EPIDEMIC MODELS ON ATTRACTORS

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Abstract: Models for epidemic processes on attractors are presented. Thresholds for disease persistence are computed and used in the study of the global behavior of solutions of simple epidemic processes. The potential dynamic complexity of an epidemic process on chaotic attractors is illustrated.

1 Introduction

Mathematical and theoretical ecologists and epidemiologists have developed models to answer questions like: When will a disease regulate a population? Extensive work using continuous-time nonlinear differential equations has been carried out by a large number of investigators (see for example, Anderson *et al.* [3]; Bailey [5]; Brauer and Castillo-Chavez [7], Castillo-Chavez *et al.* [8 - 10]; Cooke and Yorke

[12]; Hassell *et al.* [19] and Nåsell [26], to name a few). Most research with ODE models have focused on two situations: A population in a demographic steady state is invaded by a disease and its impact is assessed; or, a growing population is invaded by a disease and conditions for disease regulation are established (see Levin and Pimentel [22]).

The study of epidemics of non-fatal diseases like measles, rubella, mumps, chicken-pox, gonorrhea or herpes is typically carried out as an invasion process on populations at a demographic steady state. This assumption has proved useful in the study of diseases in human populations (see Anderson and May [3] or Brauer and Castillo-Chavez [7]) but it may not be the most appropriate in the study of disease dynamics in animal populations. Populations in the wild compete at various levels. Intraspecific competition is often fierce and as a result strong fluctuations in density may be the norm.

Here, we study disease invasions in populations that live on attractors, including chaotic attractors that result from intraspecific competition [1, 2]. In order to keep the situation as simple as possible, we focus on the study of mild pathogen invasions. We study non-fatal diseases, that is, diseases for which the disease-induced mortality is negligible.

When a disease invades, it subdivides the target population into two classes: susceptibles (non-infected) and infectives. Prior to the time of invasion, the life-history of the population is assumed to be at a demographic "steady state" modeled by a pre-selected attractor. Hence, the population dynamics remain on this attractor, and, in this sense, they are fixed. The transition from susceptible to infective is a function of the contact rate α (between individuals) and the proportion of infectives (prevalence) in the population. Will the infective population survive? And if it does, will it settle on a particular attractor? What would be the relationship between the population and epidemic attractors? Some of these questions are addressed in this article.

2 Discrete-time Epidemic Models

Nonlinear discrete-time epidemic models are built on attractors generated by the equation $T(n+1) = f(T(n)) + \gamma T(n)$ where γ $(0 \leq \gamma < 1)$ denotes the constant probability of surviving per generation; f models the typically nonlinear birth or recruitment process; $T(n)$ denotes the population size at time n; and time is measured in discrete units. Whenever new recruits arrive at the constant per-capita rate μ $(f(T(n)) = \mu T(n))$ or at the total constant rate Λ per generation $(f(T(n)) = \Lambda)$ then the $T - dynamics$ are straightforward.

Epidemics on populations with either no real demography or capable of supporting exponential growth have been extensively studied using continuous-time models. We illustrate typical epidemic results on these simple attractors using an $S - I - S$ (Susceptible-Infected-Susceptible) *discrete* epidemic process.

At time n, $S(n)$ denotes the population of susceptibles; $I(n)$ the population of infecteds, assumed infectious; $T(n) \equiv S(n) + I(n)$ the total population size; and, $T_\infty \equiv \lim_{n\to\infty} T(n)$ the "demographic" steady state for the total population, whenever it exists. Individuals survive from one generation to the next with constant probability γ; 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 $x \in [0, \infty)$. Furthermore, all recruits are susceptible, time is measured in generations, and recovery from disease does not give permanent or temporary immunity. The model 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 $(1 - \sigma)$. This is a deterministic model but *probabilistic lingo* is used to facilitate understanding.

The above assumptions and definitions lead to the following discrete-time nonlinear $S-I-S$ epidemic model:

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

where $0 < \gamma, \sigma < 1$. The constant $\alpha > 0$ weighs the impact of the prevalence $(\frac{I(n)}{T(n)})$ on disease transmission. If α is large, then it is easier to become infected (see Figure 1) while if $\alpha = 0$ there is no epidemic.

