J. Math. Biol.
DOI 10.1007/s00285-008-0188-9

**Mathematical Biology**

# Disease-induced mortality in density-dependent discrete-time S-I-S epidemic models

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Received: 5 December 2007 / Revised: 29 April 2008


**Abstract** The dynamics of simple discrete-time epidemic models without disease-induced mortality are typically characterized by global transcritical bifurcation. We prove that in corresponding models with disease-induced mortality a tiny number of infectious individuals can drive an otherwise persistent population to extinction. Our model with disease-induced mortality supports multiple attractors. In addition, we use a Ricker recruitment function in an SIS model and obtained a three component discrete Hopf (Neimark–Sacker) cycle attractor coexisting with a fixed point attractor. The basin boundaries of the coexisting attractors are fractal in nature, and the example exhibits sensitive dependence of the long-term disease dynamics on initial conditions. Furthermore, we show that in contrast to corresponding models without disease-induced mortality, the disease-free state dynamics do not drive the disease dynamics.

**Keywords** Basin of attraction · Disease-induced mortality · Multiple attractors

**Mathematics Subject Classification (2000)** 37G15 · 37G35 · 39A11 · 92B05

## 1 Introduction

The consequences of disease-induced mortality on disease dynamics have been studied quite extensively since the first development of mathematical epidemiology [4,5,

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22,30]. However, detailed studies of the interactions between density dependent birth or recruitment processes and disease-induced mortality are rare [1,2,6]. In recent papers, Hwang and Kuang [20,21,23] as well as Berezovsky et al. [6,7] illustrated surprising dynamics in a simple continuous-time susceptible-infected (SI) model with variable population size and disease-induced mortality. The SI model supports multiple attracting equilibria. Berezovsky et al. [6,7] used Briot-Bouquet (blowing-up) transformations of the singular point at the origin to show, via the existence of homoclinic orbits, a tiny number of infectious individuals igniting a disease outbreak in the simple continuous-time model. We prove that a tiny number of infectious individuals can drive an otherwise persistent population to extinction.

In this paper, we focus on discrete-time SIS epidemic models with disease-induced mortality. Viral infections, such as most infections from rhinoviruses (causative agents of the common cold) are examples of such infections. Since a mild rhinovirus infection is a non-fatal infection that occurs mostly in humans and other higher primates, it does not quite fit our discrete-time scale model framework. However, such non-fatal infections could impede the immune system and hence decrease the survivability of the infected individual. The change in survivability is illustrative of the type of biological reality being modeled. Our primary focus is on the impact of disease-induced mortality on discrete-time SIS epidemic models. Castillo-Chavez and Yakubu [9–11], Franke and Yakubu [15,16,33] as well as Rios-Soto et al. [29] have studied corresponding models without disease-induced mortality. For the model without disease induced mortality, Castillo-Chavez and Yakubu, computed the epidemic threshold parameter, $\mathcal{R}_0$, and used it to prove that if $\mathcal{R}_0 < 1$ then the disease goes extinct whereas $\mathcal{R}_0 > 1$ implies the disease is endemic. When the demographic dynamics is asymptotically constant, Castillo-Chavez and Yakubu proved that local stability of the disease-free and endemic equilibria is actually a global property. That is, the disease dynamics is characterized by a global transcritical bifurcation.

The long-term dynamics of our model with disease-induced mortality depends on three threshold parameters, the basic reproduction number ($\mathcal{R}_0$), and two demographic threshold parameters ($\mathcal{R}_{D_1}$ and $\mathcal{R}_{D_2}$). The total population is persistent whenever $\mathcal{R}_{D_2} > 1$ whereas it goes extinct at small initial population values whenever $\mathcal{R}_{D_1} < 1$. In this paper, we prove that $\mathcal{R}_0 < 1$ implies disease extinction whereas $\mathcal{R}_0 > 1$ and $\mathcal{R}_{D_2} > 1$ imply disease persistence.

In addition, we prove that our model exhibits multiple attractors. Other more realistic epidemic models, for example HIV models, are known to support multiple attractors [14,17,18,32]. When the recruitment function is the Ricker map, our simple SIS model exhibits sensitive dependence of the long-term dynamics on *positive* initial population sizes, where the disease-free state is on a globally attracting equilibrium point. In addition, we prove via the existence of a globally asymptotically stable fixed point that our model under geometric growth rate exhibits the transcritical bifurcation with or without disease-induced mortality.

The paper is organized as follows: In Sect. 2, we introduce the discrete-time SIS model with disease-induced mortality. We obtain, in Sect. 3, preliminary results on the qualitative dynamics of our model. In Sect. 4, the three threshold parameters $\mathcal{R}_0$, $\mathcal{R}_{D_1}$ and $\mathcal{R}_{D_2}$ are computed and used to study the long-term qualitative dynamics of our model. Also in Sect. 4, we use the Beverton–Holt recruitment function [8,19] to

illustrate disease induced extinction of the total population where $\mathcal{R}_0 > 1$. Conditions for the support of multiple fixed points are derived in Sect. 5. Also in Sect. 5, specific examples are used to demonstrate coexisting attracting fixed points in our model. The basins of the multiple attracting fixed points are relatively simple. Complex basin structures are highlighted in Sect. 6 [3]. In particular, we use the Ricker recruitment function [28] to illustrate two coexisting attractors, a three component Hopf (Neimark–Sacker [31]) cycle attractor and a fixed point attractor, with fractal basin boundaries. In these examples, the SIS epidemic model is under asymptotically constant disease-free dynamics and infections are modeled as Poisson processes [9]. Section 7 is on the effects of geometric growth recruitment functions in our SIS model, and concluding remarks are presented in Sect. 8.

## 2 SIS epidemic model with disease induced death

Our SIS epidemic model is built under the assumption that the dynamics of the total population size are governed by the equation

$$N(t+1) = f(N(t)) + \gamma_1 S(t) + \gamma_2 I(t), \tag{1}$$

where at generation $t$, $S(t)$ denotes the susceptible population; $I(t)$ the infected population (assumed infectious); and $N(t) \equiv S(t) + I(t)$ the total population size. The function $f(N) \in C^1(\mathbb{R}_+, \mathbb{R}_+)$ models the birth or recruitment process, and $\gamma_1, \gamma_2 \in (0, 1)$ are respectively the constant "probabilities" of the susceptible and infective individuals surviving per generation. To include disease induced mortality in our epidemic model, we assume throughout that $\gamma_1 \geq \gamma_2$. Consequently, an infected individual has a lesser chance than a susceptible individual to survive one generation.

When $\gamma = \gamma_1 = \gamma_2$, all individuals (susceptibles and infectives) have equal probability of surviving one generation and Model (1) reduces to the demographic equation

$$N(t+1) = f(N(t)) + \gamma N(t). \tag{2}$$

Beverton–Holt's model and Ricker's model are classic discrete-time single-species population models that give the population density of the species at generation $t+1$ as a function of the density at generation $t$. The Beverton–Holt and Ricker models have been used to model contest and scramble intraspecific competitions, respectively [8,12,19,24–28]. In papers that we coauthored [9–11,15,16], we studied Model (2) with the constant recruitment function,

$$f(N(t)) = \Lambda,$$

with the Beverton–Holt recruitment function,

$$f(N(t)) = \frac{\mu k N(t)}{k + (\mu - 1)N(t)},$$

and with the Ricker recruitment function,

$$f(N(t)) = N(t)e^{r\left(1-\frac{N(t)}{k}\right)},$$

where the carrying capacity of the environment is $k$. In the Beverton–Holt (respectively, Ricker) recruitment function, $\mu > 1$ (respectively, $r > 0$) is the intrinsic growth rate. With these choices of $f$, Model (2) is known to have a globally attracting positive fixed point denoted by $N_\infty$ whenever $\Lambda > 0$, $\mu > 1$, and $0 < r < \frac{2+(1-\gamma)\ln(1-\gamma)}{1-\gamma}$ [10,26]. When $r > \frac{2+(1-\gamma)\ln(1-\gamma)}{1-\gamma}$ and the recruitment function is the Ricker model, Model (2) undergoes period doubling bifurcations route to chaos [11,13,15,16,24–26].

When new recruits arrive at the positive per-capita growth rate $\mu$, then

$$f(N(t)) = \mu N(t),$$

and the solution of Eq. (2) is

$$N(t) = (\gamma + \mu)^t N(0).$$

Consequently, the *demographic basic reproductive* number is

$$\mathcal{R}_D = \frac{\mu}{1-\gamma}.$$

If $\mathcal{R}_D < 1$, the total population goes extinct at a geometric rate, and if $\mathcal{R}_D > 1$, the total population explodes at a geometric rate.

Even though our model is deterministic and does not attempt to capture stochastic phenomenon at low population levels, we use probability terminology to describe some of the model parameters. To introduce the deterministic SIS model, we assume that infective individuals recover with constant probability $(1-\sigma)$. Furthermore, assume that the *escape function*

$$\phi : [0, \infty) \to [0, 1]$$

is a monotone convex probability function with $\phi(0) = 1$ and $\phi'(x) \leq 0$ for all $x \in [0, \infty)$. Also, we assume that the susceptible individuals become infected with non-linear probability $\left(1 - \phi\left(\alpha\frac{I}{N}\right)\right)$ per generation, where the transmission constant $\alpha > 0$.

When infections are modeled as Poisson processes, for example, then $\phi\left(\alpha\frac{I}{N}\right) = e^{-\alpha\frac{I}{N}}$ [10,16,29]. When $\phi(x) = e^{-x}$ then $\phi''(x) \geq 0$ for all $x \in [0, \infty)$. To model this constraint in our SIS model, we also assume that the escape function $\phi$ is concave up and satisfies $\phi''(x) \geq 0$ for all $x \in [0, \infty)$.

Our assumptions and notation lead to the following $SIS$ epidemic model with disease induced mortality:

**Table 1** Model parameters and functions

| Parameter | Description |
|---|---|
| $\gamma_1$ | Survival "probability" of susceptible individuals per generation; |
| $\gamma_2$ | Survival "probability" of infective individuals per generation; |
| $\alpha$ | Transmission constant; |
| $(1-\sigma)$ | Recovery "probability" of infective individuals per generation; |
| $\phi$ | Frequency-dependent escape "probability" function; |
| $f$ | Birth or recruitment function; |
| $\mathcal{R}_D$ | Demographic basic reproductive number |

$$\left.\begin{aligned} S(t+1) &= f(N(t)) + \gamma_1 \phi\left(\alpha \tfrac{I(t)}{N(t)}\right) S(t) + \gamma_2 (1-\sigma) I(t) \\ I(t+1) &= \gamma_1 \left(1 - \phi\left(\alpha \tfrac{I(t)}{N(t)}\right)\right) S(t) + \gamma_2 \sigma I(t) \end{aligned}\right\}, \tag{3}$$

where $0 < \gamma_2 < \gamma_1 < 1$, $0 < \sigma < 1$ and $N(t) > 0$. Below, we summarize some of the underlying assumptions in Model (3).

| | |
|---|---|
| (a) | The disease increases mortality but does not affect fecundity; |
| (b) | There is no acquired immunity; |
| (c) | There is no latent period (or it is very short); |
| (d) | Transmission is frequency dependent rather than density dependent. |

A fair amount of theoretical and empirical work has been done to compare these assumptions. In continuous-time models, it is known that these assumptions are not universally applicable [4]. Our model parameters and functions are summarized in Table 1.

Our model is a deterministic SIS epidemic model and has no "probability" of transmission. The assumption of deterministic dynamics is valid in a large population, where stochasticity is unimportant. This assumption places a constraint on the applicability of our model. For example, stochastic transmission (including a Poisson process) in a small population (close to extinction) would not be described by our SIS model.

Model (3) reduces to the SIS epidemic model of Castillo-Chavez and Yakubu [9–11] when there is no disease induced mortality and $\gamma_1 = \gamma_2$. In Model (3), the total population in generation $t+1$ ($N(t+1) = S(t+1) + I(t+1)$), the sum of the two equations of Model (3), is Eq. (1).

