



Research article

Aggregation as a double-edged sword: Fear, Allee effects, and finite-time collapse

Kwadwo Antwi-Fordjour^{1,*} and Eric M. Takyi²

¹ Department of Mathematics and Computer Science, Samford University, Birmingham, AL 35229, USA

² Department of Mathematics, Computer Science, and Statistics, Ursinus College, Collegeville, PA 19426, USA

* **Correspondence:** Email: kantwifo@samford.edu.

Abstract: Although prey aggregation is widely regarded as an effective defense against predation, we have demonstrated that, when coupled with predator-induced fear, infectious disease, and demographic Allee effects, it can paradoxically accelerate rather than prevent ecosystem collapse. We formulated and analyzed a susceptible–infectious–predator model incorporating dual fear responses, a sublinear aggregation-based predation term, and an Allee effect. We characterized the existence and local stability of all biologically meaningful equilibria. We then proved that the non-Lipschitz aggregation term, under suitable parameter conditions, can drive the susceptible prey population to extinction in finite time under sustained predator pressure, while the Allee effect identifies the ecological regime in which populations become vulnerable to collapse. An explicit upper bound for the extinction time was derived, quantifying how predator pressure and the strength of the aggregation response jointly determine the speed of ecological collapse. Bifurcation analysis revealed transcritical, saddle-node, and Hopf bifurcations as fear intensity, aggregation strength, and the Allee threshold vary. Two-parameter continuation further identified parameter regions supporting stable coexistence, oscillatory coexistence, predator exclusion, and finite-time extinction, demonstrating that stronger aggregation progressively enlarges the finite-time extinction region while weaker aggregation promotes a richer coexistence structure. Moreover, the coexistence of alternative stable states gives rise to path-dependent ecological outcomes, indicating that identical environmental conditions may produce fundamentally different long-term dynamics depending on the initial population state. These results demonstrate that aggregation, when coupled with fear, disease dynamics, and demographic vulnerability, can fundamentally alter ecosystem resilience by accelerating collapse, generating alternative stable states, and promoting abrupt ecological tipping points.

Keywords: finite-time extinction; fear effect; Allee effect; path-dependence; tipping points

1. Introduction

Predators influence prey populations not only through direct consumption but also through behavioral changes induced by perceived predation risk. The reintroduction of gray wolves (*Canis lupus*) into Yellowstone National Park, for example, altered elk (*Cervus canadensis*) habitat use and foraging behavior, generating ecosystem-wide consequences [1,2]. Such observations illustrate that fear-mediated responses can reshape ecological interactions even in the absence of direct predation. When coupled with infectious disease, prey aggregation, and low-density demographic effects, these indirect responses may promote abrupt transitions between persistence and collapse.

Classical predator–prey models primarily emphasize consumptive effects, whereas increasing empirical and theoretical evidence demonstrates that predator-induced fear alone can substantially alter prey demography through changes in vigilance, refuge use, movement, and reproduction [3–6]. For example, Zanette et al. [7] experimentally showed that perceived predation risk reduced songbird reproduction, motivating fear-based mathematical models [8]. Subsequent studies established that fear can modify population densities, stability, oscillatory behavior, and extinction thresholds [9,10].

Fear effects have also been incorporated into eco-epidemiological models, where predator-induced behavioral responses interact with disease transmission and prey demography [11]. Since the pioneering work of Anderson and May [12], eco-epidemiological models have shown that infection within prey populations can alter predator persistence, community stability, and extinction outcomes [13–15]. However, most existing fear-based eco-epidemiological models do not simultaneously incorporate aggregation-mediated predation and an explicit Allee threshold.

Prey aggregation is a widespread anti-predator strategy that reduces individual risk through mechanisms such as dilution, collective vigilance, and predator confusion [16–18]. Mathematically, aggregation has been represented through square-root and more generally sublinear functional responses. Braza [19] introduced the square-root formulation, while later studies generalized it to S^r for $0 < r < 1$ to represent different aggregation intensities [20]. Importantly, this sublinear term is non-Lipschitz at $S = 0$, giving rise to extinction dynamics that cannot be analyzed using classical linearization techniques.

The Allee effect provides a complementary demographic mechanism by describing reduced individual fitness at low population densities arising from mate limitation, cooperative defense, group foraging, and other forms of positive density dependence [21–23]. Strong Allee effects introduce a positive extinction threshold below which population growth becomes negative, whereas weak Allee effects reduce low-density growth without creating a positive threshold [24,25]. Throughout this study, aggregation and the Allee effect are treated as distinct mechanisms: aggregation alters predator access to susceptible prey, whereas the Allee effect governs demographic performance at low population density.

Despite these advances, existing models examine only subsets of these mechanisms. Few studies combine predator-induced fear, infectious disease, aggregation-mediated predation, and an explicit Allee threshold within a single susceptible–infectious–predator framework. Consequently, it remains unclear how these interacting processes jointly influence coexistence, alternative stable states, ecological tipping points, and extinction dynamics. Our central hypothesis is that prey aggregation, traditionally viewed as a protective behavioral strategy, may instead promote ecological collapse when combined with fear-induced behavioral responses, infectious disease, and demographic vulnerability.

To investigate this hypothesis, we formulate a susceptible–infectious–predator (SIP) model in which

$S(t)$, $I(t)$, and $P(t)$ denote the densities of susceptible prey, infectious prey, and predators, respectively. Predator-induced fear independently suppresses susceptible-prey reproduction and disease transmission, aggregation is represented through a sublinear predation term, and an explicit Allee parameter captures demographic limitations at low population density. This framework enables us to systematically investigate how these mechanisms interact while preserving their distinct biological roles.

The principal contributions of this study are summarized as follows:

- (i) We prove that aggregation-mediated predation generates finite-time extinction of the susceptible prey population under suitable ecological conditions, identifying the non-Lipschitz aggregation term as the mathematical mechanism responsible for abrupt population collapse.
- (ii) We derive an explicit analytical upper bound for the extinction time, quantifying how predator pressure and aggregation strength jointly determine the speed of ecological collapse.
- (iii) We establish that finite-time extinction initiates a cascade collapse of the entire ecological community, whereby the infected prey and predator populations subsequently decay asymptotically under the biological assumptions of the model.
- (iv) We demonstrate that the interaction between fear, aggregation, and Allee dynamics produces alternative stable states, path-dependent ecological outcomes, and multiple bifurcation mechanisms, revealing that stronger aggregation progressively enlarges the finite-time extinction regime while reducing ecosystem resilience.

The remainder of the paper is organized as follows. In Section 2, we formulate the model and establish nonnegativity, boundedness, and the positively invariant region of solutions. Section 3 examines the existence and local stability of the biologically meaningful equilibria. Section 4 develops a nonclassical analysis of the extinction equilibrium, establishing finite-time extinction of susceptible prey, subsequent asymptotic cascade decay, and the dynamics on the susceptible extinction manifold. In Section 5, we investigate one- and two-parameter bifurcations and identify parameter regimes associated with qualitative changes in system dynamics. Alternative stable states, path-dependent dynamics, hysteresis, and ecological tipping points are examined in Section 6. Numerical simulations are presented throughout to illustrate the analytical findings. Finally, Section 7 summarizes the principal results, discusses their ecological implications and limitations, and outlines directions for future research.

