Mathematical Biosciences and Engineering

https://www.aimspress.com/journal/mbe

MBE, 23(3): 702–721.
DOI: 10.3934/mbe.2026027
Received: 05 September 2025
Revised: 20 October 2025
Accepted: 03 November 2025
Published: 29 January 2026

Research article

On the global stability of the discrete-time epidemic models: A new approach

Omaima Slimani[1], **Bouchra Aylaj**[1] **and Saber Elaydi**[2,*]

[1] Department of Mathematics and Computer Science, Faculty of Sciences Ain Chock, Hassan II University of Casablanca, Casablanca, Morocco

[2] Department of Mathematics, Trinity University, San Antonio, USA

* **Correspondence:** Email: selaydi@trinity.edu.

Abstract: We developed a unified analytical framework for the global dynamics of discrete-time susceptible infectious susceptible (SIS) epidemic models with nonlinear recruitment. Emphasis was placed on demographic feedback through Beverton-Holt and Ricker-type recruitment, which regulates host population size and thereby shapes transmission and long-term persistence (Persistence allows population densities to approach zero asymptotically, wheras uniform persistence requires them to remain bounded away from zero). Under minimal assumptions, we reduced non-autonomous systems to appropriately defined autonomous limiting systems and used this reduction to obtain a complete global threshold characterization: When the basic reproduction number $R_0 > 1$, the endemic equilibrium existed and was globally asymptotically stable; when $R_0 \leq 1$, solutions converged to the disease-free state. The approach extended to periodically forced SIS models, which showed that the threshold and stability conclusions persisted in the periodic non-autonomous setting. The results unified and strengthened prior work and clarify how recruitment dynamics govern persistence in discrete-time epidemic systems.

Keywords: discrete-time susceptible infectious susceptible epidemic models; non-autonomous difference equations; periodic susceptible infectious susceptible model; Beverton-Holt; Ricker; endemic equilibrium; disease-free equilibrium, net reproduction number; endemic Equilibrium; global asymptotic stability

1. Introduction

Mathematical modeling has played a fundamental role in understanding the dynamics of infectious diseases. The pioneering work of Kermack and McKendrick [1] introduced a deterministic compartmental framework that has since evolved into a cornerstone of mathematical epidemiology.

While continuous-time models remain dominant in theoretical studies, discrete-time models are particularly suited for applications involving data collected in discrete intervals, such as weekly case counts or seasonal variations and they offer increased flexibility in modeling periodic and non-autonomous effects.

Among these, the discrete-time susceptible infectious susceptible (SIS) model is of particular interest for diseases where recovered individuals return immediately to the susceptible class [2], such as gonorrhea [3, 4], hospital-acquired infections (HAIs) [5], and certain vector-borne diseases [6, 7]. Despite its apparent simplicity, the discrete SIS model becomes mathematically intricate when realistic demographic processes such as nonlinear recruitment are introduced. Traditional analyses often sidestep these complications by assuming constant or geometrically growing populations. However, in many biological contexts, recruitment is regulated by density-dependent factors such as those modeled by the Beverton and Holt [8] or Ricker [9] functions.

Incorporating such nonlinear recruitment poses significant challenges, particularly in establishing the global dynamics of the system. Although several previous works have addressed local stability and persistence [10–12], the global stability of endemic equilibrium (EE) in discrete-time models has remained unresolved in most settings, especially when the basic reproduction number is $R_0 > 1$. This paper fills that gap by establishing general and verifiable conditions under which the EE is globally asymptotically stable (GAS) in both autonomous and periodic discrete SIS models.

Our key innovation lies in a reduction technique that allows us to analyze a non-autonomous epidemic model by linking it to a suitably defined autonomous limiting system [13, 14]. This approach not only simplifies the mathematical analysis, but also yields powerful global results that have previously been accessible only in restricted cases. In particular, we prove that:

- If $R_0 \leq 1$, the disease-free equilibrium (DFE) is GAS.
- If $R_0 > 1$, the EE exists and is GAS.

These global results are obtained without relying on monotonicity or Lyapunov techniques that often require strong assumptions or restrictive functional forms. Furthermore, we apply this framework to both Beverton–Holt and Ricker recruitment structures and demonstrate that the conclusions remain valid even in periodically varying environments.

In doing so, this paper advances the theory of discrete-time epidemiological modeling by offering a comprehensive and general methodology to address a long-standing open problem. Our findings not only unify previous efforts, but also open the door to a broader class of models, including those with time-dependent interventions or seasonally varying parameters [15–18].

The paper is structured as follows. Section 2 introduces the general framework of discrete-time epidemic system and key preliminaries. Section 3 presents the discrete-time SIS epidemic model. Section 4 analyzes the global stability properties under Beverton-Holt recruitment. Section 5 investigates the analogous case with Ricker-type recruitment. Section 6 extends the results to periodic systems. The extension of our technique to susceptible infected recovered (SIR) or susceptible exposed infectious recovered (SEIR) models is still an open problem.

2. Mathematical framework for discrete-time epidemic models

Following the general framework of Elaydi and Cushing [13], we consider discrete-time compartmental infectious disease models of the form

$$\mathbf{y}(t+1) = \mathbf{g}(\mathbf{y}(t)), \quad t \in \mathbb{Z}_+, \tag{2.1}$$

where

$$\mathbf{y}(t) = (y_1(t), y_2(t), \ldots, y_n(t))^T \in \mathbb{R}^n_+.$$

We make the following assumption:

Assumption 2.1 (Smoothness and Positivity)**.** *The function* $\mathbf{g}$ *is continuously differentiable on an open set* $\Omega \subset \mathbb{R}^n$ *containing* $\mathbb{R}^n_+$*. Moreover,* $\mathbf{g}$ *maps*

$$\mathbf{g} : \mathbb{R}^n_+ \to \mathbb{R}^n_+, \quad \mathbf{g} : \text{int}(\mathbb{R}^n_+) \to \text{int}(\mathbb{R}^n_+),$$

where $\text{int}(\mathbb{R}^n_+)$ *denotes the* interior *of* $\mathbb{R}^n_+$ *(all vectors with strictly positive components).*

We are interested in equilibrium solutions $\mathbf{y}^*$ satisfying

$$\mathbf{y}^* = \mathbf{g}(\mathbf{y}^*). \tag{2.2}$$

We decompose the population into infectious and non-infectious classes:

$$\mathbf{y} = (\mathbf{y}_0, \mathbf{y}_1)^T,$$

where $\mathbf{y}_0 = (y_1, \ldots, y_m)^T$ denotes the m infectious states, and $\mathbf{y}_1 = (y_{m+1}, \ldots, y_n)^T$ denotes the $n - m$ non-infectious states. Then system (2.1) can be written as

$$\begin{aligned} \mathbf{y}_0(t+1) &= \mathbf{g}_0(\mathbf{y}_0(t), \mathbf{y}_1(t)), \\ \mathbf{y}_1(t+1) &= \mathbf{g}_1(\mathbf{y}_0(t), \mathbf{y}_1(t)). \end{aligned} \tag{2.3}$$

By Assumption 2.1, both $\mathbf{g}_0$ and $\mathbf{g}_1$ are continuously differentiable and map the positive orthant to itself:

$$\mathbf{g}_0 : \text{int}(\mathbb{R}^m_+) \to \text{int}(\mathbb{R}^m_+), \quad \mathbf{g}_1 : \text{int}(\mathbb{R}^{n-m}_+) \to \text{int}(\mathbb{R}^{n-m}_+).$$

We assume that the only source of infection comes from contacts between infectious and non-infectious individuals. Thus, if no infectious individuals are present at time t, then none will be present at time $t + 1$:

$$\mathbf{g}_0(\mathbf{0}, \mathbf{y}_1) \equiv \mathbf{0}, \quad \forall\, \mathbf{y}_1 \in \mathbb{R}^{n-m}_+. \tag{2.4}$$

An equilibrium $(\mathbf{0}, \mathbf{y}_1^*)$ in which no infectious individuals are present is called a DFE. The non-infectious component $\mathbf{y}_1^*$ satisfies

$$\mathbf{y}_1^* = \mathbf{g}_1(\mathbf{0}, \mathbf{y}_1^*), \tag{2.5}$$

which is the equilibrium of the non-infectious subsystem.