If the disease is not present ($I(t) = 0$), then $N(t) = S(t)$ and our SIS model reduces to the equation

$$S(t+1) = f(S(t)) + \gamma_1 S(t). \tag{4}$$

Equation (4) describes the population dynamics of the disease-free state. When this equation has a positive fixed point, we denote it by $S_\infty > 0$. Consequently, the point $(S_\infty, 0)$ is a disease-free equilibrium point of Model (3).

To understand the impact of disease-induced mortality on disease dynamics, we will study Model (3) under specific functional forms for the recruitment function $f$. In particular, we will study the epidemic model under constant, constant per-capita (geometric growth), Beverton–Holt and Ricker recruitment functions. These recruitment functions are commonly found in the literature [8,10,11,27,28].

## 3 Preliminary results

Here, we obtain some auxiliary results that will be used to study disease persistence and extinction in our SIS model. In the following result, we obtain one-variable bounds on the total population of our two-dimensional model.

**Lemma 1** *In Model* (3)*,*

$$f(N(t)) + \gamma_2 N(t) \leq N(t+1) \leq f(N(t)) + \gamma_1 N(t).$$

Proof of Lemma 1 is in the Appendix.

Using the substitution $S(t) = N(t) - I(t)$, the $I$-equation and the $N$-equation in Model (3) become

$$I(t+1) = \gamma_1 \left(1 - \phi\left(\alpha \frac{I(t)}{N(t)}\right)\right)(N(t) - I(t)) + \gamma_2 \sigma I(t)$$

and

$$N(t+1) = f(N(t)) + \gamma_1 (N(t) - I(t)) + \gamma_2 I(t),$$

respectively.

On the closed interval $[0, N]$, let

$$F_N(I) = \gamma_1 \left(1 - \phi\left(\alpha \frac{I}{N}\right)\right)(N - I) + \gamma_2 \sigma I$$

and

$$G_N(I) = f(N) + \gamma_1 (N - I) + \gamma_2 I.$$

When $F_N$ has a unique positive fixed point and a unique critical point, we denote them by $I_N$ and $C_N$, respectively. The sets of sequences generated by

$$I(t+1) = F_{N(t)}(I(t))$$

and

$$N(t+1) = G_{N(t)}(I(t))$$

are the sets of density sequences generated by the infective and the total population equations, respectively. Franke and Yakubu [16], used the map $F_N$ with $\gamma = \gamma_1 = \gamma_2$ to study disease dynamics in periodically forced SIS epidemic models.

To study Model (3), we need the following results on the properties of $F_N$ and $G_N$.

**Lemma 2** *$F_N(I)$ and $G_N(I)$ satisfy the following conditions.*

(a) *If $0 \leq I \leq N$, then $F_N(I) \leq \min\{N, G_N(I)\}$ with equality if and only if $(N, I) = (0, 0)$.*
(b) *$F'_N(0) = -\alpha\gamma_1\phi'(0) + \gamma_2\sigma$ and $F'_N(N) > -1$.*
(c) *$F_N(I)$ is concave down on $[0, N]$.*
(d) *$F_N(I) \leq F'_N(0)I$ on $[0, N]$.*
(e) *If $F'_N(0) > 1$, then $F_N$ has a unique positive fixed point $I_N$ in $[0, N]$.*
(f) *$F_1(\frac{I}{N}) = \frac{1}{N}F_N(I)$. That is, the frequency dependent $F_1$ is equal to the ratio of the density dependent $F_N$ and the total population size.*
(g) *If $N_0 < N_1$ and $F'_N(0) > 1$, then $I_{N_0} < I_{N_1}$ where $I_{N_i}$ is the positive fixed point of $F_{N_i}$ in $[0, N_i]$. In general, the fixed point for $F_N$ is $NI_1$.*
(h) *If $C_1$ exists, then $C_N = NC_1$.*
(i) *If $N_0 < N_1$, then $F_{N_0}(I) < F_{N_1}(I)$ for all $I \in (0, N_0]$.*

The proof of Lemma (2) is in the Appendix.

Next, we obtain the invariance of the positive quadrant.

**Lemma 3** *In Model (3),*

(a) *If $I(0) > 0$ then $I(t) > 0 \; \forall \; t \in \mathbb{Z}_+$.*
(b) *If $N(0) > 0$ then $N(t) > 0 \; \forall \; t \in \mathbb{Z}_+$.*

Proof of Lemma 3 is in the Appendix.

## 4 Disease extinction or persistence

To study the qualitative dynamics of Model (3), we define the map

$$H : \{(N, I) | 0 \leq I \leq N\} \to \{(N, I) | 0 \leq I \leq N\}$$

by

$$H(N, I) = (G_N(I), F_N(I)).$$

Lemmas (2) and (3) show that the set of iterates of the dynamical system $H$ on $\{(N, I) | 0 \leq I \leq N\}$ is equivalent to set of density sequences generated by Model (3), where $H_i^t(N, I)$ denotes the $i$th component of the $t$th iterate (under $H$) of the initial condition $(N, I)$.

The point $(N^*, I^*)$ is a *positive fixed point* of $H$ if

$$H(N^*, I^*) = (N^*, I^*)$$

and $0 < I^* \leq N^*$.

**Definition 4** The total population is *uniformly persistent* under $H$ if there exists a constant $\eta > 0$ such that

$$\liminf_{t\to\infty} H_1^t(N, I) \equiv \lim_{t\to\infty} \inf H_1^t(N, I) \geq \eta$$

for every non-zero initial condition.

The total population is said to be *persistent* under $H$ if $\liminf_{t\to\infty} H_1^t(N, I) > 0$ [16,34]. Consequently, uniform persistence implies the persistence of the total population.

**Definition 5** The total population is driven to *extinction* under $H$ if

$$\lim_{t\to\infty} H_1^t(N, I) = 0$$

for every initial condition.

For each $i \in \{1, 2\}$, define

$$D_i : [0, \infty) \to [0, \infty)$$

by

$$D_i(N) = f(N) + \gamma_i N, \tag{5}$$

and let

$$\mathcal{R}_{D_i} = \frac{f'(0)}{1 - \gamma_i} \quad \text{whenever } f(0) = 0.$$

The auxiliary functions $F_N$, $G_N$, $H$ and $D_i$ will be used throughout, and their biological meanings are summarized in Table 2.

Recall that $f(N) \in C^1(\mathbb{R}_+, \mathbb{R}_+)$. Thus, $f(0) = 0$ implies that $\mathcal{R}_{D_i} \geq 0$ for $i \in \{1, 2\}$. In our epidemic model, $\gamma_1 > \gamma_2$ implies that $\mathcal{R}_{D_1} > \mathcal{R}_{D_2}$. That is, the disease-free demographic basic reproduction number (Lemma 6) is greater than that of the total population (Lemma 7). Lemmas (6) and (7) give sufficient conditions for the persistence of the disease-free state susceptible population and the total population, respectively.

**Table 2** Auxiliary functions

| Auxiliary function | Meaning |
|---|---|
| $D_i(N) = f(N) + \gamma_i N$ | Total population of new births and survivors; |
| $F_N(I) = \gamma_1 \left(1 - \phi\left(\alpha \frac{I}{N}\right)\right)(N - I) + \gamma_2 \sigma I$ | Infective population in the next generation; |
| $G_N(I) = f(N) + \gamma_1(N - I) + \gamma_2 I$ | Total population in the next generation; |
| $H(N, I) = (G_N(I), F_N(I))$ | Vector of the total and infective populations. |

**Lemma 6** *Let $f(0) = 0$. If $\mathcal{R}_{D_1} > 1$, then the disease-free susceptible population described by Eq. (4) is persistent. However, if $\mathcal{R}_{D_1} < 1$ then $\{(0, 0)\}$ is locally asymptotically stable in Model (3), and both the susceptible and infected populations go extinct at low values of initial population sizes.*

The proof of Lemma 6 is in the Appendix.

By Lemma (6), $\mathcal{R}_{D_1}$ is the disease-free state demographic basic reproduction number.

**Lemma 7** *If either $f(0) > 0$ or $f(0) = 0$ and $\mathcal{R}_{D_2} > 1$, then the total population is uniformly persistent.*

The proof of Lemma 7 is in the Appendix.

By Lemma (7), the total population is uniformly persistent when

$$f(N) = \Lambda,$$

or

$$f(N) = \frac{\mu k N}{k + (\mu - 1)N}$$

and

$$\mu > 1 - \gamma_2,$$

or

$$f(N) = Ne^{r\left(1-\frac{N}{k}\right)}$$

and

$$r > 0 > \ln(1 - \gamma_2).$$

In Lemma (8), the object under study is the large population per capita growth rate.

**Lemma 8** *If*

$$\overline{\lim_{N\to\infty}} \frac{f(N) + \gamma_1 N}{N} \equiv \lim_{N\to\infty} \sup \frac{f(N) + \gamma_1 N}{N} < 1,$$

*then there is a compact subset, $W$, of $\{(N, I)|0 \leq I \leq N\}$ that attracts all initial conditions under $H$ iterations. That is, there is no population explosion.*

The proof of Lemma 8 is in the Appendix.

Let

$$\mathcal{R}_0 = \frac{-\gamma_1 \alpha \phi'(0)}{1 - \gamma_2 \sigma}. \tag{6}$$

The expression $\frac{1}{1-\gamma_2\sigma}$ denotes the average death-adjusted length of the infectious period in generations; $\gamma_1$ is the proportion of surviving susceptibles who can be invaded by the disease; and, $-\alpha\phi^{'}(0)$ is the maximum rate of infection per infective [11]. Thus, $R_0$ may be viewed as the average value of the expected number of secondary cases produced by a single infected individual entering the population at the infectious-free state. $R_0$ is the basic reproduction number for Model (3).

When $\gamma = \gamma_1 = \gamma_2$, our $\mathcal{R}_0$ reduces to that of the SIS epidemic model of Castillo-Chavez and Yakubu [9–11] without disease induced mortality. Next, we prove that $\mathcal{R}_0 < 1$ implies disease extinction whereas $\mathcal{R}_0 > 1$ together with the persistence of the total population implies persistence of the disease.

**Theorem 9** *In Model* (3)*, let* $N(0) \geq I(0) > 0$.

(a) *If* $\mathcal{R}_0 < 1$, *then* $\lim_{t\to\infty} I(t) = 0$. *That is, the disease goes extinct.*
(b) *If* $\mathcal{R}_0 > 1$ *and the total population is uniformly persistent, then there exists a constant* $\eta > 0$ *such that* $\underline{\lim}_{t\to\infty} I(t) \geq \eta > 0$. *That is, the disease is uniformly persistent.*

*Proof* Since $I(0) \leq N(0)$, Lemma (2) implies that $I(t) \leq N(t)$ for all $t \in \mathbb{Z}_+$.

