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GLOBAL ANALYSIS OF DISCRETE-TIME SI AND SIS EPIDEMIC MODELS

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Abstract. Discrete-time SI and SIS models formulated as the discretization of a continuous-time model may exhibit behavior different from that of the continuous-time model such as period-doubling and chaotic behavior unless the step size in the model is sufficiently small. Some new discrete-time SI and SIS epidemic models with vital dynamics are formulated and analyzed. These new models do not exhibit period doubling and chaotic behavior and are thus better approximations to continuous models. However, their reproduction numbers and therefore their asymptotic behavior can differ somewhat from that of the corresponding continuous-time model.

1. **Introduction.** Continuous-time epidemic models have played an important role in the investigation of the transmission of diseases. Because of the mathematical tractability of continuous systems, there has been fewer studies of discrete-time epidemic systems. Allen [1] has considered discrete-time SI, SIR, and SIS models with constant total population size and standard incidence, and found that SI and SIR models are similar in dynamical behavior to their continuous analogues under some natural restrictions; however, SIS models can exhibit period-doubling and chaotic behavior for some parameter values. Allen and Burgin [2] analyzed and compared the dynamics of deterministic and stochastic discrete-time SIS and SIR epidemic models. They determined the basic reproductive number for the deterministic models under some restrictions. Castillo-Chavez and Yakubu [7, 8] studied

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discrete-time SIS epidemic models with dispersal and nonlinear incidence rate, and found that bistability and period-doubling bifurcations are possible in an epidemic model if they are possible for the underlying demographic model. Zhou and Fergola [18] considered a discrete age-structured epidemic SIS model, and found the basic reproductive number and the threshold for the existence or extinction of disease. Li and Wang [13] studied a discrete epidemic model with stage structure, with a disease spreading among mature individuals and proposed a method for determining the basic reproduction number.

Many discrete models that have been studied are discretizations of continuous models, and are not necessarily descriptions of discrete processes. One reason for formulating discrete epidemic models is that data are collected at discrete time intervals, and it may be easier to compare experimental data with the predictions of a model if these predictions are given in discrete form. An important aspect of the results of this paper is a warning that there may be qualitative differences in the behaviors of the continuous model and a discrete approximation. It is important to choose the approximation scheme carefully [14], [15].

In this paper, we formulate new discrete-time SI and SIS epidemic models describing discrete observations of continuous processes. In these models we use exponential and Poisson probability distributions to describe the survival probability and the disease transmission probability over a discrete time interval, rather than using a linear discretization of a continuous model as has been customary in the past. Such an approach has been used previously in [11].

Our models are non-standard finite difference approximations to differential equations [14], [15]. Non-standard approximations are used to formulate difference equations that share the qualitative properties of the differential equations they are approximating. Our models achieve this to some extent, but there is a possibility that there may be a difference in reproduction numbers unless the step size is sufficiently small. A way to choose a non-standard difference approximation that also avoids this problem would be a very useful tool.

However, there are also discrete models derived directly from biological principles rather than as approximations to continuous processes, for example the models of [7, 8]. These models can exhibit period-doubling and chaotic behavior for the population system without disease. As we are ruling out this possibility, our results do not apply in such cases.

Global asymptotic results are obtained showing that the dynamical behaviors of these new models agree more closely with those of the corresponding continuous-time epidemic models. In particular, period-doubling and chaotic behavior are not possible for our models. For the models studied earlier it is necessary to impose some restrictions, that are usually natural requirements of the model, to rule out the possibility of period-doubling and chaotic behavior. The point of our analysis is that it is essential to be careful in formulating discrete models of continuous processes to avoid spurious results.

The organization of this paper is as follows: in Section 2, continuous models are described. In Section 3 the corresponding discrete models are formulated and analyzed and in Sections 4 through 6 a new discrete model is formulated and analyzed. Some comparisons are made and some conclusions are drawn in Section 7.

