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1 Introduction

Simple nonlinear discrete-time population models are capable of generating complex (chaotic) dynamics [4, 6, 9, 18, 21, 22]. A simple nonlinear discrete-time susceptible-infected-susceptible $(S-I-S)$ epidemic model that assumes that the population is either asymptotically bounded, experiences geometric growth or is capable of generating complex dynamics is presented.

Theoretical studies of $(S-I-S)$ epidemic models have been carried out in deterministic (discrete and continuous-time) and stochastic Markov chains for a long time (see Allen [1, 2]; Anderson et $al.$ [3]; Bailey [5]; Castillo et al. [7 - 10]; Cooke and Yorke [11]; Hassell et $al.$ [18] and Nåsell [23], to name a few). Barrera et $al.$ [6]; Velazquez [27]; and Castillo-Chavez and Yakubu [9, 10] have developed models for the study of disease dynamics in populations with discrete generations and potentially complex (chaotic) disease-free dynamics. The models in this article overlap somewhat with $(S-I-S)$ epidemic models found in the literature (see [6, 9, 10, 16, 27]).

2 Simple Discrete-time Epidemic Models

Nonlinear discrete-time epidemic models are built under the assumption that the dynamics of the total population size in generation n, denoted by $T(n)$, are governed by an equation of the form $T(n+1) = f(T(n)) + \gamma T(n)$, where $0 \leq \gamma < 1$ denotes the constant probability of surviving per generation and f models the typically nonlinear birth or recruitment process. If f is not typical, that is, if f is a constant or a linear function of the total population then the population dynamics are rather simple. Specifically, if new recruits

arrive at the constant per-capita rate μ or at the total constant rate Λ per generation then $f(T(n)) = \mu T(n)$ or $f(T(n)) = \Lambda$. An epidemic process is built on "top" of this demographic model that, by construction, has known disease-independent, and simple population dynamics for $T(n)$. Epidemics on simple demographic settings are the norm in the study of continuous-time epidemic models where the total population is typically assumed to be constant, asymptotically constant, or capable of supporting exponential growth. This framework expands the range of complexity of the underlying population dynamics (see the next section for examples).

At generation n, $S(n)$ denotes the population of susceptibles; $I(n)$ denotes the population of infecteds, assumed infectious; $T(n) \equiv S(n) + I(n)$ denotes the total population size; and, $T_\infty \equiv \lim_{n\to\infty} T(n)$ denotes the demographic steady state for the total population whenever it exists. Individuals survive from one generation to the next with constant probability γ while infected, individuals recover with constant probability $1 - \sigma$ per generation. Susceptible individuals (given that they had contacts with infectives) become infected with probability $1 - G$ or, equivalently, remain susceptible with probability G per generation. $G : [0, \infty) \to [0, 1]$ is a monotone concave function with $G(0) = 1; G'(x) < 0$ and $G''(x) \geq 0$ for all $x \in [0, \infty)$.

The population dynamics are fixed since it is assumed that the disease is not fatal. All recruits are susceptible, time is measured in generations, and recovery from the disease does not give permanent or temporary immunity. The modeling process implicitly assumes a sequential process: At the end of each generation, a fraction $(1 - \gamma)$ of each class is removed (death); surviving susceptibles become infected with probability $(1 - G)$; and, independently,

surviving infectives recover with probability $(1 - \sigma)$. It is important to note that this is a deterministic model and that *probabilistic lingo* is used *mainly* to facilitate understanding.

Our assumptions lead to the following discrete-time $S - I - S$ epidemic model:

$$\left. \begin{aligned} S(n+1) &= f(T(n)) + \gamma S(n) G(\tfrac{\alpha I(n)}{T(n)}) + \gamma I(n)(1 - \sigma), \\ I(n+1) &= \gamma(1 - G(\tfrac{\alpha I(n)}{T(n)})) S(n) + \gamma \sigma I(n), \end{aligned} \right\} \quad (1)$$

where $0 < \gamma, \sigma < 1$, and $T(n) > 0$. The constant α weighs the role of prevalence $(\tfrac{I(n)}{T(n)})$ on disease transmission. If α is large, then it is easier to become infected (see Figure 1). System (1), is an extension of the discrete - time $(S - I - S)$ epidemic models of Barrera *et al.* and Velazquez [6, 27].

