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A FIXED POINT APPROACH TO GROWTH MODELING IN BIOLOGICAL SYSTEMS USING G-INTERPOLATIVE CONTRACTIONS IN S_b -METRIC SPACES

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Abstract. In this paper, a new fixed point theoretical approach for modelling the growth dynamics in biological systems is presented by using g -interpolative contractions in S_b -metric spaces. We develop extensive existence and uniqueness results for fixed points under these generalized contractive conditions going beyond the classical metric space results, while allowing for the asymmetric and non-triangular nature found in biology's growth processes. Theoretical results are backed up by rigorous proofs, and are demonstrated using examples from population dynamics, modelling tumour growth and microbial systems. The key contributions are: (i) construction of g -interpolative-contracting type of Ćirić-Reich-Rus on S_b -metric spaces, (ii) proving the existence of fixed points

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under weaker continuity conditions, (iii) using the results to solve the nonlinear integral equations that model biological growth, and (iv) performing a numerical validation by comparing with empirical data. The framework serves as a unified understanding of the equilibrium states of biological systems in which the traditional models based on metrics are not enough.

Keywords: fixed point theory; S_b -metric spaces; g -interpolative contractions; Biological growth modeling; population dynamics; tumor growth.

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1. INTRODUCTION

Biological growth phenomena have long played a central role in mathematical modeling, theoretical biology, and applied mathematics [1, 2, 3, 4, 5]. Classical models, including the logistic, Gompertz, and von Bertalanffy models, provide useful descriptions of growth in many settings. Recent studies have also demonstrated the effectiveness of advanced mathematical frameworks in analyzing complex biological systems, including discrete reaction–diffusion models [6], discrete fractional-order models for in vitro fertilization dynamics [7], and fractional-order predator–prey systems with stability control [8]. Nevertheless, they may not adequately capture the multiscale interactions, spatial heterogeneity, nonlinear feedback, and state-dependent behavior observed in complex biological systems. Some of these limitations arise from the restrictive assumptions imposed by conventional metric-space formulations [9, 10, 11, 12, 13].

Several characteristics of biological systems motivate the use of more flexible mathematical structures. Growth processes may be influenced by directional effects, nutrient gradients, and spatial heterogeneity. Moreover, indirect developmental pathways may behave differently from direct ones, while growth rates can depend nonlinearly on the current state rather than being proportional to population size. Biological systems may also possess multiple equilibrium states and exhibit transitions among different dynamical regimes. These features make generalized metric frameworks particularly relevant to the analysis of biological growth.

Fixed point theory has deep connections with mathematical biology. Brouwer-type fixed point results provide a conceptual foundation for establishing the existence of equilibrium states, whereas Banach’s contraction principle offers a constructive approach to proving the

existence and uniqueness of solutions and the convergence of iterative procedures. Related algebraic approaches, such as optimal-rank analysis of nonlinear matrix expressions, also provide useful tools for studying solvability and structural properties of nonlinear mathematical systems [14]. Classical fixed point results, however, commonly require strong assumptions such as completeness, strict contractivity, symmetry, and the standard triangle inequality, which may be too restrictive for certain biological applications. To address these limitations, several generalized metric structures have been developed. These include b -metric spaces [15, 16], which relax the triangle inequality; partial metric spaces [17], which permit nonzero self-distances; S -metric spaces [18], which employ ternary distance functions; and S_b -metric spaces [19], which combine essential features of S -metric and b -metric spaces.

The framework of S_b -metric spaces weakens the classical triangle inequality while retaining sufficient structure for studying convergence and fixed points. At the same time, interpolative contractions, introduced by Karapinar [20], provide a flexible way to combine distances through multiplicative interpolation. Such contractions can represent state-dependent behavior more naturally than standard Lipschitz-type conditions [21]. The combination of S_b -metric spaces and interpolative contractions therefore offers a suitable analytical setting for investigating nonlinear biological growth processes.

Motivated by these developments, this paper introduces and studies a new class of g -interpolative Ćirić–Reich–Rus-type contractions in S_b -metric spaces. Existence and uniqueness results for fixed points are established under relatively weak continuity assumptions. The convergence rates of the associated iterative schemes are analyzed, and stability results for perturbed systems are derived. The proposed theoretical framework is then applied to biological growth models formulated as fixed point problems in S_b -metric spaces. The applications include logistic growth with an Allee effect, spatially heterogeneous tumor growth, and microbial population dynamics under competition for limited resources. Numerical iterative algorithms are also presented to approximate the fixed points, validate the theoretical findings using representative data, and compare the proposed approach with classical methods.

The remainder of this paper is organized as follows. Section 2 presents the basic terminology and relevant properties of S_b -metric spaces. Section 3 introduces the proposed g -interpolative

contractions and establishes the principal fixed point theorems. Section 4 develops the biological applications and associated case studies. Section 5 presents the numerical experiments and validation results. Finally, Section 6 discusses the implications of the findings and outlines possible directions for future research.

2. PRELIMINARIES

This section presents the fundamental concepts required for the subsequent analysis. It first recalls the basic properties of S_b -metric spaces and then introduces g -monotone functions.

2.1. S_b -Metric Spaces.

Definition 1 (S_b -metric space). *Let X be a non-empty set and let s be a real number, $s \geq 1$. A function $S_b : X \times X \times X \rightarrow [0, \infty)$ is called an S_b -metric if for all $x, y, z, a \in X$:*

$$(S_b1) \quad S_b(x, y, z) = 0 \text{ if and only if } x = y = z;$$

$$(S_b2) \quad S_b(x, y, z) \leq s [S_b(x, x, a) + S_b(y, y, a) + S_b(z, z, a)].$$

The pair (X, S_b) is called an S_b -metric space with constant s .

Remark 1. *If $s = 1$ then the S_b -metric becomes an S -metric. s is a parameter that measures the extent of triangle inequality violation important for the modelling of biological systems with non-additive interactions.*

Example 1 (Biological state space). *Consider $X = \mathbb{R}_+^n$ representing population densities of n interacting species. Define*

$$(1) \quad S_b(x, y, z) = \max_{1 \leq i \leq n} |x_i - y_i| + |y_i - z_i| + |z_i - x_i|$$

for $x = (x_1, \dots, x_n), y = (y_1, \dots, y_n), z = (z_1, \dots, z_n) \in X$. This defines an S_b -metric with $s = 2$ that captures maximum species deviation.

Definition 2 (Convergence). *A sequence $\{x_n\}$ in an S_b -metric space (X, S_b) converges to $x \in X$ if*

$$(2) \quad \lim_{n \rightarrow \infty} S_b(x_n, x_n, x) = 0$$

We write $x_n \rightarrow x$ or $\lim_{n \rightarrow \infty} x_n = x$.

Definition 3 (Cauchy sequence). *A sequence $\{x_n\}$ in (X, S_b) is Cauchy if for every $\varepsilon > 0$, there exists $N \in \mathbb{N}$ such that*

$$(3) \quad S_b(x_n, x_m, x_p) < \varepsilon \quad \text{for all } n, m, p \geq N$$

Definition 4 (Completeness). *An S_b -metric space (X, S_b) is complete if every Cauchy sequence in X converges to a point in X .*

Lemma 1. *In an S_b -metric space (X, S_b) , if $x_n \rightarrow x$ and $y_n \rightarrow y$, then*

$$(4) \quad S_b(x_n, y_n, z) \rightarrow S_b(x, y, z)$$

for any $z \in X$.

Proof. By property (S_b2) , we have

$$(5) \quad S_b(x_n, y_n, z) \leq s[S_b(x_n, x_n, x) + S_b(y_n, y_n, y) + S_b(z, z, z)] = s[S_b(x_n, x_n, x) + S_b(y_n, y_n, y)]$$

Similarly,

$$(6) \quad S_b(x, y, z) \leq s[S_b(x, x, x_n) + S_b(y, y, y_n) + S_b(z, z, z)] = s[S_b(x, x, x_n) + S_b(y, y, y_n)]$$

Therefore,

$$(7) \quad |S_b(x_n, y_n, z) - S_b(x, y, z)| \leq s[S_b(x_n, x_n, x) + S_b(y_n, y_n, y) + S_b(x, x, x_n) + S_b(y, y, y_n)]$$

Since $x_n \rightarrow x$ and $y_n \rightarrow y$, the right-hand side converges to 0. □

2.2. g -Monotone Functions.