(a)

$$\mathcal{R}_0 = \frac{-\gamma_1\alpha\phi'(0)}{1-\gamma_2\sigma} < 1$$

is equivalent to

$$-\alpha\gamma_1\phi'(0) + \gamma_2\sigma < 1.$$

Lemma (2) gives

$$F'_N(0) = F'_{N(t)}(0) = -\alpha\gamma_1\phi'(0) + \gamma_2\sigma < 1$$

and

$$I(t+1) = F_{N(t)}(I(t)) \leq F'_{N(t)}(0)I(t).$$

Thus, the sequence $\{I(t)\}$ is dominated by the geometrically decreasing sequence $\{\left(-\alpha\gamma_1\phi'(0) + \gamma_2\sigma\right)^t I(0)\}$ and hence,

$$\lim_{t\to\infty} I(t) = 0.$$

(b) By Lemma (3), since $I(0) > 0$ we have $I(t) > 0$ for all $t \in \mathbb{Z}_+$. Lemma (2) gives

$$F'_N(0) = F'_{N(t)}(0) = -\alpha\gamma_1\phi'(0) + \gamma_2\sigma.$$

$\mathcal{R}_0 > 1$ implies $F'_{N(t)}(0) > 1$ and the unique positive fixed point $I_{N(t)}$ exists (Lemma 2). Since

$$I(t+1) = F_{N(t)}(I(t)),\ I(t+1) > I(t)$$

on the open interval $\left(0, I_{N(t)}\right)$. If $I(t) \in (I_{N(t)}, N(t))$,

$$I(t+1) \geq \min\{I_{N(t)} = N(t)I_1,\ F_{N(t)}(N(t)) = \gamma_2\sigma N(t)\}.$$

Since the total population is uniformly persistent,

$$\underline{\lim}_{t\to\infty} H_1^t(N(0), I(0)) = \underline{\lim}_{t\to\infty} N(t) \geq \eta_1 > 0.$$

This implies

$$\underline{\lim}_{t\to\infty} \min\{I_{N(t)} = N(t)I_1, F_{N(t)}(N(t)) = \gamma_2\sigma N(t)\} > 0.$$

Thus, the orbit $\{I(t)\}$ increases when it is small, and eventually gets larger and remains larger than a fixed positive number $\eta$. Hence,

$$\underline{\lim}_{t\to\infty} I(t) \geq \eta > 0.$$

□

Without disease induced mortality, it is known that $\mathcal{R}_0 > 1$ implies disease persistence in our SIS model [9–11]. With disease induced mortality, we obtain sufficient conditions that guarantee global total population extinction, where $\mathcal{R}_0 > 1$. That is, we obtain that independent of initial population size of healthy individuals, a tiny number of infectious individuals can drive the total population to extinction.

**Theorem 10** *Let $\mathcal{R}_0 > 1$, $f(0) = 0$ and $f(N) \leq f'(0)N$ for all $N > 0$. Then there is a function, $\varsigma = \varsigma(\gamma_1, \gamma_2, \phi, \alpha, \sigma, F_1) > 1$, such that if $1 < \mathcal{R}_{D_1} < \varsigma$ then the total population goes extinct under $H$ iteration whenever $I(0) > 0$.*

*Proof* Let $0 < \beta \leq 1$. Now, we investigate the ray through the origin with slope $\beta$. If a positive initial condition $(N(0), I(0))$ is on this ray then $I(0) = \beta N(0)$. To calculate the slope of the ray that contains the image of this point under $H$, we have

$$F_N(I) = F_N(\beta N) = F_1(\beta)N,$$

and

$$G_N(I) = G_N(\beta N) = f(N) + (\gamma_1(1-\beta) + \gamma_2\beta)\, N.$$

The new slope is

$$\frac{F_N(\beta N)}{G_N(\beta N)} = \frac{F_1(\beta)N}{f(N) + (\gamma_1(1-\beta) + \gamma_2\beta)\,N}.$$

Since $f(N) \le f'(0)N$ for all $N > 0$,

$$\frac{F_N(\beta N)}{G_N(\beta N)} \ge \frac{F_1(\beta)}{f'(0) + (\gamma_1(1-\beta) + \gamma_2\beta)}. \tag{7}$$

Let

$$\epsilon_1 = \frac{(\mathcal{R}_0 - 1)(1 - \gamma_1\sigma)}{2\gamma_1}.$$

Note that $\epsilon_1$ is a function of $\gamma_1, \gamma_2, \sigma, \phi$ and $\alpha$. However, it is not a function of $f$.

We have

$$\gamma_1\epsilon_1\,(1 - \gamma_2\sigma) < \left(-\alpha\gamma_1\phi'(0) - (1 - \gamma_2\sigma)\right)(1 - \gamma_1\sigma)$$

and hence,

$$1 < -\alpha\gamma_1\phi'(0) + \gamma_2\sigma - \gamma_1\epsilon_1\frac{1 - \gamma_2\sigma}{1 - \gamma_1\sigma}.$$

Since $F_1(\beta)$ is differentiable, $F_1(0) = 0$, and $F_1'(0) = -\alpha\gamma_1\phi'(0) + \gamma_2\sigma$ there is a neighborhood $U_1 = (-a, \beta_0)$ of 0, such that if $\beta \in U_1$ then

$$F_1(\beta) > \left(-\alpha\gamma_1\phi'(0) + \gamma_2\sigma - \gamma_1\epsilon_1\frac{1 - \gamma_2\sigma}{1 - \gamma_1\sigma}\right)\beta > \beta.$$

Note that $\beta_0$ is independent of $f$. Let

$$\varsigma_1 = -\alpha\gamma_1\phi'(0) + \gamma_2\sigma - \gamma_1\epsilon_1\frac{1 - \gamma_2\sigma}{1 - \gamma_1\sigma} > 1.$$

Note that the expression $\varsigma_1$ is a function of $\gamma_1, \gamma_2, \sigma, \phi$ and $\alpha$. However, it is not a function of $f$.

$$\mathcal{R}_{D_1} < \varsigma_1$$

implies

$$0 \le f'(0) < \varsigma_1\,(1 - \gamma_1)\,, \quad \text{and}$$
$$f'(0) + \varsigma_1\gamma_1 < \varsigma_1.$$

Choose $0 < \epsilon_2 < 1$ such that

$$f'(0) + \varsigma_1\gamma_1 = (1-\epsilon_2)\varsigma_1.$$

Note that $\epsilon_2$ depends on $f$.

Thus,

$$\begin{aligned} f'(0) + \gamma_1(1-\beta) + \gamma_2\beta &\le f'(0) + \gamma_1 \\ &\le f'(0) + \varsigma_1\gamma_1 \\ &= (1-\epsilon_2)\varsigma_1. \end{aligned}$$

Hence, if $\beta \in U_1$ then the new slope

$$\begin{aligned} \frac{F_N(\beta N)}{G_N(\beta N)} &> \frac{F_1(\beta)}{f'(0) + \gamma_1(1-\beta) + \gamma_2\beta} \\ &> \frac{\varsigma_1\beta}{(1-\epsilon_2)\varsigma_1} = \frac{1}{1-\epsilon_2}\beta. \end{aligned}$$

That is, if a positive initial condition is on a ray through the origin with small slope, then the image will be on a ray through the origin with a larger slope. In fact, the slope grows by a factor larger than 1. In particular, if $0 < \beta < \beta_0$ then the new slope is bigger than the original slope; and in a finite number of iterations the slope becomes larger than $\beta_0$.

Next, let the positive initial condition be on a ray through the origin with slope $\beta \ge \beta_0$. Then the new slope satisfies Inequality (7). $\mathcal{R}_{D_1} < \varsigma_1$ implies $f'(0) < \varsigma_1(1-\gamma_1)$. Hence, $f'(0) + \gamma_1(1-\beta) + \gamma_2\beta < \varsigma_1(1-\gamma_1) + \gamma_1(1-\beta) + \gamma_2\beta$. Thus,

$$\frac{F_N(\beta N)}{G_N(\beta N)} \ge \frac{F_1(\beta)}{f'(0) + (\gamma_1(1-\beta) + \gamma_2\beta)} \ge \frac{F_1(\beta)}{\varsigma_1(1-\gamma_1) + \gamma_1(1-\beta) + \gamma_2\beta}.$$

Since $F_1(\beta)$ and $\varsigma_1(1-\gamma_1) + \gamma_1(1-\beta) + \gamma_2\beta$ are continuous and positive on $[\beta_0, 1]$, the new slopes have a positive lower bound, $\beta_m > 0$, which is independent of $f$.

If the positive initial condition is on a ray through the origin with slope

$$1 \ge \beta \ge \overline{\beta} = \min\{\beta_0, \beta_m\},$$

then the new slope either grows and is larger than $\overline{\beta}$ or it decreases but must be larger than $\beta_m$. Note that $\overline{\beta}$ is independent of $f$.

We now need to show that the total population decreases when $\beta \ge \overline{\beta}$. This will be accomplished by taking $1 < \mathcal{R}_{D_1} < \varsigma_2$ for some appropriate $\varsigma_2$ that is independent of $f$.

Let

$$\epsilon_3 = \frac{\overline{\beta}\,(\gamma_1 - \gamma_2)}{2(1-\gamma_1)} > 0$$

and

$$\varsigma_2 = 1 + \epsilon_3.$$

Note that $\epsilon_3$ and $\varsigma_2$ are independent of $f$. By hypotheses

$$f(N) \leq f'(0)N.$$

Let

$$\begin{aligned} 1 < \mathcal{R}_{D_1} < \varsigma_2 &= 1 + \epsilon_3 \\ &= 1 + \frac{\overline{\beta}\,(\gamma_1 - \gamma_2)}{2(1-\gamma_1)}. \end{aligned}$$

Hence,

$$\frac{f'(0)}{1-\gamma_1} < 1 + \epsilon_3 = 1 + \frac{\overline{\beta}\,(\gamma_1 - \gamma_2)}{2(1-\gamma_1)}$$

and

$$f'(0) < (1-\gamma_1)\left(1 + \frac{\overline{\beta}\,(\gamma_1 - \gamma_2)}{2(1-\gamma_1)}\right).$$

Consequently,

$$\begin{aligned} G_N(\beta N) &= f(N) + (\gamma_1(1-\beta) + \gamma_2\beta)\,N \\ &\leq (f'(0) + \gamma_1(1-\beta) + \gamma_2\beta)N \\ &\leq \left(f'(0) + \frac{\overline{\beta}}{2}(\gamma_1 - \gamma_2) + \gamma_1(1-\beta) + \gamma_2\beta\right)N \\ &\leq \left((1-\gamma_1)\left(1 + \frac{\overline{\beta}\,(\gamma_1 - \gamma_2)}{2(1-\gamma_1)}\right) + \frac{\overline{\beta}}{2}(\gamma_1 - \gamma_2) + \gamma_1(1-\beta) + \gamma_2\beta\right)N \\ &= \left(1 + \overline{\beta}\,(\gamma_1 - \gamma_2) - \beta\,(\gamma_1 - \gamma_2)\right)N \leq N. \end{aligned}$$

Note that if $N > 0$, then $G_N(\beta N) < N$. In fact, since $f'(0) + \gamma_1(1-\beta) + \gamma_2\beta < 1$ the total population decreases by a factor less than 1. So if $I(0) > 0$, then the slopes of the iterates increase until in a finite number of iterations it is larger than $\overline{\beta}$. Then the total population decreases at a rate less than 1. Consequently, if $1 < \mathcal{R}_{D_1} < \varsigma = \min\{\varsigma_1, \varsigma_2\}$ all positive initial conditions converge to the origin and the total population is driven to extinction, where $\varsigma = \varsigma(\gamma_1, \gamma_2, \phi, \alpha, \sigma, F_1)$ and is independent of $f$. □

*Remark 11* From the proof of Theorem (10), $1 < \mathcal{R}_{D_1} < \varsigma = \min\{\varsigma_1, \varsigma_2\}$ implies $f'(0) < \varsigma_2(1-\gamma_1) = (1-\gamma_1) + \frac{\overline{\beta}(\gamma_1-\gamma_2)}{2} \leq (1-\gamma_2)$. That is, $1 < \mathcal{R}_{D_1} < \varsigma = \min\{\varsigma_1, \varsigma_2\}$ implies $\mathcal{R}_{D_2} < 1$.

*Remark 12* Since $\varsigma = \varsigma(\gamma_1, \gamma_2, \phi, \alpha, \sigma, F_1)$ in Theorem (10) is independent of $f$ whereas $\mathcal{R}_{D_1}$ is dependent on $f$, there are possible values of $f^{'}(0)$ such that $1 < \mathcal{R}_{D_1} < \varsigma = \min\{\varsigma_1, \varsigma_2\}$ (see Example 13).

