \tilde{x}_i(\infty)$, then $\tilde{z}_i(\infty) = 1 - \hat{x}_i$, and it follows from (5.6) that

$$\ln\left(\frac{\hat{x}_i}{n_i}\right) + \beta\gamma^{-1} \sum_{j=1}^{\infty} (1 - \hat{x}_j) p_{ji} = 0, \quad i = 1, 2, \dots \quad (5.7)$$

The set of equations (5.7) determines the final outcome of the deterministic general contact distribution epidemic.

THEOREM 5.1

If the general dynamic population and contact distribution epidemics are matched according to the criterion of Section 4, then they have the same deterministic final outcome.

Proof. We simply show that the sets of equations (5.2) and (5.7) are identical. It is convenient to introduce vector notation. Let $\hat{\mathbf{x}} = (\hat{x}_1, \hat{x}_2, \dots)^T$, $\mathbf{1} = (1, 1, \dots)^T$, and $\hat{\mathbf{u}} = [\ln(x_1/n_1), \ln(x_2/n_2), \dots]^T$. Index $\hat{\mathbf{x}}$ and $\hat{\mathbf{u}}$ by D or C to denote dynamic population or contact distribution model, respectively. Then (5.2) and (5.7) become

$$\hat{\mathbf{x}}_D - \mathbf{1} + \beta^{-1}(\mathbf{Q}^T - \gamma\mathbf{I})\hat{\mathbf{u}}_D = \mathbf{0}, \quad (5.8)$$

$$\hat{\mathbf{u}}_C + \beta\gamma^{-1}\mathbf{P}^T(\mathbf{1} - \hat{\mathbf{x}}_C) = \mathbf{0}. \quad (5.9)$$

But from Theorem 4.1, $\mathbf{P}^T = \gamma(\gamma\mathbf{I} - \mathbf{Q}^T)^{-1}$, and it is easily verified that Equations (5.8) and (5.9) are identical.

Remark 1. The fact that the two epidemics have the same deterministic final outcome admits a simple intuitive explanation. The density of initial susceptibles ultimately infected in a given group is directly proportional to the total amount of infection that group is exposed to during the course of the epidemic; the temporal pattern of such exposure to infection does not matter. The two models are matched so that a given infective yields identical patterns of mean total exposure to infection in the two. Thus the final outcomes of the two epidemics will be identical.

Remark 2. Although the two epidemics have the same final outcome, they have different temporal patterns of spread, as can be seen from their governing differential equations. This is because infectives in the two

epidemics give rise to different temporal patterns of mean exposure to infection.

5.2. STOCHASTIC MODELS

When the number of groups m is finite, the final outcome of the stochastic general dynamic population epidemic can be studied using techniques similar to those of Ball [5]. The details are rather lengthy and will be presented elsewhere. In short, it is possible to derive a triangular set of linear equations governing the total size distribution and recursive expressions for the joint probability generating function (and hence for moments of all orders) of the numbers of susceptibles surviving the epidemic in each of the m groups. The results are similar in form to those obtained by Ball [5] for the contact distribution epidemic. However, the final size distributions are not identical for the two epidemics when matched by the criterion of Section 4. This is because although the mean profiles of the total number of contacts made by given infectives in the two epidemics are identical, their distributions are not.

5.3. EFFECT OF SPEED AT WHICH INFECTIVES MOVE

Suppose that in the general dynamic population epidemic the transition rate matrix Q of $\{Z(t); t \geq 0\}$ takes the form $Q = \lambda Q_0$, where $\lambda > 0$ can be viewed as the speed at which infectives move around the groups. A natural question to ask is, how does the speed λ affect the final size of the epidemic? We examine this in the context of a two-group model with