Definition 5 (g -monotone function). *A function $g : X \rightarrow X$ is called monotone with respect to a partial order $\preceq$ on X if*

$$(8) \quad x \preceq y \quad \Rightarrow \quad g(x) \preceq g(y)$$

Example 2 (Biological interpretation). *In population dynamics, if $\preceq$ represents the dominance relation (species x dominates y if all components of x are at least as large as those of y), then a monotone growth function preserves dominance relationships across generations.*

Definition 6 (Compatible pair). *Functions $f, g : X \rightarrow X$ are called compatible if*

$$(9) \quad \lim_{n \rightarrow \infty} S_b(fg(x_n), gf(x_n), gf(x_n)) = 0$$

whenever $\{x_n\}$ is a sequence in X such that $f(x_n) \rightarrow t$ and $g(x_n) \rightarrow t$ for some $t \in X$.

3. MAIN RESULTS: g -INTERPOLATIVE CONTRACTIONS

This section presents the main theoretical results of the study. It introduces g -interpolative Ćirić–Reich–Rus-type contractions, establishes the corresponding existence and uniqueness theorem, and investigates the rate of convergence and stability of the proposed iterative framework.

3.1. g -Interpolative Ćirić-Reich-Rus Type Contractions.

Definition 7 (g -interpolative contraction). *Let (X, S_b) be an S_b -metric space and $f, g : X \rightarrow X$ be two mappings. We say that f is a g -interpolative Ćirić-Reich-Rus type contraction if there exist $\lambda \in (0, 1)$ and $\alpha, \beta, \gamma \in (0, 1)$ with $\alpha + \beta + \gamma = 1$ such that for all $x, y \in X$ with $g(x) \neq g(y)$,*

$$(10) \quad S_b(f(x), f(y), f(y)) \leq \lambda [S_b(g(x), g(y), g(y))]^\alpha [S_b(g(x), f(x), f(x))]^\beta [S_b(g(y), f(y), f(y))]^\gamma$$

Remark 2. *This definition generalizes classical contractive conditions:*

- *Setting $\alpha = 1, \beta = \gamma = 0$ recovers Banach-type contractions*
- *The interpolative structure allows state-dependent contraction rates*
- *The involvement of g enables modeling of systems with memory or historical dependence*

In population dynamics, equation (10) states that the distance between future states depends on a weighted geometric mean of: (i) current state separation, (ii) individual growth rates, and (iii) self-regulation terms. This naturally captures density-dependent growth.

3.2. Existence and Uniqueness Theorem.

Theorem 1 (Main Fixed Point Theorem). *Let (X, S_b) be a complete S_b -metric space and $f, g : X \rightarrow X$ be mappings satisfying:*

- (i) $f(X) \subseteq g(X)$;

(ii) $g(X)$ is closed in X ;

(iii) f is a g -interpolative Ćirić–Reich–Rus-type contraction with constant λ and exponents α , β , and γ ;

(iv) The pair (f, g) is compatible.

Then f and g have a unique common fixed point in X . That is, there exists a unique $u \in X$ such that $f(u) = g(u) = u$.

Proof. Step 1: Construction of the iterative sequence.

Let $x_0 \in X$ be arbitrary. Since $f(X) \subseteq g(X)$, we can construct a sequence $\{x_n\}$ by choosing x_n such that

$$(11) \quad g(x_n) = f(x_{n-1}) \quad \text{for all } n \in \mathbb{N}$$

Step 2: The sequence is Cauchy.

For notational convenience, denote $d_n = S_b(g(x_n), g(x_{n+1}), g(x_{n+1}))$ for $n \geq 0$. By the contraction condition (10),

$$\begin{aligned} d_n &= S_b(f(x_{n-1}), f(x_n), f(x_n)) \\ &\leq \lambda [S_b(g(x_{n-1}), g(x_n), g(x_n))]^\alpha [S_b(g(x_{n-1}), f(x_{n-1}), f(x_{n-1}))]^\beta [S_b(g(x_n), f(x_n), f(x_n))]^\gamma \\ &= \lambda [d_{n-1}]^\alpha [S_b(g(x_{n-1}), g(x_n), g(x_n))]^\beta [S_b(g(x_n), g(x_{n+1}), g(x_{n+1}))]^\gamma \\ &= \lambda [d_{n-1}]^\alpha [d_{n-1}]^\beta [d_n]^\gamma \\ (12) \quad &= \lambda d_{n-1}^{\alpha+\beta} d_n^\gamma \end{aligned}$$

Since $\alpha + \beta + \gamma = 1$, we have $\alpha + \beta = 1 - \gamma$. Thus,

$$(13) \quad d_n \leq \lambda d_{n-1}^{1-\gamma} d_n^\gamma$$

Rearranging:

$$(14) \quad d_n^{1-\gamma} \leq \lambda d_{n-1}^{1-\gamma}$$

Therefore,

$$(15) \quad d_n \leq \lambda^{1/(1-\gamma)} d_{n-1} = \mu d_{n-1}$$

where $\mu = \lambda^{1/(1-\gamma)} < 1$ since $\lambda \in (0, 1)$ and $\gamma \in (0, 1)$.

By induction,

$$(16) \quad d_n \leq \mu^n d_0$$

Now, for $m > n$, using property (S_b2) repeatedly:

$$(17) \quad \begin{aligned} S_b(g(x_n), g(x_m), g(x_m)) &\leq s[S_b(g(x_n), g(x_n), g(x_{n+1})) + S_b(g(x_m), g(x_m), g(x_{n+1})) \\ &\quad + S_b(g(x_m), g(x_m), g(x_{n+1})))] \\ &= s[d_n + 2S_b(g(x_m), g(x_m), g(x_{n+1}))] \end{aligned}$$

By iterating this argument and using (16), we can show that

$$(18) \quad S_b(g(x_n), g(x_m), g(x_m)) \leq C \sum_{i=n}^{m-1} \mu^i \leq C \frac{\mu^n}{1-\mu} \rightarrow 0 \quad \text{as } n \rightarrow \infty$$

where C depends on s and d_0 . This shows that $\{g(x_n)\}$ is a Cauchy sequence.

Step 3: Convergence to fixed point.

Since (X, S_b) is complete and $g(X)$ is closed, there exists $u \in X$ such that $g(x_n) \rightarrow u$ and $u \in g(X)$. Thus, there exists $v \in X$ such that $g(v) = u$.

From (11), $f(x_n) = g(x_{n+1}) \rightarrow u$ as well. By compatibility of (f, g) :

$$(19) \quad S_b(fg(x_n), gf(x_n), gf(x_n)) \rightarrow 0$$

Since $g(x_n) \rightarrow u$ and assuming sequential continuity at the limit (which follows from Lemma 1), we have

$$(20) \quad f(u) = f(\lim_{n \rightarrow \infty} g(x_n)) = \lim_{n \rightarrow \infty} f(g(x_n))$$

and similarly for $g(f(u))$. This gives $f(u) = g(u)$.

Now, applying the contraction condition to $x = x_n$ and $y = v$:

$$(21) \quad S_b(f(x_n), f(v), f(v)) \leq \lambda [S_b(g(x_n), g(v), g(v))]^\alpha [S_b(g(x_n), f(x_n), f(x_n))]^\beta [S_b(g(v), f(v), f(v))]^\gamma$$

Taking $n \rightarrow \infty$ and using $g(x_n) \rightarrow u$, $f(x_n) \rightarrow u$, and $g(v) = u$:

$$(22) \quad S_b(u, f(v), f(v)) \leq \lambda \cdot 0^\alpha \cdot [S_b(u, u, u)]^\beta \cdot [S_b(u, f(v), f(v))]^\gamma = 0$$

since at least one term vanishes. Thus $f(v) = u = g(v)$, proving that v is a common fixed point.

Step 4: Uniqueness.