2. Model formulation and basic mathematical properties

2.1. Model formulation

We develop a novel eco-epidemiological model inspired by the works in [11, 26] to investigate the coupled dynamics of disease transmission and predator-prey interactions, explicitly incorporating fear-induced behavioral responses, prey aggregation, and the Allee effect. The model is grounded in the following biologically motivated assumptions:

- (a) **Prey population structure:** The prey population is stratified into two epidemiological compartments: susceptible prey (S) and infectious prey (I). This division captures disease spread within the prey species and allows for epidemiological feedback mechanisms.
- (b) **Disease transmission dynamics:** Disease transmission occurs solely within the prey population through direct contact between susceptible and infectious individuals. The pathogen is assumed

to be prey-specific, and predator infection is neglected. Although predators may occasionally be exposed to pathogens carried by infected prey in natural systems, such spillover events are assumed to be epidemiologically insignificant and do not contribute to disease persistence. Therefore, predators affect the epidemiological dynamics only indirectly through predation and fear-induced behavioral changes, while disease transmission remains restricted to the prey population.

- (c) **Pathogen-induced fitness impairment:** Infected prey are assumed to be reproductively inactive over the ecological timescale of interest. Consequently, they do not contribute to prey recruitment. In addition, disease reduces their physiological condition, making them less effective at foraging and competing for resources than susceptible individuals.
- (d) **Fear-driven behavioral modifications:** Predator-induced fear influences susceptible prey through two distinct behavioral pathways. The first pathway acts on reproduction. Individuals experiencing elevated predation risk allocate more time to vigilance, refuge use, and anti-predator behavior, thereby reducing reproductive investment. Following Wang et al. [8], this effect is represented by the fear-modified reproductive factor

$$f(k_1, P) = \frac{1}{1 + k_1 P}.$$

The second pathway acts on disease transmission. Fear-induced behavioral changes often reduce movement, aggregation size, and social contact rates, thereby decreasing opportunities for pathogen transmission. To capture this mechanism, we assume that effective disease transmission decreases with perceived predation risk according to

$$f(k_2, P) = \frac{1}{1 + k_2 P}.$$

Although both pathways are modeled using saturating decreasing functions of predator density, they represent biologically distinct processes. Parameter k_1 quantifies reproductive sensitivity to fear, whereas k_2 measures the sensitivity of disease transmission to fear-induced behavioral modifications. The reduction in disease transmission represented by the parameter k_2 is introduced as a biologically motivated modeling assumption. Unlike the fear-induced reduction in reproduction represented by k_1 , which is supported by experimental observations such as those of Zanette et al. [7], there is currently limited direct empirical evidence quantifying the influence of predator-induced fear on disease transmission. The parameter k_2 therefore provides a mechanism for investigating the potential epidemiological consequences of fear-mediated behavioral changes. The use of the same functional form reflects the assumption that both responses saturate at high levels of predation risk while retaining separate biological interpretations; see [11] and the references therein.

The functions can be generalized as

$$f(k_i, P) = \frac{1}{1 + k_i P}, \quad i = 1, 2.$$

This function exhibits key ecological properties:

- (i) *No fear effect* ($k_i = 0$): $f(0, P) = 1$, recovering baseline dynamics.
- (ii) *No predators* ($P = 0$): $f(k_i, 0) = 1$, indicating no suppression in reproduction or contact.

- (iii) *High level of fear or large predator density*: $\lim_{k_i \rightarrow \infty} f(k_i, P) = 0$ and $\lim_{P \rightarrow \infty} f(k_i, P) = 0$, capturing fear-induced behavioral inhibition.
- (e) **Irreversible infection**: Infection is assumed to be effectively irreversible over the ecological timescale of interest. Infected prey either remain chronically infectious or experience disease-induced mortality before recovery can significantly influence population dynamics. Consequently, recovery is neglected and infected individuals remain within the infectious class for the duration of their infection period. This assumption is commonly adopted for persistent or slowly recovering diseases where transmission and mortality processes dominate recovery dynamics.
- (f) **Carrying capacity constraint**: Resource availability imposes a density-dependent limitation on prey growth. Since both susceptible and infected prey utilize the same environmental resources, the total prey population ($S + I$) contributes to intraspecific competition. Accordingly, population growth is regulated through a logistic-type carrying capacity K , which represents the maximum sustainable prey density supported by the environment.
- (g) **Predator-prey interactions**: Predator-prey dynamics are governed by several ecological mechanisms:
- (i) **Logistic growth**: In the absence of predation and disease, the susceptible prey population exhibits logistic growth.
 - (ii) **Prey aggregation**: In models of prey aggregation, predators often interact primarily with individuals located on the boundary of a prey group rather than with all individuals within the group. Motivated by this observation, Braza [19] proposed a square-root functional response in which the effective number of vulnerable prey scales as $\sqrt{S}$. The square-root dependence reflects the fact that only a fraction of the prey population is directly exposed to predation when individuals aggregate into herds or groups [27, 28]. Following this idea, we consider the more general aggregation function employed in [11],

$$S^r, \quad 0 < r < 1,$$

where r quantifies the intensity of prey aggregation. The classical square-root formulation corresponds to $r = \frac{1}{2}$, whereas other values of r represent different degrees of aggregation. Biologically, values of r close to one correspond to relatively weak aggregation, for which the effective number of prey accessible to predators increases almost proportionally with prey abundance, thereby recovering dynamics close to the classical mass-action formulation. In contrast, smaller values of r represent stronger aggregation, reflecting situations in which an increasingly smaller fraction of the prey population is directly exposed to predation because individuals remain tightly clustered. Consequently, the effective prey available to predators grows much more slowly than the total prey population as aggregation intensifies.

Accordingly, the predation term

$$d_0 S^r P$$

models aggregation-mediated predation in which predator access to susceptible prey increases sublinearly with prey abundance. Moreover, the parameter r serves as a biologically meaningful measure of aggregation intensity throughout this study: decreasing r corresponds to progressively stronger aggregation, whereas increasing r corresponds to weaker aggregation. This interpretation provides the biological basis for the numerical continuation studies presented later, where changes in r are used to investigate how increasing aggregation intensity

alters the qualitative dynamics of the eco-epidemiological system, including the onset of bistability, ecological tipping points, and finite-time extinction.