An important stability tool for determining diagnostic quantity that is often more analytically tractable is the inherent or basic reproduction number R_0, which provides insight when designing

prevention and control strategies for established disease infections [19, 20]. A next generation matrix method for defining and computing the basic reproduction number R_0 for discrete-time compartmental epidemic models with a stable fixed point was first developed by Allen, van den Driessche and Watmough in [19, 21].

In epidemiology, R_0 is biologically defined as the expected number of infections produced by a single infectious individual introduced into a totally susceptible population [22]. Consequently, values of $R_0 < 1$ are expected to imply that the number of infections will decrease over time and that the disease will eventually die out as the chain of transmission cannot be maintained. However, values of $R_0 > 1$ imply that a low level of infection will increase infections in the population and a disease outbreak will occur. We will use the next-generation approach to compute R_0.

Our analytical framework relies on a limiting autonomous system. If the host population remains asymptotically constant and converges to a positive state, we can study the global stability of the original system using the following theorem:

Theorem 2.1 (Global Stability via Limiting Equation)**.** *[14] Consider the non-autonomous system* $\mathbf{x}(t+1) = \mathbf{g}_t(\mathbf{x}(t))$, $\mathbf{x}(t) \in \mathbb{R}^n_+$. *Assume that* $\mathbf{g}_t$ *converges uniformly to* $\mathbf{g}$ *as* $t \to \infty$ *and* g_t, g *are continuous on* $\text{int}(\mathbb{R}^n_+)$. *If* $\mathbf{x}^* \in \text{int}(\mathbb{R}^n_+)$ *is a GAS equilibrium of the limiting system* $\mathbf{x} = \mathbf{g}(\mathbf{x})$, *then all orbits of the original system with* $\mathbf{x}(0) \in \text{int}(\mathbb{R}^n_+)$ *converge to* $\mathbf{x}^*$ *as* $t \to \infty$.

Remark 2.1. *Theorem 2.1 does not assert uniqueness of positive equilibria; uniqueness must be verified for each specific model (e.g., discrete-time SIS with nonlinear recruitment often has a unique EE under certain conditions).*

3. Discrete-time susceptible infectious susceptible model

In this section, we show how to apply the general framework of non-autonomous difference equations to the discrete-time SIS epidemic model. While discrete-time SIS models have been extensively studied, establishing the *global stability* of the EE remains challenging.

The total population is divided into two compartments:

- **Susceptible (S):** individuals who are disease-free or have recovered from prior infection. Population density at time t is $S(t)$.
- **Infectious (I):** individuals who are actively infected and capable of transmitting the disease. Population density at time t is $I(t)$.

The total population density is $N(t) = S(t) + I(t)$. Its temporal dynamics follow a demographic equation $N(t+1) = h(N(t))$ (see Section 4), with a limiting positive steady state

$$\lim_{t\to\infty} N(t) = N^*,$$

whenever it exists.

The transitions between the susceptible (S) and infected (I) states occur through the following mechanisms:

- **Infection:** Susceptible individuals become infected upon contact with infectious individuals. The transition from susceptible to infected is governed by the contact rate between susceptibles and the prevalence of infectives in the population.

- **Survival:** Susceptible and infected individuals survive each generation with constant probabilities γ_S and γ_I, respectively.
- **Recovery:** Infected individuals may recover and return to the susceptible class, as recovery does not confer permanent immunity. This is represented by a recovery probability σ.
- **Recruitment:** New susceptibles are recruited per unit time according to a function $f(N(t))$, representing the typically nonlinear birth or recruitment process.

These assumptions and notations lead to the following SIS epidemic model:

$$\begin{cases} S(t+1) = f(N(t)) + \gamma_S \varphi\left(\dfrac{I(t)}{N(t)}\right) S(t) + \gamma_I \sigma I(t), \\ I(t+1) = \gamma_S \left[1 - \varphi\left(\dfrac{I(t)}{N(t)}\right)\right] S(t) + \gamma_I (1-\sigma) I(t). \end{cases} \tag{3.1}$$

with initial conditions $S(0) \geq 0$, $I(0) \geq 0$, and parameters $0 < \gamma_S, \gamma_I, \sigma < 1$. The right-hand sides are sums of non-negative terms, ensuring $S(t), I(t), N(t) \geq 0$ for all t.

The model dynamics are illustrated in Figure 1.

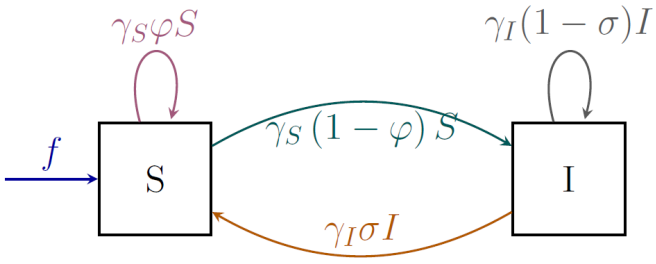

Figure 1. Compartmental diagram of the discrete-time SIS model. Parameters: γ_S / γ_I (survival probability), σ (recovery probability), f (recruitment function), φ (escape function).

The *escape function* $\varphi\colon [0,1] \to [0,1]$ represents the probability that a susceptible individual escapes infection during a time step, depending on the proportion of infectious individuals I/N.

Assumption 3.1 (Escape Function)**.** *The function φ satisfies the following conditions:*
1) $0 \leq \varphi(I/N) \leq 1$ *for* $I \geq 0$, *and* $\varphi(0) = 1$.
2) φ *is concave, monotone decreasing, and continuously differentiable.*
3) $\varphi''(x) > 0$ *for all* $x \in [0,1]$.

Our study generalizes any function φ that satisfies all the above conditions.

Remark 3.1. *When infections are modeled using Poisson processes, φ takes the form*

$$\varphi\left(\frac{I(t)}{N(t)}\right) = \exp\left(-\beta \frac{I(t)}{N(t)}\right),$$

where $\beta > 0$ is the transmission parameter. In other words, β reflects how the level of prevalence shapes the function φ.

The Poisson approximation is often preferred because it provides a simple and analytical way to model the random number of infections occurring within a given time interval. It is particularly suitable when the probability of transmission per contact is low but the number of potential contacts is high, which is typically the case for soil-borne diseases [23]. In such settings, infection events can be considered rare, independent, and scattered among a large number of susceptible hosts. This approach has therefore been adopted in several modelling studies, such as [24]. Moreover, the Poisson process is widely used in continuous-time epidemic models as well [25].

In what follows, we consider $\varphi\left(\frac{I(t)}{N(t)}\right) = e^{-\frac{\beta I(t)}{N(t)}}$, and examine discrete-time SIS models, assuming that both susceptible and infected individuals have the same probability of surviving from one generation to the next, i.e. $\gamma_S = \gamma_I = \gamma$.

Remark 3.2. *For example, in HAIs in a short-stay facility, both susceptible and infected patients have a similar probability of remaining in the facility from one discrete time step to the next [6] (see also [11] and the references cited therein).*

In the following sections, we will consider specific recruitment functions f (Beverton-Holt and Ricker typest) and extend the analysis to periodic environments.

4. Discrete SIS model with Beverton-Holt demography

The Beverton-Holt recruitment function is widely used to model density-dependent population growth in ecology and epidemiology [8]. It is typically expressed as

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

where $a > 0$ represents the maximum per capita recruitment rate at low population densities, and $b > 0$ measures the strength of density dependence. As N increases, the recruitment rate saturates at the carrying capacity $K = \frac{a-1}{b}$, reflecting limited resources or space that constrain population growth. This functional form allows the population to approach a positive equilibrium even when recruitment is nonlinear, which is particularly useful for modeling host populations in epidemic models.