In Model (3), it is known that when $\gamma_1 = \gamma_2$, there is no disease induced mortality, and $\mathcal{R}_{D_1} > 1$ implies the persistence of the infective population, where $\mathcal{R}_0 > 1$ [9–11]. Next, we use the Beverton–Holt recruitment function to illustrate a disease induced extinction of the total population in Model (3), where $\mathcal{R}_0 > 1$, $\mathcal{R}_{D_1} > 1$, $\mathcal{R}_{D_2} < 1$ and $\gamma_1 > \gamma_2$.

*Example 13* Consider Model (3) with the Beverton–Holt recruitment function

$$f(N) = \frac{aN}{1 + bN}$$

and

$$\phi\left(\frac{\alpha I}{N}\right) = e^{-\frac{\alpha I}{N}},$$

where

$$0.1 < a < 0.2, \quad b = 1, \quad \alpha = 5, \quad \gamma_1 = 0.9, \quad \gamma_2 = 0.8, \quad \text{and} \quad \sigma = 0.9.$$

In Example (13),

$$\mathcal{R}_{D_1} = \frac{a}{1 - \gamma_1} > \frac{0.1}{1 - 0.9} = 1$$

implies the persistence of the susceptible population in the absence of the disease (Lemma 6), where

$$\mathcal{R}_{D_2} = \frac{a}{1 - \gamma_2} < \frac{0.2}{1 - 0.8} = 1.$$

With our choice of parameters, the disease-free dynamics are governed by the Beverton–Holt model with survivors. That is, in the absence of the disease, the susceptible population live on a globally attracting positive fixed point. Moreover, $\mathcal{R}_0 = \frac{\alpha\gamma_1}{1-\gamma_2\sigma} = 16.071 > 1$, $f'(0) = a$, $f(0) = 0$, and $f$ is concave down so $f(N) \leq f'(0)N$ for all $N$. Hence, all of the hypotheses of Theorem (10) are satisfied and our numerical results show that $0.1 < a < 0.177$ gives the extinction of the total population predicted by the theorem (see Fig. 1).

To illustrate the region that leads to extinction in $(a, \gamma_2) - \textit{parameter space}$ of the Beverton–Holt model, we continuously vary the intrinstic growth rate $a$ between 0 and 1, and $\gamma_2$ between 0 and 0.9 where all the other parameters are kept fixed at their current values in Example (13) with $\gamma_2 < \gamma_1$. Figure 2 shows that in $(a, \gamma_2)$-space, the species goes extinct at low values of the intrinsic growth rate whenever $\mathcal{R}_{D_2} < 1$.

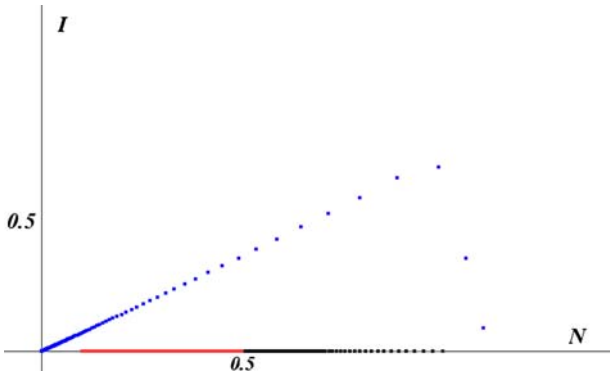


**Fig. 1** The initial condition (1.1, 0.1) converges to (0, 0), while the initial conditions (0.1, 0) and (1, 0) converge to (0.5, 0). All the parameters are exactly as in Example (13) with $a = 0.15$

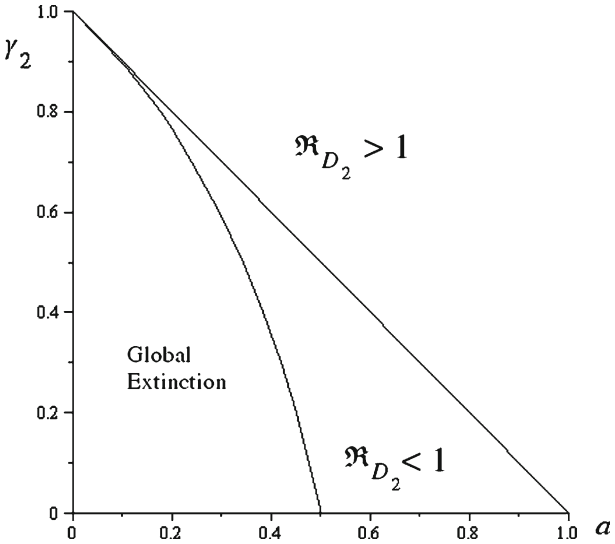


**Fig. 2** Region that leads to extinction in $(a, \gamma_2)$ parameter space of Example (13), where on the horizontal axis $0 < a < 1$ and on the vertical axis $0 < \gamma_2 < 0.9$

However, the species persists at high values of the intrinsic growth rate whenever $\mathcal{R}_{D_2} < 1$ and the survival "probability" of infective individuals, $\gamma_2$, is low.

## 5 Multiple attractors

Discrete-time SIS epidemic models with no disease induced mortality are known to exhibit single global attractors (not coexisting multiple attractors), whenever the disease-free state dynamics supports a single global attractor. Age structure, dispersal and periodic forcing are examples of biological factors that are known to generate multiple attractors in ecological and epidemiological models. Here, we demonstrate

that disease induced mortality can generate multiple attractors in simple SIS epidemic models.

Next, we provide sufficient conditions that guarantee the occurrence of more than one positive fixed point in our model.

**Theorem 14** *Let* $\overline{\lim}_{N\to\infty}\frac{f(N)+\gamma_1 N}{N} < 1$. *If* $\mathcal{R}_{D_2} > 1$ *and there are two positive numbers* $N_0 < N_1$ *with* $G_{N_0}(I_1N_0) < N_0$ *and* $G_{N_1}(I_1N_1) > N_1$, *then* $H$ *has at least two positive fixed points. That is,* $H$ *has multiple fixed points when* $G_N$ *"decreases" at low population sizes while it "increases" at high population values.*

*Proof* Let $L(N) = G_N(I_1N) - N$, a continuous function of $N$. Then $L^{'}(N) = f^{'}(N) + \gamma_1(1 - I_1) + \gamma_2 I_1 - 1$, and $L^{'}(0) = f^{'}(0) + \gamma_1(1 - I_1) + \gamma_2 I_1 - 1 > f^{'}(0) + \gamma_2 - 1$. Thus, $\mathcal{R}_{D_2} > 1$ implies $L^{'}(0) > 0$. Since $L(0) = 0$ and $L(N_0) < 0$, $L$ has a positive zero, denoted by $N_0^*$, between 0 and $N_0$. Recall that $F_N(I_1N) = I_1N$. Therefore, $H(N_0^*, I_1N_0^*) = (N_0^*, I_1N_0^*)$.

By Lemma (8), there are no population explosions under $H$ iterations, and there is an $\overline{N} > N_1$where $L(\overline{N}) < 0$. Hence, by continuity there is an $N_1^* > N_1$ where $L(N_1^*) = 0$. Again, $H(N_1^*, I_1N_1^*) = (N_1^*, I_1N_1^*)$ and we have our two positive fixed points. □

**Corollary 15** *Let* $\overline{\lim}_{N\to\infty}\frac{f(N)+\gamma_1 N}{N} < 1$. *If* $\mathcal{R}_{D_1} < 1$ *and there is* $0 < N_0$ *with* $G_{N_0}(I_1N_0) > N_0$, *then the origin is not a global attractor and* $H$ *has at least two positive fixed points.*

*Proof* As in the proof of Theorem (14), let $L(N) = G_N(I_1N) - N$, a continuous function of $N$. $L^{'}(0) = f^{'}(0) + \gamma_1(1 - I_1) + \gamma_2 I_1 - 1 < f^{'}(0) + \gamma_1 - 1$. Thus, $\mathcal{R}_{D_1} < 1$ implies $L^{'}(0) < 0$. Since $L(0) = 0$ and $L(N_0) > 0$, there is an $N_1 < N_0$ with $L(N_1) < 0$. By continuity, $L(N)$ has a positive zero, denoted by $N_0^*$, between $N_1$ and $N_0$. Hence, $H(N_0^*, I_1N_0^*) = (N_0^*, I_1N_0^*)$.

The second fixed point is produced just as in the proof of the last theorem. □

When the recruitment function is the Beverton–Holt model, then $G_N$ is a monotone function. In this case, our SIS epidemic model exhibits at most one positive fixed point. Next, we use a "simple" modified Beverton–Holt model to illustrate multiple positive fixed points (two coexisting positive attracting fixed points) in a specific SIS epidemic model with disease-induced mortality. To destroy the monotonicity in the classic Beverton–Holt model, we use a step function to force a peak in the modified model (see Fig. 3). In fisheries models, the step function could be a harvesting term. The harvesting term is proportional to the population size at small population levels. However, it is constant at high population levels.

*Example 16* Consider Model (3) with the modified Beverton–Holt recruitment function

$$f(N) = \frac{aN}{1+bN} - \frac{P}{Q}N(1 - \text{step}(N - Q)) - \text{Pstep}(N - Q),$$

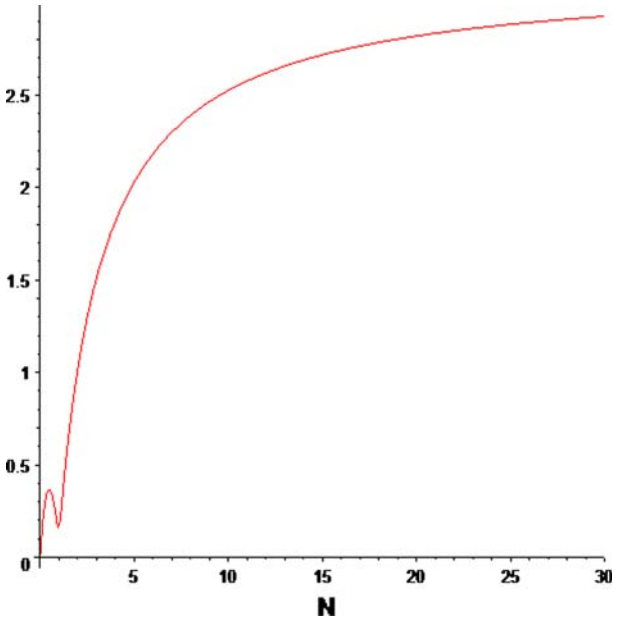


**Fig. 3** Modified Beverton–Holt map with parameters as in Example (16)

where

$$\text{step}(N-Q) = \begin{cases} 0 & \text{if } N-Q<0, \\ 1 & \text{if } N-Q\geq 0, \end{cases}$$
$$\phi\left(\frac{\alpha I}{N}\right) = e^{-\frac{\alpha I}{N}},$$

and

$$a=4, \quad b=0.75, \quad \alpha=5, \quad \gamma_1=0.9, \quad \gamma_2=0.8,$$
$$P=2.18, \quad Q=1, \quad \text{and} \quad \sigma=0.9.$$

In Example (16), the recruitment function has the basic structure of the Beverton–Holt map except for a local maximum and minimum near zero (see Fig. 3). The disease-free state has a globally attracting positive fixed point at $S_\infty = 29.2$.

Since $\lim_{N\to\infty} f(N)$ is a real number, $\overline{\lim}_{N\to\infty} \frac{f(N)+\gamma_1 N}{N} = \gamma_1 < 1$. With our choice of parameters, $\mathcal{R}_{D_2} = 9.1 > 1$ and $I_1 = 0.7586$. Set $N_0 = 1.02$ and $N_1 = 10$. Then $G_{N_0}(I_1 N_0) = 0.972 < N_0$ and $G_{N_1}(I_1 N_1) = 10.767 > N_1$. By Theorem (14), Example (16) has at least two positive fixed points.

Figure 4 shows that Example (16) exhibits two coexisting (multiple) positive attractors, a "black" and "blue" positive attracting fixed points at (0.93, 0.71) and (15.53, 11.78) respectively, coexisting with a "red" disease-free fixed point at (29.2, 0) that is globally attracting in the disease-free state.