- (iii) **Mass-action predation and conversion:** Predators consume both susceptible and infected prey at rates proportional to prey density and gain energy based on a conversion efficiency. The predation terms are modeled via bilinear mass-action kinetics.
 - (iv) **Natural mortality:** Each population experiences baseline mortality at constant per capita rates.
 - (v) **Predator resource dependence:** Predators are assumed to depend exclusively on the susceptible and infected prey populations for food. Alternative prey or external food resources are neglected over the ecological timescale considered. Consequently, predator persistence is entirely determined by the availability of the focal prey population. This assumption is standard in closed predator–prey systems and is essential for establishing the cascade-collapse result following finite-time extinction of the susceptible prey population.
 - (h) **Allee effects and aggregation dynamics:** The model incorporates an Allee effect to account for demographic limitations that arise when population densities become sufficiently small. Such effects may result from difficulties in mate finding, cooperative defense, social interactions, or group foraging, all of which reduce individual fitness at low population density. Depending on the magnitude of the threshold parameter L , the system may exhibit either weak or strong Allee effects. When $L \leq 0$, the population experiences a weak Allee effect, whereas $L > 0$ corresponds to a strong Allee effect with a critical density threshold.
- Although aggregation and Allee effects may arise from related ecological processes in social species, they are represented here as distinct mechanisms. The aggregation term S^r describes the reduction in predation vulnerability arising from herd formation, whereas the parameter L captures demographic limitations operating at low population densities. The interaction between these mechanisms constitutes one of the principal focuses of the present study.

Collectively, these assumptions yield a biologically consistent framework for exploring how disease transmission and predator pressure interact under behavioral and demographic constraints. Based on these principles, we propose the following susceptible-infectious-predator (SIP) model:

$$\begin{aligned}\frac{dS}{dt} &= \frac{b_0 S}{1 + k_1 P} \left(1 - \frac{S + I}{K}\right) (S - L) - d_0 S^r P - \frac{e_0 S I}{1 + k_2 P}, \\ \frac{dI}{dt} &= \frac{e_0 S I}{1 + k_2 P} - d_1 I P - a_1 I, \\ \frac{dP}{dt} &= d_2 S^r P + d_3 I P - a_2 P.\end{aligned}\tag{2.1}$$

Throughout this paper, we assume that

$$0 < r < 1, \quad L \in \mathbb{R},$$

while all remaining model parameters are strictly positive unless otherwise stated. The restriction $0 < r < 1$ ensures that the aggregation-based predation term $S^r P$ is sublinear and introduces a non-Lipschitz singularity at $S = 0$, which plays a fundamental role in the finite-time extinction dynamics. Allowing $L \in \mathbb{R}$ enables the model to encompass both weak ($L \leq 0$) and strong ($L > 0$) Allee effects

within a unified framework. The state variables $S(t)$, $I(t)$, and $P(t)$ denote normalized population densities. Accordingly, the carrying capacity K and Allee threshold L are expressed on the same normalized scale, and the aggregation term S^r is dimensionally consistent for all $0 < r < 1$.

Table 1. Biological description of parameters from model (2.1), along with representative values and references.

Parameter	Biological description	Value	Reference
b_0	Intrinsic growth rate of susceptible prey	3	[26]
r	Aggregating exponent of susceptible prey	0.5	[19]
e_0	Disease transmission rate	4	[11]
K	Carrying capacity of susceptible and infected prey populations	4	[11]
L	Allee threshold of susceptible prey	-1	Variable
a_1	Infectious prey natural death rate	0.4	[11]
a_2	Predator natural death rate	0.8	[11]
d_0	Attack rate of the predator on the susceptible prey	0.7	[11]
d_1	Attack rate of the predator on infectious prey	0.7	[11]
d_2	Biomass conversion efficiency $\times$ attack rate on susceptible prey	0.4	[11]
d_3	Biomass conversion efficiency $\times$ attack rate on infected prey	0.4	[26]
k_1	Level of fear that suppresses the growth rate of susceptible prey	0.1	[11]
k_2	Level of fear that reduces disease transmission	0.85	[11]

2.2. Biological feasibility

We first show preliminary results concerning the biological feasibility and mathematical meaningfulness of system (2.1). We examine the well-posedness of the system. This includes showing that solutions to system (2.1) are nonnegative and bounded. Negative solutions do not make biological sense since population sizes are never negative. The boundedness property ensures that population densities do not grow unbounded due to a limitation in resources.

2.3. Nonnegativity of the solution

Theorem 2.1. *Let the initial conditions satisfy $S(0) \geq 0$, $I(0) \geq 0$, $P(0) \geq 0$. Then all solutions $(S(t), I(t), P(t))$ of model (2.1) remain nonnegative for all $t \geq 0$.*

Proof. We show that the right-hand sides of model (2.1) preserve nonnegativity for all time, provided the initial conditions are nonnegative.

(i) Nonnegativity of $S(t)$: For $S > 0$, the right-hand side of the susceptible-prey equation is locally Lipschitz, and therefore the solution is unique while $S(t) > 0$. Observe that

$$\left. \frac{dS}{dt} \right|_{S=0} = 0,$$

since every term in the susceptible equation contains the factor S . Hence, the hypersurface

$$S_0 = \{(S, I, P) \in \mathbb{R}_+^3 : S = 0\}$$

is positively invariant. Consequently, a trajectory with $S(0) \geq 0$ cannot cross from the nonnegative region into $S < 0$. Therefore,

$$S(t) \geq 0, \quad t \geq 0.$$

(ii) Nonnegativity of $I(t)$: The equation for I is

$$\frac{dI}{dt} = I \cdot \Psi(S, P), \quad \text{where} \quad \Psi(S, P) = \frac{e_0 S}{1 + k_2 P} - d_1 P - a_1.$$

Hence,

$$I(t) = I(0) \exp\left(\int_0^t \Psi(S(s), P(s)) ds\right) \geq 0,$$

since $I(0) \geq 0$ and the exponential is nonnegative.

(iii) Nonnegativity of $P(t)$: Similarly, the equation for P takes the form

$$\frac{dP}{dt} = P \cdot \Gamma(S, I), \quad \text{where} \quad \Gamma(S, I) = d_2 S^r + d_3 I - a_2.$$

It follows that

$$P(t) = P(0) \exp\left(\int_0^t \Gamma(S(s), I(s)) ds\right) \geq 0.$$

Therefore, the solution trajectory remains in the nonnegative orthant

$$\mathbb{R}_+^3 = \{(S, I, P) \in \mathbb{R}^3 : S \geq 0, I \geq 0, P \geq 0\}$$

for all $t \geq 0$. □

2.4. Boundedness of solutions

Theorem 2.2. Let $0 < r < 1$, $L \in \mathbb{R}$, and assume that all remaining parameters of model (2.1) are strictly positive. Suppose further that the initial prey densities satisfy

$$S(0) + I(0) \leq K.$$

Then every nonnegative solution $(S(t), I(t), P(t))$ of model (2.1) is uniformly bounded for all $t \geq 0$. More precisely,

$$0 \leq S(t) + I(t) \leq K, \quad t \geq 0,$$

and there exists a constant $M_Q > 0$ such that

$$AS(t) + BI(t) + P(t) \leq M_Q, \quad t \geq 0,$$

where

$$A = \frac{d_2}{d_0} + \frac{d_3}{d_1}, \quad B = \frac{d_3}{d_1}.$$

Proof. Let

$$N(t) = S(t) + I(t).$$

Adding the susceptible and infectious prey equations of model (2.1) gives

$$\frac{dN}{dt} = \frac{b_0 S}{1 + k_1 P} \left(1 - \frac{S + I}{K}\right) (S - L) - d_0 S^r P - d_1 I P - a_1 I.$$