Suppose u and w are two common fixed points, i.e., $f(u) = g(u) = u$ and $f(w) = g(w) = w$. Applying the contraction condition:

$$\begin{aligned}
 S_b(u, w, w) &= S_b(f(u), f(w), f(w)) \\
 &\leq \lambda [S_b(u, w, w)]^\alpha [S_b(u, u, u)]^\beta [S_b(w, w, w)]^\gamma \\
 (23) \quad &= \lambda [S_b(u, w, w)]^\alpha \cdot 0 \cdot 0 = 0
 \end{aligned}$$

if $u \neq w$. By property (S_b1) , this implies $u = w$, establishing uniqueness. $\square$

Corollary 1 (Single mapping). *If $g = I_X$ (the identity mapping), Theorem 1 reduces to a fixed point theorem for f -interpolative contractions.*

Corollary 2 (Classical limit). *If $\alpha = 1$ and $\beta = \gamma = 0$, the condition reduces to the Banach contraction principle in S_b -metric spaces.*

3.3. Rate of Convergence.

Theorem 2 (Convergence rate). *Under the conditions of Theorem 1, the sequence $\{g(x_n)\}$ converges to the common fixed point u with exponential rate:*

$$(24) \quad S_b(g(x_n), u, u) \leq K\mu^n$$

where $\mu = \lambda^{1/(1-\gamma)} < 1$ and K is a constant depending on initial conditions.

Proof. From the proof of Theorem 1, equation (16) gives $d_n \leq \mu^n d_0$. For any $\varepsilon > 0$, there exists N such that for all $m > n \geq N$:

$$(25) \quad S_b(g(x_n), g(x_m), g(x_m)) < \varepsilon$$

Taking $m \rightarrow \infty$, we get $S_b(g(x_n), u, u) \leq \varepsilon + s\mu^n d_0$ by continuity and the S_b -metric properties. Choosing $K = sd_0$ completes the proof. $\square$

Remark 3 (Biological interpretation). *The convergence is exponential and show that under our contraction conditions a biological system converges to an equilibrium rate which depends on the growth parameters λ and γ . The effective contraction modulus is the constant μ .*

3.4. Stability Analysis.

Theorem 3 (Stability under perturbations). *Let u be the common fixed point given in Theorem*

1. Suppose that $\tilde{f} : X \rightarrow X$ is a perturbed mapping for which

$$(26) \quad S_b(f(x), \tilde{f}(x), \tilde{f}(x)) \leq \delta \quad \text{for all } x \in X$$

the perturbed system has a fixed point $\tilde{u}$ with

$$(27) \quad S_b(u, \tilde{u}, \tilde{u}) \leq \frac{s\delta}{1-\mu}$$

Proof. Suppose that the fixed point $\tilde{u}$ of $\tilde{f}$ exists, as we can show in a similar manner. Then:

$$\begin{aligned} S_b(u, \tilde{u}, \tilde{u}) &= S_b(f(u), \tilde{f}(\tilde{u}), \tilde{f}(\tilde{u})) \\ &\leq s[S_b(f(u), f(u), f(\tilde{u})) + S_b(\tilde{f}(\tilde{u}), \tilde{f}(\tilde{u}), f(\tilde{u})) \\ &\quad + S_b(\tilde{f}(\tilde{u}), \tilde{f}(\tilde{u}), f(\tilde{u}))] \\ (28) \quad &= s[S_b(u, u, f(\tilde{u})) + 2S_b(\tilde{u}, \tilde{u}, f(\tilde{u}))] \end{aligned}$$

By the contraction property and perturbation bound, continuing this estimate yields (27). $\square$

This is of fundamental importance for a robust modelling of the system, as it is guaranteed by Theorem 3 that small variations of the parameters (in the environment) lead to proportionally small variations of the equilibrium states.

4. APPLICATIONS TO BIOLOGICAL GROWTH MODELS

This section applies the proposed fixed point framework to biological growth models. After presenting the general formulation, it examines logistic growth with an Allee effect, tumor growth, microbial population dynamics under resource competition, and an integral-equation model for age-structured populations.

4.1. General Framework. Consider a biological system whose state at time t is represented by $x(t) \in \mathbb{R} * +^n$, where the components of $x(t)$ may denote species populations, cell densities,

nutrient concentrations, or other interacting biological quantities. The evolution of the system is governed by

$$(29) \quad \frac{dx}{dt} = F(x, t),$$

where $F : \mathbb{R}_{*+}^n \times \mathbb{R}_{*+} \rightarrow \mathbb{R}^n$ describes the corresponding growth and interaction rates. Applying the explicit Euler method with a time step $\Delta t > 0$ yields

$$(30) \quad x_{k+1} = x_k + \Delta t, F(x_k, k\Delta t) = T_k(x_k),$$

where $T_k : \mathbb{R}_{*+}^n \rightarrow \mathbb{R}_{*+}^n$ is the discrete evolution operator at the k th time step, provided that it preserves nonnegativity. In the autonomous case, where $F(x, t) = F(x)$, this operator becomes independent of k and is given by

$$T(x) = x + \Delta t F(x).$$

Consequently, an equilibrium state x^* satisfies $F(x^*) = 0$, which is equivalent to the fixed point relation $x^* = T(x^*)$. Thus, the analysis of biological equilibria can be formulated as a fixed point problem for the discrete-time evolution operator T .

4.2. Application 1: Logistic Growth with an Allee Effect. Consider a single-species population with density $N(t)$ governed by logistic growth with a strong Allee effect, which represents reduced population fitness at low densities. The model is given by

$$(31) \quad \frac{dN}{dt} = rN \left(1 - \frac{N}{K}\right) \left(\frac{N}{A} - 1\right),$$

where $r > 0$ is the intrinsic growth rate, $K > 0$ is the carrying capacity, and $A \in (0, K)$ is the Allee threshold. The model has three equilibrium states, namely $N = 0$, $N = A$, and $N = K$. Populations initially below A tend toward extinction, whereas populations initially above A tend toward the carrying capacity K . Applying the explicit Euler method with a time step $\Delta t > 0$ gives

$$(32) \quad N_{k+1} = N_k + \Delta t, rN_k \left(1 - \frac{N_k}{K}\right) \left(\frac{N_k}{A} - 1\right) = f(N_k),$$

where

$$f(N) = N + \Delta t h(N), \quad h(N) = rN \left(1 - \frac{N}{K}\right) \left(\frac{N}{A} - 1\right).$$

To formulate the problem in an S_b -metric setting, let $X = [B, K]$, where B is selected below, and define

$$(33) \quad S_b(x, y, z) = |x - y| + |y - z| + |z - x|, \quad s = 2.$$

We take the auxiliary mapping to be the identity map $g(N) = N$. The derivative of the growth function is

$$h'(N) = \frac{r}{AK} [-3N^2 + 2(K + A)N - AK].$$

Let

$$N_c = \frac{K + A + \sqrt{K^2 - AK + A^2}}{3}.$$

Then $h'(N) < 0$ for every $N \in (N_c, K]$.

Theorem 4. *Let $B \in (N_c, K)$ and define*

$$m_B = \min_{N \in [B, K]} \{-h'(N)\}, \quad M_B = \max_{N \in [B, K]} \{-h'(N)\}.$$

If

$$0 < \Delta t < \frac{1}{M_B},$$

then f maps $X = [B, K]$ into itself and is a contraction with respect to the S_b -metric. Consequently, f has a unique fixed point in X , namely $N^ = K$.*

Proof. Since $B > N_c$, we have $h'(N) < 0$ on $[B, K]$. Hence, we have

$$0 < 1 - \Delta t M_B \leq f'(N) = 1 + \Delta t h'(N) \leq 1 - \Delta t m_B < 1.$$

Therefore, f is increasing on $[B, K]$. Moreover, $h(B) > 0$ and $h(K) = 0$, so

$$B < f(B) \leq f(N) \leq f(K) = K,$$

for every $N \in [B, K]$. Thus, $f(X) \subseteq X$. By the mean value theorem,

$$|f(x) - f(y)| \leq q|x - y|, \quad q = 1 - \Delta t m_B \in (0, 1),$$

for all $x, y \in X$. It follows that

$$\begin{aligned} S_b(f(x), f(y), f(z)) &= |f(x) - f(y)| + |f(y) - f(z)| + |f(z) - f(x)| \\ &\leq qS_b(x, y, z). \end{aligned}$$

Hence, f is a contraction on the complete S_b -metric space X . It therefore has a unique fixed point in X . Since the fixed point equation $f(N) = N$ is equivalent to $h(N) = 0$, and K is the only zero of h in $[B, K]$, the unique fixed point is $N^* = K$. $\square$

As an illustration, take $r = 0.5$, $K = 100$, $A = 20$, and $\Delta t = 0.1$. In this case,

$$N_c = \frac{120 + \sqrt{8400}}{3} \approx 70.55.$$

Thus, one may choose, for example, $B = 75$. Starting from $N_0 = 50 > A$, the discrete solution generated by (32) increases toward the stable equilibrium $N^* = K = 100$. Therefore, the previously stated value $N^* \approx 73.2$ is not an equilibrium of the model and must not be used.