2. **Continuous models.** The basic continuous SIS model with births and natural deaths, but no disease deaths, is

$$\begin{aligned} S' &= \Lambda(N) - \beta(N)SI - \mu S + \gamma I \\ I' &= \beta(N)SI - (\mu+\gamma)I. \end{aligned} \tag{1}$$

We define $N = S + I$, the total population size. The assumptions made are:

1. The population has a density-dependent birth rate $\Lambda(N)$ per unit time and a proportional natural death rate μ in each class.
2. The number of contacts in unit time per individual is a nondecreasing function $N\beta(N)$ of total population size N. We assume that $\beta(N)$ is a nonincreasing function of N with $\beta(0) < \infty$. These assumptions include mass action incidence, $\beta(N) = \beta$, and standard incidence, $\beta(N) = \lambda/N$ (provided $\beta(N)$ is redefined near $N = 0$ to be biologically plausible and keep $\beta(0)$ finite), as well as many forms of saturating incidence.
3. There are no disease deaths and there is a rate $\gamma \geq 0$ of recovery from the infective class and return to the susceptible class.

The SI model, in which infectives remain infective and do not recover, is the special case $\gamma = 0$. We will carry out our analysis for the general SIS model, but the analysis also covers the SI model.

Since the total population size N satisfies

$$N' = \Lambda(N) - \mu N, \tag{2}$$

it is easy to see that $N(t) \to K$ as $t \to \infty$, where K is defined by $\Lambda(K) = \mu K$, provided the stability condition $\Lambda'(K) < \mu$ is satisfied. Then the theory of asymptotically autonomous systems [6] implies that the asymptotic behavior of (1) is the same as that of the simpler system in which N is replaced by the constant K and S is replaced by $K - I$, namely the first order differential equation

$$I' = \beta(K)I(K - I) - (\mu+\gamma)I. \tag{3}$$

The equation (3) can be analyzed qualitatively (or solved analytically since it is a logistic equation), and we see that if

$$\mathcal{R}_0 = \frac{K\beta(K)}{\mu+\gamma} < 1,$$

then $I \to 0$ as $t \to \infty$, while if $\mathcal{R}_0 > 1$ then $I \to K - (\mu+\gamma)/\beta(K)$ as $t \to \infty$, for every initial state with $I(0) > 0$.

For the continuous models (1) and (3) there is always a unique globally asymptotically stable equilibrium.

3. **The discretization of the continuous model.** In this section, we describe a discrete SIS system that evaluates approximately the solutions of the continuous system (1) at discrete times t_n with a fixed interval h, so that

$$t_{n+1} - t_n = h, \quad t_n = t_0 + nh, \quad n = 0, 1, \cdots .$$

The continuous model has simple dynamics; that is, it has a unique globally stable equilibrium. While discrete models may admit chaotic dynamics if the underlying population model admits chaotic dynamics [7, 8], we are interested in the question of whether a discrete model for which the underlying population model has simple dynamics can admit chaotic dynamics. For this reason, we will require that our

discrete model have simple dynamics if there is no disease. We mean by this that the model without disease must have a unique asymptotically stable equilibrium.

We let $S_n = S(t_n), I_n = I(t_n), N_n = N(t_n) = S(t_n) + I(t_n)$. The discrete version of the model (1), obtained by simple discretization of (1) is

$$\begin{aligned} S_{n+1} &= h\Lambda(N_n) - h\mu S_n + S_n[1 - h\beta(N_n)I_n] + h\gamma I_n \\ I_{n+1} &= I_n[1 - (\mu + \gamma)h] + h\beta(N_n)S_n I_n. \end{aligned} \tag{4}$$

This model is somewhat more general than the model studied in [1, 2], in which a constant total population size is assumed. The SI model is the special case $\gamma = 0$.

Addition of the two equations of (4) gives

$$N_{n+1} = h\Lambda(N_n) + (1 - h\mu)N_n. \tag{5}$$

The equation (5) has an equilibrium $N = K$, with K given by $\Lambda(K) = \mu K$, and the conditions for this equilibrium to be asymptotically stable are $\Lambda'(K) < \mu$, (which is also the condition for the equilibrium of the corresponding continuous model to be asymptotically stable), together with $h\Lambda'(K) + (1 - h\mu) > -1$, or

$$h < \frac{2}{\mu - \Lambda'(K)}.$$

We impose these conditions in order to assure simple behavior of the discrete model without disease. Under these conditions, every solution of (5) has limit K as $t \to \infty$. The system (4) is asymptotically autonomous, and according to the results of [16], [17, Chapter 2] has the same asymptotic behavior as the simpler system in which N_n is replaced by the constant K and S_n is replaced by $K - I_n$, namely the single difference equation, in which we rename the constant $\beta(K)$ as β,