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. The $T-$ *dynamics* are independent of disease dynamics–a consequence of the fact that the disease is assumed to be non-fatal. The function f could "literally" be replaced by any biologically reasonable birth or recruitment function [6, 30]. Hence, it is possible to fix the long-term $T-$*dynamics* to "any" type of attractor—including chaotic attractors.

3 Simple Population Dynamics and SIS Epidemics

Two simple examples are used to introduce epidemiological and demographic concepts. When $f(T(n)) = \mu T(n)$, Equation (2) reduces to

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

and, consequently,

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

The demographic basic reproductive number

$$\Re_d = \frac{\mu}{1-\gamma},$$

a dimensionless quantity, gives the average number of descendants produced by a (typically small) pioneer population ($T(0)$) over its life-time. If $\Re_d > 1$ then the population invades at a geometric rate while if $\Re_d < 1$ the population dies out.

Letting

$$x(n) = \frac{S(n)}{T(n)} \text{ and } y(n) = \frac{I(n)}{T(n)}, \qquad (3)$$

reduces System (1) to the one-dimensional autonomous "system" for $y(n)$:

$$y(n+1) = \frac{\gamma}{\gamma+\mu}(1-y(n))(1-G(\alpha y(n))) + \frac{\gamma\sigma}{\gamma+\mu}y(n). \qquad (4)$$

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

$\Re_0$, the number of secondary infections generated by a "typical" infected (assumed infectious) individual over his or her lifetime in a population of (mostly) susceptibles (at a demographic steady state), determines whether or not a disease can invade. From Equation (4), we find that

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

after noticing that, near the demographic steady state $(x_\infty, y_\infty) \equiv (1,0)$,

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

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 [10, 11]. Since $\frac{1}{(1-\gamma)(\Re_d-1)+(1-\gamma\sigma)}$ gives the demographic death-adjusted infectious period measured in generations, $\Re_0$ decreases with population growth ($\Re_d > 1$) and increases with population decay ($0 < \Re_d < 1$). This is not surprising since all recruits are susceptible. At any rate, whenever $\Re_d \neq 1$ demography plays a role. We summarize these findings below:

Result 1. *(a) Suppose that* $\Re_d > 1$. *Then*

(i) $\Re_0 \leq 1$, *implies that the proportion* $\frac{I}{T}$ *of infectives in the total population tends to* 0 *as* $n \to \infty$. $(\frac{S}{T}, \frac{I}{T})$ *tends to the disease-free equilibrium* $(1, 0)$ *while* S *and* T *increase at the geometric rate* $(\mu + \gamma)$.

(ii) $\Re_0 > 1$, *implies that the proportion* $\frac{I}{T}$ *of infectives in the total population tends to a positive number,* $\overline{\frac{I}{T}}$ *as* $n \to \infty$. $(\frac{S}{T}, \frac{I}{T})$ *tends to an endemic equilibrium while* I, S *and* T *increase at the geometric rate* $(\mu + \gamma)$.

(b) Suppose that $\Re_d < 1$. *Then*

(i) $\Re_0 \leq 1$, *implies that the proportion* $\frac{I}{T}$ *of infectives tends to* 0 *as* $n \to \infty$. $(\frac{S}{T}, \frac{I}{T})$ *tends to the disease-free equilibrium* $(1, 0)$ *while* S *and* T *decrease to zero at the geometric rate* $(\mu + \gamma)$.

(ii) $\Re_0 > 1$, *implies that the proportion* $\frac{I}{T}$ *of infectives tends to a positive number* $\overline{\frac{I}{T}}$ *as* $n \to \infty$. $(\frac{S}{T}, \frac{I}{T})$ *tends to an endemic equilibrium while* I, S *and* T *increase at the geometric rate* $(\mu + \gamma)$.