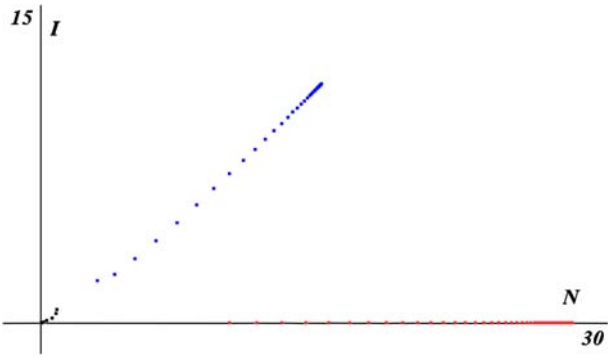


**Fig. 4** The initial condition (3, 2) converges to (15.53, 11,78), the initial condition (0.01, 0.01) converges to (0.93, 0.71) and the initial conditions (10, 0) converges to (29.2, 0). All the parameters are exactly as in Example (16)

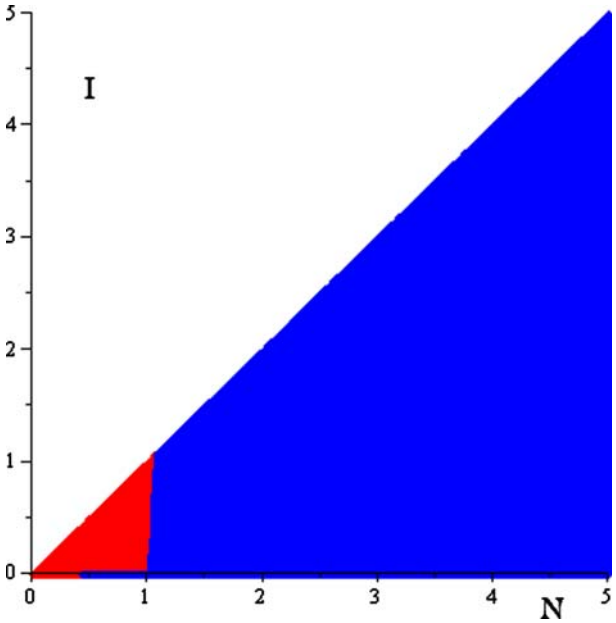


**Fig. 5** Basin of attraction of (15.53, 11.78) and basin of attraction of (0.93, 0.71), where all parameters are exactly as in Fig. 4

In Fig. 5, we illustrate the basins of attraction for the two coexisting positive fixed points in Fig. 4, where the "red" and "blue" regions are respectively subsets of the basins of attraction of the positive fixed points (0.93, 0.71) and (15.53, 11.78).

In Example (16), the origin is a repellor. Next, we demonstrate a situation where the origin is an attractor (Corollary 15). Set $P = 0.0395$ and $Q = 0.01$ and keep all the other parameters in Example (16) fixed at their current values. With our choice of parameters, $\mathcal{R}_{D_1} = 0.5 < 1$ and $I_1 = 0.7586$. Set $N_0 = 1.00$. Then $G_{N_0}(I_1 N_0) = 3.07 > N_0$. By Corollary (15), the origin is not a global attractor and the system has

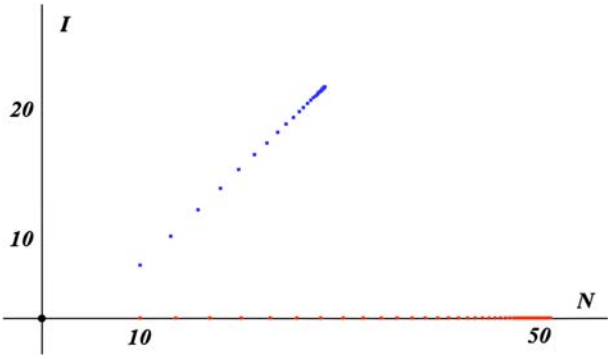


**Fig. 6** The initial condition (10, 5) converges to (28.76, 21.82), the initial condition (0.01, 0.01) converges to (0, 0) and the initial conditions (10, 0) converges to (51.59, 0). All the parameters are exactly as in Example (16), except $P = 0.0395$ and $Q = 0.01$

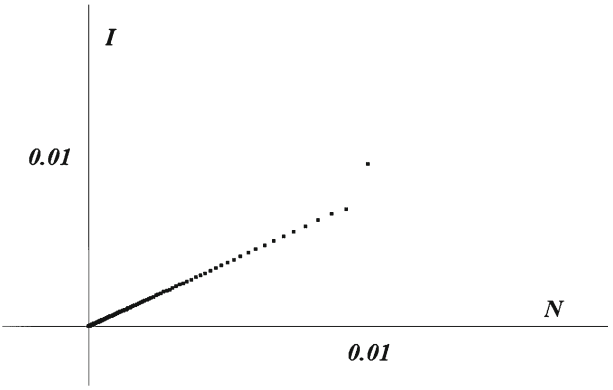


**Fig. 7** This is a zoom around the origin of Fig. 6. The initial condition (0.01, 0.01) converges to (0, 0)

at least two positive fixed points. Figures 6 and 7 show that the origin, "black", attracts an open subset of the interior, while coexisting with an attracting positive "blue" fixed point. Figure 8 shows the basin of attraction for the origin.

Our examples have highlighted some of the multiple attractors generated by our model when the disease-free dynamics are compensatory and the susceptible population (in the absence of the disease) are on a globally attracting fixed point. In these examples, the basins of the coexisting attractors are relatively simple and there is no evidence of sensitive dependence of the long-term disease dynamics on the initial population sizes. In the next section, we demonstrate multiple attractors with basin structures that show evidence of sensitive dependence on initial population sizes.

## 6 Complex disease dynamics

When the recruitment function is the Beverton–Holt model, our SIS epidemic dynamics are *compensatory* (equilibrium dynamics). However, when the recruitment function

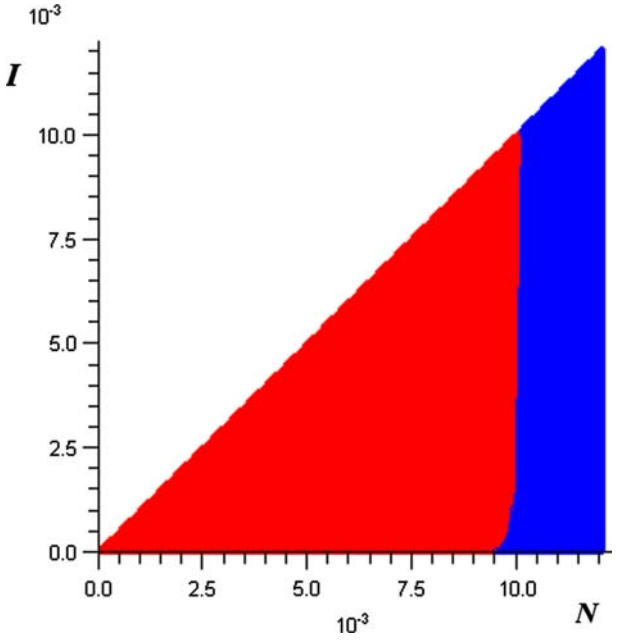


**Fig. 8** Basin of attraction of the origin where all parameters are exactly as in Fig. 6

is a one-hump map, our SIS dynamics are *overcompensatory* (oscillatory dynamics) such as periodic attractors and discrete Hopf (Neimark–Sacker) bifurcations. Our examples exhibit all these complex structures while the disease-free dynamics is a relatively simple globally attracting fixed point (compensatory dynamics). We use the Ricker map in the following example to highlight these phenomena.

*Example 17* Consider Model (3) with the Ricker recruitment function

$$f(N) = N \exp(r - N)$$

and

$$\phi\left(\frac{\alpha I}{N}\right) = e^{-\frac{\alpha I}{N}},$$

where

$$\alpha = 5, \quad \gamma_1 = 0.9, \quad \gamma_2 \in (0, 0.9), \quad r = 4 \quad \text{and} \quad \sigma = 0.9.$$

With our choice of parameters, in the absence of the disease, the susceptible population is on a globally attracting positive fixed point at

$$S_\infty = r - \ln(1 - \gamma_1) = 6.303.$$

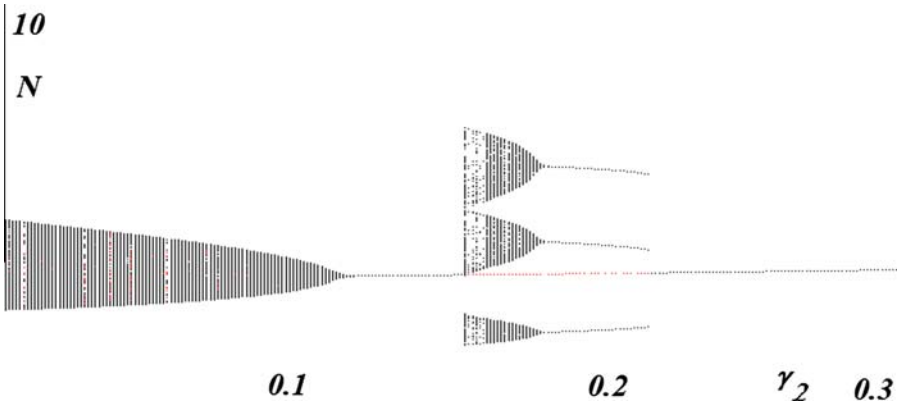


**Fig. 9** Bifurcation diagram of Example (17) where $\gamma_2$ is varied between 0 and 0.3

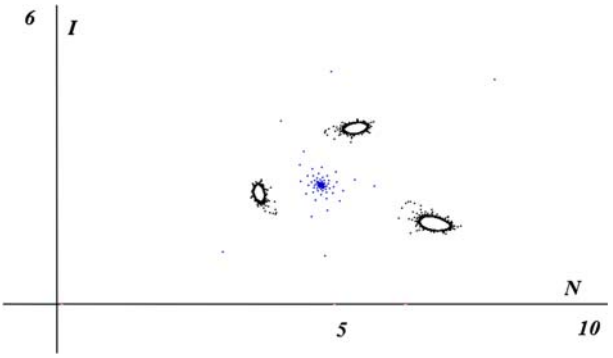


**Fig. 10** The initial condition (20, 18) converges to the fixed point (4.78, 2.37), the initial conditions (6.4, 1.4) converges to the three component limit cycle and the initial condition (0.1, 0) converges to (6.30, 0). All the parameters are exactly as in Example (17), except $\gamma_2 = 0.176$

In Fig. 9, we use the two initial population sizes, (6, 1) and (5.2, 3.6), to generate a bifurcation diagram, where $\gamma_2$ is varied between 0 and 0.3. At very low values of $\gamma_2$, Fig. 9 shows that Example (17) has a complex discrete Hopf (Neimark–Sacker) cycle attractor, while at high values of $\gamma_2$, it exhibits a stable fixed point attractor. For intermediate values of $\gamma_2$, the system has either a single stable fixed point attractor or a fixed point attractor coexisting with either a three component complex Neimark–Sacker cycle attractor or a period 3 attractor.

Figure 10 shows that at $\gamma_2 = 0.176$, Example (17) has an attracting positive "blue" fixed point at (4.78, 2.37) coexisting with a three component complex "black" Neimark–Sacker cycle attractor. The basins of attraction of these multiple attractors have fractal structures that illustrate sensitive dependence on initial conditions (see Fig. 11).

Example (17) and Figs. 9, 10, and 11 have demonstrated some of the complex, non-intuitive interactions between the recruitment function, disease induced mortality and disease dynamics.