Substituting the Beverton-Holt recruitment function into model (3.1), we obtain the following discrete-time SIS epidemic model with Beverton-Holt type recruitment :

$$\begin{cases} S(t+1) = \dfrac{aN(t)}{1 + bN(t)} + \gamma\varphi\left(\dfrac{I(t)}{N(t)}\right) S(t) + \gamma\sigma I(t), \\ I(t+1) = \gamma\left[1 - \varphi\left(\dfrac{I(t)}{N(t)}\right)\right] S(t) + \gamma(1-\sigma) I(t). \end{cases} \tag{4.1}$$

4.1. Demographic equation

Our simple deterministic discrete-time epidemic model is formulated under the assumptions that the dynamics of the total population size in generation t, denoted by $N(t)$, are governed by a one-dimensional equation of the form:

$$N(t+1) = f(N(t)) + \gamma N(t), \tag{4.2}$$

where $\gamma \in (0,1)$ is the constant probability of surviving per generation, and $f : \mathbb{R}^+ \to \mathbb{R}^+$ represents the typically non-linear birth or recruitment process, and $f(N) \in C^1(\mathbb{R}_+, \mathbb{R}_+)$. Let the map $h : \mathbb{R}^+ \to \mathbb{R}^+$ be defined by: $h(N) = f(N) + \gamma N$, then the set of iterates of the map h is equivalent to the set of density sequences generated by Eq (4.2), and is used to describe its dynamics.

In this section, we briefly review the demographic equation with the Beverton-Holt recruitment function, highlighting key results relevant to our study. The Ricker recruitment function will be discussed later in the paper.

If the recruitment function is the classic Beverton-Holt model, then the demographic Eq (4.2) reduces to the nonlinear difference equation: $N(t+1) = h(N(t)) = \frac{aN(t)}{1+bN(t)} + \gamma N(t)$. This demographic equation has a positive equilibrium, denoted by N^*, which is GAS.

This equilibrium is obtained by solving $N(t+1) = N(t)$, yielding $N^* = \frac{a-(1-\gamma)}{b(1-\gamma)} = \frac{1}{b}\left(\frac{a}{1-\gamma} - 1\right)$ Let

$$h(N) = \frac{aN}{1+bN} + \gamma N.$$

So, $h'(N) = \frac{a}{(1+bN)^2} + \gamma$ with $h'(0) = a + \gamma$ and $h'(N^*) = \frac{(1-\gamma)^2}{a} + \gamma$.

We have two cases:

a) $a + \gamma > 1$ that is implies that $N^* > 0$ exists and $h'(0) > 1$.

b) $a + \gamma \leq 1$ that is implies that $N^* > 0$ does not exist and $N^* = 0$ is the only equilibrium point. We have $0 < h'(0) \leq 1$.

Lemma 4.1. *$h(N)$ is a monotone function.*

Proof. Let $N_1 < N_2$ then $h(N_1) = \frac{aN_1}{1+bN_1} + \gamma N_1$, $h(N_2) = \frac{aN_2}{1+bN_2} + \gamma N_2$ clearly $\gamma N_1 < \gamma N_2$.

Now, $aN_1 + abN_1N_2 < aN_2 + abN_1N_2$ which imply that $\frac{aN_1}{1+bN_1} + \gamma N_1 < \frac{aN_2}{1+bN_2} + \gamma N_2$.

Thus $h(N_1) < h(N_2)$. Consequently, h is a monotone function. □

Theorem 4.1. *The following statements hold true:*

i) if $a + \gamma \leq 1$, then $\lim_{t\to\infty} N(t) = 0$ (biologically unrealistic).

ii) if $a + \gamma > 1$, then $\lim_{t\to\infty} N(t) = N^*$.

Proof. (*i*) Assume that $a + \gamma \leq 1$ then $0 < h'(0) \leq 1$. Since $h'(N) > 0$ and $h''(N) < 0$, $h(N) < N$ for all $N > 0$. Thus for any $N_0 > 0$, the sequence $\{N_0, h(N_0), ..., h^t(N_0), ...\}$ is decreasing and bounded below by 0. Hence it must converge to a point $\widehat{N}$ which must be an equilibrium point. But since 0 is the only equilibrium point, $\widehat{N} = 0$.

(*ii*) Assume that $a + \gamma > 1$. Then $h'(0) > 1$. Since $h'(N) > 0$ and $h''(N) < 0$, $h(N) > N$ for $N < N^*$ and $h(N) < N$ for $N > N^*$.

$$
\begin{aligned}
N < N^* &\Leftrightarrow N < \frac{a-1+\gamma}{b(1-\gamma)} \\
&\Leftrightarrow bN(1-\gamma) < a-1+\gamma \\
&\Leftrightarrow (a+\gamma-1) + bN(\gamma-1) > 0 \Leftrightarrow h(N) - N > 0
\end{aligned}
$$

$$
\begin{aligned}
N > N^* &\Leftrightarrow N > \frac{a-1+\gamma}{b(1-\gamma)} \\
&\Leftrightarrow bN(1-\gamma) > a-1+\gamma \\
&\Leftrightarrow (a+\gamma-1) + bN(\gamma-1) < 0 \Leftrightarrow h(N) - N < 0.
\end{aligned}
$$

Using a similar argument as before, for $N > N^*$ the sequence $\{N_0, h(N_0), ..., h^t(N_0), ...\}$ is decreasing and bounded below by N^*. Hence it must converge to a point which must be an equilibrium point. But since N^* is the only equilibrium point, then $\lim_{t\to\infty} N(t) = N^*$ and for $N < N^*$ the sequence $\{N_0, h(N_0), ..., h^t(N_0), ...\}$ is increasing and bounded above by N^*. Hence it must converge to a point which must be an equilibrium point. Since N^* is the only one, all orbits converges to N^*. □

Using the demographic Eq (4.2), we showed that if $a + \gamma > 1$, then the total population converges to a positive steady state

$$\lim_{t\to\infty} N(t) = N^* = \frac{a-(1-\gamma)}{b(1-\gamma)}.$$

This means that the dynamics are compensatory in $(0, \infty)$ and the total population is uniformly persistent (and hence, persistent) in $(0, \infty)$. They conclude that when the recruitment function is the Beverton-Holt type the population dynamics are compensatory[†] and the total population is uniformly persistent and lives on a globally attracting positive fixed point.

Remark 4.1. *The survival probability, denoted γ, replaces natural mortality via the expression $1-\gamma$, since this quantity represents the probability of death due to natural causes. Thus, the population dynamics account for mortality through this survival probability, making explicit removal unnecessary. Indeed, including recruitment, with or without natural removal, leads the population to a positive asymptotic state in all cases studied.*

We can then define the *associated limiting model* by replacing $N(t)$ with its asymptotic value N^* in the SIS equations.

$$
\begin{cases}
S(t+1) = \dfrac{aN^*}{1+bN^*} + \gamma\,\varphi\left(\dfrac{I(t)}{N^*}\right) S(t) + \gamma\sigma\, I(t), \\
I(t+1) = \gamma\left[1 - \varphi\left(\dfrac{I(t)}{N^*}\right)\right] S(t) + \gamma(1-\sigma)\, I(t).
\end{cases}
\tag{4.3}
$$

[†]Compensatory means the per capita growth decreases smoothly with density without oscillations. This is observed in certain insects or fish.