Since $T(n+1) \equiv S(n+1) + I(n+1)$, then

$$T(n+1) = f(T(n)) + \gamma T(n), \qquad (2)$$

that is, Equation (2) models the dynamics (demography) of the total population.

3 Simple Population Dynamics and SIS Epidemics

If $f(T(n)) = \mu T(n)$ then Equation (2), the demographic equation, reduces to the linear difference equation

$$T(n+1) = (\mu + \gamma)T(n), \qquad (3)$$

and, consequently,

$$T(n) = (\mu + \gamma)^n T(0). \qquad (4)$$

4

The demographic basic reproductive number,

$$\mathfrak{R}_d = \frac{\mu}{1 - \gamma},$$

is a dimensionless quantity. It gives the average number of descendants produced by a (typically) small pioneer population $(T(0))$ over its life-time. Hence, if $\mathfrak{R}_d > 1$, the population invades at a geometric rate and if $\mathfrak{R}_d < 1$ the population dies geometrically. The epidemic process constructed then lives in a population experiencing geometric dynamics.

If we let

$$x(n) = \frac{S(n)}{T(n)} \text{ and } y(n) = \frac{I(n)}{T(n)}, \qquad (5)$$

then System (1) with $f(T(n)) = \mu T(n)$ reduces to the one-dimensional autonomous "system" for $y(n)$:

$$y(n+1) = \frac{\gamma}{\gamma + \mu}(1 - y(n))(1 - G(\alpha y(n))) + \frac{\gamma \sigma}{\gamma + \mu}y(n). \qquad (6)$$

Since $x(n) + y(n) = 1$ for all n then all solutions live on the invariant line $\{(x, y) \in [0, \infty) \times [0, \infty) \mid x + y = 1\}$.

$\mathfrak{R}_0$, the number of secondary infections generated by an infected (assumed infectious) individual over his or her lifetime in a population of susceptibles, determines whether or not the disease can invade. From Equation (6), we find that

$$\mathfrak{R}_0 = \frac{-\alpha \gamma G'(0)}{(1 - \gamma)(\mathfrak{R}_d - 1) + (1 - \gamma \sigma)},$$

after noticing that near the equilibrium point $(x_\infty, y_\infty) \equiv (1, 0)$,

$$y(n+1) \approx \frac{\gamma}{\gamma + \mu}(-\alpha G'(0) + \frac{\gamma \sigma}{\gamma + \mu})y(n).$$

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Whenever $\Re_d = 1$ (no demographic impact), the basic reproductive number, $\Re_0$, reduces to $\Re_0 = \frac{-\alpha\gamma G'(0)}{1-\gamma\sigma}$, where $\frac{1}{1-\gamma\sigma}$ denotes the average death-adjusted length of the infectious period in generations; γ is the proportion of surviving susceptibles who can be invaded by the disease; and, $-\alpha G'(0)$ is the maximum rate of infection per infective [9, 10]. Since $\frac{1}{(1-\gamma)(\Re_d-1)+(1-\gamma\sigma)}$ gives the demographic death-adjusted infectious period measured in generations then $\Re_0$ decreases with population growth ($\Re_d > 1$) and increases with population decay ($0 < \Re_d < 1$), that is, whenever $\Re_d \neq 1$ demography has an impact.

Result 1. *(a) Suppose that $\Re_d > 1$, that is, assume T is increasing at the geometric rate, $(\mu + \gamma)$. Then*

(i) $\Re_0 \leq 1$, implies that the proportion $\frac{I}{T}$ of infectives in the total population tends to 0 as $n \to \infty$. Hence, $\left(\frac{S}{T}, \frac{I}{T}\right)$ tends to the disease-free equilibrium $(1,0)$, while S is increasing at the same geometric rate as T.

(ii) $\Re_0 > 1$, implies that the proportion $\frac{I}{T}$ of infectives in the total population tends to a positive number, $\frac{\overline{I}}{T}$ as $n \to \infty$. Hence, $\left(\frac{S}{T}, \frac{I}{T}\right)$ tends to an endemic equilibrium while I, S and T are increasing at the same geometric rate.