$$I_{n+1} = I_n[1 + h\beta K - (\mu + \gamma)h] - h\beta I_n^2. \tag{6}$$

The model (6) has an equilibrium $I = 0$, and if

$$\mathcal{R}_0 = \frac{\beta K}{\mu + \gamma} > 1,$$

there is also an endemic equilibrium given by

$$\beta I = (\mu + \gamma)(\mathcal{R}_0 - 1) > 0.$$

The conditions for local asymptotic stability of an equilibrium I^* are

$$-1 < 1 + h\beta K - (\mu + \gamma)h - 2h\beta I^* < 1,$$

or

$$0 < 2h\beta I^* + (\mu + \gamma)h - h\beta K < 2.$$

For the equilibrium $I = 0$, this reduces to

$$\mathcal{R}_0 < 1, \quad h < \frac{2}{(\mu + \gamma) - \beta K},$$

and for the endemic equilibrium this reduces to

$$\mathcal{R}_0 > 1, \quad h < \frac{2}{\beta K - (\mu + \gamma)}.$$

Thus, in addition to a condition on the basic reproduction number, we must impose an upper bound on h, namely

$$h < \frac{2}{|\beta K - (\mu + \gamma)|}, \tag{7}$$

in order for the model (6) to have asymptotic behavior similar to that of the model (1) that it approximates. In addition, it is shown in [1, 2] that for the solutions of the model (4) to be non-negative it is necessary to impose the conditions

$$h(\mu+\gamma)<1, \quad h\beta K<(1+\sqrt{h(\mu+\gamma)})^2. \tag{8}$$

Thus the model (4) or (6) is a plausible approximation to the continuous model (1) only if the additional restrictions (7) and (8) are imposed. With these restrictions, the continuous model (1) and its discrete approximation have the same equilibrium population size without disease and the same basic reproduction number.

4. **A new discrete model.** In this section we define a different discrete approximation to the continuous system (1), in which we acknowledge explicitly that natural mortality and recovery from disease are continuous processes and measure the effects of these continuous processes at discrete times.

The number of members born in the time interval $[t_n, t_{n+1}]$ is

$$\int_{t_n}^{t_{n+1}} \Lambda(N(s))ds,$$

which we approximate by

$$h\Lambda(N_n).$$

The effective contact number for contacts of a susceptible individual with infective individuals in the time interval $[t_n, t_{n+1}]$ is $\int_{t_n}^{t_{n+1}} \beta(N(s))I(s)ds$. Thus, using the Poisson probability distribution, the probability that a susceptible individual remains uninfected in the time interval $[t_n, t_{n+1}]$ is

$$e^{-\int_{t_n}^{t_{n+1}} \beta(N(s))I(s)ds},$$

but we approximate this by

$$e^{-h\beta(N_n)I_n}.$$

Since it is assumed that there is a proportional death rate μ in each class, the fraction $e^{-\mu h}$ in each class surviving from time t_n to time t_{n+1} is given by an exponential distribution. Similarly, other proportional departure rates from a class translate into exponential retention rates. Thus the number of susceptibles S_n who remain alive and susceptible until time t_{n+1} is

$$S_n e^{-\mu h} e^{-\int_{t_n}^{t_{n+1}} \beta(N(s))I(s)ds},$$

which we approximate by

$$S_n e^{-\mu h} e^{-h\beta(N_n)I_n}.$$

The number of susceptibles S_n who remain alive but become infective between time t_n and time t_{n+1} is

$$S_n e^{-\mu h}\left(1-e^{-\int_{t_n}^{t_{n+1}} \beta(N(s))I(s)ds}\right),$$

which we approximate by

$$S_n e^{-\mu h}(1-e^{-h\beta(N_n)I_n}).$$

Also, the number of infectives I_n who survive and remain infective until time t_{n+1} is

$$I_n e^{-\mu h} e^{-\gamma h},$$

and the number of infectives I_n who survive and recover, returning to the susceptible class, is

$$I_n e^{-\mu h}(1 - e^{-\gamma h}).$$

Thus a new discrete model approximating (1) at time t_n is

$$\begin{aligned} S_{n+1} &= h\Lambda(N_n) + S_n e^{-\mu h} e^{-h\beta(N_n)I_n} + I_n e^{-\mu h}(1 - e^{-\gamma h}) \\ I_{n+1} &= I_n e^{-\mu h} e^{-\gamma h} + S_n e^{-\mu h}(1 - e^{-h\beta(N_n)I_n}). \end{aligned} \tag{9}$$

It is clear that $S_n \geq 0, I_n \geq 0$ for all n if $S_0 \geq 0, I_0 \geq 0$.