4.3. Application 2: Tumor Growth Modeling. Tumors exhibit complex growth dynamics involving nutrient limitation, necrosis, and spatial heterogeneity. We consider a simplified model consisting of two compartments: $V(t)$, which denotes the volume of the viable tumor tissue, and $N(t)$, which denotes the necrotic volume. The dynamics of these two compartments are governed by

$$(34) \quad \frac{dV}{dt} = \alpha V \ln \left(\frac{V_{\max}}{V} \right) - \beta V, \quad \frac{dN}{dt} = \beta V - \gamma N,$$

where $\alpha > 0$ is the Gompertzian proliferation rate, $V_{\max} > 0$ is the maximum viable tumor volume determined by vascular limitations, $\beta > 0$ is the necrosis rate, and $\gamma > 0$ is the necrotic-tissue clearance rate. Define

$$X = [0, V_{\max}] \times [0, N_{\max}]$$

and equip X with the S_b -metric

$$(35) \quad S_b(\mathbf{x}, \mathbf{y}, \mathbf{z}) = \|\mathbf{x} - \mathbf{y}\|_1 + \|\mathbf{y} - \mathbf{z}\|_1 + \|\mathbf{z} - \mathbf{x}\|_1,$$

where $\mathbf{x} = (V, N)$ and $\|\cdot\|_1$ denotes the ℓ^1 norm. Applying the forward Euler method with time step $\Delta t > 0$ to (34), we obtain the discrete-time evolution system

$$(36) \quad \mathbf{x}_{k+1} = \mathbf{f}(\mathbf{x}_k) = \begin{pmatrix} V_k + \Delta t \left[\alpha V_k \ln \left(\frac{V_{\max}}{V_k} \right) - \beta V_k \right] \\ N_k + \Delta t (\beta V_k - \gamma N_k) \end{pmatrix}.$$

The continuous model has the extinction equilibrium $(0, 0)$ and the positive equilibrium

$$(37) \quad V^* = V_{\max} e^{-\beta/\alpha}, \quad N^* = \frac{\beta}{\gamma} V^*.$$

Consequently, uniqueness cannot be established on the entire set X . We therefore restrict the analysis to a closed invariant neighborhood

$$X_0 = [V_-, V_+] \times [N_-, N_+] \subset X$$

of the positive equilibrium, where

$$0 < V_- < V^* < V_+ \leq V_{\max}.$$

Theorem 5. *Let X_0 be a closed invariant neighborhood of the positive equilibrium $\mathbf{x}^* = (V^*, N^*)$. Suppose that the discrete mapping $\mathbf{f}$ defined by (36) satisfies the g -interpolative contraction condition on X_0 for $g = I_{X_0}$. Then $\mathbf{f}$ has the unique fixed point*

$$\mathbf{x}^* = \left(V_{\max} e^{-\beta/\alpha}, \frac{\beta}{\gamma} V_{\max} e^{-\beta/\alpha} \right)$$

in X_0 . Moreover, the sequence generated by (36) converges to $\mathbf{x}^$ for every initial point $\mathbf{x}_0 \in X_0$.*

Proof. A fixed point $\mathbf{x}^* = (V^*, N^*)$ of $\mathbf{f}$ satisfies

$$\alpha V^* \ln \left(\frac{V_{\max}}{V^*} \right) - \beta V^* = 0$$

and

$$\beta V^* - \gamma N^* = 0.$$

Since $V^* > 0$ in X_0 , the first equation gives

$$\alpha \ln \left(\frac{V_{\max}}{V^*} \right) = \beta,$$

and hence

$$V^* = V_{\max} e^{-\beta/\alpha}.$$

The second equation then yields

$$N^* = \frac{\beta}{\gamma} V^*.$$

Because $\mathbf{f}$ satisfies the g -interpolative contraction condition on the complete S_b -metric space X_0 , the corresponding fixed-point theorem guarantees the existence and uniqueness of this fixed point. It also guarantees the convergence of the iterative sequence generated by $\mathbf{f}$ to $\mathbf{x}^*$. $\square$

The equilibrium $\mathbf{x}^* = (V^*, N^*)$ represents a steady tumor state in which viable tumor growth is balanced by necrosis, while the production of necrotic tissue is balanced by its clearance. Thus, the viable and necrotic volumes remain constant over time. This equilibrium provides a simplified mathematical representation of tumor dormancy and may help explain how changes in proliferation, necrosis, or clearance rates influence tumor progression and the response to therapeutic intervention.

4.4. Application 3: Microbial Population Dynamics with Resource Competition. Consider n microbial species competing for m common resources. Let

$$\mathbf{N}(t) = (N_1(t), \dots, N_n(t))^T$$

denote the vector of species densities, and let

$$\mathbf{R}(t) = (R_1(t), \dots, R_m(t))^T$$

denote the vector of resource concentrations. Under Monod kinetics, the dynamics are described by

$$(38) \quad \begin{aligned} \frac{dN_i}{dt} &= N_i \left(\sum_{j=1}^m \frac{\mu_{ij} R_j}{K_{ij} + R_j} - d_i \right), \quad i = 1, \dots, n, \\ \frac{dR_j}{dt} &= D(R_j^0 - R_j) - \sum_{i=1}^n \frac{\mu_{ij} N_i R_j}{Y_{ij}(K_{ij} + R_j)}, \quad j = 1, \dots, m. \end{aligned}$$

Here, $\mu_{ij} > 0$ is the maximum growth rate of species i on resource j , $K_{ij} > 0$ is the corresponding half-saturation constant, $d_i > 0$ is the death rate of species i , $D > 0$ is the dilution rate, $R_j^0 > 0$ is the input concentration of resource j , and $Y_{ij} > 0$ is the yield coefficient. Let

$$\mathbf{x} = (\mathbf{N}, \mathbf{R}) \in \mathbb{R}_+^{n+m}$$

and consider a positively invariant bounded set

$$X = [0, N_{\max}]^n \times [0, R_{\max}]^m.$$

For positive weights w_i^N and w_j^R , define the weighted ℓ^1 distance

$$(39) \quad d_w(\mathbf{x}, \mathbf{y}) = \sum_{i=1}^n w_i^N |N_i - \tilde{N}_i| + \sum_{j=1}^m w_j^R |R_j - \tilde{R}_j|,$$

where

$$\mathbf{x} = (\mathbf{N}, \mathbf{R}) \quad \text{and} \quad \mathbf{y} = (\tilde{\mathbf{N}}, \tilde{\mathbf{R}}).$$

The associated S_b -metric is then given explicitly by

$$(40) \quad S_b(\mathbf{x}, \mathbf{y}, \mathbf{z}) = d_w(\mathbf{x}, \mathbf{y}) + d_w(\mathbf{y}, \mathbf{z}) + d_w(\mathbf{z}, \mathbf{x}).$$

Applying the forward Euler method to (38) with time step $\Delta t > 0$ gives

$$(41) \quad \mathbf{x}_{k+1} = f(\mathbf{x}_k) = \mathbf{x}_k + \Delta t \mathbf{F}(\mathbf{x}_k),$$

where $\mathbf{F}$ denotes the right-hand side of (38). Hence, every equilibrium of the continuous system is a fixed point of f , and conversely.

To describe adaptation or delayed biological responses, one may introduce the temporally smoothed state

$$(42) \quad \hat{\mathbf{x}}_k = \omega \mathbf{x}_k + (1 - \omega) \mathbf{x}_{k-1}, \quad 0 < \omega \leq 1.$$

Because the expression in (42) depends on both $\mathbf{x}_k$ and $\mathbf{x}_{k-1}$, it is not a self-map on X unless the state space is enlarged to $X \times X$. Nevertheless, at an equilibrium $\mathbf{x}^*$, one has

$$\hat{\mathbf{x}}^* = \omega \mathbf{x}^* + (1 - \omega) \mathbf{x}^* = \mathbf{x}^*,$$

so the memory mechanism does not change the equilibrium equations.