4.2. Basic reproduction number

We have

$$I(t+1) = \gamma\left[1 - \varphi\left(\frac{I(t)}{N^*}\right)\right] S(t) + \gamma(1-\sigma)I(t),$$

With the vector of new infections is $\mathfrak{F}(t) = \gamma\left[1 - \varphi\left(\frac{I(t)}{N^*}\right)\right] S(t)$ and the vector of other transitions is $\mathfrak{T}(t) = \gamma(1-\sigma)I(t)$

$$F = \frac{\partial \mathfrak{F}(t)}{\partial I}\Big|_{(S^*,0)} = \frac{\partial}{\partial I}\Big[\gamma\left(1 - \varphi(I/N^*)\right) S\Big]\Big|_{(S^*,0)} = \beta\gamma$$

$$T = \frac{\partial \mathfrak{T}(t)}{\partial I}\Big|_{(S^*,0)} = \frac{\partial}{\partial I}\left[\gamma(1-\sigma)I\right]\Big|_{(S^*,0)} = \gamma(1-\sigma).$$

Using the next-generation matrix approach [19, 21], we have

$$R_0 = \frac{\beta\gamma}{1-\gamma(1-\sigma)}.$$

The product $\beta\gamma$ represents the effective transmission rate of the infection. The denominator, $1 - \gamma(1-\sigma)$, combines the effects of the recovery probability (σ) and the survival probability (γ). A larger value of σ (i.e., faster removal of infected individuals) or a smaller value of γ (i.e., lower survival) increases the denominator, thereby reducing R_0.

Mathematically, R_0 acts as a critical threshold that governs the dynamics of both the system and the disease.

Epidemiologically, it represents the expected number of secondary infections generated by a single infectious individual in a fully susceptible population [22]. If $R_0 \leq 1$ the number of infected individuals progressively diminishes until the disease is eradicated. Conversely, if $R_0 > 1$ the disease persists within the population, leading to an epidemic.

Remark 4.2. *An effective way to reduce the basic reproduction number R_0. Figure 2 illustrates how R_0 influences the dynamics of the disease: when $R_0 < 1$, the infection dies out, whereas for $R_0 > 1$, the infection can spread and establish itself in the population.*

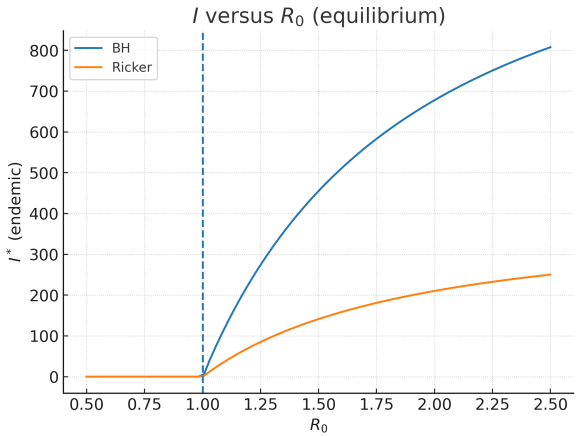

Figure 2. Illustration of the role of R_0 in determining whether a disease dies out or persists.

For the SIS model considered here, R_0 is given by

$$R_0 = \frac{\beta\gamma}{1-\gamma(1-\sigma)}.$$

The sensitivity of R_0 to changes in survival probability γ and recovery probability σ can be computed as

$$\frac{\partial R_0}{\partial \gamma} = \frac{\beta}{(1-\gamma(1-\sigma))^2} > 0, \qquad \frac{\partial R_0}{\partial \sigma} = -\frac{\beta\gamma^2}{(1-\gamma(1-\sigma))^2} < 0.$$

This analysis shows that:

- *R_0 increases with γ, indicating that higher host survival may promote disease transmission.*
- *R_0 decreases with increasing σ, meaning that faster recovery reduces transmission and helps control the epidemic.*

Thus, Figure 2 provides a visual representation of these effects, highlighting how adjusting parameters such as β, γ, or σ can influence the overall epidemic outcome.

4.3. Global asymptotic stability of the equilibria

In this part, we will apply the new approach that is based on a simple tool, that is, we consider the limiting Eq (4.4) as we have the population in the case $a + \gamma > 1$ is asymptotically constant and converges to N^* then by $S(t) = N^* - I(t)$ we have:

$$I(t+1) = \gamma(1 - e^{\frac{-\beta I(t)}{N^*}})(N^* - I(t)) + \gamma(1-\sigma)I(t). \tag{4.4}$$

For $I \in \mathbb{R}^+$, let

$$g(I) = \gamma(1 - e^{\frac{-\beta I}{N^*}})(N^* - I) + \gamma(1-\sigma)I.$$

Corollary 4.1. *If $R_0 \leq 1$, then $(S^*, 0)$ is GAS in the limiting Eq (4.4). Moreover, if $R_0 > 1$, $(S^*, 0)$ is unstable.*

Proof. Using the limiting equation with $a + \gamma > 1$ and $\lim_{t\to\infty} N(t) = N^* = \dfrac{a+\gamma-1}{b(1-\gamma)}$,

$$\begin{aligned} g'(I) &= -\gamma(1 - e^{\frac{-\beta I}{N^*}}) + \frac{\gamma\beta}{N^*} e^{\frac{-\beta I}{N^*}}(N^* - I) + \gamma(1-\sigma) \\ &= \gamma\left[e^{\frac{-\beta I}{N^*}}\left(\beta\left(1 - \frac{I}{N^*}\right) + 1\right) - \sigma\right]. \end{aligned}$$

Recall that,

$$R_0 = \frac{\beta\gamma}{1-\gamma(1-\sigma)} \text{ and } g'(0) = \gamma(\beta + 1 - \sigma).$$

If $R_0 \leq 1$, then $0 < g'(0) \leq 1$. Moreover, $g'(I) = \gamma\beta e^{\frac{-\beta I}{N^*}}\left(1 - \frac{I}{N^*}\right) + \gamma(1-\sigma) < \gamma\beta + \gamma(1-\sigma) \leq 1$. Thus by integrating between 0 and I: $g(I) < I$ for all $I > 0$.

This implies that $g^t(I) \longmapsto \widehat{I}$ as $t \to \infty$. But since $I^* = 0$ is the only equilibrium point so $\widehat{I} = 0$. Hence, $\lim_{t\to\infty} I(t) = 0$.

Now, we have $S(t) = N(t) - I(t)$. Hence $\lim_{t\to\infty} S(t) = \lim_{t\to\infty} N(t) - \lim_{t\to\infty} I(t) = N^* - 0 = N^*$.

Therefore, $\lim_{t\to\infty}(S(t), I(t)) = (S^*, 0)$. □

By invoking Theorem 2.1, we may now establish the global stability stated in the following theorem.

Theorem 4.2. *If $R_0 \leq 1$, then $(S^*, 0)$ is GAS in the SIS model. Moreover, if $R_0 > 1$, $(S^*, 0)$ is unstable.*

Next, we investigate the case when $R_0 > 1$. In this case, $g'(0) > 1$. Moreover,

$$g''(I) = \frac{-\beta^2\gamma}{N^*} e^{\frac{-\beta I}{N^*}} \left(1 - \frac{I}{N^*}\right) - \frac{2\beta\gamma}{N^*} e^{\frac{-\beta I}{N^*}} < 0.$$

Hence, $g(I)$ is concave down. Now, $g(N^*) = \gamma(1-\sigma)N^* < N^*$. So, there exists a unique positive fixed point I^*.

Theorem 4.3. *[13, 14, 26, 27]*

Assume that g is continuous on an interval I such that all the orbits of the equation $x(t+1) = g(x(t))$ are bounded. Then every orbit converges to an equilibrium point if and only if there are no $2-$ periodic cycles (that is, the only solutions of the equation $g(g(x)) = x$ are the equilibrium points of g).

One of the challenges in using Theorem 4.3 is to show that the difference equation has no periodic orbits of minimal period 2. The following result may help in this.