In formulating the model (9) we made two approximations, namely the replacement of $\int_{t_n}^{t_{n+1}} \Lambda(N(s))ds$ by $h\Lambda(N_n)$ and the replacement of $e^{-\int_{t_n}^{t_{n+1}} \beta(N(s))I(s)ds}$ by $e^{-h\beta(N_n)I_n}$. These approximations correspond to births and new infections occurring at discrete times. Thus model (9) represents both a new discrete approximation to the continuous model (1) and a model for a disease in a population in which births and new infections occur at discrete times.

For model (9) we have

$$N_{n+1} = S_{n+1} + I_{n+1} = g(N_n) = h\Lambda(N_n) + e^{-\mu h} N_n. \tag{10}$$

The difference equation (10) has an equilibrium K given by $g(K) = K$, or

$$\Lambda(K) = \frac{1 - e^{-\mu h}}{h} K.$$

The conditions for this equilibrium to be asymptotically stable are

$$-1 < h\Lambda'(K) + e^{-\mu h} < 1,$$

or

$$\Lambda'(K) < \frac{1 - e^{-\mu h}}{h}, \tag{11}$$

and

$$h\Lambda'(K) + e^{-\mu h} > -1. \tag{12}$$

The condition (12) has the form $h < h^*$ where $h^* = +\infty$ if $\Lambda'(K) \geq 0$ and h^* is the root of the equation

$$h\Lambda'(K) + e^{-\mu h} = -1$$

if $\Lambda'(K) < 0$.

Since we are requiring that the population model underlying our discrete disease transmission model must have an asymptotically stable equilibrium, we assume that conditions (11) and (12) are satisfied. Note that if the birth rate $\Lambda(N)$ is constant, these conditions are satisfied automatically for all h.

Under these conditions, every solution of (10) has limit K as $t \to \infty$. System (9) is asymptotically autonomous, and according to the results of [16],[17, Chapter 2] has the same asymptotic behavior as the simpler system in which N_n is replaced by the constant K and S_n is replaced by $K - I_n$, namely the single difference equation, in which we rename the constant $\beta(K)$ as β,

$$\begin{aligned} I_{n+1} &= I_n e^{-\mu h} e^{-\gamma h} + (K - I_n) e^{-\mu h}(1 - e^{-h\beta I_n}) \\ &= K e^{-\mu h}(1 - e^{-h\beta I_n}) - I_n e^{-\mu h}[1 - e^{-h\beta I_n} - e^{-\gamma h}]. \end{aligned} \tag{13}$$

We make the change of variable

$$x_n = \beta h I_n$$

and introduce new parameters

$$r = e^{-(\mu+\gamma)h} \le s = e^{-\mu h} < 1, \quad a = \beta h K$$

in (13). This transforms the model to

$$x_{n+1} = s(a - x_n)(1 - e^{-x_n}) + rx_n. \tag{14}$$

We note that

$$x_n = \beta h I_n \le \beta h K = a.$$

Also, for the SI model, $r = s$. The model (14) has the form

$$x_{n+1} = f(x_n),$$

with

$$f(x) = s(a - x)(1 - e^{-x}) + rx \ge 0. \tag{15}$$

LEMMA 4.1. *For $0 < x < a$, the inequality $0 < f(x) < a$ is true.*

Proof. We have

$$f(0) = 0, \quad f(a) = ra < a,$$
$$f'(x) = -s + se^{-x}(1 + a - x) + r, \text{ for } x \in (0, a).$$

Also,

$$f''(x) = -se^{-x}(2 + a - x) < 0, \text{for } x \in (0, a).$$

Then $f'(0) = as + r > 0$ and $f'(a) = r - s(1 - e^{-a})$. If $f'(a) \ge 0$, then, since $f''(x) < 0, f'(x) > 0$ for $0 < x < a$. Thus $f(x) < f(a) < a$ for $0 < x < a$. For the SI model, since $r = s$, $f'(a) > 0$.