Proof: Define the reproduction function for the infected individuals of System (4), $h : [0, 1] \to [0, 1]$ by $h(y) = \frac{\gamma}{\gamma+\mu}(1 - y)(1 - G(\alpha y)) + \frac{\gamma\sigma}{\gamma+\mu}y$, where $h(0) = 0$ and $0 \leq y \leq 1$. 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 $h'(0) = \frac{\gamma}{\gamma+\mu}(-\alpha G'(0) + \sigma) \leq 1$, and, 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 $\overline{y}$, the nonexistence of non-trivial two-cycles for h is established. 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 $y \in [0, 1]$, and, h has no non-trivial 2-cycles. Why? 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. Sharkovskii's Theorem and $1 + h'(y) \neq 0$ imply the nonexistence of any $m - cycles$ for $m > 1$ in $[0, 1]$. From a result of Cull [13], 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 $n \to \infty$.

When $f(T(n)) = \Lambda$ then $T(n+1) = \Lambda + \gamma T(n)$, $T(n) = (T(0) - \frac{\Lambda}{1-\gamma})\gamma^n + \frac{\Lambda}{1-\gamma}$, and $T_\infty = \frac{\Lambda}{1-\gamma}$. The total population is asymptotically constant, that is, demographic effects "eventually" disappear.

Letting $S(n) = T_\infty - I(n)$ in the $I - equation$ of System (1) gives a one-dimensional dynamically equivalent autonomous "system" for $I(n)$ [28, 31] :

$$I(n+1) = \gamma(1 - G(\frac{\alpha I(n)}{T_\infty}))(T_\infty - I) + \gamma\sigma I(n). \qquad (5)$$

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 [10, 11]:

Result 2: *Suppose* $f(T(n)) = \Lambda$ *in System (1)* then

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

(b) $\Re_0 > 1$ *implies that 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 following numerical example illustrates the dynamics in these "extreme" but simple demographic situations:

Let $e^{-d} = \gamma$, $e^{-\frac{\alpha I(n)}{T(n)}} = G(\frac{\alpha I(n)}{T(n)})$ and $e^{-\beta} = \sigma$ then

$$\left.\begin{array}{rcl} S(n+1) & = & f(T(n)) + e^{-d}S(n)e^{-\frac{\alpha I(n)}{T(n)}} + e^{-d}I(n)(1-e^{-\beta}), \\ I(n+1) & = & e^{-d}(1-e^{-\frac{\alpha I(n)}{T(n)}})S(n) + e^{-d}e^{-\beta}I(n), \end{array}\right\} \quad (6)$$

where α, β and d are positive constants. $f(T(n)) = \mu T(n)$ implies that $T(n+1) = (e^{-d} + \mu)T(n)$ and $\Re_d = \frac{\mu}{1-e^{-d}}$. Using proportions reduces System (6) to

$$\left.\begin{array}{rcl} y(n+1) & = & \frac{e^{-d}}{e^{-d}+\mu}(1-y(n))(1-e^{-\alpha y(n)}) + \frac{e^{-d}e^{-\beta}}{e^{-d}+\mu}y(n). \end{array}\right\} (7)$$

The basic reproductive number

$$\Re_0 = \frac{\alpha^2}{1 + \mu e^d - e^{-\beta}},$$

determines the asymptotic behavior of System (6) [see Result 1].

The constants $\beta = 0.1$, $d = \ln 2$ and $\mu = 0.1$ are fixed and the transmission coefficient α is varied. As α increases, the basic reproductive number, $\Re_0$, increases from values less than 1 to numbers bigger than 1. A transcritical bifurcation occurs while the demographic basic reproductive number remains fixed at $\Re_d = \frac{\mu}{1-e^{-d}} = 0.25 < 1$. The asymptotic dynamics of the system with proportions changes: from a globally stable disease-free equilibrium becomes unstable, a globally stable endemic equilibrium is born, and, the total population decreases to zero (see Figure 1). In particular, $\alpha = 0.6$ gives $\Re_0 = 1.22 > 1$ and the system with proportions has a globally stable "endemic" equilibrium at $(0.5598, 0.4402)$. Similar results are obtained with $f(T) = \Lambda$.