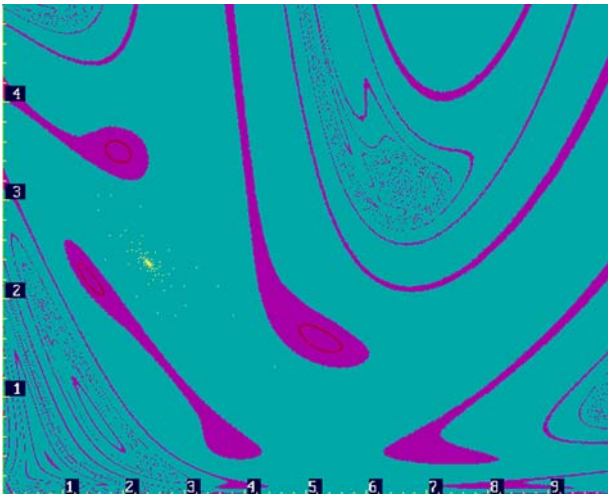


**Fig. 11** On the horizontal region, $0 \leq S \leq 10$. On the vertical axis, $0 \leq I \leq 5$. The initial condition in the $(S, I)$-plane is approaching the attracting fixed point. The three circles are the three component attracting Neimark–Sacker cycle. The large region is the basin of attraction of the cycle and the other region is that of the fixed point. All parameters are exactly as in Fig. 10

## 7 Geometric growth

When we assume that the birth or recruitment processes are governed by the geometric recruitment function

$$f(N) = \mu N,$$

then, in the absence of the disease, the susceptible (disease-free state) equation becomes

$$S(t+1) = \mu S(t) + \gamma_1 S(t) = (\mu + \gamma_1) S(t).$$

The solution of this equation is

$$S(t) = (\mu + \gamma_1)^t S(0),$$

and

$$\mathcal{R}_{D_1} = \frac{\mu}{1 - \gamma_1}.$$

If $\mathcal{R}_{D_1} < 1$, the population goes extinct at a geometric rate and if $\mathcal{R}_{D_1} > 1$, the population explodes at a geometric rate. Under geometric recruitment function, $\mathcal{R}_0 < 1$ implies the extinction of the infective population while $\mathcal{R}_{D_2} > 1$ and $\mathcal{R}_0 > 1$ implies its persistence (Theorem 9).

Next, we use proportions to gain more understanding of our epidemic model under geometric growth rate. That is, we let

$$i = \frac{I}{N}$$

and

$$s = \frac{S}{N}.$$

Consequently, $i$ denotes the fraction of the population that is infected and $s$ denotes the fraction that is susceptible. Since $i(t) + s(t) = 1$, our SIS model reduces to the single equation,

$$i(t+1) = \frac{\gamma_1 (1 - \phi(\alpha i(t)))(1 - i(t)) + \gamma_2 \sigma i(t)}{\mu + \gamma_1 + (\gamma_2 - \gamma_1) i(t)} = \frac{F_1(i(t))}{\mu + \gamma_1 + (\gamma_2 - \gamma_1) i(t)}. \tag{8}$$

The ability to reduce the geometric growth model to a single equation relies on the assumption of the frequency-dependent transmission.

The set of iterates of

$$h(i) = \frac{\gamma_1 (1 - \phi(\alpha i))(1 - i) + \gamma_2 \sigma i}{\mu + \gamma_1 + (\gamma_2 - \gamma_1) i}$$

is equivalent to the set of density sequence generated by Model (8). Equation (8), the one-dimensional $i$-equation is not capable of exhibiting two-dimensional bifurcations such as the discrete Hopf (Neimark–Sacker) bifurcations of Example (17).

Let

$$\mathcal{R}_0 = \frac{-\gamma_1 \alpha \phi'(0)}{(1 - \gamma_1)(\mathcal{R}_{D_1} - 1) + 1 - \gamma_2 \sigma}.$$

If $\mathcal{R}_{D_1} = 1$, then the demography has no impact on $\mathcal{R}_0$ and $\mathcal{R}_0 = \frac{-\gamma_1 \alpha \phi'(0)}{1 - \gamma_2 \sigma}$ as in Eq. (6). However, if $\mathcal{R}_{D_1} \neq 1$ then the demography impacts $\mathcal{R}_0$ and $\frac{1}{(1-\gamma_1)(\mathcal{R}_{D_1} - 1) + 1 - \gamma_2 \sigma}$ gives the demographic death-adjusted infectious period measured in generations. Consequently, $\mathcal{R}_0$ decreases with increasing values of $\mathcal{R}_{D_1}$.

Next, we obtain that independent of initial population sizes $\mathcal{R}_0 \leq 1$ implies that the proportion of the infected eventually decreases to zero.

**Theorem 18** *If* $\mathcal{R}_0 \leq 1$, *then* $\lim_{t \to \infty} i(t) = 0$. *That is, the proportion of the infected population decreases to* $\{0\}$.

*Proof* Recall that

$$h(i) = \frac{\gamma_1 (1 - \phi(\alpha i))(1 - i) + \gamma_2 \sigma i}{\mu + \gamma_1 + (\gamma_2 - \gamma_1) i}.$$

Since $\phi(0) = 1$, $\phi'(i) \leq 0$ and $\phi''(i) \geq 0$,

$$0 \leq 1 - \phi(\alpha i) \leq -\alpha\phi'(0)\, i.$$

Hence,

$$h(i) \leq \frac{\gamma_1\left(-\alpha\phi'(0)\right)(1-i) + \gamma_2\sigma}{\mu + \gamma_1 + (\gamma_2 - \gamma_1)i} i.$$

Let

$$M(i) = \frac{\gamma_1\left(-\alpha\phi'(0)\right)(1-i) + \gamma_2\sigma}{\mu + \gamma_1 + (\gamma_2 - \gamma_1)i}.$$

We will show that $M(i) < 1$ for all $i > 0$. This will force $\{i(t)\}_{t\geq 0}$ to be a decreasing sequence which converges to 0.

$$M(0) = \frac{-\gamma_1\alpha\phi'(0) + \gamma_2\sigma}{\mu + \gamma_1}$$

and

$$M(1) = \frac{\gamma_2\sigma}{\mu + \gamma_2} < 1.$$

Since $\mathcal{R}_0 \leq 1$,

$$\begin{aligned}
\frac{-\gamma_1\alpha\phi'(0)}{(1-\gamma_1)(\mathcal{R}_{D_1} - 1) + 1 - \gamma_2\sigma} \leq 1 &\iff \\
\frac{-\gamma_1\alpha\phi'(0)}{\mu - 1 + \gamma_1 + 1 - \gamma_2\sigma} \leq 1 &\iff \\
\frac{-\gamma_1\alpha\phi'(0) + \gamma_2\sigma}{\mu + \gamma_1} \leq 1 &\iff \\
M(0) \leq 1.&
\end{aligned}$$

We next show that $M(i)$ is monotone on $[0, 1]$.

$$M'(i) = \frac{\gamma_1\alpha\phi'(0)(\mu + \gamma_2) - \gamma_2\sigma(\gamma_2 - \gamma_1)}{(\mu + \gamma_1 + (\gamma_2 - \gamma_1)i)^2}.$$

Since the numerator is a constant and the denominator is positive, $M'(i)$ does not change signs. Hence, $M$ is monotone and takes on values between $M(0) = \frac{-\gamma_1\alpha\phi'(0)+\gamma_2\sigma}{\mu+\gamma_1}$ and $M(1) = \frac{\gamma_2\sigma}{\mu+\gamma_2}$. That is, the maximum value of $M$ on $[0, 1]$ is less than or equal to 1. Since $M(1) < 1$, $M(i) < 1$ for all $i > 0$. Consequently, $h(i) < i$ whenever $i > 0$. Therefore, the sequence $\{i(t)\}_{t\geq 0}$ decreases to zero as $t \to \infty$. □

Now, we obtain that $\mathcal{R}_0 > 1$ implies the persistence of the proportion of infected population. This result is independent of whether $\mathcal{R}_{D_2}$ is bigger than 1 or less than 1.

**Theorem 19** *If $\mathcal{R}_0 > 1$, then $\{0\}$ is an unstable fixed point of Model (8) and the proportion of infected population is uniformly persistent.*

*Proof*

$$h(i) = \frac{\gamma_1 (1 - \phi(\alpha i))(1 - i) + \gamma_2 \sigma i}{\mu + \gamma_1 + (\gamma_2 - \gamma_1) i}$$

and

$$h'(0) = \frac{-\alpha \gamma_1 \phi'(0) + \gamma_2 \sigma}{\mu + \gamma_1}.$$

Thus,

$$\mathcal{R}_0 > 1$$

is equivalent to

$$\frac{-\gamma_1 \alpha \phi'(0)}{(1 - \gamma_1)(\mathcal{R}_{D_1} - 1) + 1 - \gamma_2 \sigma} > 1$$

and

$$\frac{-\alpha \gamma_1 \phi'(0) + \gamma_2 \sigma}{\mu + \gamma_1} > 1.$$

Therefore, there is a repelling neighborhood to the right of the unstable fixed point $\{0\}$, $(0, \zeta_1)$. The continuous function $h(i)$ is positive on the compact interval $[\zeta_1, 1]$ and must obtain its positive minimum $\zeta_2$. Let

$$\zeta = \min\{\zeta_1, \zeta_2\} > 0.$$

Then

$$\underline{\lim}_{t \to \infty} i(t) \geq \zeta$$

whenever $i(0) > 0$ and the proportion of infected population is uniformly persistent. □

By Corollary (15) and Example (16), our epidemic model is capable of supporting multiple positive equilibrium points. However, under geometric growth, our (proportions) model has a globally attracting positive fixed point. To establish this, we need the following auxiliary results.

**Lemma 20** *Model* (8) *has either no critical points or a closed interval of critical points.*

The proof of Lemma 20 is in the Appendix.

*Remark 21* If $\phi' \leq 0$, then Model (8) has at most one critical point.

*Remark 22* If Model (8) has a closed non-single point interval of critical points, then $h$ is a non-decreasing function.

**Theorem 23** *If* $\mathcal{R}_0 > 1$ *and* $\phi'''$ *is non-positive, then Model* (8) *has a unique positive equilibrium.*

*Proof* The fixed point for our system is the solution of the equation

$$h(i) = \frac{\gamma_1 (1 - \phi(\alpha i))(1 - i) + \gamma_2 \sigma i}{\mu + \gamma_1 + (\gamma_2 - \gamma_1) i} = i.$$

Finding the fixed points is equivalent to finding where the two functions

$$F_1(i) = \gamma_1 (1 - \phi(\alpha i))(1 - i) + \gamma_2 \sigma i$$

and

$$P(i) = (\mu + \gamma_1 + (\gamma_2 - \gamma_1) i)\, i$$

intersect. $F_1(0) = P(0) = 0$ and $F_1(1) = \gamma_2 \sigma < \mu + \gamma_2 = P(1)$. $P(i)$ is a quadratic function with constant negative second derivative $2(\gamma_2 - \gamma_1)$.

$$F_1'(i) = -\gamma_1 (1 - \phi(\alpha i)) - \alpha \gamma_1 \phi'(\alpha i)(1 - i) + \gamma_2 \sigma.$$

$$F_1''(i) = 2\alpha \gamma_1 \phi'(\alpha i) - \alpha^2 \gamma_1 \phi''(\alpha i)(1 - i).$$

$$F_1'''(i) = 3\alpha^2 \gamma_1 \phi''(\alpha i) - \alpha^3 \gamma_1 \phi'''(\alpha i)(1 - i).$$