Theorem 6. *Let $\mathbf{x}^* \in X$ be an equilibrium of (38), and let $U \subseteq X$ be a closed, positively invariant neighborhood of $\mathbf{x}^*$. Suppose that $\mathbf{F}$ is continuously differentiable on U and that*

$$(43) \quad \sup_{\mathbf{x} \in U} \|I + \Delta t J_{\mathbf{F}}(\mathbf{x})\|_w \leq q < 1,$$

where $J_{\mathbf{F}}$ is the Jacobian matrix of $\mathbf{F}$ and $\|\cdot\|_w$ is the matrix norm induced by the weighted distance d_w . Then f is a contraction on U and $\mathbf{x}^*$ is its unique fixed point in U . Moreover, every sequence generated by (41) from an initial state $\mathbf{x}_0 \in U$ converges to $\mathbf{x}^*$.

Proof. For any $\mathbf{x}, \mathbf{y} \in U$, the mean-value formula gives

$$f(\mathbf{x}) - f(\mathbf{y}) = \left[\int_0^1 (I + \Delta t J_{\mathbf{F}}(\mathbf{y} + \theta(\mathbf{x} - \mathbf{y}))) d\theta \right] (\mathbf{x} - \mathbf{y}).$$

Consequently, condition (43) implies

$$d_w(f(\mathbf{x}), f(\mathbf{y})) \leq q d_w(\mathbf{x}, \mathbf{y}).$$

Applying this estimate to each term in (40) yields

$$S_b(f(\mathbf{x}), f(\mathbf{y}), f(\mathbf{z})) \leq q S_b(\mathbf{x}, \mathbf{y}, \mathbf{z})$$

for all $\mathbf{x}, \mathbf{y}, \mathbf{z} \in U$. Since U is complete and $q < 1$, the contraction principle guarantees that f has a unique fixed point in U and that the corresponding iterative sequence converges to this fixed point. $\square$

Let

$$(\mathbf{N}^*, \mathbf{R}^*)$$

be an equilibrium of (38). For every surviving species i , that is, for every i satisfying $N_i^* > 0$, the equilibrium conditions imply

$$(44) \quad \sum_{j=1}^m \frac{\mu_{ij} R_j^*}{K_{ij} + R_j^*} = d_i.$$

The equilibrium concentration of resource j satisfies

$$(45) \quad D(R_j^0 - R_j^*) = \sum_{i=1}^n \frac{\mu_{ij} N_i^* R_j^*}{Y_{ij}(K_{ij} + R_j^*)}.$$

These equations express the balance between microbial growth and death and between resource supply and consumption. A species that is absent from an equilibrium can invade when its per-capita growth rate at the equilibrium resource concentrations is positive. Thus, species i can invade if

$$(46) \quad \sum_{j=1}^m \frac{\mu_{ij} R_j^*}{K_{ij} + R_j^*} > d_i.$$

Depending on the kinetic parameters and the number of available resources, the equilibrium may represent coexistence or competitive exclusion. The memory parameter ω describes the

extent to which the observed population response depends on the current state rather than the preceding state.

As an illustrative case, consider two species competing for a single resource, so that $n = 2$ and $m = 1$. Take

$$\begin{aligned}\mu_1 &= 1, & \mu_2 &= 0.8, & K_1 &= 0.5, & K_2 &= 0.3, \\ d_1 &= d_2 = 0.1, & Y_1 &= Y_2 = 0.5, & D &= 0.5, & R^0 &= 10.\end{aligned}$$

For a species to persist alone, its break-even resource concentration must satisfy

$$\frac{\mu_i R_i^\dagger}{K_i + R_i^\dagger} = d_i.$$

Therefore,

$$(47) \quad R_i^\dagger = \frac{d_i K_i}{\mu_i - d_i}, \quad \mu_i > d_i.$$

For the given parameter values,

$$R_1^\dagger = \frac{0.1(0.5)}{1 - 0.1} = \frac{1}{18} \approx 0.05556,$$

whereas

$$R_2^\dagger = \frac{0.1(0.3)}{0.8 - 0.1} = \frac{3}{70} \approx 0.04286.$$

Since species 2 has the lower break-even resource concentration, it outcompetes species 1. The corresponding single-species equilibrium is

$$N_1^* = 0, \quad R^* = R_2^\dagger = \frac{3}{70},$$

and

$$(48) \quad N_2^* = \frac{DY_2(R^0 - R^*)}{d_2} = \frac{0.5(0.5)(10 - \frac{3}{70})}{0.1} \approx 24.893.$$

At this equilibrium, the invasion growth rate of species 1 is

$$\frac{\mu_1 R^*}{K_1 + R^*} - d_1 = \frac{\frac{3}{70}}{\frac{1}{2} + \frac{3}{70}} - 0.1 \approx -0.02105 < 0.$$

Thus, species 1 cannot invade the equilibrium dominated by species 2. For a single limiting resource, stable coexistence generally requires the two species to have the same break-even resource concentration, together with additional conditions that prevent competitive exclusion.

4.5. Application 4: Integral Equation Model for Age-Structured Populations. Many biological populations are naturally described by integral equations because their reproductive and survival characteristics depend on age. Let $n(a, t)$ denote the population density of individuals of age a at time t , and let $b(a) \geq 0$ and $s(a) \in [0, 1]$ denote the age-specific fertility rate and the probability of surviving from birth to age a , respectively. The total population at time t is

$$(49) \quad P(t) = \int_0^\infty n(a, t) da.$$

It is important to distinguish the total population $P(t)$ from the birth flux

$$B(t) = n(0, t).$$

Individuals of age a at time t were born at time $t - a$ and survived to age a . Thus, for $0 \leq a \leq t$,

$$n(a, t) = s(a)B(t - a).$$

Consequently, the birth flux satisfies the renewal equation

$$(50) \quad B(t) = \int_0^t b(a)s(a)B(t - a) da + \psi(t), \quad 0 \leq t \leq T,$$

where $\psi(t)$ represents births produced at time t by individuals who were already alive at the initial time. Let

$$X = C([0, T], \mathbb{R})$$

be equipped with the supremum norm

$$\|\phi\|_\infty = \sup_{t \in [0, T]} |\phi(t)|.$$

Define the operator $\mathcal{T} : X \rightarrow X$ by

$$(51) \quad (\mathcal{T}\phi)(t) = \int_0^t b(a)s(a)\phi(t - a) da + \psi(t).$$

A function $B \in X$ solves the renewal equation (50) if and only if it is a fixed point of $\mathcal{T}$.

For $\phi_1, \phi_2, \phi_3 \in X$, define

$$(52) \quad \begin{aligned} S_b(\phi_1, \phi_2, \phi_3) &= \|\phi_1 - \phi_2\|_\infty + \|\phi_2 - \phi_3\|_\infty \\ &\quad + \|\phi_3 - \phi_1\|_\infty. \end{aligned}$$

This metric is induced by the supremum norm and provides the required ternary distance structure on X .

Theorem 7 (Existence and uniqueness of the renewal solution). *Suppose that b and s are continuous and nonnegative and that $\psi \in C([0, T], \mathbb{R})$. If*

$$(53) \quad q_T := \int_0^T b(a)s(a) da < 1,$$

then the operator $\mathcal{T}$ defined by (51) is a contraction on X . Consequently, the renewal equation (50) has a unique continuous solution on $[0, T]$.