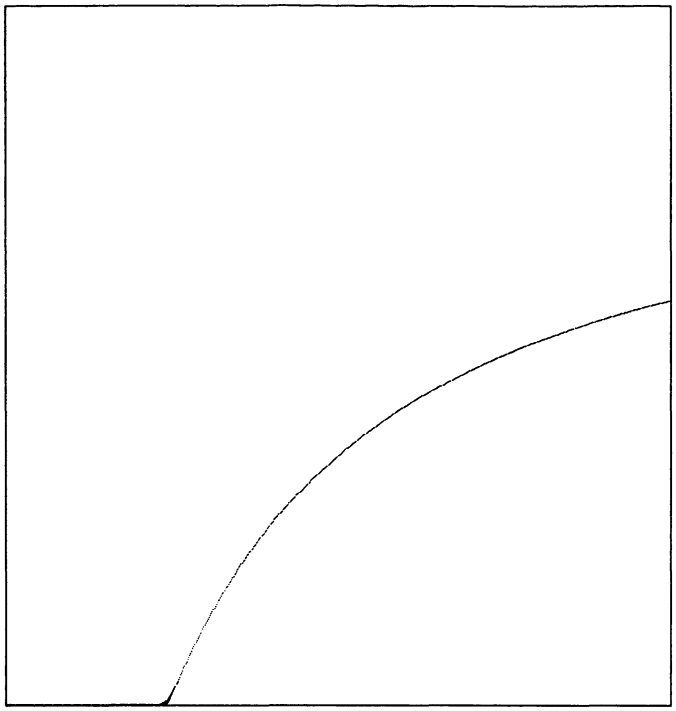

Figure 1: Infecteds increase as α increases. Horizontal axis $0 \leq \alpha \leq 1$, vertical axis $0 \leq \frac{I_\infty}{T_\infty} \leq 1$.

The prior example gives "typical" epidemic results. However, the outcomes are not always this obvious. If one assumes that the infection rate α is a function of the prevalence, $\frac{I(n)}{T(n)}$, that is, if $\alpha \equiv \alpha(\frac{I(n)}{T(n)})$ then $\Re_0 > 1$ is not always *the threshold.* In fact, multiple epidemic attractors can coexist when $\Re_{critical} < \Re_0 < 1$, where $\Re_{critical}$ depends on the function α. We do not pursue this direction here. For examples of this type (backward bifurcation) arising from discrete and continuous time models see [14-21, 29].

4 Epidemic Models in Complex Attractors

Since the $T-dynamics$ are forced to live on the attractor generated by $T(n+1) = f(T(n)) + \gamma T(n)$ then the selection of a flexible f makes it possible to increase the type of demographic "steady states" available. To illustrate the role of f, it

is assumed that f is governed by the Smith-Slatkin model [14, 27], that is,

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

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

$$T(n+1) = \frac{aT(n)}{1 + (bT(n))^l} + \gamma T(n), \qquad (8)$$

where a is the maximal per-capita intrinsic growth rate of the population; l reflects the type and strength of intraspecific competition; and b scales the carrying capacity of the population [14, 27]. Whenever $0 < l < \frac{2(a+\gamma)}{a+\gamma-1}$, $T_\infty = \frac{(a+\gamma-1)^{\frac{1}{l}}}{b}$, approaches a simple demographic attractor (fixed point); Theorem 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). Whenever T is on a $2 - cycle$, then either the typical definition of $\Re_0$ breaks down or it must be computed on the corresponding steady state of T^2. This process of period doubling bifurcation continues indefinitely. This f is capable of supporting complex (including chaotic) attractors. Figure 2 illustrates the existence of a chaotic regime in the demographic model, we notice that when $l = 29.9$ the chaotic attractor is a two-piece attractor. For illustrative purposes we have chosen to focus on this region.

Building epidemic models on the attractors generated by Equation (8) (or using any other function that supports complex dynamics) provides the set up for the study of non-fatal epidemics on attractors. What is the structure of disease dynamics on these attractors? Do the $I - orbits$ have the same structure as the $T - orbits$? Some possibilities are presented with an example.