Hence,

$$\frac{dN}{dt} \leq \frac{b_0 S}{K(1 + k_1 P)} (K - N)(S - L).$$

On the boundary $N = K$, the demographic term vanishes and therefore

$$\left. \frac{dN}{dt} \right|_{N=K} = -d_0 S^r P - d_1 I P - a_1 I \leq 0.$$

Consequently, the region

$$\mathcal{D}_K = \{(S, I, P) \in \mathbb{R}_+^3 : S + I \leq K\}$$

is positively invariant. Since $S(0) + I(0) \leq K$, it follows that

$$0 \leq S(t) + I(t) \leq K, \quad t \geq 0.$$

In particular,

$$0 \leq S(t) \leq K, \quad 0 \leq I(t) \leq K.$$

We next establish a uniform bound for the predator population. Define

$$Q(t) = AS(t) + BI(t) + P(t).$$

Differentiating Q along solutions of model (2.1) yields

$$\begin{aligned} \frac{dQ}{dt} = & A \frac{b_0 S}{1 + k_1 P} \left(1 - \frac{S + I}{K}\right) (S - L) - A d_0 S^r P - A \frac{e_0 S I}{1 + k_2 P} \\ & + B \frac{e_0 S I}{1 + k_2 P} - B d_1 I P - B a_1 I + d_2 S^r P + d_3 I P - a_2 P. \end{aligned}$$

Since

$$A - B = \frac{d_2}{d_0}, \quad A d_0 - d_2 = \frac{d_0 d_3}{d_1}, \quad B d_1 = d_3,$$

we obtain

$$\begin{aligned} \frac{dQ}{dt} = & A \frac{b_0 S}{1 + k_1 P} \left(1 - \frac{S + I}{K}\right) (S - L) - \frac{d_2 e_0 S I}{d_0 (1 + k_2 P)} \\ & - \frac{d_0 d_3}{d_1} S^r P - B a_1 I - a_2 P. \end{aligned}$$

The second and third terms on the right-hand side are nonpositive, and therefore

$$\frac{dQ}{dt} \leq A \frac{b_0 S}{K(1 + k_1 P)} (K - (S + I))(S - L) - B a_1 I - a_2 P.$$

Because $(S, I, P) \in \mathcal{D}_K$, the demographic term is uniformly bounded above. Define

$$M = \max_{\substack{S, I \geq 0 \\ S+I \leq K \\ P \geq 0}} \left[\frac{b_0 S}{K(1+k_1 P)} (K - (S + I))(S - L) \right].$$

Then $M < \infty$ and

$$\frac{dQ}{dt} \leq AM - Ba_1 I - a_2 P.$$

Let

$$\beta = \min\{a_1, a_2\} > 0.$$

Since

$$Ba_1 I + a_2 P \geq \beta(BI + P),$$

we obtain

$$\frac{dQ}{dt} \leq AM - \beta(BI + P).$$

Using

$$BI + P = Q - AS$$

and $S(t) \leq K$, we have

$$\frac{dQ}{dt} \leq AM - \beta Q + \beta AS \leq AM + \beta AK - \beta Q.$$

Define

$$C = AM + \beta AK.$$

Thus,

$$\frac{dQ}{dt} \leq C - \beta Q.$$

By the application of the comparison theorem, we obtain

$$Q(t) \leq Q(0)e^{-\beta t} + \frac{C}{\beta} (1 - e^{-\beta t}),$$

and hence

$$Q(t) \leq \max \left\{ Q(0), \frac{C}{\beta} \right\} = \max \left\{ Q(0), AK + \frac{AM}{\beta} \right\} =: M_Q.$$

Since $A > 0$ and $B > 0$, it follows that $S(t)$, $I(t)$, and $P(t)$ are uniformly bounded for all $t \geq 0$. $\square$

Remark 2.1. The constant M may be characterized explicitly. Since $1 + k_1 P \geq 1$ and $S + I \leq K$, the maximum positive contribution from the demographic term occurs for $P = 0$ and $I = 0$. Hence,

$$M = \frac{b_0}{K} \max_{0 \leq S \leq K} [S(K - S)(S - L)]_+,$$

where $[x]_+ = \max\{x, 0\}$. If $L < K$, the positive maximum occurs at

$$S^\dagger = \frac{K + L + \sqrt{K^2 - KL + L^2}}{3},$$

and therefore

$$M = \frac{b_0}{K} S^\dagger (K - S^\dagger)(S^\dagger - L).$$

If $L \geq K$, the demographic term is nonpositive throughout $0 \leq S \leq K$, and one may take $M = 0$.

2.5. Positively invariant feasible region

Corollary 1. As an immediate consequence of Theorems 2.1 and 2.2, the set

$$\Omega = \left\{ (S, I, P) \in \mathbb{R}_+^3 : S + I \leq K, Q \leq AK + \frac{AM}{\beta} \right\},$$

where Q, A, B, β , and M are defined in Theorem 2.2, is positively invariant under the flow of model (2.1).

3. Existence and local stability analysis of equilibria

3.1. Feasible equilibria

Let $E(S, I, P)$ denote a nonnegative equilibrium point. To obtain $E(S, I, P)$, we solve the system

$$\frac{b_0 S}{1 + k_1 P} \left(1 - \frac{S + I}{K} \right) (S - L) - d_0 S^r P - \frac{e_0 S I}{1 + k_2 P} = 0, \quad (3.1)$$

$$\frac{e_0 S I}{1 + k_2 P} - d_1 I P - a_1 I = 0, \quad (3.2)$$

$$d_2 S^r P + d_3 I P - a_2 P = 0. \quad (3.3)$$

The positively invariant region established in Corollary 1 implies that every biologically feasible equilibrium of model (2.1) must satisfy $S + I \leq K$. Accordingly, in addition to the positivity requirements for each equilibrium, we require all equilibrium points to lie within the feasible region Ω .