Since $\phi' \leq 0, \phi'' \geq 0$ and $\phi''' \leq 0$, we have $F_1'' \leq 0$ and $F_1''' \geq 0$ on $[0, 1]$. $F_1'(0) = -\alpha \gamma_1 \phi'(0) + \gamma_2 \sigma$ and $P'(0) = \mu + \gamma_1$. Since

$$\mathcal{R}_0 > 1$$

is equivalent to

$$\frac{-\alpha \gamma_1 \phi'(0) + \gamma_2 \sigma}{\mu + \gamma_1} > 1.$$

That is,

$$\mathcal{R}_0 > 1$$

is equivalent to

$$F_1'(0) > P'(0).$$

Thus, there must be at least one more intersection which must be positive. Let $i_\infty$ be the smallest positive point of intersection. Since $F_1(i) > P(i)$ on $(0, i_\infty)$, $F_1'(i_\infty) \leq P'(i_\infty)$. However, there exists a point $i_c \in (0, i_\infty)$ where $F_1'(i_c) < P'(i_c)$. If $F_1'(i_\infty) = P'(i_\infty)$, then $F_1'$ has decreased less than $P'$ on $(i_c, i_\infty)$. Hence, $F_1'' > P''$ at some point in $(i_c, i_\infty)$. Since $F_1''' \geq 0$ and $P''' = 0$, once $F_1'' > P''$ is true it must stay true. Thus $F_1'' > P''$ would be true on $(i_\infty, 1]$. So with $F_1'(i_\infty) = P'(i_\infty)$, we have $F_1 > P$ on $(i_\infty, 1]$ which contradicts $F_1(1) < P(1)$. Thus,

$$F_1'(i_\infty) < P'(i_\infty). \tag{9}$$

Suppose there is a second point of intersection, $i_{2\infty}$. Since $F_1(i) < P(i)$ on $(i_\infty, i_{2\infty})$, we have $F_1'(i_{2\infty}) \geq P'(i_{2\infty})$. Thus, $F_1'$ has decreased less than $P'$ on $(i_\infty, i_{2\infty})$. Hence, $F_1'' > P''$ at some point in $(i_\infty, i_{2\infty})$. Since $F_1''' \geq 0$ and $P''' = 0$, once $F_1'' > P''$ is true it must stay true. Thus, $F_1'' > P''$ would be true on $(i_{2\infty}, 1]$. So with $F_1'(i_\infty) \geq P'(i_\infty)$, we have $F_1 > P$ on $(i_{2\infty}, 1]$ which contradicts $F_1(1) < P(1)$. Therefore, there can be no second point of intersection and Model (8) has a unique positive equilibrium. □

**Lemma 24** *Let the linear rational function*

$$L(i) = \frac{ai + b}{ci + d},$$

*where $a, b, c$ and $d$ are constants. Then*

1. *The composition map $L(L(i))$ is a linear rational function.*
2. *If $L$ has a point of prime period 2, then $L$ has a self inverse.*
3. *If*

$$L[0, 1] \subset (0, 1]$$

*is a decreasing rational function, then $L(L(i)) > i$ for every $i \in [0, i_\infty)$.*

The proof of Lemma (24) is in the Appendix.

Now, we state a definition of *envelopes on compact intervals* that is similar to the one used by Cull [12].

**Definition 25** Let

$$F : [0, 1] \to [0, 1]$$

have a unique critical point, $i_c$, and a unique positive fixed point, $i_\infty$, where $0 < i_c < i_\infty < 1$. Also, let $\{0\}$ be an unstable fixed point of $F$. A function

$$E : [0, 1] \to [0, 1]$$

envelopes the function $F$ if and only if

$$E(i) \geq F(i) \text{ on } [0, i_\infty] \text{ and}$$
$$E(i) \leq F(i) \text{ on } [i_\infty, 1].$$

A slight modification to a corollary of Theorem 1 of Cull in [12] gives the following result.

**Theorem 26** *If $E$ envelopes $F$ on $[0, 1]$ and $E(E(i)) > i$ for all $i \in [i_c, i_\infty)$, then $i_\infty$ is a globally asymptotically stable positive fixed point of $F$ on $(0, 1]$.*

The proof of Theorem (26) is omitted. Next, we use Theorem (26) to show that in stark contrast to the epidemic model under either the modified Beverton–Holt or the Ricker recruitment functions, the model under geometric growth does not exhibit multiple attractors.

**Theorem 27** *If $\mathcal{R}_0 > 1$ and $\phi'''$ is non-positive, then Model (8) has a unique positive globally asymptotically stable equilibrium. That is, independent of positive initial population sizes, the proportion of the infected population approaches a unique positive equilibrium point.*

*Proof* By Theorem (23), Model (8),

$$h(i) = \frac{\gamma_1 (1 - \phi(\alpha i))(1 - i) + \gamma_2 \sigma i}{\mu + \gamma_1 + (\gamma_2 - \gamma_1) i},$$

has a unique positive fixed point, $i_\infty$. By Theorem (19), $\{0\}$ is an unstable fixed point of $h$. If $h$ is a non-decreasing function, then $i < h(i) \leq i_\infty$ for all $i \in (0, i_\infty)$ and $i_\infty \leq h(i) < i$ for all $i \in (i_\infty, 1]$. Thus, every positive initial condition generates a monotone sequence that converges to the positive unique fixed point, $i_\infty$. Hence, $i_\infty$ is globally asymptotically stable in $(0, 1]$.

Next, we consider the case when $h$ is not a non-decreasing function. That is, $h$ has critical points. By Lemma (20) and Remark (22), $h$ has a unique critical point, $i_c$. If $i_\infty \leq i_c$, then $i < h(i) \leq i_\infty$ for all $i \in (0, i_\infty)$. Consequently, every point in $(0, i_\infty)$ converges monotonically to $i_\infty$. Furthermore, $h(i) < i$ for all $i \in (i_\infty, 1]$. Thus, every positive initial condition either generates a monotone sequence that converges to the positive unique fixed point or an iterate of the point gets mapped below $i_\infty$ and then monotonically increases to $i_\infty$. Hence, $i_\infty$ is globally asymptotically stable in $(0, 1]$.

Now, we consider the situation when $i_\infty > i_c$. Let

$$h_\infty(i) = \frac{\gamma_1 (1 - \phi(\alpha i_\infty))(1 - i) + \gamma_2 \sigma i}{\mu + \gamma_1 + (\gamma_2 - \gamma_1) i}.$$

Notice that $h_\infty(i)$ is a linear rational function with vertical asymptote larger than 1. Also, $0 \leq h_\infty(0) = \frac{\gamma_1(1-\phi(\alpha i_\infty))}{\mu+\gamma_1} < 1$ and $0 < h_\infty(1) = \frac{\gamma_2 \sigma}{\mu+\gamma_2} < 1$. Thus, $h_\infty([0, 1]) \subset [0, 1]$. Since $\phi$ is a non-increasing function, $h_\infty(i) \geq h(i)$ whenever $i \in [0, i_\infty]$ and $h_\infty(i) \leq h(i)$ whenever $i \in [i_\infty, 1]$. By Definition (25), $h_\infty$

envelopes $h$ on $[0, 1]$. Notice that $h_\infty(i_c) \geq h(i_c) > h(i_\infty) = h_\infty(i_\infty)$. Hence, $h_\infty$ is a decreasing function on $[0, 1]$ and $h_\infty([0, 1]) \subset (0, 1]$.

Using Lemma (24) and the decreasing nature of $h_\infty$ we obtain that $h_\infty(h_\infty(i)) > i$ for all $i \in (0, i_\infty)$. Now, apply Theorem (26), to obtain that $i_\infty$ is globally asymptotically stable in $(0, 1]$. □

## 8 Conclusion

We analyzed a discrete-time deterministic SIS epidemic model in a dynamic population. Our model allowed the population dynamics and disease transmission to be fairly general. Within this framework, we highlighted the role of disease-induced mortality, and the complexity of the interaction between infectives and susceptibles in discrete-time models.

Our results show that disease-induced death can force the extinction of the population with $\mathcal{R}_0 > 1$, where the population persists without disease-induced death. In [6,7], Berezovsky et al. used a continuous-time SI model with disease-induced mortality to show that for some initial population sizes, a tiny number of infectious individuals can drive an otherwise persistent population to extinction. We obtain a similar result for arbitrary initial population sizes. Furthermore, our model illustrates that disease-induced mortality can generate multiple stable equilibria whereas the corresponding model without disease-induced mortality has only one stable equilibrium point [4,14].

The emergence of fractal basin boundaries and the associated sensitive dependence on initial population sizes in simple deterministic epidemic models indicate the need for further investigations in our deterministic model and corresponding stochastic models, both theoretical and experimental, on the impact of disease-induced mortality on the persistence and control of infectious disease dynamics. Are our results on extinction and persistence different in corresponding stochastic SIS epidemic models? The important assumptions of our model are: discrete-time; deterministic dynamics of both the host population and the spread of the disease; the disease increases mortality but does not affect fecundity; no vertical transmission or acquired immunity; and frequency-dependent transmission. What are the consequences of these assumptions on corresponding stochastic models?

In the absence of disease-induced mortality, Castillo-Chavez and Yakubu obtained that disease-free dynamics drives the disease dynamics in deterministic SIS epidemic models [9–11]. That is, when the dynamics of the susceptible population in the absence of the disease are cyclic and non-chaotic, then the disease dynamics are cyclic and non-chaotic. Similarly, when the dynamics of the susceptible population in the absence of the disease are chaotic, then the disease dynamics are chaotic. In the current paper, we show that when the recruitment function is not the geometric growth rate then it is possible for the infective population to exhibit multiple attractors with complicated basin structures while the susceptible population in the absence of the disease are on a globally attracting fixed point (compensatory disease-free dynamics [27]). That is, in epidemic models with disease-induced mortality the disease-free dynamics do not drive the disease dynamics.

**Acknowledgments** We thank the referees for useful comments and suggestions that improved our manuscript. Abdul-Aziz Yakubu was partially supported by funds from NOAA-NEFSC in Woods Hole, MA.

## Appendix

*Proof of Lemma 1* Since $\gamma_2 < \gamma_1$,

$$\begin{aligned} f(N(t)) + \gamma_2 N(t) &= f(N(t)) + \gamma_2(S(t) + I(t)) \le f(N(t)) + \gamma_1 S(t) + \gamma_2 I(t) \\ &= N(t+1) \le f(N(t)) + \gamma_1(S(t) + I(t)) = f(N(t)) + \gamma_1 N(t). \end{aligned}$$

□

*Proof of Lemma 2* (a) Since

$$\begin{aligned} F_N(I) &= \gamma_1 \left(1 - \phi\left(\alpha \frac{I}{N}\right)\right)(N - I) + \gamma_2 \sigma I, \\ F_N(I) &= \gamma_1 \left(1 - \phi\left(\alpha \frac{I}{N}\right)\right)(N - I) + \gamma_2 \sigma I \\ &\le \gamma_1(N - I) + \gamma_2 I \le N \max\{\gamma_1, \gamma_2\} \le N. \end{aligned}$$

Since

$$\begin{aligned} G_N(I) &= f(N) + \gamma_1(N - I) + \gamma_2 I \\ G_N(I) - F_N(I) &= f(N) + \gamma_1 \phi\left(\alpha \frac{I}{N}\right)(N - I) + \gamma_2(1 - \sigma) I \ge 0. \end{aligned}$$

Hence,

$$F_N(I) \le \min\{N, G_N(I)\}.$$

It is easy to check that the equality holds if and only if $(N, I) = (0, 0)$.