Proof. Let $\phi_1, \phi_2 \in X$. For every $t \in [0, T]$, we have

$$\begin{aligned} |(\mathcal{T}\phi_1)(t) - (\mathcal{T}\phi_2)(t)| &= \left| \int_0^t b(a)s(a) [\phi_1(t-a) - \phi_2(t-a)] da \right| \\ &\leq \int_0^t b(a)s(a) |\phi_1(t-a) - \phi_2(t-a)| da \\ &\leq \left(\int_0^t b(a)s(a) da \right) \|\phi_1 - \phi_2\|_\infty \\ (54) \quad &\leq q_T \|\phi_1 - \phi_2\|_\infty. \end{aligned}$$

Taking the supremum over $t \in [0, T]$ yields

$$(55) \quad \|\mathcal{T}\phi_1 - \mathcal{T}\phi_2\|_\infty \leq q_T \|\phi_1 - \phi_2\|_\infty.$$

Since $q_T < 1$, the operator $\mathcal{T}$ is a contraction. Moreover, applying (55) to the three terms in (52) gives

$$(56) \quad S_b(\mathcal{T}\phi_1, \mathcal{T}\phi_2, \mathcal{T}\phi_3) \leq q_T S_b(\phi_1, \phi_2, \phi_3).$$

Because X is complete, the contraction principle guarantees that $\mathcal{T}$ has a unique fixed point in X . This fixed point is the unique continuous solution of (50). $\square$

The net reproductive rate associated with the model is

$$(57) \quad R_0 = \int_0^\infty b(a)s(a) da.$$

When $R_0 < 1$, each generation produces fewer individuals than the preceding generation. Under the standard integrability and irreducibility assumptions of renewal theory, the population

therefore tends asymptotically to extinction. In the homogeneous case $\psi = 0$, the unique fixed point is the zero solution $B^* = 0$.

The long-term growth or decline of the population can be described by a stable age profile. Suppose that the birth flux has the exponential form

$$B(t) = B_0 e^{rt},$$

where r is the intrinsic population growth rate. Then

$$n(a, t) = B(t - a)s(a) = B(t)e^{-ra}s(a).$$

Thus, apart from normalization, the stable age profile is

$$(58) \quad n_r(a) = e^{-ra}s(a).$$

Substitution into the renewal equation gives the Lotka–Euler equation

$$(59) \quad 1 = \int_0^\infty b(a)s(a)e^{-ra} da.$$

If $R_0 < 1$, the corresponding solution of the Lotka–Euler equation typically satisfies $r < 0$, indicating population decline. If $R_0 > 1$, then $r > 0$, indicating asymptotic population growth rather than a stationary equilibrium. The critical case $R_0 = 1$ corresponds to $r = 0$ and represents exact generational replacement. This formulation extends the fixed-point framework to biologically structured populations while preserving the distinction between birth flux, total population size, and stable age distribution.

5. NUMERICAL EXPERIMENTS AND VALIDATION

This section presents the computational procedure used to approximate the common fixed points arising in the biological models and describes the quantities employed to monitor numerical convergence.

5.1. Computational Algorithm. Based on Theorem 1, the iterative sequence is defined by

$$(60) \quad g(x_{k+1}) = f(x_k), \quad k = 0, 1, 2, \dots$$

The condition $f(X) \subseteq g(X)$ guarantees that, for each $x_k \in X$, there exists at least one $x_{k+1} \in X$ satisfying (60). If g is invertible, the iteration can be written explicitly as

$$(61) \quad x_{k+1} = g^{-1}(f(x_k)).$$

In the particular case $g = I_X$, this reduces to the standard Picard iteration

$$x_{k+1} = f(x_k).$$

Algorithm 1 Common Fixed-Point Iteration for Biological Models

Require: Initial state x_0 , mappings f and g , tolerance $\varepsilon > 0$, and maximum number of iterations

$K_{\max}$

Ensure: An approximate common fixed point x^*

```

1:  $k \leftarrow 0$ 
2: while  $k < K_{\max}$  do
3:   Compute  $y_k \leftarrow f(x_k)$ 
4:   Find  $x_{k+1} \in X$  such that  $g(x_{k+1}) = y_k$ 
5:    $r_k \leftarrow S_b(x_{k+1}, x_k, x_k)$ 
6:    $c_k \leftarrow S_b(f(x_{k+1}), g(x_{k+1}), g(x_{k+1}))$ 
7:   if  $r_k < \varepsilon$  and  $c_k < \varepsilon$  then
8:     return  $x^* \leftarrow x_{k+1}$ 
9:   end if
10:   $x_k \leftarrow x_{k+1}$ 
11:   $k \leftarrow k + 1$ 
12: end while
13: return  $x^* \leftarrow x_k$ 

```

When g is invertible, the step requiring a solution of $g(x_{k+1}) = y_k$ is implemented directly as

$$x_{k+1} = g^{-1}(y_k).$$

If $g = I_X$, one simply sets $x_{k+1} = y_k = f(x_k)$.

To assess numerical convergence, we monitor the successive-iterate residual

$$(62) \quad r_k = S_b(x_{k+1}, x_k, x_k),$$

the common fixed-point residual

$$(63) \quad c_k = S_b(f(x_{k+1}), g(x_{k+1}), g(x_{k+1})),$$

and the normalized change

$$(64) \quad \delta_k = \frac{r_k}{\max\{1, S_b(x_k, x_0, x_0)\}}.$$

For $k \geq 1$ and $r_{k-1} > 0$, the observed convergence factor is defined by

$$(65) \quad \rho_k = \frac{r_k}{r_{k-1}}.$$

If the theoretical estimate in Theorem 2 has the form

$$r_k \leq \mu r_{k-1}, \quad 0 < \mu < 1,$$

then

$$\rho_k \leq \mu$$

whenever the estimate applies. The computed values of ρ_k may approach μ when the theoretical bound is asymptotically sharp; however, they need not be equal to μ , since μ may only be an upper bound for the actual convergence factor. The iteration is declared convergent when both r_k and c_k are smaller than the prescribed tolerance ε .

5.2. Experiment 1: Logistic Growth with Allee Effects. We consider the logistic growth model with an Allee effect introduced in Section 4. The parameter values are

$$r = 0.5 \text{ day}^{-1}, \quad K = 1000, \quad A = 200, \quad \Delta t = 0.1 \text{ day},$$

and the initial populations are selected as

$$N_0 \in \{50, 150, 250, 500, 800\}.$$

The corresponding forward Euler iteration is

$$(66) \quad N_{k+1} = N_k + \Delta t r N_k \left(1 - \frac{N_k}{K}\right) \left(\frac{N_k}{A} - 1\right).$$

The iteration is terminated when

$$r_k = S_b(N_{k+1}, N_k, N_k) = 2|N_{k+1} - N_k| < 10^{-6}.$$

TABLE 1. Numerical convergence results for the logistic growth model with an Allee effect.

N_0	Limiting equilibrium	Iterations	Final residual	Asymptotic factor
50	0	309	9.91×10^{-7}	0.95
150	0	358	9.88×10^{-7}	0.95
250	1000	133	8.36×10^{-7}	0.80
500	1000	99	8.39×10^{-7}	0.80
800	1000	86	9.09×10^{-7}	0.80

The continuous model has three equilibria, obtained from

$$(67) \quad rN^* \left(1 - \frac{N^*}{K}\right) \left(\frac{N^*}{A} - 1\right) = 0.$$

Therefore,

$$(68) \quad N^* \in \{0, A, K\} = \{0, 200, 1000\}.$$

The equilibria $N^* = 0$ and $N^* = K$ are locally asymptotically stable, whereas the Allee threshold $N^* = A$ is unstable. Consequently, initial populations satisfying $0 < N_0 < A$ converge to extinction, while initial populations satisfying $A < N_0 < K$ converge to the carrying capacity K . If $N_0 = A$, the population remains at the unstable equilibrium A in exact arithmetic.

To explain the observed convergence factors, define

$$F(N) = N + \Delta t rN \left(1 - \frac{N}{K}\right) \left(\frac{N}{A} - 1\right).$$

The local convergence factor near a fixed point N^* is $|F'(N^*)|$. At the extinction equilibrium,

$$F'(0) = 1 - r\Delta t = 1 - 0.5(0.1) = 0.95,$$

whereas at the carrying capacity,

$$F'(K) = 1 - r\Delta t \left(\frac{K}{A} - 1\right) = 1 - 0.5(0.1)(5 - 1) = 0.80.$$

These values agree with the asymptotic convergence factors reported in Table 1. They also explain why convergence toward K is faster than convergence toward extinction for the chosen parameter values.

The forward Euler discretization preserves all three equilibria of the continuous model exactly. For example, starting from $N_0 = 250$, the computed value at termination is $N_{133} \approx 999.9999983$, which agrees with the analytical attracting equilibrium $N^* = K = 1000$ within the prescribed stopping tolerance. Thus, the numerical results correctly reproduce the threshold behavior associated with the Allee effect.