- (i) Extinction state $E_0 = (0, 0, 0)$,
- (ii) Susceptible prey-only $E_1 = (K, 0, 0)$,
- (iii) Susceptible prey-only $E_2 = (L, 0, 0)$. Since $S = L, I = 0$, biological feasibility additionally requires $0 < L \leq K$,
- (iv) Infectious prey-free $E_3 = \left(\left(\frac{a_2}{d_2} \right)^{\frac{1}{r}}, 0, P_3 \right)$, where P_3 is a positive root of

$$\underbrace{\frac{d_0 a_2 k_1}{d_2} P^2 + \frac{d_0 a_2}{d_2} P - b_0 \left(\frac{a_2}{d_2} \right)^{\frac{1}{r}} \left(1 - \frac{1}{K} \left(\frac{a_2}{d_2} \right)^{\frac{1}{r}} \right) \left(\frac{1}{K} \left(\frac{a_2}{d_2} \right)^{\frac{1}{r}} - L \right)}_{=B^*} = 0 \quad (3.4)$$

and is given by

$$P_3 = \frac{-\frac{d_0 a_2}{d_2} + \sqrt{\left(\frac{d_0 a_2}{d_2} \right)^2 + \frac{4 d_0 a_2 k_1 B^*}{d_2}}}{\frac{2 d_0 a_2 k_1}{d_2}}, \quad (3.5)$$

which is feasible when $B^* > 0$. This further requires $(a_2/d_2)^{1/r} \leq K$.

- (v) The predator-free equilibrium is $E_4 = \left(\frac{a_1}{e_0}, I_4, 0 \right)$, where

$$I_4 = \frac{b_0 (K e_0 - a_1) (a_1 - L e_0)}{e_0 [b_0 a_1 + e_0 (K e_0 - b_0 L)]}.$$

The positivity condition $I_4 > 0$ is satisfied whenever any of the following conditions hold:

- (a) $\frac{a_1}{L} < e_0 < \frac{a_1}{K}$ and $L < a_1 + \frac{e_0^2 K}{b_0}$;
- (b) $\frac{a_1}{K} < e_0 < \frac{a_1}{L}$ and $L < a_1 + \frac{e_0^2 K}{b_0}$;
- (c) $e_0 < \frac{a_1}{K}$, $e_0 < \frac{a_1}{L}$, and $L > a_1 + \frac{e_0^2 K}{b_0}$;
- (d) $e_0 > \frac{a_1}{K}$, $e_0 > \frac{a_1}{L}$, and $L > a_1 + \frac{e_0^2 K}{b_0}$.

Biological feasibility further requires $\frac{a_1}{e_0} + I_4 \leq K$, which guarantees that $E_4 \in \Omega$.

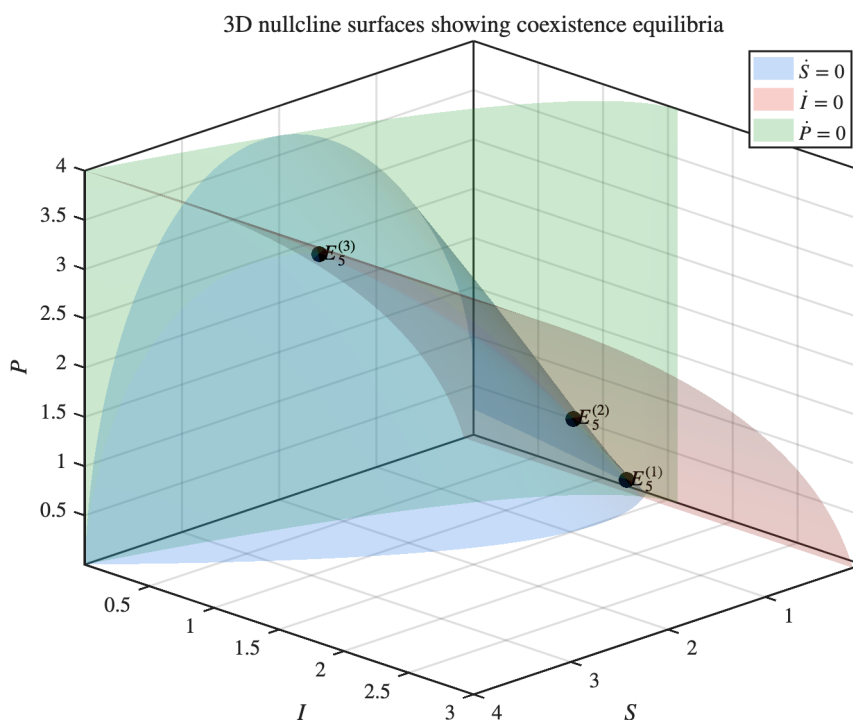


Figure 1. Three-dimensional nullcline surfaces of model (2.1) in the (S, I, P) -phase space. Parameter values are taken from Table 1 with $k_1 = 1.5$. The black markers denote the three coexistence equilibria obtained numerically: $E_5^{(1)} = (0.1472, 1.2931, 0.1561)$, $E_5^{(2)} = (0.4192, 1.0820, 0.7768)$, and $E_5^{(3)} = (2.1543, 0.4258, 2.7294)$.

(vi) For $S^* > 0, I^* > 0, P^* > 0$, the coexistence state $E_5 = (S^*, I^*, P^*)$, where

$$S^* = \left(\frac{a_2 - d_3 I^*}{d_2} \right)^{\frac{1}{r}} \quad (3.6)$$

is obtained by solving Eq (3.3). However, $S^* > 0$ when $I^* < \frac{a_2}{d_3}$. Next, we substitute S^* into Eq (3.2) and solve for P^* . This results in the following quadratic equation in P^* :

$$d_1 k_2 P^{*2} + (d_1 + a_1 k_2) P^* + a_1 - e_0 \left(\frac{a_2 - d_3 I^*}{d_2} \right)^{\frac{1}{r}} = 0. \quad (3.7)$$

By Descartes' rule of signs, there is only one positive root to Eq (3.7) given by

$$P^* = \frac{-(d_1 + a_1 k_2) + \sqrt{a_1^2 k_2^2 + d_1^2 - 2d_1 k_2 \left(a_1 - 2e_0 \left(\frac{a_2 - d_3 I^*}{d_2}\right)^{\frac{1}{r}}\right)}}{2d_1 k_2}. \quad (3.8)$$

For $P^* > 0$, we require that $I^* < \frac{1}{d_3} \left(a_2 - d_2 \left(\frac{a_1}{e_0}\right)^r\right)$. To find I^* explicitly, we substitute P^* in Eq (3.8) and S^* in Eq (3.6) into Eq (3.1). Notably, it is difficult to find the explicit form of I^* due to the algebraic complexity of the resulting equation. However, we provide a sufficient condition for the feasibility of I^* , which is

$$0 < I^* < \min\left\{\frac{a_2}{d_3}, \frac{1}{d_3} \left(a_2 - d_2 \left(\frac{a_1}{e_0}\right)^r\right)\right\} = \frac{1}{d_3} \left(a_2 - d_2 \left(\frac{a_1}{e_0}\right)^r\right). \quad (3.9)$$

In addition, biological feasibility requires $(S^* + I^* \leq K)$, which follows from Corollary 1. We provide a numerical example to illustrate the existence of three biologically feasible coexistence equilibria obtained as intersections of the nullcline surfaces for model (2.1). See Figure 1.