Lemma 4.2. *Let $T(x) = x + g(x)$. If T is monotone, then g has no points of minimal period 2. Equivalently, if $1 + g'(x) \neq 0$ for all x, then g has no periodic orbits of period 2.*

Corollary 4.2. *If $R_0 > 1$ all orbits in the interior of $\mathbb{R}^{2+}$ converge to (S^*, I^*) in the limiting Eq (4.4).*

Proof. First, we will show that all orbits are bounded.

$$\begin{aligned} I(t+1) &< N^* + (1-\sigma)I(t) \\ &< (1-\sigma)^t I_0 + \frac{N^*(1-(1-\sigma)^t)}{\sigma} \\ &< I_0 + \frac{N^*}{\sigma}. \end{aligned}$$

Second, we will show that there are no periodic orbit of period 2. Assume not then let $\overline{I_1}, \overline{I_2}$ be a two periodic cycle, i.e., $g(\overline{I_1}) = \overline{I_2}$ and $g(\overline{I_2}) = \overline{I_1}$ with $\overline{I_1} < \overline{I_2}$.

Now, $T(I) = I + g(I)$ is monotone. Since, $T'(I) = 1 + g'(I) = 1 + \gamma(1-\sigma) + \gamma\beta e^{\frac{-\beta I}{N^*}} \left(1 - \frac{I}{N^*}\right) > 0$ because of $1 + \gamma(1-\sigma) > 1$ and $\gamma\beta e^{\frac{-\beta I}{N^*}} \left(1 - \frac{I}{N^*}\right) > 0$. Therefore, $1 + g'(I) \neq 0$.

$$T(\overline{I_1}) = \overline{I_1} + g(\overline{I_1}) = \overline{I_1} + \overline{I_2} \text{ and } T(\overline{I_2}) = \overline{I_2} + g(\overline{I_2}) = \overline{I_2} + \overline{I_1}.$$

Contradiction since $T(\overline{I_1}) < T(\overline{I_2})$.

It follows by Theorem 4.3 that every orbit converge to an equilibrium point. Since 0 is unstable, all orbits must converged to $I^* > 0$, and I^* is GAS.

We have $\lim_{t\to\infty} S(t) = \lim_{t\to\infty} N(t) - \lim_{t\to\infty} I(t) = N^* - I^*$. □

According to Theorem 2.1, we can now establish the global stability result stated below.

Corollary 4.3. *If $R_0 > 1$ all orbits in the interior of $\mathbb{R}^{2+}$ converge to (S^*, I^*) in the SIS model 4.1.*

All of the above analysis is illustrated in Figure 3.

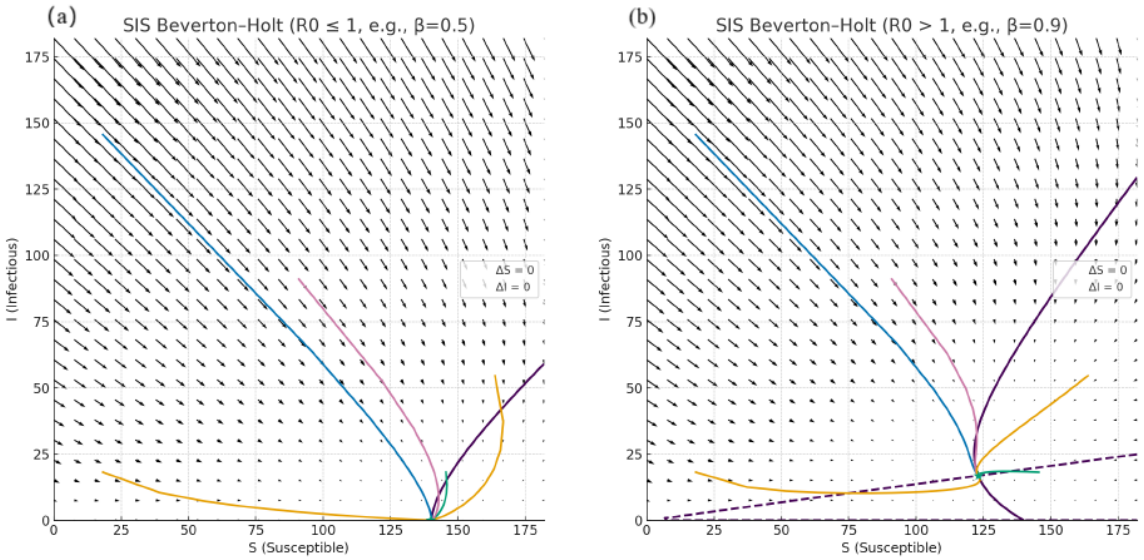

Figure 3. (a) Shows the global stability of the disease free equilibrium $(S^*, 0)$ when $R_0 \leq 1$ ($\beta = 0.5$, $\gamma = 0.8$, $\sigma = 0.5$). (b) Shows the global stability of the EE (S^*, I^*) when $R_0 > 1$ ($\beta = 0.9$, $\gamma = 0.8$, $\sigma = 0.5$). Both graphs are for the models SIS when the recruitment function is the Beverton-Holt.

5. Discrete SIS model with Ricker demography

The Ricker recruitment function, widely used in population dynamics [9], is represented by

$$f(N(t)) = r\,N(t)\,e^{-cN(t)}, \tag{5.1}$$

where $r > 0$ is the intrinsic growth rate of the population and $c > 0$ is the density-dependence coefficient, which captures the strength of competition among individuals. This function models overcompensatory dynamics, meaning that at high population densities, recruitment decreases due to limited resources, while at low densities, recruitment increases approximately exponentially.

Substituting this recruitment function into model (3.1), we obtain the discrete-time SIS epidemic model with Ricker-type recruitment:

$$\begin{cases} S(t+1) = rN(t)e^{-cN(t)} + \gamma\varphi\left(\dfrac{I(t)}{N(t)}\right)S(t) + \gamma\sigma I(t), \\ I(t+1) = \gamma\left[1 - \varphi\left(\dfrac{I(t)}{N(t)}\right)\right]S(t) + \gamma(1-\sigma)I(t). \end{cases} \tag{5.2}$$

The function φ denotes the escape function, as introduced in the previous sections, i.e., $\varphi = \varphi\left(\frac{I}{N}\right)$.

In this case, the demographic Eq (4.2) reduces to the nonlinear difference equation: $N(t+1) = k(N(t)) = rN(t)e^{-cN(t)} + \gamma N(t)$. Then the demographic equation has a positive equilibrium which is GAS.

This equilibrium is obtained by solving $N(t+1) = N(t)$, which gives $N^* = \frac{1}{c} ln\left(\frac{r}{1-\gamma}\right)$ Let

$$k(N) = rNe^{-cN} + \gamma N.$$

So, $k'(N) = (1 - cN)re^{-cN} + \gamma$ with $k'(0) = r + \gamma$ and $k'(N^*) = 1 - (1-\gamma)ln\left(\frac{r}{1-\gamma}\right)$.

We have two cases:

a) $r + \gamma > 1$ that is implies that $N^* > 0$ exists and $k'(0) > 1$.

b) $r + \gamma \leq 1$ that is implies that $N^* > 0$ does not exist and $N^* = 0$ is the only equilibrium point. We have $0 < k'(0) \leq 1$.

Before we start the next theorem we need to show that $N(t)$ is bounded above. Let us write $N(t+1) = rN(t)e^{-cN(t)} + \gamma N(t) = h(N(t) + \gamma N(t)$. Now $h'(N) = (1 - cN)re^{-cN} = 0$ if $N = \frac{1}{c}$. Moreover $h''(\frac{1}{c}) < 0$. Hence $\max(h(N)) = h(\frac{1}{c}) = \frac{r}{ce}$ Thus $N(t+1) \leq \frac{r}{ce} + \gamma N(t)$. This implies that $N(t) \leq \gamma^t N_0 + \frac{r}{ce}\frac{1-\gamma^t}{1-\gamma} \leq M$, for some constant M.

Theorem 5.1. *The following statements hold true:*

i) If $r + \gamma \leq 1$ *, then* $\lim_{t\to\infty} N(t) = 0$ *(not relevant biologically).*

ii) If $r + \gamma > 1$ *and* $c < \frac{1}{M}$ *, then* $\lim_{t\to\infty} N(t) = N^*$.