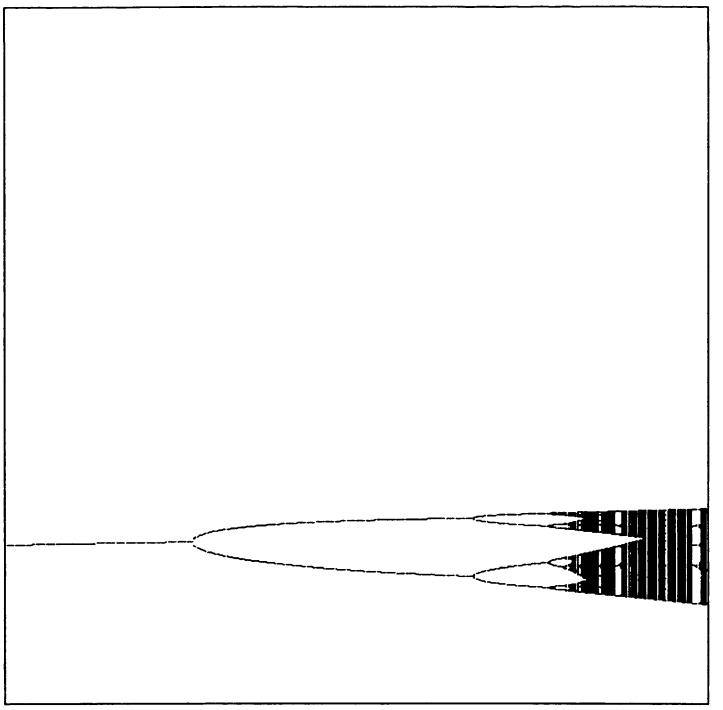

Figure 2: Period-doubling bifurcations route to chaos in the demographic equation under Smith-Slatkin model with $a = 1.5$, $b = 1$, $\gamma = 0.9$ and $0 \leq l \leq 30$. On the horizontal axis, $0 \leq l \leq 30$.

5 Illustrative Example

Using Ricker's equation for the $T - dynamics$, Barrera *et al.*[6] constructed a numerical example where it appears that T and S are on a $2 - cycle$, while I is on a fixed value. They used γ (the probability of survival) as their bifurcation parameter. Unfortunately, varying γ alters the $T - dynamics$. Hence, in order to keep the $T - dynamics$ fixed, we vary α, an implicit measure of the contact process between $S-$ and $I-$ individuals. We keep all other parameters in System (6) with $f(T) = \frac{aT}{1+(bT)^l}$ fixed at

$$a = 1.5, b = 1, l = 29.9, \beta = \ln 10 \text{ and } d = \ln \frac{10}{9},$$

that is, the $T-$ *dynamics* are kept at a fixed two-piece chaotic attractor. When the infection (contact) rate α is small, the disease dies out ($I(t) \to 0$) (see Figure 5). As α increases the situation changes (see Figures 3, 4 and 5). The $T-$ and $S-$ *dynamics* appear to be the same (both live on two-piece chaotic attractors) while those of $I-$ differ but probably only for a relatively small range of values of α; that is, as long as $0 \leq \alpha < \alpha_{critical}$. We have not been able to compute, even for this example, the exact value of $\alpha_{critical}$. However, our simulations support an $\alpha_{critical} > 0$ and the possibility that the $I-, S-$, and $T-$ *dynamics* are *qualitatively* equivalent whenever $\alpha > \alpha_{critical}$. That is, numerical simulations support the possibility that the $I-, S-$, and $T-$ populations live on (what appear to be self-similar) chaotic attractors. Are there any quantitative differences?

Numerical simulations show that the "amplitude" (range of values) of each attractor differ and are functions of α (contact rate). The "amplitude" of the $I-$ *attractor* can be so small (for some values of α) that detailed (chaotic) dynamics seem like mild fluctuations around a fixed point (or "average value") when α is near $\alpha_{critical}$. However, the "amplitude" of the I fluctuations increases dramatically with α(see Figure 5).