3.2. Local stability analysis

The Jacobian of model (2.1) for any nonnegative equilibrium point $E(S^*, I^*, P^*)$ is computed as

$$J_E = \begin{pmatrix} J_{11} & J_{12} & J_{13} \\ J_{21} & J_{22} & J_{23} \\ J_{31} & J_{32} & J_{33} \end{pmatrix}, \quad (3.10)$$

where

$$\begin{aligned} J_{11} &= \frac{b_0 \left(I^*(L - 2S^*) + 2S^*(K + L) - KL - 3S^{*2}\right)}{K(1 + k_1 P^*)} - d_0 r P^* S^{*(r-1)} - \frac{e_0 I^*}{1 + k_2 P^*}, \\ J_{12} &= \frac{b_0 S^*(L - S^*)}{K(1 + k_1 P^*)} - \frac{e_0 S^*}{1 + k_2 P^*}, \\ J_{13} &= \frac{b_0 k_1 S^*(S^* - L)(I^* - K + S^*)}{K(1 + k_1 P^*)^2} - d_0 S^{*r} + \frac{k_2 e_0 I^* S^*}{(1 + k_2 P^*)^2}, \\ J_{21} &= \frac{e_0 I^*}{1 + k_2 P^*}, \\ J_{22} &= -a_1 - d_1 P^* + \frac{e_0 S^*}{1 + k_2 P^*}, \\ J_{23} &= -d_1 I^* - \frac{k_2 e_0 I^* S^*}{(1 + k_2 P^*)^2}, \\ J_{31} &= d_2 r P^* S^{*(r-1)}, \\ J_{32} &= d_3 P^*, \\ J_{33} &= -a_2 + d_3 I^* + d_2 S^{*r}. \end{aligned}$$

Theorem 3.1. *Let $K > 1$ and $a_2 > d_2$. Then the susceptible prey-only equilibrium E_1 is locally stable if $L < K < \frac{a_1}{e_0}$ and $r < \frac{\ln\left(\frac{a_2}{d_2}\right)}{\ln K}$.*

Proof. Suppose $L < K < \frac{a_1}{e_0}$ and $r < \frac{\ln\left(\frac{a_2}{d_2}\right)}{\ln K}$. Then an evaluation of the Jacobian at E_1 yields

$$J_{E_1} = \begin{pmatrix} b_0(L-K) & b_0(L-K) - Ke_0 & -d_0K^r \\ 0 & Ke_0 - a_1 & 0 \\ 0 & 0 & d_2K^r - a_2 \end{pmatrix}. \quad (3.11)$$

The eigenvalues associated with J_{E_1} are $\lambda_1 = -b_0(K-L)$, $\lambda_2 = d_2K^r - a_2$, and $\lambda_3 = Ke_0 - a_1$, and are all negative. Hence E_1 is locally stable. However, E_1 is locally unstable if at least one of the following conditions are met: $L > K$ or $K > \frac{a_1}{e_0}$ or $r > \frac{\ln\left(\frac{a_2}{d_2}\right)}{\ln K}$. $\square$

Theorem 3.2. *The susceptible prey-only equilibrium E_2 is locally stable if $K < L < \frac{a_1}{e_0}$ and $r < \frac{\ln\left(\frac{a_2}{d_2}\right)}{\ln L}$.*

Proof. Assume $K < L < \frac{a_1}{e_0}$ and $r < \frac{\ln\left(\frac{a_2}{d_2}\right)}{\ln L}$. The Jacobian matrix evaluated at E_2 is given by

$$J_{E_2} = \begin{pmatrix} \frac{b_0L(K-L)}{K} & -Le_0 & -d_0L^r \\ 0 & Le_0 - a_1 & 0 \\ 0 & 0 & d_2L^r - a_2 \end{pmatrix}. \quad (3.12)$$

The eigenvalues of J_{E_2} are $\lambda_1 = \frac{b_0L(K-L)}{K}$, $\lambda_2 = d_2L^r - a_2$, and $\lambda_3 = Le_0 - a_1$. Based on earlier assumptions, each eigenvalue is negative and hence E_2 is locally stable. This susceptible prey-only state loses its stability when either $K > L$ or $L > \frac{a_1}{e_0}$ or $r > \frac{\ln\left(\frac{a_2}{d_2}\right)}{\ln L}$. $\square$

With regards to the stability of E_3 , an evaluation of J_{E_3} results in the matrix

$$J_{E_3} = \begin{pmatrix} A_{11} & A_{12} & A_{13} \\ 0 & A_{22} & 0 \\ A_{31} & A_{32} & 0 \end{pmatrix}, \quad (3.13)$$

where

$$\begin{aligned} A_{11} &= \frac{b_0 \left(2(K+L) \left(\frac{a_2}{d_2} \right)^{1/r} - 3 \left(\frac{a_2}{d_2} \right)^{2/r} - KL \right)}{K(1+k_1P_3)} - d_0r \left(\left(\frac{a_2}{d_2} \right)^{1/r} \right)^{r-1} P_3, \\ A_{12} &= \left(\frac{a_2}{d_2} \right)^{1/r} \left(\frac{b_0 \left(L - \left(\frac{a_2}{d_2} \right)^{1/r} \right)}{K(1+k_1P_3)} - \frac{e_0}{1+k_2P_3} \right), \\ A_{13} &= \frac{b_0k_1 \left(\frac{a_2}{d_2} \right)^{1/r} \left(\left(\frac{a_2}{d_2} \right)^{1/r} - K \right) \left(\left(\frac{a_2}{d_2} \right)^{1/r} - L \right)}{K(1+k_1P_3)^2} - \frac{d_0a_2}{d_2}, \\ A_{22} &= \frac{e_0 \left(\frac{a_2}{d_2} \right)^{1/r}}{1+k_2P_3} - a_1 - d_1P_3, \\ A_{31} &= d_2r \left(\left(\frac{a_2}{d_2} \right)^{1/r} \right)^{r-1} P_3, \end{aligned}$$

$$A_{32} = d_3 P_3.$$

One of the eigenvalues of J_{E_3} is $\lambda_1 = A_{22}$ and the other two eigenvalues are roots of the characteristic equation

$$\lambda^2 - A_{11}\lambda - A_{13}A_{31} = 0, \quad (3.14)$$

which satisfy $\lambda_2 + \lambda_3 = A_{11}$ and $\lambda_2\lambda_3 = -A_{31}A_{13}$. By the Routh–Hurwitz stability criteria, the infectious prey-free state is locally stable provided $A_{22} < 0$, $A_{11} < 0$, and $-A_{31}A_{13} > 0$. (This happens only if $A_{13} < 0$.) Next, we state a theorem concerning the local stability of the infectious prey-free state.