$$Q_0 = \begin{bmatrix} -1 & 1 \\ 1 & -1 \end{bmatrix},$$

that is, infectives change groups at rate λ . The final outcome of the deterministic version of this epidemic is determined by (5.2), although no explicit solution is available. Figure 1 shows the total number of susceptibles ultimately infected by the epidemic for various values of λ when $N = 10$, $\gamma = 8.0$, $\beta = 1.0$, and initially one of the susceptibles in group 1 becomes infected. Note that the overall total size increases with λ . I conjecture that this will happen for all sets of parameter values, though it does not seem straightforward to prove from (5.2). (This conjecture has been proved by Damian Clancy. The proof will be presented elsewhere.) I conjecture also that a similar result will hold for the stochastic model, that is, that the overall total size will be stochastically increasing in λ , though again a proof seems elusive. These conjectures seem plausible, even in a more general setting, because as λ increases, the probability of a given infective contacting the same person more than once decreases. Note, however, that these conjectures do not hold for certain heterogeneous

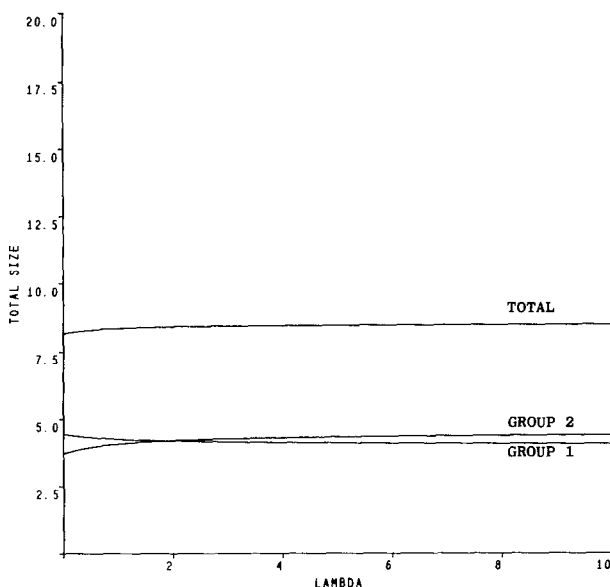


FIG. 1. Final outcome of deterministic dynamic population epidemic model with two groups. Further details are given in the text.

populations. For example, consider the above two-group model with initial susceptible population sizes $N_1 = 9$ and $N_2 = 0$ and an initial infective in group 1. Then any time spent in group 2 is wasted as far as the epidemic is concerned, so the overall total size is decreasing with λ .

6. VELOCITIES

In this section we compare the velocity of spread of infection in a spatial dynamic population epidemic model with that of an equivalent contact distribution model. For ease of calculation and simulation we consider only one-dimensional epidemics. We shall compute the asymptotic wave-front velocities for the deterministic models and use simulations to estimate the speed of propagation of infection in the stochastic models. There is a difficulty with the stochastic models because the infection ultimately goes extinct with probability 1, so we shall estimate the velocity given that the epidemic has yet to die out.

There is a considerable literature on the asymptotic velocity of spread of the deterministic contact distribution epidemic (see, e.g., [1], [8], [11], [12], [20], [24], [25], [30], [31], [36]). A general approach is given in [36] (see also [11] and [25]), which we now outline in our present context of a

population concentrated on $\mathbb{Z}$. The method involves calculating the velocity for the linear model in which all contacts result in infection. Let $\tilde{B}(a, i, j)$ be the density of newborns produced per unit time in group i by an individual of age a born in group j . We assume that the population is spatially homogeneous, so

$$\tilde{B}(a, i, j) = B(a, i - j).$$

Let $b(t, i)$ be the number of births per unit time in group i at time t . As we are interested in asymptotic behavior, the population equation takes the form

$$b(t, i) = \int_0^\infty \sum_{j=-\infty}^\infty b(t-a, j) B(a, i-j) da. \quad (6.1)$$

We seek traveling wave solutions of (6.1) having the form

$$b(t, i) = \tilde{b}(ct - i).$$

Substitution of the trial solution

$$\tilde{b}(ct - i) = \exp[\mu(ct - i)]$$

leads to the characteristic equation

$$L(c, \mu) = 1, \quad (6.2)$$

where

$$L(c, \mu) = \int_0^\infty \sum_{j=-\infty}^\infty \exp[-\mu(ca - j)] B(a, j) da. \quad (6.3)$$