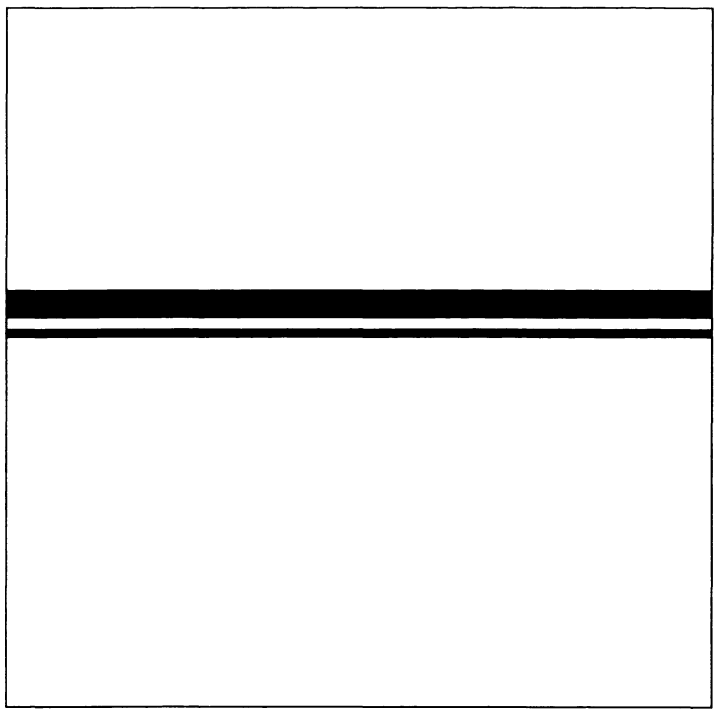

Figure 3: Demographic process on a two-piece chaotic attractor. The dynamics is independent of the infection rate α. On the horizontal axis, $0 \leq \alpha \leq 30$. On the vertical axis, $0 \leq T \leq 2$.

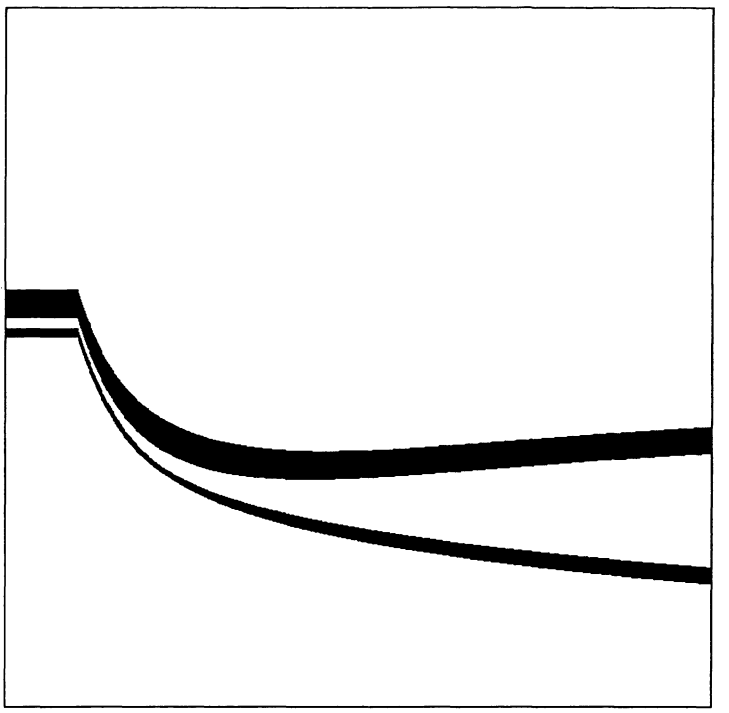

Figure 4: Susceptible population remains on a chaotic attractor as the infection rate α increases from zero. The two chaotic branches separate as α increases, the "amplitude" of S increases with α. On the horizontal axis, $0 \leq \alpha \leq 30$. On the vertical axis, $0 \leq T \leq 2$.