(b)

$$\begin{aligned} F_N'(I) &= -\frac{\alpha\gamma_1}{N}\phi'\left(\alpha \frac{I}{N}\right)(N - I) - \gamma_1 \left(1 - \phi\left(\alpha \frac{I}{N}\right)\right) + \gamma_2 \sigma. \\ F_N'(0) &= -\frac{\alpha\gamma_1}{N}\phi'(0)(N - 0) - \gamma_1(1 - \phi(0)) + \gamma_2 \sigma \\ &= -\alpha\gamma_1 \phi'(0) + \gamma_2 \sigma. \\ F_N'(N) &= -\frac{\alpha\gamma_1}{N}\phi'\left(\alpha \frac{N}{N}\right)(N - N) - \gamma_1 \left(1 - \phi\left(\alpha \frac{N}{N}\right)\right) + \gamma_2 \sigma \\ &= -\gamma_1(1 - \phi(\alpha)) + \gamma_2 \sigma > -\gamma_1 > -1. \end{aligned}$$

(c)

$$F_N''(I) = -\left(\frac{\alpha}{N}\right)^2 \gamma_1 \phi''\left(\alpha \frac{I}{N}\right)(N - I) + 2\frac{\alpha\gamma_1}{N}\phi'\left(\alpha \frac{I}{N}\right).$$

Since $\phi' \leq 0$ and $\phi'' \geq 0$ on $[0, \infty)$, we have

$$F_N''(I) \leq 0 \text{ on } [0, N].$$

(d) $F_N(0) = 0$ implies that $y = F_N'(0)I$ is the tangent line to the graph of $F_N(I)$ at 0. Since $F_N$ is concave down on $[0, N]$, its graph is below the tangent line at the origin on $[0, N]$. Hence,

$$F_N(I) \leq F_N'(0)I \text{ on } [0, N].$$

(e) $F_N(N) = \gamma_2 \sigma N < N$. Since $F_N'(0) > 1$, the graph of $F_N(I)$ starts out higher than the diagonal and must cross it before $I = N$. The concavity property of $F_N(I)$ (see (c)) implies that there is a unique positive fixed point.

(f) Let $\Psi_N(I) = \frac{I}{N}$.
$F_1(I) = \gamma_1 (1 - \phi(\alpha I))(1 - I) + \gamma_2 \sigma I$. Thus,

$$\begin{aligned} F_1(\Psi_N(I)) &= \gamma_1 \left(1 - \phi\left(\alpha \frac{I}{N}\right)\right)\left(1 - \frac{I}{N}\right) + \gamma_2 \sigma \frac{I}{N} \\ &= \frac{1}{N} F_N(I) = \Psi_N(F_N(I)). \end{aligned}$$

(g) Since

$$F_{N_0}'(0) = \left(-\alpha \gamma_1 \phi'(0) + \gamma_2 \sigma\right) > 1,$$

$I_{N_0}$ exists with $F_{N_0}(I_{N_0}) = I_{N_0}$. Thus,

$$\Psi_{N_0}\left(F_{N_0}(I_{N_0})\right) = \Psi_{N_0}\left(I_{N_0}\right) = F_1\left(\Psi_{N_0}\left(I_{N_0}\right)\right).$$

That is. $\Psi_{N_0}\left(I_{N_0}\right) = I_1$, the unique positive fixed point of $F_1$ and $I_{N_0} = N_0 I_1$. Similarly, $I_{N_1} = N_1 I_1$. Hence, $N_0 < N_1$ implies $I_{N_0} < I_{N_1}$. In general, the fixed point for $F_N$ is $N I_1$.

(h) Topological conjugacy preserves critical points. The result follows from $(f)$.

(i) Let $N_0 < N_1$ and $I \in (0, N_0]$.The topological conjugacy in Part $(f)$ shows that

$$F_{N_0}(I) = N_0 F_1\left(\frac{I}{N_0}\right)$$

and

$$F_{N_1}(I) = N_1 F_1\left(\frac{I}{N_1}\right).$$

Note that $\frac{I}{N_1} < \frac{I}{N_0}$. Since the graph of $F_1$ goes through the origin with positive slope and is concave down, the ray through the origin and $\left(\frac{I}{N_1}, F_1\left(\frac{I}{N_1}\right)\right)$ has a

larger slope than the ray through the origin and $\left(\frac{I}{N_0}, F_1\left(\frac{I}{N_0}\right)\right)$. The first ray contains the point $\left(I, N_1 F_1\left(\frac{I}{N_1}\right)\right)$, while the second ray contains $\left(I, N_0 F_1\left(\frac{I}{N_0}\right)\right)$. Hence, $F_{N_1}(I) = N_1 F_1\left(\frac{I}{N_1}\right) < N_0 F_1\left(\frac{I}{N_0}\right) = F_{N_0}(I)$. □

*Proof of Lemma 3* (a) $I(t+1) = \gamma_1\left(1-\phi\left(\alpha\frac{I(t)}{N(t)}\right)\right)(N(t)-I(t)) + \gamma_2\sigma I(t)$. By Lemma (2*a*), $N(t)-I(t) \geq 0\,\forall t \in \mathbb{Z}_+$. Therefore, $\gamma_1\left(1-\phi\left(\alpha\frac{I(t)}{N(t)}\right)\right)(N(t)-I(t)) \geq 0$. $I(0) > 0$ implies $\gamma_2\sigma I(0) > 0$ and hence, $I(1) > 0$. By induction, $I(t) > 0$ and $\gamma_2\sigma I(t) > 0$. Hence, $I(t+1) > 0$.

(b) Use $N(t+1) = f(N(t)) + \gamma_1(N(t) - I(t)) + \gamma_2 I(t)$ and proceed as in part (a). □

*Proof of Lemma 6* $\mathcal{R}_{D_1} > 1$ implies $\{0\}$ is a repelling fixed point. Since $\gamma_1 S$ is an increasing function, the persistence of the susceptible population governed by Eq. (4) is immediate.

Since $\gamma_2 < \gamma_1$ implies $0 \leq \mathcal{R}_{D_2} < \mathcal{R}_{D_1}$. $\mathcal{R}_{D_2} < 1$ whenever $\mathcal{R}_{D_1} < 1$. Hence, $\{0\}$ is a locally asymptotically stable fixed point of the functions $D_1(N) = f(N) + \gamma_1 N$ and $D_2(N) = f(N) + \gamma_2 N$ whenever $\mathcal{R}_{D_1} < 1$. By Lemma (1), small positive initial values of $N$ lead to the extinction of the total population. $N = S + I$ implies the extinction of the susceptible and infected populations at positive small initial values of $S$ and $I$. □

*Proof of Lemma 7* First, we consider the case $f(0) > 0$. Since $\lim_{N\to\infty} f(N) + \gamma_2 N = \infty$, $D_2(N) = f(N) + \gamma_2 N$ has a positive minimum on $[0, \infty)$. Lemma (1) gives $D_2(N) \leq H_1(N, I)$. Thus, $H_1^t(N, I)$ is larger than this positive minimum for every $t > 0$ and the total population is uniformly persistent.

Now, we consider the case $f(0) = 0$ and $\mathcal{R}_{D_2} > 1$. In this case, $f'(0) > 1 - \gamma_2$ and there is a $\kappa > 0$ such that $N \in (0, \kappa)$ implies that

$$N < D_2(N) \leq H_1(N, I).$$

If $N > \frac{\kappa}{\gamma_2}$, then $\kappa < D_2(N) \leq H_1(N, I)$. Let

$$A_\kappa = \left\{(N, I) | 0 \leq I \leq N, \kappa \leq N \leq \frac{\kappa}{\gamma_2}\right\}.$$

$H_1$ is positive on the compact set $A_\kappa$, and it has a minimum $\overline{\kappa} > 0$ on $A_\kappa$. Consequently,

$$\underline{\lim_{t\to\infty}} H_1^t(N, I) \geq \min\{\kappa, \overline{\kappa}\} = \eta > 0$$

and the total population is uniformly persistent. □

*Proof of Lemma 8* Let

$$\delta = \overline{\lim_{N\to\infty}} \frac{f(N) + \gamma_1 N}{N} < 1.$$

There is an $\overline{N} > 0$ such that $N \geq \overline{N}$ implies

$$\frac{f(N) + \gamma_1 N}{N} = \frac{D_1(N)}{N} < \delta + \frac{1-\delta}{2} = \frac{1+\delta}{2} < 1.$$

Lemma (1) gives $H_1(N, I) \leq D_1(N)$. Thus, if $N \geq \overline{N}$, then $H_1(N, I) < \frac{1+\delta}{2} N$. Let

$$B_{\overline{N}} = \{(N, I) | 0 \leq I \leq N \leq \overline{N}\}.$$

$H_1$ is continuous on the compact set $B_{\overline{N}}$, and hence has a maximum $\overline{\eta} > 0$ on $B_{\overline{N}}$. As a result, the region

$$W = \{(N, I) | 0 \leq I \leq N \leq \max\{\overline{N}, \overline{\eta}\}\},$$

a compact subset of $\{(N, I) | 0 \leq I \leq N\}$, attracts all initial conditions under $H$ iterations. Hence, all orbits are bounded. □

*Proof of Lemma 20* Recall that

$$h(i) = \frac{\gamma_1 (1 - \phi(\alpha i))(1 - i) + \gamma_2 \sigma i}{\mu + \gamma_1 + (\gamma_2 - \gamma_1) i},$$

and

$$h'(i) = \frac{H(i)}{(\mu + \gamma_1 + (\gamma_2 - \gamma_1) i)^2},$$

where

$$F_1(i) = \gamma_1 (1 - \phi(\alpha i))(1 - i) + \gamma_2 \sigma i,$$

and

$$H(i) = F_1'(i)(\mu + \gamma_1 + (\gamma_2 - \gamma_1) i) - (\gamma_2 - \gamma_1) F_1(i).$$

Then

$$\begin{aligned} H'(i) &= F_1''(i)(\mu + \gamma_1 + (\gamma_2 - \gamma_1) i) \\ &= 2\alpha\gamma_1 (\mu + \gamma_1 + (\gamma_2 - \gamma_1) i) \phi'(\alpha i) \\ &\quad -\alpha^2 \gamma_1 (1 - i)(\mu + \gamma_1 + (\gamma_2 - \gamma_1) i) \phi''(\alpha i). \end{aligned}$$

Next, we show that $H'$ is a non-positive function. Recall that $\phi' \leq 0$ and $\phi'' \geq 0$. Notice that $2\alpha\gamma_1 (\mu + \gamma_1 + (\gamma_2 - \gamma_1) i) \geq 2\alpha\gamma_1 (\mu + \gamma_2) > 0$ on $[0, 1]$. Hence, the first term of $H'$ is a non-positive function. Since, $\alpha^2 \gamma_1 (1 - i)(\mu + \gamma_1 + (\gamma_2 - \gamma_1) i) \geq 0$ on $[0, 1]$, we have that both terms of $H'$ are non-positive functions. Therefore, $H'$

is a non-positive function. If $H'(i) = 0$, let $i_c^*$ be the first critical point of $H$. Then $\phi'\left(\alpha i_c^*\right) = 0$. Since $\phi' \leq 0$ and $\phi'' \geq 0$, $\phi'(\alpha i) = 0$ for all $i \geq i_c^*$. Hence, $H$ is a constant on $[i_c^*, 1]$.

If $H(1) > 0$, then $H(i) > 0$ for all $i \in [0, 1]$. In this case, $h$ is an increasing function with no critical points.

If $H(1) < 0$, then $H$ has a unique zero, $i_c$. So $h'$ has $i_c$ as a unique zero, the unique critical point of $h$.

Now, we consider the last case, $H(1) = 0$. In this case, $h'$ is positive on $[0, i_c^*)$ and zero on $[i_c^*, 1]$. Thus, $h$ is increasing on $[0, i_c^*)$ and constant on $[i_c^*, 1]$. Its set of critical points is the closed interval $[i_c, 1] = [i_c^*, 1]$. □

*Proof of Lemma 24* The proofs of (1) and (2) are straightforward and are omitted. Now, we prove (3).

Since $L$ is decreasing and $L([0, 1]) \subset (0, 1]$, $L$ has a unique fixed point $i_\infty \in (0, 1]$ and the vertical asymptote of $L$ is outside of $[0, 1]$. Hence, $i < i_\infty \leq L(i)$ and $L^2(i) \leq i_\infty$ for all $i \in [0, i_\infty)$. The zeroes of the continuous function $L^2(i) - i$ are the period 2 points of $L$. $L^2(0) > 0$. That is, $L^2(i) - i > 0$ when $i = 0$. Hence, $\{0\}$ is not a point of period 2. By part (2), $L$ has no points of prime period 2. Hence, $L^2(i) - i$ has no zeroes in $[0, i_\infty)$. Thus, $L^2(i) - i > 0$ for all $i \in [0, i_\infty)$.

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