Proof.

- (i) If $r+\gamma \leq 1$ then $0 < k'(0) \leq 1$. Now $N(t+1) = rN(t)e^{-cN(t)} + \gamma N(t) = N(t)(\gamma + re^{-cN(t)}) < N(t)(\gamma + r) \leq N(t)$.

  Hence $N(t)$ is decreasing and bounded below by 0. Thus it must converge, and the limit must be a fixed point of the map. But the only fixed point in this case is 0. This implies that $\lim_{t\to\infty} N(t) = 0$

- (ii) If $r + \gamma > 1$, then $k'(0) = r + \gamma > 1$.

  Now $k'(N) = \gamma + (1 - cN)re^{-cN}$. Since $c < \frac{1}{M}$, $c < \frac{1}{N}$, and consequently, $k'(N) > 0$. Hence $N(t)$ is increasing and bounded above by M Hence, it must converge to a limit which must be a fixed point of the map k. But the only positive fixed point is N^* and thus $\lim_{t\to\infty} N(t) = N^*$.

□

The associated limiting model, obtained by replacing $N(t)$ by N^*, is given below.

$$\begin{cases} S(t+1) = rN^*e^{-cN^*} + \gamma\varphi\left(\dfrac{I(t)}{N^*}\right)S(t) + \gamma\sigma I(t), \\ I(t+1) = \gamma\left[1 - \varphi\left(\dfrac{I(t)}{N^*}\right)\right]S(t) + \gamma(1-\sigma)I(t). \end{cases} \tag{5.3}$$

We have the same analysis that was presented before in the previous section, Concerning the study of the existence and stability of the equilibria, the same approach will be applied. All the above analysis is illustrated in Figure 4.

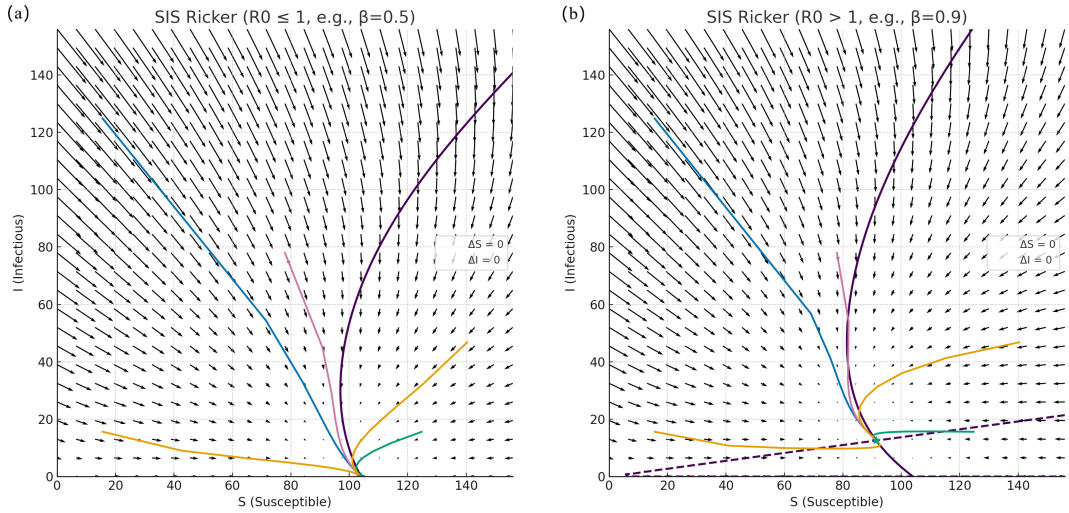

Figure 4. (a) Shows the global stability of the disease free equilibrium $(S^*, 0)$ when $R_0 \leq 1$ ($\beta = 0.5,\ \gamma = 0.8,\ \sigma = 0.5$). (b) Shows the global stability of the EE (S^*, I^*) when $R_0 > 1$ ($\beta = 0.9,\ \gamma = 0.8,\ \sigma = 0.5$). Both graphs are for the models SIS when the recruitment function is the Ricker.

Although the Ricker recruitment function can exhibit non-monotone dynamics for certain parameter values, we impose a sufficient condition ensuring that the population update map remains monotone. Under this assumption, the stability thresholds remain fully characterized by the basic reproduction number R_0 : The DFE is GAS whenever $R_0 \leq 1$, while the EE is GAS whenever $R_0 > 1$, paralleling the Beverton-Holt case.

6. Non-autonomous periodic SIS model with Beverton-Holt

To present the non-autonomous p-periodic SIS epidemic model with period $p \geq 2$, we consider a host population divided into two compartments: susceptibles, denoted by $S(t)$, and infectives, denoted by $I(t)$. The demographic dynamics follow a p-periodic Beverton-Holt model to account for seasonal or other regularly varying environmental effects, which makes the model particularly relevant for diseases exhibiting seasonal patterns or other periodic fluctuations in population or transmission rates.

The system is described by the following difference equations:

$$\begin{cases} S(t+1) = \dfrac{aN(t)}{1+bN(t)} + \gamma\varphi\left(\dfrac{I(t)}{N(t)}\right)S(t) + \gamma\sigma I(t), \\ I(t+1) = \gamma\left[1-\varphi\left(\dfrac{I(t)}{N(t)}\right)\right]S(t) + \gamma(1-\sigma)I(t). \end{cases} \tag{6.1}$$

where $a(t), b(t), \gamma(t), \sigma(t), \beta(t)$ are all p-periodic functions, i.e., $a_{t+p} = a_t, b_{t+p} = b_t, \gamma_{t+p} = \gamma_t, \sigma_{t+p} = \sigma_t$, $\beta_{t+p} = \beta_t$ for all $t \in \mathbb{Z}^+$.

The function φ denotes the escape function. While we could keep it general, here we use the commonly applied exponential form:

$$\varphi\left(\frac{I(t)}{N(t)}\right) = \exp\left(-\frac{\beta(t)I(t)}{N(t)}\right),$$

which arises naturally when infections are modeled as Poisson processes. This form simplifies computations while retaining the essential nonlinear dependence on the prevalence of infection.

By summing the two compartments, the total population $N(t) = S(t) + I(t)$ evolves according to:

$$N(t+1) = \frac{a(t)N(t)}{1 + b(t)N(t)} + \gamma(t)N(t), \tag{6.2}$$

which can be written compactly as $N(t+1) = g_t(N(t))$, with $g_{t+p} = g_t$ for all $t \in \mathbb{Z}^+$. Define

$$\phi_t = g_{t-1} \circ g_{t-2} \circ \cdots \circ g_0,$$

where $g_i(N(i)) = N(i+1)$.

Lemma 6.1. *The composition of monotone maps is a monotone map.*

Corollary 6.1. *The composition map ϕ_p is a monotone map.*

Theorem 6.1. *The following statements hold:*

i) If $\prod_{i=0}^{p-1}(a_i + \gamma_i) < 1$, *then* $\lim_{t\to\infty} N(t) = 0$ *(case of limited biological relevance).*

ii) If $\prod_{i=0}^{p-1}(a_i + \gamma_i) > 1$, *then all the orbits converge to a unique positive p-periodic cycle* $\{\overline{N_0}, \overline{N_1}, ..., \overline{N_{p-1}}\} = N_p^*$.

Proof. (*i*) From the (Section 4), it was shown that $0 < g_i'(0) \leq 1$, $g_i'(N) > 0$ and $g_i''(N) < 0$, $g_i(N) < N$ for all $N > 0$. It follows that

$$\phi_p'(0) = g_{p-1}'(0) \circ g_{p-2}'(0) \circ .... \circ g_0'(0) = \prod_{i=0}^{p-1}(a_i + \gamma_i) < 1.$$

Since $\phi_p'(N) > 0$ and $\phi_p''(N) < 0$ we have $\phi_p(N) < N$ for all $N > 0$ (see Section 4), one may show that $\lim_{t\to\infty} N(t) = 0$.