(b) Suppose that $\Re_d < 1$, that is, assume T is decreasing at the geometric rate, $(\mu + \gamma)$. Then

(i) $\Re_0 \leq 1$, implies that the proportion $\frac{I}{T}$ of infectives in the total population tends to 0 as $n \to \infty$. Hence, $\left(\frac{S}{T}, \frac{I}{T}\right)$ tends to the disease-free equilibrium $(1,0)$ while S is decreasing to zero at the same geometric rate as T.

(ii) $\Re_0 > 1$, implies that the proportion $\frac{I}{T}$ of infectives in the total population tends to a positive number $\frac{\overline{I}}{T}$ as $n \to \infty$. Hence, $\left(\frac{S}{T}, \frac{I}{T}\right)$ tends to an endemic equilibrium while I, S and T are decreasing at the same geometric

rate.

Proof: Define the reproduction function for the infected individuals of System (6), $h : [0,1] \to [0,1]$ by $h(y) = \frac{\gamma}{\gamma+\mu}(1-y)(1-G(\alpha y)) + \frac{\gamma\sigma}{\gamma+\mu}y$, where $h(0) = 0$ and $0 \leq y \leq 1$. The set of iterates of h is equivalent to the set of density sequence generated by the second equation in System (6). Differentiation with respect to y gives

$$
\begin{aligned}
h'(y) &= \tfrac{\gamma}{\gamma+\mu}(-1 + G(\alpha y) - \alpha(1-y)G'(\alpha y) + \sigma), \\
h''(y) &= \tfrac{\gamma}{\gamma+\mu}(2\alpha G'(\alpha y) - \alpha^2(1-y)G''(\alpha y)).
\end{aligned}
$$

$\Re_0 \leq 1$ implies that $h'(0) = \frac{\gamma}{\gamma+\mu}(-\alpha G'(0) + \sigma) \leq 1$. Therefore, the fixed point $\{0\}$ is locally stable under h-iteration. Since $G' < 0$ and $G'' \geq 0$ we have that, $h''(y) < 0$ for $y \in [0, 1]$. The monotonicity condition on h' and the fact that $h'(0) \leq 1$ imply that $h'(y) < 1$ or $h(y) < y$ for $y \in (0, 1]$. Hence, $\{y(n)\}_{n\geq 0}$, a strictly decreasing sequence bounded below by zero, converges to the only fixed point of h in the interval $[0, 1]$, $\{0\}$. If $\Re_0 \leq 1$, we have proved that $\frac{I}{T} \to 0$ as $n \to \infty$, while the proportion $\frac{S}{T} \to 1$ as $n \to \infty$.

$\Re_0 > 1$ implies that $h'(0) = \frac{\gamma}{\gamma+\mu}(-\alpha G'(0) + \sigma) > 1$ and, therefore, the fixed point $\{0\}$ is locally unstable under h-iteration. Let $\overline{y}$ denote the smallest positive fixed point of h in $[0, 1]$, and note that $h(1) = \frac{\gamma\sigma}{\gamma+\mu} < 1$. The Intermediate Value Theorem guarantees the existence of the positive fixed point $\overline{y} \in (0,1)$ satisfying $h(\overline{y}) = \overline{y}$ and $h(y) > y$ for $y \in (0,\overline{y})$ and, consequently, $h'(\overline{y}) \leq 1$. Since $h''(y) < 0$ implies that $h'(y) < h'(\overline{y}) \leq 1$ for $y \in (\overline{y}, 1)$, then $\int_{\overline{y}}^{y} h'(u)du < \int_{\overline{y}}^{y} du$ and, we have $h(y) < y$ for $y > \overline{y}$. Hence, h has a unique positive fixed point $\overline{y} \in (0,1)$. Furthermore, $h(y) > y$ for $y \in (0,\overline{y})$ and $h(y) < y$ for $y \in (\overline{y}, 1]$.