5.3. Experiment 2: Tumor Growth Dynamics. We consider the two-compartment tumor growth model introduced in Section 4. The parameter values are

$$\alpha = 0.1 \text{ day}^{-1}, \quad V_{\max} = 5000 \text{ mm}^3, \quad \beta = 0.05 \text{ day}^{-1},$$

$$\gamma = 0.02 \text{ day}^{-1}, \quad \Delta t = 0.5 \text{ day}.$$

The initial viable and necrotic tumor volumes are taken as

$$V_0 = 100 \text{ mm}^3, \quad N_0 = 10 \text{ mm}^3.$$

The forward Euler discretization is given by

$$(69) \quad \begin{aligned} V_{k+1} &= V_k + \Delta t \left[\alpha V_k \ln \left(\frac{V_{\max}}{V_k} \right) - \beta V_k \right], \\ N_{k+1} &= N_k + \Delta t (\beta V_k - \gamma N_k). \end{aligned}$$

According to (37), the positive equilibrium is

$$(70) \quad \begin{aligned} V^* &= V_{\max} e^{-\beta/\alpha} = 5000 e^{-0.5} \approx 3032.6533 \text{ mm}^3, \\ N^* &= \frac{\beta}{\gamma} V^* = \frac{0.05}{0.02} (3032.6533) \approx 7581.6332 \text{ mm}^3. \end{aligned}$$

For the numerical implementation, the residual is defined by

$$(71) \quad r_k = S_b(\mathbf{x}_{k+1}, \mathbf{x}_k, \mathbf{x}_k) = 2(|V_{k+1} - V_k| + |N_{k+1} - N_k|).$$

The iteration is terminated when $r_k < 10^{-6}$. The resulting numerical values are

$$(72) \quad V_{1916} \approx 3032.6532986 \text{ mm}^3, \quad N_{1916} \approx 7581.6331970 \text{ mm}^3,$$

with

$$r_{1915} \approx 9.98 \times 10^{-7}.$$

Thus, the computed state agrees with the analytical equilibrium (70) within the prescribed tolerance.

The viable tumor volume initially undergoes rapid Gompertzian growth and approaches its equilibrium substantially faster than the necrotic volume. The necrotic compartment responds more slowly because its rate of adjustment is controlled by the smaller clearance rate γ . For example, after 200 days, the numerical solution is approximately

$$V(200) \approx 3032.6533 \text{ mm}^3, \quad N(200) \approx 7377.1800 \text{ mm}^3.$$

Therefore, the viable component is already effectively at equilibrium, whereas the necrotic component is still approaching its limiting value.

The different convergence speeds can also be explained analytically. At the positive equilibrium, the Jacobian matrix of the continuous system is

$$(73) \quad J^* = \begin{pmatrix} -\alpha & 0 \\ \beta & -\gamma \end{pmatrix}.$$

Consequently, the Jacobian matrix of the Euler mapping is

$$I + \Delta t J^* = \begin{pmatrix} 1 - \alpha \Delta t & 0 \\ \beta \Delta t & 1 - \gamma \Delta t \end{pmatrix},$$

whose eigenvalues are

$$1 - \alpha \Delta t = 0.95 \quad \text{and} \quad 1 - \gamma \Delta t = 0.99.$$

Hence, the viable volume has an asymptotic convergence factor of approximately 0.95, while the full two-compartment system is eventually dominated by the slower convergence factor 0.99 of the necrotic compartment.

To examine parameter sensitivity, the necrosis rate β is varied by $\pm 20\%$, while all other parameters remain unchanged. Since

$$V^*(\beta) = V_{\max} e^{-\beta/\alpha},$$

the resulting equilibrium values are

$$\begin{aligned}
 \beta = 0.04 : \quad V^* &\approx 3351.6002 \text{ mm}^3, \\
 \beta = 0.05 : \quad V^* &\approx 3032.6533 \text{ mm}^3, \\
 \beta = 0.06 : \quad V^* &\approx 2744.0582 \text{ mm}^3.
 \end{aligned}
 \tag{74}$$

Relative to the baseline value, decreasing β by 20% increases the equilibrium viable volume by approximately 10.52%, whereas increasing β by 20% decreases it by approximately 9.52%. These results demonstrate that the equilibrium viable volume responds continuously, but not symmetrically, to changes in the necrosis rate.

The present experiment constitutes a numerical verification of the analytical equilibrium and convergence behavior. A clinical validation cannot be claimed without identifying the xenograft dataset, describing the parameter-estimation procedure, and providing the observed tumor-volume measurements. Accordingly, quantities such as the mean absolute percentage error, correlation coefficient, and residual sum of squares should only be reported after they have been calculated from a clearly cited dataset. Differences between model predictions and clinical measurements may then be attributed to tumor-cell heterogeneity, unmodeled treatment interventions, and measurement uncertainty.

5.4. Experiment 3: Microbial Competition. We consider the two-species, one-resource chemostat model introduced in Section 4. The parameter values are

$$\mu_1 = 1.0 \text{ hr}^{-1}, \quad K_1 = 0.5 \text{ g/L}, \quad d_1 = 0.1 \text{ hr}^{-1}, \quad Y_1 = 0.5,$$

for species 1, and

$$\mu_2 = 0.8 \text{ hr}^{-1}, \quad K_2 = 0.3 \text{ g/L}, \quad d_2 = 0.1 \text{ hr}^{-1}, \quad Y_2 = 0.5,$$

for species 2. The chemostat parameters are

$$D = 0.5 \text{ hr}^{-1}, \quad R^0 = 10 \text{ g/L}, \quad \Delta t = 0.01 \text{ hr}.$$

The initial state is chosen as

$$(N_{1,0}, N_{2,0}, R_0) = (1, 1, 9).$$

For these parameter values, the discrete system is

$$\begin{aligned}
 (75) \quad N_{1,k+1} &= N_{1,k} + \Delta t N_{1,k} \left(\frac{\mu_1 R_k}{K_1 + R_k} - d_1 \right), \\
 N_{2,k+1} &= N_{2,k} + \Delta t N_{2,k} \left(\frac{\mu_2 R_k}{K_2 + R_k} - d_2 \right), \\
 R_{k+1} &= R_k + \Delta t \left[D(R^0 - R_k) - \frac{\mu_1 N_{1,k} R_k}{Y_1 (K_1 + R_k)} - \frac{\mu_2 N_{2,k} R_k}{Y_2 (K_2 + R_k)} \right].
 \end{aligned}$$

The break-even resource concentration of species i is determined by

$$\frac{\mu_i R_i^\dagger}{K_i + R_i^\dagger} = d_i,$$

which gives

$$(76) \quad R_i^\dagger = \frac{d_i K_i}{\mu_i - d_i}, \quad \mu_i > d_i.$$

For the two species, these concentrations are

$$R_1^\dagger = \frac{0.1(0.5)}{1.0 - 0.1} = \frac{1}{18} \approx 0.05556 \text{ g/L}$$

and

$$R_2^\dagger = \frac{0.1(0.3)}{0.8 - 0.1} = \frac{3}{70} \approx 0.04286 \text{ g/L}.$$

Since $R_2^\dagger < R_1^\dagger$, species 2 can survive at a lower resource concentration and therefore competitively excludes species 1.

The predicted attracting equilibrium is consequently

$$(77) \quad N_1^* = 0, \quad R^* = R_2^\dagger = \frac{3}{70},$$

and

$$\begin{aligned}
 (78) \quad N_2^* &= \frac{DY_2(R^0 - R^*)}{d_2} \\
 &= \frac{(0.5)(0.5) \left(10 - \frac{3}{70}\right)}{0.1} \\
 &= \frac{697}{28} \approx 24.89286.
 \end{aligned}$$

Thus, the attracting equilibrium is

$$(79) \quad (N_1^*, N_2^*, R^*) = (0, 24.89286, 0.04286).$$

The numerical residual is defined by

$$(80) \quad \begin{aligned} r_k &= S_b(\mathbf{x}_{k+1}, \mathbf{x}_k, \mathbf{x}_k) \\ &= 2(|N_{1,k+1} - N_{1,k}| + |N_{2,k+1} - N_{2,k}| + |R_{k+1} - R_k|). \end{aligned}$$

The iteration is terminated when $r_k < 10^{-6}$.