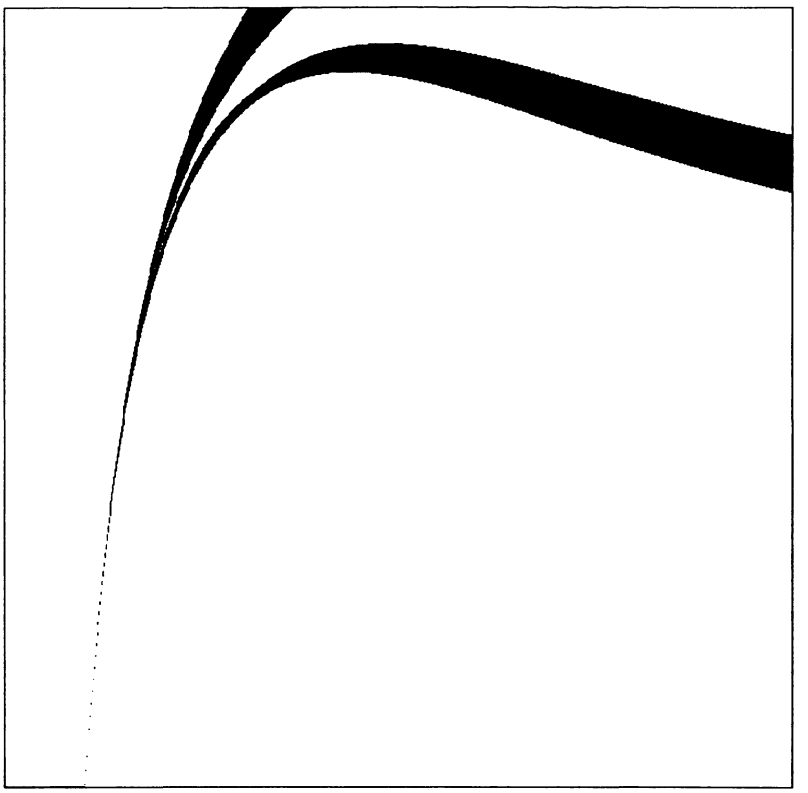

Figure 5: Infective population increases from zero and catches up with the demographic process on a two-piece chaotic attractor as the infection rate α increases from zero. The two pieces of the chaotic attractor separate as α increases. On the horizontal axis, $0 \leq \alpha \leq 30$. On the vertical axis, $0 \leq T \leq 0.1$.

6 Conclusions

Classical (continuous-time) epidemiological models have focused on the study of disease dynamics in populations at simple demographic steady states (fixed point or exponential or geometric growth). This assumption has been quite useful in the study of diseases in humans. Animal populations often experience wild fluctuations. Population fluctuations are the result of a variety of factors that include but are not limited to intraspecific competition. Scramble competition is typically modeled with a one-hump map. Such maps are capable of generating

complex (including chaotic) dynamics [23-25].

In this article, we study disease dynamics in populations with potentially complex intrinsic (disease-free) population dynamics. The populations of interest include those where fierce competition (scramble competition) for resources is the norm.

In the tradition of classical epidemiology, we fix the population dynamics to its corresponding (disease-free) demographic steady state which now includes "arbitrary" attractors–the type of attractors generated by one-hump maps [23-25].

Disease dynamics typically depend on contact rates (here modeled by α) and hence, the $I-dynamics$ on the $T-attractor$ are followed as α increases from 0 (no disease) to ∞ (where the $I-$ and $T-dynamics$ are closer as $S-$individuals become $I-individuals$ very fast). Numerical simulations show that as α increases a fast transition from no-disease to complex I (endemic) dynamics takes place. It appears (except for a tiny interval of $\alpha-$values) that the $T-$ and $I-dynamics$ are *qualitatively* equivalent. However, the range of values of I ("amplitude") increases as α crosses $\alpha_{critical}$. The contact rate plays a key role on disease survival ($I > 0$) and the severity of the epidemic. In other words, α drives the quantitative differences.

Numerical simulations suggest that the differences between $I-$ and $T-dynamics$ are limited to variations in "amplitude". Numerical simulations suggest the possibility that the $I-$ and $T-$populations may actually have self-similar dynamics in the chaotic regime.

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