To establish the global stability of $\overline{y}$, we first prove the nonexistence of

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non-trivial two-cycles for h. Note that $1 + h'(y) = 1 + \frac{\gamma}{\gamma+\mu}(-1 + G(\alpha y) - \alpha(1 - y)G'(\alpha y) + \sigma) \geq 1 - \frac{\gamma}{\gamma+\mu} + \frac{\gamma\sigma}{\gamma+\mu} > 0$. Hence, $1 + h'(y) \neq 0$ for $y \in [0, 1]$, that is, h has no non-trivial 2-cycles. Suppose h has a non-trivial 2-cycle $\{p, q\}$ where $p, q \in [0, 1]$, then $h(p) = q$ and $h(q) = p$ where $p \neq q$. The Mean Value Theorem guarantees the existence of a point ξ between p and q such that $h'(\xi) = \frac{h(p)-h(q)}{p-q} = -1$, and $1 + h'(\xi) = 0$, a contradiction. Hence, h has no non-trivial 2-cycles in $[0, 1]$. Sharkovskii's Theorem and $1 + h'(y) \neq 0$ imply the nonexistence of any $m - cycles$ for $m > 1$. From a result of Cull [12], the nonexistence of non-trivial 2-cycles for h implies global stability of the positive fixed point $\overline{y}$. Hence, $\Re_0 > 1$, implies that $\frac{I}{T} \to \frac{\overline{I}}{T} \in (0,1)$ as $n \to \infty$, and $\frac{S}{T} \to \frac{\overline{S}}{T} \in (0,1)$ as $n \to \infty$.

A second simple example is provided with $f(T(n)) = \Lambda$. The demographic equation, reduces to the linear difference equation

$$T(n+1) = \Lambda + \gamma T(n) \qquad (7)$$

and, hence

$$T(n) = (T(0) - \frac{\Lambda}{1-\gamma})\gamma^n + \frac{\Lambda}{1-\gamma}. \qquad (8)$$

Since $0 < \gamma < 1$, the total population is asymptotically constant, that is, $T_\infty = \frac{\Lambda}{1-\gamma}$, that is, the demographic effects "eventually" disappear.

To study disease epidemics on (7), we assume that the total population $T(n) \equiv S(n) + I(n)$ has reached the positive steady state T_∞. If we redefine $S(n) = T_\infty - I(n)$ then from System (1) we obtain the following associated one-dimensional dynamically equivalent autonomous "system" for $I(n)$ [25,

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28] :

$$S(n+1) = f(T_\infty) + c\gamma(T_\infty - I)G(\tfrac{\alpha I(n)}{T_\infty}) + \gamma I(n)(1-\sigma) \Bigg\}$$
$$I(n+1) = \gamma(1 - G(\tfrac{\alpha I(n)}{T_\infty}))(T_\infty - I) + \gamma\sigma I(n). \Bigg\} \quad (9)$$

Its basic reproductive number is

$$\Re_0 = \frac{-\alpha\gamma G'(0)}{1 - \gamma\sigma}$$

and a simple application of earlier results gives the following the result [9, 10]:

Result 2: *Suppose* $f(T(n)) = \Lambda$ *in system (9) then*

(a) If $\Re_0 \le 1$, *then all solutions* $(S(n), I(n))$ *approach the globally asymptotically stable disease free equilibrium,* $(\tfrac{\Lambda}{1-\gamma}, 0)$, *as* $n \to \infty$.

(b) If $\Re_0 > 1$, *then all solutions* $(S(n), I(n))$ *approach a unique positive and globally asymptotically stable endemic equilibrium,* $(\overline{S}, \overline{I}) \in (0, \infty) \times (0, \infty)$, *as* $n \to \infty$.

The proof of Result (2) is the same as that of Theorem (1) in [9].

Consider the following particular version of Model (1):

$$S(n+1) = f(T(n)) + e^{-d}S(n)e^{-\frac{\alpha I(n)}{T(n)}} + e^{-d}I(n)(1 - e^{-\beta}), \Bigg\}$$
$$I(n+1) = e^{-d}(1 - e^{-\frac{\alpha I(n)}{T(n)}})S(n) + e^{-d}e^{-\beta}I(n), \Bigg\} \quad (10)$$

where α, β and d are positive constants. Here, we have taken $e^{-d} = \gamma$, $e^{-\frac{\alpha I(n)}{T(n)}} = G(\tfrac{\alpha I(n)}{T(n)})$ and $e^{-\beta} = \sigma$. $f(T(n)) = \mu T(n)$ implies that $T(n+1) = (e^{-d} + \mu)T(n)$ and $\Re_d = \frac{\mu}{1 - e^{-d}}$. Using proportions reduces System (10) to the following system of equations (see, [6, 9, 10]):

$$x(n+1) = \frac{\mu}{e^{-d}+\mu} + \frac{e^{-d}}{e^{-d}+\mu}x(n)e^{-\alpha y(n)} + \frac{e^{-d}(1-e^{-\beta})}{e^{-d}+\mu}y(n), \Bigg\}$$
$$y(n+1) = \frac{e^{-d}}{e^{-d}+\mu}x(n)(1 - c^{-\alpha y(n)}) + \frac{e^{-d}e^{-\beta}}{e^{-d}+\mu}y(n). \Bigg\} \quad (11)$$