Let

$$R_0 = \int_0^\infty \sum_{j=-\infty}^\infty B(a, j) da$$

be the reproductive ratio for the process. Then, provided $R_0 > 1$, (6.2) admits solutions for all $c \geq c_0$, for some minimal velocity c_0 . Further, if the initial population has bounded support, then c_0 is the asymptotic velocity of population expansion. It is conjectured in [36] that the asymptotic velocity of expansion for certain nonlinear models equals that of its linear variant. This is proved in [11] for the one-dimensional continuous-space contact

distribution epidemic, subject to certain conditions on the contact distribution. We shall assume that this is also true for our discrete-space epidemic models.

THEOREM 6.1

Suppose that time is linearly rescaled so that $\beta = 1$ and the initial infectives have bounded support. Then, if $\gamma < 1$, the one-dimensional deterministic dynamic population epidemic has asymptotic velocity

$$c = \mu^{-1} [1 - \gamma - \lambda(1 - \cosh \mu)], \quad (6.4)$$

where μ is the positive root of

$$1 - \gamma - \lambda(1 - \cosh \mu + \mu \sinh \mu) = 0. \quad (6.5)$$

The equivalent contact distribution epidemic has asymptotic velocity

$$c = \frac{\gamma [1 - \gamma - \lambda(1 - \cosh \mu)]}{\mu [\gamma + \lambda(1 - \cosh \mu)]}, \quad (6.6)$$

where μ is the positive root of

$$\mu \lambda \sinh \mu - [\gamma + \lambda(1 - \cosh \mu)] [1 - \gamma - \lambda(1 - \cosh \mu)] = 0. \quad (6.7)$$

Proof. Consider first the dynamic population epidemic. We need to determine the corresponding $B(a, i)$. Let

$$p_i(t) = P[Z(t) = i \mid Z(0) = 0], \quad i \in \mathbb{Z}; t \geq 0.$$

The probability that an infective is still infectious a units of time after its infection is $e^{-\gamma a}$, so

$$B(a, i) = e^{-\gamma a} p_i(a).$$

Thus,

$$L(c, \mu) = \int_0^\infty \exp[-(\gamma + \mu c)a] \sum_{j=-\infty}^\infty \exp(\mu j) p_j(a) da.$$

By a standard argument $p_i(t)$ satisfies the forward equation

$$\frac{dp_i}{dt} = \frac{\lambda}{2} (p_{i-1} + p_{i+1} - p_i), \quad i \in \mathbb{Z}.$$

Thus if we let

$$h(s, t) = \sum_{i=-\infty}^{\infty} s^i p_i(t), \quad t \geq 0; |s| \leq 1,$$

it follows that

$$\frac{\partial h}{\partial t} = \frac{\lambda}{2} (s + s^{-1} - 2) h$$

and $h(0, s) \equiv 1$. Thus,

$$h(s, t) = \exp \left[\frac{\lambda}{2} (s + s^{-1} - 2) t \right],$$

and hence

$$L(c, \mu) = [\mu c + \gamma + \lambda(1 - \cosh \mu)]^{-1}.$$

Elementary calculus now shows that the asymptotic wave-front velocity is given by (6.4) and (6.5).

Turning to the equivalent contact distribution model, with contact distribution $\{p_i; i \in \mathbb{Z}\}$ given by Theorem 4.2. We have $B(a, i) = e^{-\gamma a} p_i$, so

$$\begin{aligned} L(c, \mu) &= \int_0^{\infty} \exp[-(\gamma + \mu c)a] \sum_{j=-\infty}^{\infty} \exp(\mu j) p_j da \\ &= (\gamma + \mu c)^{-1} \sum_{j=-\infty}^{\infty} \exp(\mu j) p_j. \end{aligned}$$

Let $h(s) = \sum_{j=-\infty}^{\infty} s^j p_j$. Then it follows from (4.4) and (4.5) that

$$h(s) = (\gamma + \lambda)^{-1} \left[\frac{\lambda}{2} (s + s^{-1}) h(s) + \gamma \right],$$

so

$$h(s) = \gamma \left[\gamma + \frac{\lambda}{2} (2 - s - s^{-1}) \right]^{-1}, \quad |s| \leq 1.$$