Theorem 3.3. *The infectious prey-free equilibrium E_3 is locally stable if $A_{22} < 0$, $A_{11} < 0$ and $A_{13} < 0$.*

Next, an evaluation of the Jacobian at E_4 yields

$$J_{E_4} = \begin{pmatrix} B_{11} & B_{12} & B_{13} \\ B_{21} & B_{22} & B_{23} \\ 0 & 0 & B_{33} \end{pmatrix}, \quad (3.15)$$

where

$$\begin{aligned} B_{11} &= \frac{b_0(2a_0e_0(-I_4 + K + L) - 3a_0^2 + Le_0^2(I_4 - K))}{Ke_0^2} - e_0I_4, \\ B_{12} &= \left(\frac{b_0(Le_0 - a_0)}{Ke_0^2} - 1 \right), \\ B_{13} &= \frac{a_0b_0k_1(a_0 - Le_0)(a_0 + e_0(I_4 - K))}{Ke_0^3} - d_0\left(\frac{a_0}{e_0}\right)^r + a_0k_2I_4, \\ B_{21} &= e_0I_4, \\ B_{22} &= a_0 - a_1, \\ B_{23} &= -(a_0k_2 + d_1)I_4, \\ B_{33} &= d_2\left(\frac{a_0}{e_0}\right)^r - a_2 + d_3I_4. \end{aligned}$$

The characteristic equation for J_{E_4} is

$$\lambda^3 + \zeta_1\lambda^2 + \zeta_2\lambda + \zeta_3 = 0, \quad (3.16)$$

where

$$\begin{aligned} \zeta_1 &= -(B_{11} + B_{22}), \\ \zeta_2 &= B_{11}B_{22} - B_{12}B_{21}, \\ \zeta_3 &= B_{12}B_{21}B_{33} - B_{11}B_{22}B_{33}. \end{aligned}$$

We can ascertain the local stability of E_4 by applying the Routh–Hurwitz stability criteria in the following theorem:

Theorem 3.4. *The predator-free equilibrium E_4 is locally stable if $\zeta_1 > 0, \zeta_2 > 0, \zeta_3 > 0$, and $\zeta_1\zeta_2 - \zeta_3 > 0$.*

Finally, we evaluate J_{E_5} and obtain the following characteristic equation:

$$\lambda^3 + \sigma_1\lambda^2 + \sigma_2\lambda + \sigma_3 = 0, \quad (3.17)$$

where

$$\begin{aligned} \sigma_1 &= -(J_{11} + J_{22} + J_{33}), \\ \sigma_2 &= J_{22}J_{33} + J_{11}(J_{22} + J_{33}) - J_{12}J_{21} - J_{13}J_{31} - J_{23}J_{32}, \\ \sigma_3 &= J_{13}J_{22}J_{31} - J_{12}J_{23}J_{31} - J_{13}J_{21}J_{32} + J_{11}J_{23}J_{32} + J_{12}J_{21}J_{33} - J_{11}J_{22}J_{33}. \end{aligned}$$

Similarly, we apply the Routh–Hurwitz stability criteria for local stability of the coexistence equilibrium via the next theorem.

Theorem 3.5. *The coexistence equilibrium E_5 is locally stable if $\sigma_1 > 0, \sigma_2 > 0, \sigma_3 > 0$, and $\sigma_1\sigma_2 - \sigma_3 > 0$.*

4. Dynamics near the extinction equilibrium

The extinction equilibrium E_0 requires a nonstandard analysis because the aggregation term S^r , with $0 < r < 1$, is not differentiable at $S = 0$. Consequently, the Jacobian matrix of model (2.1) is not defined at E_0 , and classical linearization results, including the Hartman–Grobman theorem, cannot be applied. We therefore analyze the dynamics near E_0 directly by means of comparison arguments.

The behavior near the extinction state depends essentially on whether predators are initially present. When $P(0) > 0$, the non-Lipschitz predation term d_0S^rP may drive a sufficiently small susceptible prey population to zero in finite time. When $P(0) = 0$, the predator-free face is invariant and the susceptible population cannot reach zero in finite time. Its asymptotic behavior is then determined by the sign of the Allee threshold L .

Theorem 4.1. *Assume that*

$$0 < r < 1, \quad S(0) > 0, \quad I(0) \geq 0, \quad P(0) > 0.$$

Let M_S, M_I , and M_P denote uniform upper bounds for $S(t), I(t)$, and $P(t)$, respectively. Define

$$\alpha = \max \left\{ 0, \sup_{\substack{0 \leq S \leq M_S \\ 0 \leq I \leq M_I \\ 0 \leq P \leq M_P}} \left[\frac{b_0}{1 + k_1 P} \left(1 - \frac{S + I}{K} \right) (S - L) - \frac{e_0 I}{1 + k_2 P} \right] \right\},$$

and let

$$\gamma = (1 - r)\alpha + a_2.$$

If

$$S(0)^{1-r} < \frac{(1-r)d_0P(0)}{\gamma}, \quad (4.1)$$

then the susceptible prey population reaches zero in finite time. More precisely, there exists $T_{\text{ext}} < \infty$ such that

$$S(t) > 0 \quad \text{for} \quad 0 \leq t < T_{\text{ext}}, \quad S(T_{\text{ext}}) = 0,$$

and

$$T_{\text{ext}} \leq -\frac{1}{\gamma} \ln \left(1 - \frac{\gamma S(0)^{1-r}}{(1-r)d_0P(0)} \right). \quad (4.2)$$

Following extinction of the susceptible prey, the infected prey and predator populations decay exponentially to zero. Consequently,

$$\lim_{t \rightarrow \infty} (S(t), I(t), P(t)) = (0, 0, 0).$$

Proof. The predator equation can be written as

$$\frac{dP}{dt} = P(d_2S^r + d_3I - a_2).$$

Since $S, I \geq 0$, we have

$$\frac{dP}{dt} \geq -a_2P.$$

Because $P(0) > 0$, the comparison theorem gives

$$P(t) \geq P(0)e^{-a_2t}, \quad t \geq 0. \quad (4.3)$$

The susceptible prey equation may be written as

$$\frac{dS}{dt} = S \left[\frac{b_0}{1+k_1P} \left(1 - \frac{S+I}{K} \right) (S-L) - \frac{e_0I}{1+k_2P} \right] - d_0S^rP.$$

By the definition of α ,

$$\frac{dS}{dt} \leq \alpha S - d_0S^rP.$$

Using (4.3), we obtain

$$\frac{dS}{dt} \leq \alpha S - d_0P(0)e^{-a_2t}S^r \quad (4.4)$$

for as long as $S(t) > 0$.