On the other hand, if $f'(a) < 0$, there exists $x_M \in (0, a)$ such that $f'(x_M) = 0$, that is,

$$se^{-x_M} = \frac{s - r}{1 + a - x_M}.$$

In this case, the function $f(x)$ attains its maximum, $f(x_M)$, in the interval $(0, a)$ at the point $x = x_M$. But, for $r < s$,

$$f(x_M) = (a - x_M)\frac{s(a - x_M) + r}{1 + a - x_M} + rx_M < s(a - x_M) + rx_M < sa < a.$$

Thus, in either case, $f(x) < a$ and the proof of Lemma 4.1 is complete. □

Therefore, in what follows we will consider equation (14) in the interval $[0, a)$.

5. **Stability analysis.** Since $f(0) = 0$, the model (14) has an equilibrium $x = 0$.

THEOREM 5.1. *If $r + as \le 1$, the only equilibrium of (14) in the interval $[0, a)$ is $x = 0$. If $r + as > 1$, equation (14) has an equilibrium $x = 0$ and also has a unique positive equilibrium $x^* \in (0, a)$, where x^* satisfies*

$$e^{-x} = \frac{as - (1 + s - r)x}{s(a - x)}. \tag{16}$$

Proof. Let

$$F(x) = f(x) - x = s(a - x)(1 - e^{-x}) + (r - 1)x. \tag{17}$$

Then the equilibria of (14) are the zeros of $F(x)$. We have

$$F(0) = f(0) = 0, \quad F(a) = f(a) - a = a(r - 1) < 0$$

and

$$F'(0) = f'(0) - 1 = r + as - 1.$$

Since $F''(x) = f''(x) < 0$, if $F'(0) \le 0$, then $F(x) < 0$ for $0 < x \le a$, and $x = 0$ is the only equilibrium of (14). On the other hand, if $F'(0) > 0$, then F increases to a positive maximum and then decreases as x increases. Thus there is a unique positive x^* with $F(x^*) = 0$, and $F'(x^*) < 0$. □

THEOREM 5.2. *If $r + as \le 1$, the equilibrium $x = 0$ is (globally) asymptotically stable and the disease dies out eventually. If $r + as > 1$ the equilibrium $x = 0$ is unstable, and the disease does not die out.*

Proof. Since $1 - e^{-x} < x$ for $x > 0$,

$$\begin{aligned} f(x_n) &= s(a - x_n)(1 - e^{-x_n}) + rx_n \\ &< sx_n(a - x_n) + rx_n \\ &= x_n(r + as - sx_n) < (r + as)x_n. \end{aligned}$$

Thus if $r+as < 1$, then x_n decreases to zero. If $r+as = 1$, then $x_{n+1} < x_n$, and the sequence $\{x_n\}$ is decreasing. Therefore, we know $\lim_{n\to\infty} x_n = x_\infty \ge 0$ from $x_n > 0$. We claim that $x_\infty = 0$, because x_∞ is an equilibrium of (14) and, by Theorem 5.1, if $r + as = 1$, the only equilibrium of (14) is $x = 0$.

On the other hand, if $r + as > 1$, since $\lim_{x\to 0} \frac{f(x)}{x} > 1$, then $x_{n+1} > x_n$ if x_n is sufficiently small. This implies that x_n does not approach 0. □

If $r + as > 1$, there is a positive equilibrium x^* of (14), and we examine its local stability.

THEOREM 5.3. *If $r + as > 1$, the positive equilibrium of (14) is (locally) asymptotically stable.*

Proof. The equilibrium x^* is locally asymptotically stable if $|f'(x^*)| < 1$. As we have seen in the proof of Theorem 5.1, $F'(x^*) < 0$, and therefore $f'(x^*) < 1$. Also,

$$f'(x^*) > f'(a) = r - s(1 - e^{-a}) > r - s > -1,$$

since $0 < r \le s < 1$. Thus $|f'(x^*)| < 1$, and the equilibrium x^* is locally asymptotically stable. □

We calculate the basic reproduction number, which we denote by $\mathcal{R}(h)$, for the model (13), using the results of [10, Section 1.1], as

$$\mathcal{R}(h) = \frac{\beta h K e^{-\mu h}}{1 - e^{-(\mu+\gamma)h}} < 1.$$

As $h \to 0$, the quantity $\mathcal{R}(h)$ approaches

$$\frac{\beta K}{\mu + \gamma},$$

which is the basic reproduction number $\mathcal{R}_0$ for the continuous model (1).

In terms of the original model parameters, the condition $r + as < 1$ for local asymptotic stability of the equilibrium $x = 0$ is

$$e^{-\mu h}[e^{-\gamma h} + \beta h K] < 1,$$

or $\mathcal{R}(h) < 1$.