(*ii*) If $\prod_{i=0}^{p-1}(a_i + \gamma_i) > 1$. Then $\phi_p'(0) > 1$. From Section 4, we have shown that $g_i'(N) > 0$, $i = 0, 1, 2..., p-1$. Hence $\phi_p'(N) > 0$ and thus ϕ_p is increasing. Moreover, since $\phi_p''(N) < 0$, ϕ_p is concave down. Since each g_i is bounded above ϕ_p is also bounded above. Thus, there exists $\overline{N_0}$ such that $\phi_p(\overline{N_0}) = \overline{N_0}$, and thus $\{\overline{N_0}, \overline{N_1}, ..., \overline{N_{p-1}}\}$ is a $p-$periodic cycle, which is GAS in the interior of $\mathbb{R}^{2+}$. □

In the sequel, we assume that $\prod_{i=0}^{p-1}(a_i + \gamma_i) > 1$. In this case, we have $\lim_{t\to\infty} N(t) = N_p^* = \{\overline{N_0}, \overline{N_1}, ..., \overline{N_{p-1}}\}$ where $N_p^*(t+p) = N_p^*(t)$.

Since $S(t) + I(t) = N_p^*$ and $S(t) = N_p^* - I(t)$. Hence we have the limiting equation:

$$I(t+1) = \gamma(t)(1 - e^{\frac{-\beta(t)I(t)}{N_p^*}})(N_p^* - I(t)) + \gamma(t)(1 - \sigma(t))I(t). \tag{6.3}$$

Let $I(t+1) = h_t(I(t))$ and $\psi_p(I) = h_{p-1} \circ h_{p-2} \circ .... \circ h_0(I)$ is the composition map. And $\psi_p^{'}(0) = \prod_{i=0}^{p-1} \gamma_i(\beta_i + (1-\sigma_i))$.

The net reproduction number is given by

$$R_0 = \prod_{i=0}^{p-1} \frac{\beta_i \gamma_i}{1 - \gamma_i(1-\sigma_i)}.$$

The quantity R_0 represents the expected number of secondary infections produced by a single infectious individual introduced into a fully susceptible population over one full period of environmental or demographic fluctuations. If $R_0 > 1$, then each infected individual generates, on average, more than one new case across a complete cycle, leading to the persistence of the disease. Conversely, if $R_0 < 1$, transmission is insufficient to sustain infection and the disease eventually dies out. Hence, R_0 plays the role of a global invasion threshold even in non-autonomous systems.

Theorem 6.2. *If $R_0 \leq 1$, then $\lim_{t\to\infty} I(t) = 0$.*

Proof. Since $R_0 \leq 1$ it follows that $0 < \psi_p^{'}(0) \leq 1$. Moreover,

$$\psi_p^{'}(I) = \prod_{i=0}^{p-1} \left[\gamma_i \beta_i e^{\frac{-\beta_i I}{N_p^*}} \left(1 - \frac{I}{N_p^*}\right) + \gamma_i(1-\sigma_i) \right] < \prod_{i=0}^{p-1} (\gamma_i \beta_i + \gamma_i(1-\sigma_i)) \leq 1.$$

Thus $\psi_p(I) < I$ for all $I > 0$, this implies that $\lim_{t\to\infty} I(t) = c_p$, where c_p is a $p-$periodic cycle. But, since $I = 0$ is the only $p-$periodic (fixed) point it follows that $\lim_{t\to\infty} I(t) = 0$. □

Corollary 6.2. *If $R_0 \leq 1$, then the DFE $(S_p^*, 0)$ is GAS in the limiting Eq (6.3). Moreover, if $R_0 > 1$, $(S_p^*, 0)$ is unstable.*

Proof. Since $S(t) = N(t) - I(t)$ it follows that

$$\lim_{t\to\infty} S(t) = \lim_{t\to\infty} N(t) - \lim_{t\to\infty} I(t) = N_p^* - 0 = N_p^*.$$

So, $\lim_{t\to\infty}(S(t), I(t)) = (S_p^*, 0)$. □

By Theorem 2.1 we summarize,

Corollary 6.3. *If $R_0 \leq 1$, then the DFE $(S_p^*, 0)$ is GAS in the original SIS model (6.1). Moreover, if $R_0 > 1$, $(S_p^*, 0)$ is unstable.*

We now turn our attention to the case of, $R_0 > 1$, that is, $\psi_p^{'}(0) > 1$.

Recall that $\psi_p^{'}(I) = \prod_{i=0}^{p-1} \left[\gamma_i \beta_i e^{\frac{-\beta_i I}{N_p^*}} \left(1 - \frac{I}{N_p^*}\right) + \gamma_i(1-\sigma_i) \right] = \prod_{i=0}^{p-1} H_i(t)$.

As well,

$$\psi_p^{''}(I) = \sum_{j=0}^{p-1} \left[H_j^{'}(I) \prod_{i=0}^{p-1} H_i(I) \right] < 0.$$

Because of $H_i^{'}(I) = \gamma_i\beta_i\left(\frac{-\beta_i}{N_p^*}e^{\frac{-\beta_i I}{N_p^*}}\left(1-\frac{I}{N_p^*}\right)-\frac{1}{N_p^*}e^{\frac{-\beta_i I}{N_p^*}}\right)$ hence $\psi_p(I)$ is concave down.

Besides, $\psi_p(N_p^*) = \prod_{i=0}^{p-1}\gamma_i(1-\sigma_i)N_p^* < N_p^*$. Then, there exists a unique p-periodic cycle (fixed) point I_p^*.

Corollary 6.4. *If $R_0 > 1$ all orbits converge to the p-periodic cycle (S_p^*, I_p^*) in the SIS model (6.1).*

Proof. ψ_p is continuous as a composition of continuous maps, assume that $\gamma(t), \sigma(t) \in]0,1[$, $\beta(t) > 0$, for all $I(t) \in [0, max(\overline{N_i})]$, $0 \le i < p$ we have

$$I(t+1) < \gamma(t)max(\overline{N_i}) + \gamma(t)I(t) < 2max(\overline{N_i}).$$

So each composition is bounded and has values in $(0, 2max(\overline{N_i}))$. Therefore, the composition remains within this interval. Thus by recurrence,

$$\psi_1(I) = h_0(I) < 2max(\overline{N_i}), \psi_2(I) = h_1(h_0)(I) < 2max(\overline{N_i}), ..., \psi_p(I) < 2max(\overline{N_i}).$$

The sequence of compositions is uniformly bounded on $[0, max(\overline{N_i})]$. Assume that $\overline{I_1} \neq \overline{I_2}$ are two periodic cycles then $h(\overline{I_1}) = \overline{I_2}$ and $h(\overline{I_2}) = \overline{I_1}$.

Now, for $F(I) = h(I) + I$ is monotone: $F(\overline{I_1}) = h(\overline{I_1}) + \overline{I_1} = \overline{I_2} + \overline{I_1}$ and $F(\overline{I_2}) = h(\overline{I_2}) + \overline{I_2} = \overline{I_1} + \overline{I_2}$ which is false. Hence, all orbits should converge to I_p^*, which is the only fixed point of the composition map. □

7. Conclusions

In this paper we have developed a new and unified analytical framework for the global dynamics of discrete-time SIS epidemic models with nonlinear recruitment functions, concentrating on the Beverton-Holt and Ricker demographic structures. The main contribution is the establishment of rigorous threshold results: The DFE is GAS whenever $R_0 \le 1$, while the EE is GAS whenever $R_0 > 1$. This completes a longstanding gap in the theory of discrete epidemic models and extends earlier approaches in discrete dynamical systems and population biology [13, 14, 27].