Thus,

$$L(c, \mu) = \gamma (\gamma + \mu c)^{-1} [\gamma + \lambda(1 - \cosh \mu)]^{-1},$$

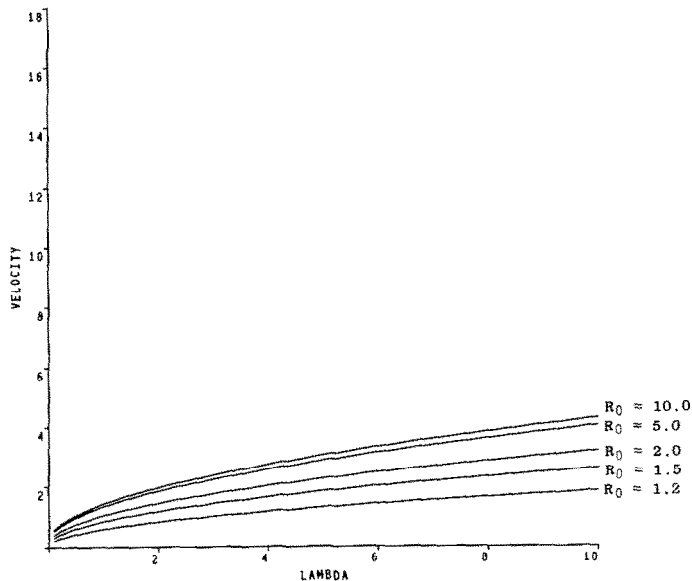
and again elementary calculus shows that the asymptotic wave-front velocity is given by (6.6) and (6.7).

Computed velocities for the two models for various values of R_0 and λ are shown in Figure 2, which displays two immediately striking features. First, the velocity of a dynamic population epidemic is smaller than that of its equivalent contact distribution epidemic, the difference increasing with both λ and R_0 . Second, for fixed R_0 the velocity increases faster with λ for the contact distribution model than for the dynamic population model, with the effect becoming more marked with increasing R_0 . Both of these phenomena can be explained by the fact that in the contact distribution epidemic, long-distance contacts are possible during the early stages of an infective's infectious period, an event that is extremely unlikely in the dynamic population epidemic. A formal proof of the first phenomenon is given in [25].

We next consider velocities for the stochastic versions of our models. It is not possible with current methodology to obtain theoretical results, so we resorted to simulation. We considered one-dimensional models with groups of size N ($= 1, 2, 3, 4$, or 5) located at each of the integers $-500, -499, \dots, 500$. The epidemics were initiated by one of the susceptibles at the origin becoming infected. As already noted, there is a problem in defining velocities for these one-dimensional models because extinction is almost sure. However, those simulated epidemics that did not die out quickly tended to propagate at a roughly constant velocity until either they eventually went extinct or the boundary of the study area was reached. We estimated the velocity of such epidemics as follows. We considered only those epidemics that reached at least as far as the group at 100, and to diminish the effect of the initial condition we used only the spread to the right of the group at 50 to estimate velocity. Let $d(t)$ be the maximum positive spread of the epidemic at time t , $t_0 = \inf_{t > 0} [t: d(t) \geq 50]$, $d_0 = d(t_0)$, and for $i = 1, 2, \dots$, $t_i = \inf_{t > t_{i-1}} [t: d(t) > d_{i-1}]$, $d_i = d(t_i)$, with the implicit assumption that the process terminates as soon as the epidemic dies out or $d(t) > 500$. Thus we obtain a set of points $\{(t_i, d_i); i = 0, 1, \dots, p\}$, and, provided $p \geq 2$, the velocity c is estimated by fitting the straight line $d - d_0 = c(t - t_0)$ by least squares. The results are shown in Table 1. The two phenomena displayed by the deterministic models, commented on earlier, are still present in the stochastic models. However, the velocities of the stochastic models are considerably less than those of the corresponding deterministic models. Further, the velocities of the stochastic models increase with group size N . This latter fact can be proved formally, because epidemics for different N can be coupled so that they are stochastically increasing with N .