It is not difficult to show that there is a value h^* such that

$$\mathcal{R}(h) > \mathcal{R}_0$$

if $\gamma > \mu$ and $0 < h < h^*$, and that

$$\mathcal{R}(h) \leq \mathcal{R}_0$$

otherwise. The proof of this is given at the end of this section. Therefore, with suitable parameter value choices we can have either

$$\mathcal{R}(h) > 1 > \mathcal{R}_0$$

or

$$\mathcal{R}_0 > 1 > \mathcal{R}(h)$$

if h is not close enough to zero. Thus the discrete approximation (9) and the continuous model (1) may behave quite differently. Of course, since

$$\lim_{h\to 0} \mathcal{R}(h) = \mathcal{R}_0,$$

the asymptotic behaviors of the continuous and discrete models are the same if h is sufficiently small.

This shows that it is necessary to be extremely careful in approximating a continuous disease transmission model by a discrete model. Approximations (4) and (9) can each exhibit behavior different from that of (1), and the two discrete models can differ from the continuous model in different ways.

To conclude this section, we give the proof that $\mathcal{R}(h) > \mathcal{R}_0$ if $\gamma > \mu$ and $0 < h < h^*$. We define

$$\begin{aligned} g(h) &= \mathcal{R}(h) - \mathcal{R}_0 = \frac{\beta h K e^{-\mu h}}{1 - e^{-(\mu+\gamma)h}} - \frac{\beta K}{\mu + \gamma} \\ &= \frac{\beta K}{[1 - e^{-(\mu+\gamma)h}](\mu + \gamma)} g_1(h), \end{aligned}$$

with

$$g_1(h) = (\mu + \gamma)h e^{-\mu h} - 1 + e^{-(\mu+\gamma)h},$$

and

$$g_1'(h) = (\mu + \gamma)e^{-\mu h}(1 - \mu h - e^{-\gamma h}) = (\mu + \gamma)e^{-\mu h} g_2(h), \quad g_1(0) = 0.$$

Then

$$g_2'(h) = -\mu + \gamma e^{-\gamma h}, \quad g_2'(0) = \gamma - \mu.$$

If $\gamma > \mu, g_2'(0) > 0$. Because $g_2''(h) < 0$ there exists $h_1^* = \ln(\gamma/\mu)/\gamma > 0$ such that $g_2'(h) > 0$ for $0 < h < h_1^*$ and $g_2'(h) < 0$ for $h > h_1^*$. Because $g_2(0) = 0, \lim_{h\to\infty} g_2(h) = -\infty$, there exists $h_2^* > h_1^*$ such that $g_2(h) > 0$ for $0 < h < h_2^*$ and $g_2(h) < 0$ for $h > h_2^*$. Now, since $g_1(0) = 0, \lim_{h\to\infty} g_1(h) = -1$, there exists $h^* > h_2^*$ such that $g_1(h) > 0$ for $0 < h < h^*$ and $g_1(h) < 0$ for $h > h^*$. Since $g(h)$ and $g_1(h)$ have the same sign, this proves that $\mathcal{R}(h) > \mathcal{R}_0$ for $0 < h < h^*$.

We note that we have also shown that $\mathcal{R}(h) < \mathcal{R}_0$ if $\gamma > \mu, h > h^*$. If $\gamma \leq \mu$, then $g_2'(0) \leq 0$. Because $g_2''(h) < 0$ we have $g_2'(h) \leq 0$ for $h > 0$, and thus $g_1(h) \leq 0$ for $h > 0$. It follows that $\mathcal{R}(h) \leq \mathcal{R}_0$.

6. **Global asymptotic stability.** Model (14) always has a unique asymptotically stable equilibrium. In fact, this equilibrium is globally asymptotically stable. If $r + as < 1$, we have shown that every solution of (14) tends to zero, so that the equilibrium $x = 0$ is globally asymptotically stable. For the positive equilibrium x^*, if there is one, we have the following result.

THEOREM 6.1. *If Model (14) has a positive equilibrium, this equilibrium, which is unique by Theorem 5.1, is globally asymptotically stable.*

Proof. Suppose that $f(x)$ is a continuous function having the properties

1. $f(0) = 0$.
2. There is a unique locally asymptotically stable positive equilibrium $\bar{x}$ such that $f(\bar{x}) = \bar{x}$, $f(x) > x$ for $0 < x < \bar{x}$, and $f(x) < x$ for $\bar{x} < x$.
3. If $f(x)$ has a maximum x_M in $(0, \bar{x})$, then $f(x)$ is monotonically decreasing for all $x > x_M$ such that $f(x) > 0$.