The basic reproductive number,

$$\mathfrak{R}_0 = \frac{\alpha^2}{1 + \mu e^d - e^{-\beta}},$$

determines the asymptotic behavior of System (11) [see Result 1].

We fix the constants $\beta = 0.1$, $d = \ln 2$ and $\mu = 0.1$ while we vary the transmission coefficient α. As we increase α, the basic reproductive number, $\mathfrak{R}_0$, increases from values less than 1 to numbers bigger than 1. A transcritical bifurcation occurs. The demographic basic reproductive number remains fixed at $\mathfrak{R}_d = \frac{\mu}{1 - e^{-d}} = 0.25 < 1$. The system with proportions changes from a globally stable disease-free equilibrium to a globally stable endemic equilibrium while the total population is decreasing to zero at a geometric rate (see Figure 1). In particular, at $\alpha = 0.6$, $\mathfrak{R}_0 = 1.22 > 1$ and the system with proportions has a globally stable endemic equilibrium at $(0.5598, 0.4402)$.
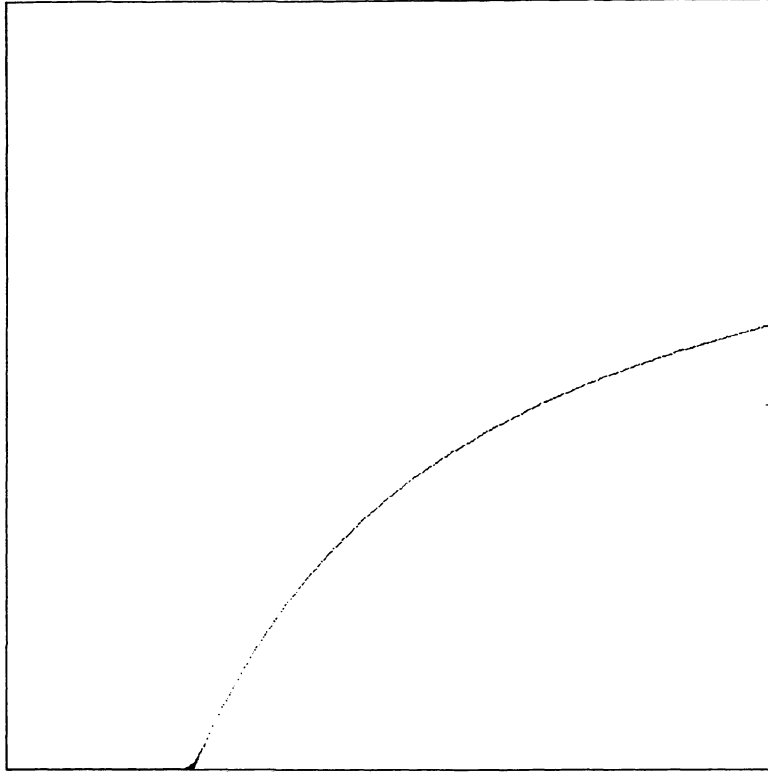
Figure 1: The size of infecteds increases from zero as α increases.