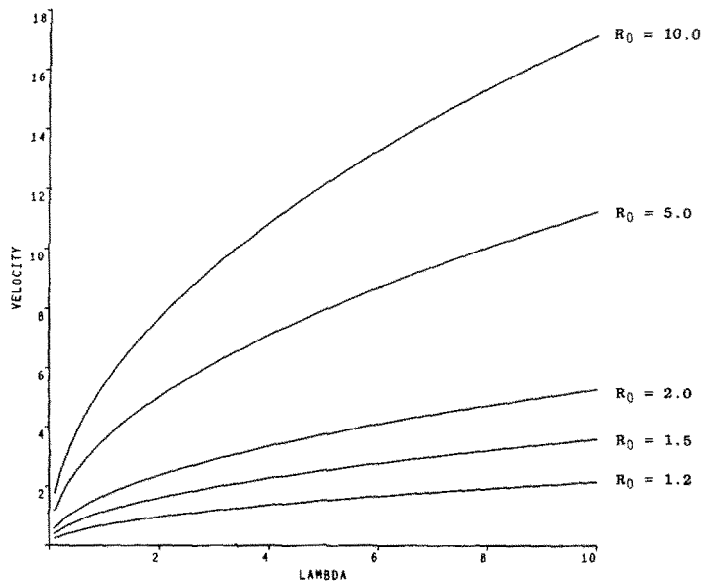
Recall from Section 3.1 that as $N \rightarrow \infty$ a stochastic dynamic population epidemic converges almost surely to an appropriate branching process; a similar conclusion holds for contact distribution models. Velocities for the limiting branching processes can be derived, as special cases, from [9] and

DYNAMIC POPULATION EPIDEMIC



(a)

CONTACT DISTRIBUTION EPIDEMIC



(b)

FIG. 2. Asymptotic wave-front velocities for deterministic spatial dynamic population and contact distribution epidemics. For all epidemics, $\beta = 1$ and $R_0 = \gamma^{-1}\beta$. Further details are given in the text.

TABLE 1

Velocities of Spread of Infection for Stochastic Spatial Epidemics with $\beta = 1, \gamma = 0.2$, and Various Values of λ and N^a

		Dynamic Population Epidemic			Contact Distribution Epidemic		
		Mean	σ	n	Mean	σ	n
$\lambda = 2,$	$N = 1$						
	2				1.03646	0.23408	3
	3	0.83603	0.09172	10	1.24733	0.24232	27
	4	0.94921	0.15338	35	1.39491	0.22898	53
	5	1.00128	0.09137	56	1.54750	0.19034	68
	∞	1.84256			5.02811		
$\lambda = 4,$	$N = 1$						
	2	1.26071	0.21328	16	1.58560	0.36541	20
	3	1.30170	0.16369	51	1.86158	0.36386	58
	4	1.43557	0.14253	75	2.19527	0.26018	65
	5	1.56108	0.09752	76	2.40160	0.26060	71
	∞	2.56970			7.10201		
$\lambda = 6,$	$N = 1$	1.33454	0.22150	3			
	2	1.56671	0.25501	38	1.85853	0.39435	46
	3	1.75222	0.15116	66	2.49757	0.47080	72
	4	1.90140	0.13265	73	2.84838	0.30732	80
	5	2.01306	0.11741	80	3.16273	0.34578	81
	∞	3.13131			8.69454		
$\lambda = 8,$	$N = 1$	1.63473	0.38522	6	1.60911	0.11834	2
	2	1.89033	0.23504	46	2.21822	0.43635	52
	3	2.13729	0.22267	70	2.88280	0.36149	72
	4	2.27716	0.13992	80	3.36346	0.43924	67
	5	2.40789	0.16054	80	3.77048	0.38145	75
	∞	3.60666			10.03550		
$\lambda = 10,$	$N = 1$	1.71210	0.44544	11	2.26073	0.84639	15
	2	2.15095	0.30778	59	2.72213	0.79964	56
	3	2.45036	0.19809	74	3.48865	0.59546	70
	4	2.64122	0.18131	80	3.95142	0.54076	74
	5	2.77066	0.13407	77	4.41357	0.54237	69
	∞	4.02604			11.22086		

^aThe $N = \infty$ values are theoretical, the remainder estimated from simulations. For the simulated velocities, the mean, standard deviation, and number of runs for each set of parameter values are shown. Further details are given in the text.