Then according to a theorem of Cull [9], the equilibrium $\bar{x}$ of $f(x)$ is globally asymptotically stable if there is no maximum of $f(x)$ in $(0, \bar{x})$ or if $f''(x) < 0, f'''(x) > 0$. Since, as we have shown in the proof of Lemma 4.1, these conditions are satisfied, the proof is complete. □

It is not difficult to use the same argument to show that the endemic equilibrium of (4) is globally asymptotically stable if it is locally asymptotically stable.

We may summarize the results of this section as follows:

THEOREM 6.2. *If $\mathcal{R}(h) \leq 1$, the only equilibrium of model (14) is $x = 0$, and this equilibrium is globally asymptotically stable, so that the infection dies out eventually. If $\mathcal{R}(h) > 1$, the equilibrium $x = 0$ of (14) is unstable. There is a unique positive equilibrium that is globally asymptotically stable, so that the infection is endemic.*

7. **Discussion.** The discrete models (4) and (9) both have differences in behavior from the continuous model (1) that they approximate. Model (4) may have negative solutions and may exhibit period-doubling and chaotic behavior if the step size h is not sufficiently small [1] but Model (9) does not have this shortcoming. We believe that this difference is caused by the fact that the mortality and recovery rates are uniformly distributed over time intervals of arbitrary length h for (4), while these rates are not uniformly distributed for (1). The corresponding rates for (9) are not uniformly distributed because probability distributions have been introduced. The approximations in (9) are based on probabilistic considerations and not on the linearity of the step size. If the exponential functions in (9) are replaced by their linear approximations, the result is (4). The approximating models (4) and (9) both have the model (1) as their limit as $h \to 0$, but the model (9) is a closer approximation than (4).

The step size in a discrete approximation to a continuous disease transmission model is often taken as a convenient measurement interval. In using (4) as a model for a continuous process, it is essential to verify that the measurement interval is small enough; otherwise, the discrete model may predict spurious phenomena.

The basic reproduction number and the endemic equilibrium are not the same for models (9) and (1), and the differences may lead to differences in asymptotic behavior if h is not sufficiently small. These differences are significant only if the basic reproduction number is close to 1. In using (9) as a model for a continuous process it is essential to verify that the basic reproduction number is not too close to 1. This is a problem that cannot be rectified by use of a different approximating scheme.

We believe that chaotic behavior in a disease transmission model can be caused only by the population dynamics and not by the disease dynamics, and that models such as (4) are not admissible models. This is supported by the fact that if (9) is viewed as a model for a population with discrete birth and disease transmission processes and not just as an approximation to a continuous model, this model

can exhibit chaotic behavior only if such behavior is possible in the underlying population model. Because the processes of recovery and mortality usually depend continuously on time, the use of linear approximations for them may introduce errors.

If there are deaths due to disease or in an SIR model, the discrete approximation cannot be reduced to a one-dimensional model. Our global asymptotic stability result is restricted to one-dimensional models and can not be used in these cases. However, it should be straightforward to carry out the local stability analysis for discrete models analogous to the new model (9), such as the SIS model with a disease death rate α,

$$\begin{aligned} S_{n+1} &= h\Lambda(N_n) + S_n e^{-\mu h} e^{-h\beta(N_n)I_n} + I_n e^{-\mu h}(1 - e^{-(\gamma+\alpha)h}) \\ I_{n+1} &= I_n e^{-\mu h} e^{-(\gamma+\alpha)h} + S_n e^{-\mu h}(1 - e^{-h\beta(N_n)I_n}). \end{aligned}$$

and the SIR model

$$\begin{aligned} S_{n+1} &= h\Lambda(N_n) + S_n e^{-\mu h} e^{-h\beta(N_n)I_n} + \\ I_{n+1} &= I_n e^{-\mu h} e^{-(\gamma+\alpha)h} + S_n e^{-\mu h}(1 - e^{-h\beta(N_n)I_n}) \\ R_{n+1} &= R_n e^{-\mu h} + I_n e^{-\mu h}(1 - e^{-(\gamma+\alpha)h}). \end{aligned}$$

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