TABLE 2. Selected numerical results for the two-species chemostat model.

Iteration	Time (hr)	$N_{1,k}$	$N_{2,k}$	R_k
1247	12.47	9.13099	9.78845	0.06625
10000	100.00	2.85572	22.03257	0.04407
47463	474.63	0.00119	24.89167	0.04286

At iteration 47463, the computed state is

$$(N_{1,47463}, N_{2,47463}, R_{47463}) \approx (0.001186, 24.891669, 0.042858),$$

and the residual is

$$r_{47462} \approx 9.9994 \times 10^{-7}.$$

The numerical solution therefore approaches the equilibrium (79). The first species approaches zero slowly, while the second species approaches its positive equilibrium density.

The asymptotic decay of species 1 is determined by its per-capita growth rate at the equilibrium established by species 2:

$$(81) \quad \begin{aligned} \frac{\mu_1 R^*}{K_1 + R^*} - d_1 &= \frac{\frac{3}{70}}{\frac{1}{2} + \frac{3}{70}} - 0.1 \\ &\approx -0.02105 < 0. \end{aligned}$$

Thus, species 1 cannot invade the equilibrium occupied by species 2. For the forward Euler iteration, the associated local factor is approximately

$$1 + \Delta t(-0.02105) \approx 0.999789.$$

This factor explains the relatively slow convergence toward complete competitive exclusion.

The trajectory does not spiral toward a coexistence equilibrium. Instead, both populations initially increase while consuming the abundant resource. As the resource becomes limited, species 2 gains a competitive advantage because it has the lower break-even concentration. Its density then approaches a positive equilibrium, whereas the density of species 1 gradually decreases toward zero.

If the smoothed state

$$\hat{\mathbf{x}}_k = \omega \mathbf{x}_k + (1 - \omega) \mathbf{x}_{k-1}, \quad \omega = 0.8,$$

is used for convergence monitoring, it reduces short-term numerical fluctuations but does not alter the equilibrium or generate coexistence. A genuine biological memory effect would require a delay or hereditary term to be included explicitly in the governing equations.

The numerical results agree with the classical competitive exclusion principle: when two species compete for one limiting resource under constant chemostat conditions, the species with the lower break-even resource concentration excludes the other. Different half-saturation constants alone do not guarantee niche differentiation or coexistence in a homogeneous single-resource environment.

No experimental chemostat dataset has been specified in the present experiment. Therefore, an observed species ratio or a claim of agreement with experimental uncertainty cannot be reported without a cited dataset and a clearly described comparison procedure. For the present model, the predicted limiting ratio is not 1.61; instead,

$$\frac{N_1^*}{N_2^*} = 0,$$

because species 1 is asymptotically excluded.

5.5. Experiment 4: Age-Structured Population. We consider the renewal model introduced in Section 4. The age-specific fertility and survival functions are chosen as

$$(82) \quad b(a) = \begin{cases} 0, & 0 \leq a < 1, \\ 2e^{-0.5a}, & a \geq 1, \end{cases} \quad s(a) = e^{-0.2a}.$$

The condition $b(a) = 0$ for $a < 1$ represents a maturation delay of one year. For the numerical evaluation, the age interval $[1, 50]$ is discretized using 500 equally spaced points.

The net reproductive rate is

$$\begin{aligned}
 R_0 &= \int_0^{\infty} b(a)s(a) da \\
 &= 2 \int_1^{\infty} e^{-0.7a} da \\
 &= \frac{2}{0.7} e^{-0.7} \\
 (83) \quad &\approx 1.41881.
 \end{aligned}$$

Since $R_0 > 1$, the population is in the supercritical regime. Therefore, it exhibits asymptotic exponential growth rather than convergence to the extinction solution.

The intrinsic growth rate r is determined by the Lotka–Euler equation

$$(84) \quad 1 = \int_0^{\infty} b(a)s(a)e^{-ra} da.$$

Substituting the functions in (82) gives

$$\begin{aligned}
 1 &= 2 \int_1^{\infty} e^{-(0.7+r)a} da \\
 (85) \quad &= \frac{2e^{-(0.7+r)}}{0.7+r}.
 \end{aligned}$$

Setting $q = 0.7 + r$, we obtain

$$qe^q = 2.$$

Hence,

$$(86) \quad r = W_0(2) - 0.7 \approx 0.1526055 \text{ year}^{-1},$$

where W_0 denotes the principal branch of the Lambert W function.

To verify this value numerically, the Lotka–Euler integral is truncated at age 50 and approximated by the composite trapezoidal rule using 500 points on $[1, 50]$. The numerical growth rate is

$$(87) \quad r_{\text{num}} \approx 0.1528744 \text{ year}^{-1}.$$

The corresponding relative error is

$$(88) \quad \frac{|r_{\text{num}} - r|}{|r|} \times 100 \approx 0.176\%.$$

The small discrepancy is caused by the finite truncation of the age domain and the quadrature discretization.

The asymptotic birth flux has the form

$$B(t) \sim B_0 e^{rt},$$

and the associated stable age profile is proportional to

$$(89) \quad n_r(a) = e^{-ra} s(a) = e^{-(r+0.2)a}.$$

After normalization, the stable age-density function is

$$(90) \quad p_r(a) = (r+0.2)e^{-(r+0.2)a}, \quad a \geq 0.$$

Its mean age is

$$(91) \quad \bar{a} = \frac{1}{r+0.2} \approx 2.836 \text{ years.}$$

Thus, the stable age distribution is approached asymptotically over time; it does not correspond to an age structure of approximately 150 years.

For comparison with a discrete age-structured model having a one-year projection interval, the continuous growth rate corresponds to the annual multiplication factor

$$(92) \quad \lambda = e^r = e^{0.1526055} \approx 1.16487.$$

The numerical growth rate gives

$$e^{r_{\text{num}}} = e^{0.1528744} \approx 1.16518.$$

Therefore, a consistently constructed Leslie matrix based on the same fertility, survival, and age-discretization assumptions should have a dominant annual eigenvalue approaching 1.16487 as the age-grid resolution is increased. The numerical value 1.16518 differs from the continuous prediction by approximately 0.027%. An explicit Leslie-matrix eigenvalue should only be reported after the age classes and fertility coefficients of that matrix have been fully specified.

6. DISCUSSION

This study develops a fixed-point framework for discrete biological models in S_b -metric spaces. The proposed g -interpolative contractions extend classical contraction principles by allowing more flexible relationships among mappings and their iterates. Theorem 1 establishes existence and uniqueness under the stated contractive and compatibility assumptions without requiring continuous differentiability, while Theorems 2 and 3 provide convergence estimates and robustness results under perturbations.

The biological applications illustrate several forms of long-term behavior. The logistic model exhibits a strong Allee effect: populations initially below A approach extinction, whereas those above A approach the carrying capacity K . In the tumor model, the positive steady state represents a balance among proliferation, necrosis, and necrotic-tissue clearance. The microbial model illustrates competitive exclusion in a single-resource chemostat, where species 2 excludes species 1 because it has the lower break-even resource concentration. The age-structured model connects fertility, survival, and population growth through the net reproductive rate and the Lotka–Euler equation.

The fixed-point formulation is particularly suitable for discrete-time biological models and provides constructive procedures for approximating equilibria. It may also accommodate problems for which classical differentiability-based methods are difficult to apply. Moreover, a verified contraction condition provides convergence throughout the invariant set on which it holds. Nevertheless, these conclusions require the explicit verification of invariance, completeness, and the relevant contractive inequalities; they cannot be inferred solely from the form of the biological model.

7. CONCLUSION

This study developed a fixed-point framework for discrete biological models using g -interpolative contractions in S_b -metric spaces. Under suitable invariance, completeness, compatibility, and contractivity assumptions, the framework provides results concerning the existence, uniqueness, convergence, and stability of fixed points. Its applicability was illustrated

through models of logistic growth with an Allee effect, tumor growth, microbial resource competition, and age-structured populations. The analytical and numerical results demonstrate how fixed-point methods can support the study of equilibria and long-term behavior in biological systems. Future work may extend this framework to delayed, stochastic, and spatially distributed models and investigate its validation using experimental data.

CONFLICT OF INTERESTS

The author(s) declare that there is no conflict of interests.

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