Our method, based on limiting equations for non-autonomous systems, provides an alternative to traditional techniques such as Lyapunov functions and monotone dynamical systems. Unlike those methods, which often require strong assumptions and do not easily extend to non-autonomous or periodic settings, the limiting equation approach yields global stability in both autonomous and time-dependent environments. This not only generalizes earlier results but also situates our analysis within the broader theory of asymptotically autonomous difference equations [11, 28, 29].

From a modeling perspective, the results highlight the decisive role of demographic feedback. In the Beverton-Holt case, density dependence produces smooth monotonic convergence toward equilibrium. In contrast, the classical Ricker recruitment function is known to generate oscillations and even chaotic fluctuations under suitable parameter regimes. However, in the present work we restrict our attention to a parameter range in which the population map remains monotone, so that complex oscillatory behavior does not arise. Within this regime, our theory guarantees that the basic reproduction number R_0 still acts

as a sharp threshold separating extinction from persistence. Thus, the central epidemiological insight–that controlling R_0 below one guarantees eradication-remains valid even in discrete-time, nonlinear, and demographically structured settings.

Beyond resolving this fundamental stability problem, the framework opens several avenues for future research. It can be extended to multi-compartment epidemic models (such as SIR and SEIR), to host-vector systems, and to metapopulation models with spatial dispersal. Another promising direction is the study of stochastic perturbations and environmental noise, where the limiting equation method may yield novel insights into persistence and extinction. These extensions would broaden the scope of discrete-time epidemiology and connect it with current developments in ecological and evolutionary dynamics.

Use of AI tools declaration

The authors declare they have not used Artificial Intelligence (AI) tools in the creation of this article.

Conflict of interest

The authors declare there are no conflicts of interest.

References

1. W. O. Kermack, A. G. Mckendrick, A contribution to the mathematical theory of epidemics, *Proc. R. Soc. A*, **115** (1927), 700–721. https://doi.org/10.1098/rspa.1927.0118
2. L. J. S. Allen, Some discrete-time SI, SIR and SIS epidemic models, *Math. Biosci.*, **124** (1994), 83–105. https://doi.org/10.1016/0025-5564(94)90025-6
3. H. W. Hethcote, J. A. Yorke, *Gonorrhea Transmission Dynamics and Control*, 1st edition, Springer, 1984. https://doi.org/10.1007/978-3-662-07544-9
4. A. Lajmanovich, J. A. Yorke, A deterministic model for gonorrhea in a nonhomogeneous population, *Math. Biosci.*, **28** (1976), 221–236. https://doi.org/10.1016/0025-5564(76)90125-5
5. D. Clancy, A stochastic SIS infection model incorporating indirect transmission, *J. Appl. Probab.*, **42** (2005), 726–737. https://doi.org/10.1239/jap/1127322023
6. M. Martcheva, *An Introduction to Mathematical Epidemiology*, 1st edition, Springer, 2015. https://doi.org/10.1007/978-1-4899-7612-3
7. L. Cai, X. Li, Analysis of a simple vector-host epidemic model with direct transmission, *Discrete Dyn. Nat. Soc.*, **2010** (2010). https://doi.org/10.1155/2010/679613
8. R. J. H. Beverton, S. J. Holt, *On the Dynamics of Exploited Fish Populations*, 1st edition, Springer, 1993. https://doi.org/10.1007/978-94-011-2106-4
9. W. E. Ricker, Stock and recruitment, *J. Fish. Res. Board Can.*, **11** (1954), 559–623. http://doi.org/10.1139/f54-039
10. X. Ma, Y. Zhou, H. Cao, Global stability of the endemic equilibrium of a discrete SIR epidemic model, *Adv. Differ. Equations* , **2013** (2013), 42. https://doi.org/10.1186/1687-1847-2013-42

11. C. Castillo-Chavez, A. Yakubu, Dispersal, disease and life-history evolution, *Math. Biosci.*, **173** (2001), 35–53. https://doi.org/10.1016/S0025-5564(01)00065-7
12. J. E. Franke, A. A. Yakubu, Disease-induced mortality in density-dependent discrete-time S-I-S epidemic models, *J. Math. Biol.*, **57** (2008), 755–790. https://doi.org/10.1007/s00285-008-0188-9
13. S. N. Elaydi, J. M. Cushing, *Discrete Mathematical Models in Population Biology: Ecological, Epidemic, and Evolutionary Dynamics*, 1st edition, Springer, 2024. https://doi.org/10.1007/978-3-031-64795-6
14. E. D'Aniello, S. Elaydi, The structure of ω-limit sets of asymptotically non-autonomous discrete dynamical systems, *Discrete Contin. Dyn. Syst. Ser. B*, **25** (2020), 903–915. https://doi.org/10.3934/dcdsb.2019195
15. S. Elaydi, R. J. Sacker, Global stability of periodic orbits of non-autonomous difference equations and population biology, *J. Differ. Equations*, **208** (2005), 258–273. https://doi.org/10.1016/j.jde.2003.10.024
16. J. Li, Z. Ma, F. Brauer, Global analysis of discrete-time SI and SIS epidemic models, *Math. Biosci. Eng.*, **4** (2007), 699–710. https://doi.org/10.3934/mbe.2007.4.699
17. J. E. Franke, A. Yakubu, Periodically forced discrete-time SIS epidemic model with disease-induced mortality, *Math. Biosci. Eng.*, **8** (2011), 385–408. https://doi.org/10.3934/mbe.2011.8.385
18. I. Papst, D. J. D. Earn, Invariant predictions of epidemic patterns from radically different forms of seasonal forcing, *J. R. Soc. Interface*, **16** (2019), 20190202. https://doi.org/10.1098/rsif.2019.0202
19. L. J. S. Allen, P. van den Driessche, The basic reproduction number in some discrete-time epidemic models, *J. Differ. Equations Appl.*, **14** (2008), 1127–1147. https://doi.org/10.1080/10236190802332308
20. F. Brauer, Z. Feng, C. Castillo-Chávez, Discrete epidemic models, *Math. Biosci. Eng.*, **7** (2010), 1–15. http://doi.org/10.3934/mbe.2010.7.1
21. P. van den Driessche, J. Watmough, Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission, *Math. Biosci.*, **180** (2002), 29–48. https://doi.org/10.1016/S0025-5564(02)00108-6
22. O. Diekmann, J. A. P. Heesterbeek, J. A. J. Metz, On the definition and the computation of the basic reproduction ratio R_0 in models for infectious diseases in heterogeneous populations, *J. Math. Biol.*, **28** (1990), 365–382. https://doi.org/10.1007/bf00178324
23. N. J. Cunniffe, C. A. Gilligan, A theoretical framework for biological control of soil-borne plant pathogens: Identifying effective strategies, *J. Theor. Biol.*, **278** (2011), 32–43. https://doi.org/10.1016/j.jtbi.2011.02.023
24. N. G. Becker, *Analysis of Infectious Disease Data*, 1st edition, Chapman and Hall/CRC, 1989. https://doi.org/10.1201/9781315137407
25. N. T. J. Bailey, *The Elements of Stochastic Processes with Applications to the Natural Sciences*, Wiley, 1964.
26. S. N. Elaydi, *Discrete Chaos: With Applications in Science and Engineering*, 2nd edition, Chapman and Hall/CRC, 2007. https://doi.org/10.1201/9781420011043

27. K. Mokni, S. Elaydi, M. CH-Chaoui, A. Eladdadi, Discrete evolutionary population models: A new approach, *J. Biol. Dyn.*, **14** (2020), 454–478. https://doi.org/10.1080/17513758.2020.1772997

28. C. Castillo-Chavez, A. Yakubu, Discrete-time S-I-S models with complex dynamics, *Nonlinear Anal. Theory Methods Appl.*, **47** (2001), 4753–4762. https://doi.org/10.1016/S0362-546X(01)00587-9

29. A. Yakubu, J. E. Franke, Discrete-time SIS epidemic model in a seasonal environment, *SIAM J. Appl. Math.*, **66** (2006), 1563–1587. https://doi.org/10.1137/050638345

AIMS Press