4 Complex Dynamics In S-I-S Models

Equations (2) is capable of supporting complex (chaotic) dynamics. For example, if new recruits are governed by Smith-Slatkin model [11, 22],

$$f(T(n)) = \frac{aT(n)}{1 + (bT(n))^l},$$

where a, b and l are positive constants and $a > 1$ then

$$T(n+1) = \frac{aT(n)}{1 + (bT(n))^l} + \gamma T(n), \qquad (12)$$

where a is maximal per-capita intrinsic growth rate of the population. The parameter l reflects the type and strength of intraspecific competition while

the parameter b scales the carrying capacity of the population [13, 24]. Whenever $0 < l < \frac{2(a+\gamma)}{a+\gamma-1}$, the total population assumes a positive steady state at $T_\infty = \frac{(a+\gamma-1)^{\frac{1}{l}}}{b}$ and Result 2 applies. However, as l increases past $\frac{2(a+\gamma)}{a+\gamma-1}$, the positive equilibrium point undergoes period-doubling bifurcation resulting in a stable $2-cycle$ (see Figure 2). This process of period doubling bifurcation continues indefinitely.
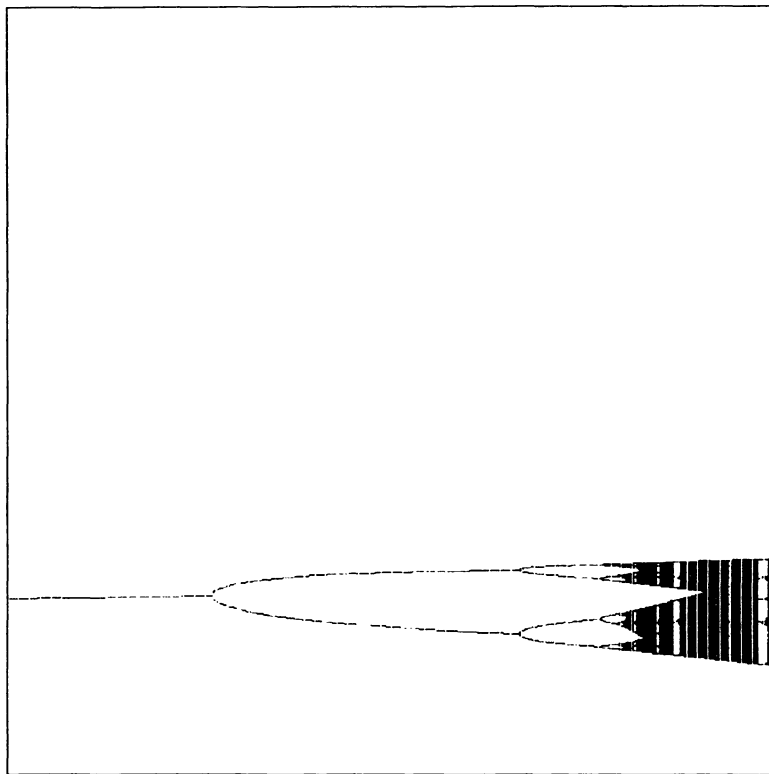

Figure 2: Period-doubling bifurcations route to chaos in the demographic equation under Smith-Slatkin model with $a = 1.5$, $b = 1$, $\gamma = 0.1$ and $4.5 < l < 8.5$.

Figure 2 illustrates the existence of a chaotic regime in the demographic model. Do complex population dynamics drive disease dynamics? Exam-

ples in [6] demonstrate that demography does not always drive an epidemic process even in the absence of disease induced mortality.

5 Backward Bifurcations

Typically, epidemic models have a unique endemic equilibrium with the reproductive number of the disease serving as a threshold parameter (transcritical bifurcation). If the reproduction number is less than one, the disease dies out while if the reproduction number is bigger than one, the disease persists (a forward transcritical bifurcation). If the transmission rate $\alpha_i \equiv \alpha_i(\frac{I_i}{T_i})$ is non-constant but rather a function of the prevalence then multiple endemic equilibria for System (1) are possible even when the basic reproductive number is less than one (backward bifurcation).

Examples of epidemic models exhibiting this behavior were first established for continuous-time epidemic models by Castillo-Chavez et al. [7, 8, 14] and Huang et al. [19]. Recently, examples using simpler continuous-time models were constructed by Castillo-Chavez and Hadeler [17], Feng et al. [15], Kribs et al. [20] and P. van den Driessche et al.[26]. The results of Feng et al. [15], Huang et al. [19] and Castillo-Chavez and Hadeler [17] have far reaching implications for the implementation of effective public health policies for HIV and Tuberculosis. The results of P. van den Driessche and P. Watmough have important theoretical implications as they illustrate the possibility of backward bifurcations for simple epidemic processes (S-I-S epidemic models based on a Volterra integral equation [26]). Backward bifurcations are possible in classical discrete-time epidemic models, that is, when α is constant. An example using a classical (constant α) discrete-time

susceptible-exposed-infective-susceptible (S-E-I-S) epidemic model has been recently developed [4].