[10] and are identical to those for the corresponding deterministic models given in Theorem 6.1. Note that this supports the case that the asymptotic velocities of the deterministic epidemic models are equal to those of the corresponding deterministic linear models. For as $N \rightarrow \infty$, the velocity of the stochastic epidemic converges to that of the corresponding branching process. One would expect a similar result to hold for deterministic models.

The dynamic equations for the deterministic epidemics, in density form, are independent of N , so if the wave-front velocity is independent of the initial set of infectives (provided it has bounded support), then it will also be independent of N . It follows that the wave-front velocity must equal that of the linear model, otherwise the above convergence will not hold.

In [25] a contact distribution epidemic is derived that has the same deterministic wave-front velocity as our one-dimensional spatial dynamic population epidemic. The argument, which was also suggested to me independently by John Biggins, goes as follows. Consider the stochastic linear model corresponding to the one-dimensional spatial dynamic population epidemic. We may view migration of an individual as a death and instantaneous birth. This will yield a "contact distribution" type of process but with correlated births and deaths. However, this correlation is not reflected in the deterministic model, so our dynamic population epidemic will have the same deterministic velocity as a contact distribution model with infection rate $\beta' = \beta + \lambda$, removal rate $\gamma' = \gamma + \lambda$, and contact distribution

$$p_i = \begin{cases} \beta/(\beta + \lambda) & \text{if } i = 0, \\ \frac{1}{2} \lambda/(\beta + \lambda) & \text{if } i = \pm 1, \\ 0 & \text{otherwise.} \end{cases} \quad (6.8)$$

The argument extends to any dynamic population epidemic. One might hope that the above equivalence can be used to show that a dynamic population epidemic has smaller velocity than its corresponding contact distribution epidemic, as the contact distribution (6.8) is likely to be less diffuse than that of Theorem 4.2. However, the infection and removal rates for the model corresponding to (6.8) are greater than they were earlier, so no simple comparison exists.

7. CONNECTION BETWEEN DETERMINISTIC AND STOCHASTIC MODELS

There has been considerable debate in the literature on the relationship between stochastic and deterministic epidemic models. Here I will mainly reiterate the principal points in the context of our dynamic population epidemic models. The spread of an epidemic is essentially a stochastic phenomenon, at least as far as our current understanding of the process is concerned, so deterministic models should be viewed as approximations to more realistic stochastic models. The usual justification for using a deterministic model is that if the population sizes are large the probabilistic effects will tend to cancel each other out. However, such an argument requires *all* the subpopulations to be large, and this is generally not the case

for our dynamic population epidemics, where the group sizes are usually small. Moreover, even if the group sizes are large, the number of infectives at the start of an epidemic is small, so the deterministic model still does not necessarily provide a good guide to the behavior of the stochastic model. Indeed it does not, as borne out by the difference in interpretation of the threshold theorems for the two models. What the deterministic model does provide in such circumstances is a good approximation in the event of the epidemic taking off; see the central limit theorems of [28], [29], [37], [39], [40], and [41], which show that for certain epidemic models the total size of a major epidemic is asymptotically normally distributed about the total size of the corresponding deterministic epidemic.

It is well known that for nonlinear models, such as the dynamic population epidemic, the deterministic equations do *not* describe the mean of the stochastic model. Indeed, for contact distribution epidemics the deterministic model is closely related to the *linear* stochastic model, in that the one-dimensional simple epidemic (i.e., no removal of infectives) describes the survivor function of the furthest individual to the right in the corresponding linear stochastic birth process [24, 26]. We now investigate whether a similar relationship holds for our one-dimensional spatial dynamic population epidemic.