To illustrate these results $\alpha(y) = y$, that is, $G(\alpha(y)y) = e^{-y^2}$. To make the example transparent, we use the approximation $e^{-y^2} \approx 1 - y^2$. Equation (6) becomes

$$y(t+1) = \frac{\gamma}{\mu + \gamma}(1 - y(t))y^2(t) + \frac{\gamma}{\mu + \gamma}\sigma y(t). \qquad (13)$$

Since $\alpha(y) = y$ is a non-constant transmission rate, prior results do not apply. Linearization of Equation (13) near the disease-free equilibrium $(x_\infty, y_\infty) \equiv (1, 0)$ gives

$$y(t+1) \approx \frac{\gamma}{\mu + \gamma}(-\alpha(0)G'(0) + \sigma)y(t).$$

Hence, $\Re_0 = \frac{\gamma\sigma}{(1-\Re_d)\gamma + \Re_d}$ where $\Re_d = \frac{\mu}{1-\gamma}$. A backward bifurcation using Equation (13) is illustrated numerically. We vary $\Re_0$ while the survival probability γ and the variable coefficient μ remain fixed at $\gamma = 0.98$ and $\mu = 0.01$. The following bifurcation curve (Figure 3) is generated:
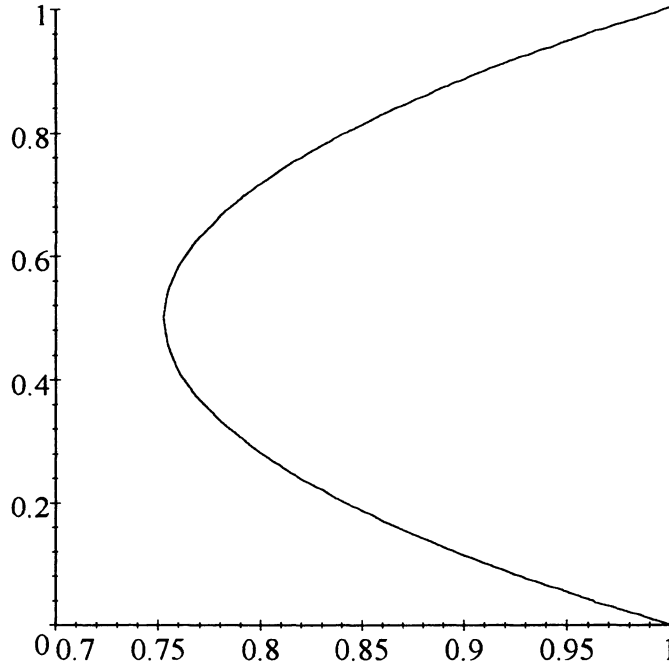
Figure 3: The relationship between the proportion of susceptibles and the basic reproductive number $\mathfrak{R}_0$. The horizontal axis, $0.7 \leq \mathfrak{R}_0 \leq 1$, and the vertical axis, $0 \leq y_\infty \leq 1$.

The disease free equilibrium $(1,0)$ is locally asymptotically stable for all values of the parameters. It is globally stable whenever $\mathfrak{R}_0 < 0.7525$. A backward bifurcation occurs at $\mathfrak{R}_0 \approx 0.7525$. When $\mathfrak{R}_0 \approx 0.7525$, an unstable endemic equilibrium appears, and for values of $\mathfrak{R}_0$ in the interval $(0.7525, 1)$ the system has 2 endemic equilibria coexisting with the disease free equilibrium (see Figure 3). hence, the future of the disease depends on initial conditions, that is, the model supports multiple attractors.

6 Conclusions

Simple nonlinear discrete - time population models are capable of generating complex (chaotic) dynamics. Nonlinear discrete-time $(S - I - S)$ epidemic models built on bounded or geometrically growing populations do not generate complex (chaotic) dynamics but could generate multiple attractors. We derived thresholds for the persistence of the disease and analyzed the global and local behavior of solutions. Extensions to populations with complex population dynamics and multiple attractors were briefly discussed.

7 ACKNOWLEDGMENTS

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