Consider a one-dimensional birth process in which individuals live on $\mathbb{Z}$, move according to a continuous-time simple symmetric random walk with step rate λ , and give birth at rate β . At time $t = 0$ there is one individual, located at the origin. For $t \geq 0$, let $R(t)$ be the location of the rightmost individual in the population at time t and $p_k(t) = P[R(t) \leq k]$, $k \in \mathbb{Z}$. Let U and V be the times at which the initial ancestor first gives birth and moves, respectively, and $W = \min(U, V)$. Conditioning on W , we have

$$\begin{aligned} p_k(t) &= \int_0^t (\beta + \lambda) e^{-(\beta + \lambda)u} \\ &\quad \times \left\{ \frac{\beta}{\beta + \lambda} p_k(t - u)^2 + \frac{\lambda}{2(\beta + \lambda)} [p_{k-1}(t - u) \right. \\ &\quad \left. + p_{k+1}(t - u)] \right\} du + e^{-(\beta + \lambda)t} 1_{\{k \geq 0\}} \\ &= \int_0^t e^{-(\beta + \lambda)(t - u)} \\ &\quad \times \left\{ \beta p_k(u)^2 + \frac{\lambda}{2} [p_{k-1}(u) + p_{k+1}(u)] \right\} du + e^{-(\beta + \lambda)t} 1_{\{k \geq 0\}}. \end{aligned}$$

Differentiating with respect to t , we obtain

$$\frac{dp_k}{dt} = -(\beta + \lambda)p_k + \beta p_k^2 + \frac{\lambda}{2}(p_{k-1} + p_{k+1}), \quad k \in \mathbb{Z}. \quad (7.1)$$

Now let $\alpha_k(t) = P[R(t) > k] = 1 - P_k(t)$, $k \in \mathbb{Z}$. Then (7.1) implies that

$$\frac{d\alpha_k}{dt} = \beta\alpha_k(1 - \alpha_k) - \lambda\alpha_k + \frac{\lambda}{2}(\alpha_{k-1} + \alpha_{k+1}), \quad k \in \mathbb{Z}. \quad (7.2)$$

We also have the initial condition

$$\alpha_k(0) = 1_{\{k < 0\}}. \quad (7.3)$$

Comparing (7.2) with (2.6) (with $\lambda = 0$), we see that $\alpha_k(t)$ is governed by an appropriate deterministic simple epidemic provided that in the latter $\tilde{x}_i + \tilde{y}_i \equiv 1$ for all i . This condition clearly holds for contact distribution epidemics, but it fails in dynamic population epidemics, because the movement of infectives implies that the group sizes do not remain constant. Thus the present connection between deterministic epidemic and linear stochastic models does not hold for dynamic population models. Nevertheless, we have seen already that, as far as velocities are concerned, deterministic dynamic population epidemic models are related to their linear, rather than nonlinear, stochastic counterparts.

The advantage of deterministic models over stochastic models is that they are easier to analyze or simulate. Their results are also easier to assimilate because there is only one possible outcome for any given initial condition! However, there can be difficulties in interpreting the results of a deterministic model, owing in part to the fact that discrete quantities are being modeled continuously. This is notably the case when deterministic spatial models are used to evaluate the efficacy of control policies that reduce the susceptible population within a control zone (see, e.g., [27] in the context of fox rabies), because unless the susceptible density is reduced to zero the infection will survive, albeit at a very low level, in the control zone and take off again outside. This problem is often addressed by imposing the assumption that the infection goes extinct in a group whenever the density of infectives drops below a cutoff value. However, the choice of cutoff value often seems rather arbitrary, and clearly a stochastic model is to be preferred. The main use of a deterministic model is to provide a broad indication as to how *qualitative* behavior of an epidemic model might depend on model assumptions and parameter values (e.g., the dependence of velocities on model type and λ in Section 6). However, there is no guarantee that such conclusions carry over to the corresponding stochastic model (e.g., the threshold behavior of one-dimensional spatial models), so they should always be checked, if necessary by resorting to simulation.

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