Define

$$W(t) = S(t)e^{-\alpha t}.$$

Differentiating and using (4.4) gives

$$\begin{aligned} \frac{dW}{dt} &= e^{-\alpha t} \left(\frac{dS}{dt} - \alpha S \right) \\ &\leq -d_0P(0)e^{-(\alpha+a_2)t}S^r. \end{aligned}$$

Since $S(t) = W(t)e^{\alpha t}$, it follows that

$$\frac{dW}{dt} \leq -d_0 P(0) W^r e^{-[(1-r)\alpha + a_2]t}.$$

With

$$\gamma = (1-r)\alpha + a_2,$$

we therefore have

$$W^{-r} \frac{dW}{dt} \leq -d_0 P(0) e^{-\gamma t}.$$

Multiplication by $1-r > 0$ yields

$$\frac{d}{dt} W^{1-r} \leq -(1-r)d_0 P(0) e^{-\gamma t}.$$

Integrating from 0 to t , and observing that $W(0) = S(0)$, gives

$$W(t)^{1-r} \leq S(0)^{1-r} - \frac{(1-r)d_0 P(0)}{\gamma} (1 - e^{-\gamma t}). \quad (4.5)$$

Under condition (4.1), the right-hand side of (4.5) becomes zero in finite time. Solving

$$S(0)^{1-r} - \frac{(1-r)d_0 P(0)}{\gamma} (1 - e^{-\gamma t}) = 0$$

gives

$$t = -\frac{1}{\gamma} \ln \left(1 - \frac{\gamma S(0)^{1-r}}{(1-r)d_0 P(0)} \right).$$

Thus $W(t)$, and hence $S(t)$, must reach zero no later than the time specified in (4.2).

By Theorem 2.1, the susceptible-extinction face $\mathcal{S}_0$ is positively invariant. Hence,

$$S(t) \equiv 0, \quad t \geq T_{\text{ext}}.$$

Moreover, on the invariant manifold $S = 0$, the reduced (I, P) -system

$$\frac{dI}{dt} = -(d_1 P + a_1)I, \quad \frac{dP}{dt} = P(d_3 I - a_2)$$

is smooth and locally Lipschitz. Therefore, the post-extinction dynamics admit a unique continuation from $t = T_{\text{ext}}$.

The infected prey equation then satisfies

$$\frac{dI}{dt} = -d_1 I P - a_1 I \leq -a_1 I.$$

Therefore,

$$I(t) \leq I(T_{\text{ext}}) e^{-a_1(t-T_{\text{ext}})}, \quad t \geq T_{\text{ext}}, \quad (4.6)$$

and hence $I(t) \rightarrow 0$ exponentially.

For $t \geq T_{\text{ext}}$, the predator equation becomes

$$\frac{dP}{dt} = P(d_3I - a_2).$$

Using (4.6), we obtain

$$\begin{aligned} P(t) &= P(T_{\text{ext}}) \exp \left(\int_{T_{\text{ext}}}^t [d_3I(s) - a_2] ds \right) \\ &\leq P(T_{\text{ext}}) \exp \left(\frac{d_3I(T_{\text{ext}})}{a_1} - a_2(t - T_{\text{ext}}) \right). \end{aligned}$$

Thus $P(t) \rightarrow 0$ exponentially. It follows that

$$(S(t), I(t), P(t)) \longrightarrow (0, 0, 0), \quad \text{as } t \rightarrow \infty.$$

□

Remark 4.1. Condition (4.1) may equivalently be written as

$$S(0) < \left[\frac{(1-r)d_0P(0)}{(1-r)\alpha + a_2} \right]^{\frac{1}{1-r}}.$$

It therefore identifies an explicit region of initial conditions from which predator pressure drives susceptible prey to extinction in finite time. This region expands as the initial predator density $P(0)$ or attack rate d_0 increases. The result holds for any $L \in \mathbb{R}$; thus, finite-time extinction caused by the non-Lipschitz predation term is not restricted to the strong Allee regime.

The finite-time extinction mechanism established above is a direct consequence of the non-Lipschitz aggregation-based predation term S^r , $0 < r < 1$, which causes the per capita predation pressure d_0PS^{r-1} to increase as the susceptible prey density approaches zero. Biologically, this idealizes situations in which individuals become increasingly vulnerable once aggregation can no longer provide effective protection against predators. Such behavior should therefore be interpreted as an idealized representation of severe low-density vulnerability rather than as a literal prediction that predation rates become unbounded in natural populations. The resulting finite-time extinction is thus a property of the proposed model formulation, while its ecological significance lies in demonstrating that strong aggregation-mediated nonlinearities can generate abrupt population collapse rather than gradual asymptotic decline.

We next examine the case in which predators are initially absent. Define the predator-free face

$$\mathcal{P}_0 = \{(S, I, P) \in \mathbb{R}_+^3 : P = 0\}.$$

Proposition 1. *Suppose that*

$$P(0) = 0, \quad S(0) > 0, \quad I(0) \geq 0.$$

Then

$$P(t) = 0 \quad \text{and} \quad S(t) > 0$$

for every finite $t \geq 0$. Consequently, susceptible prey cannot undergo finite-time extinction on the predator-free face.

Proof. Because

$$\frac{dP}{dt} = P(d_2 S^r + d_3 I - a_2),$$

the condition $P(0) = 0$ implies

$$P(t) = 0, \quad \text{for all } t \geq 0.$$

Thus $\mathcal{P}_0$ is positively invariant.

On $\mathcal{P}_0$, the susceptible prey equation reduces to

$$\frac{dS}{dt} = S \left[b_0 \left(1 - \frac{S + I}{K} \right) (S - L) - e_0 I \right].$$

The expression in brackets is continuous and bounded on every bounded time interval. Hence,

$$S(t) = S(0) \exp \left\{ \int_0^t \left[b_0 \left(1 - \frac{S(s) + I(s)}{K} \right) (S(s) - L) - e_0 I(s) \right] ds \right\}.$$

Since $S(0) > 0$, it follows that

$$S(t) > 0$$

for every finite $t \geq 0$. Thus finite-time extinction cannot occur in the absence of predators. $\square$

The preceding proposition does not determine whether $S(t)$ converges to zero asymptotically. That behavior depends on the sign of the Allee threshold.

Proposition 2. *Suppose that $L \leq 0$. Then the extinction equilibrium E_0 is unstable.*

Proof. Consider the susceptible-only axis

$$\mathcal{A} = \{(S, I, P) \in \mathbb{R}_+^3 : I = P = 0\}.$$

Since the equations for I and P contain I and P , respectively, as multiplicative factors, $\mathcal{A}$ is positively invariant.

On $\mathcal{A}$, model (2.1) reduces to

$$\frac{dS}{dt} = b_0 S \left(1 - \frac{S}{K} \right) (S - L).$$

If $L < 0$, then for all sufficiently small $S > 0$,

$$1 - \frac{S}{K} > 0 \quad \text{and} \quad S - L > 0.$$

Therefore,

$$\frac{dS}{dt} > 0.$$

Hence trajectories starting arbitrarily close to E_0 on $\mathcal{A}$ move away from the extinction state.

If $L = 0$, then

$$\frac{dS}{dt} = b_0 S^2 \left(1 - \frac{S}{K} \right) > 0$$

whenever $0 < S < K$. These trajectories also move away from E_0 . Consequently, E_0 is unstable for every $L \leq 0$. $\square$

Theorem 4.2. Suppose that $L > 0$. Then E_0 is locally asymptotically stable relative to the predator-free face $\mathcal{P}_0$. More precisely, there exists $\delta > 0$ such that every solution satisfying

$$0 < S(0) < \delta, \quad 0 \leq I(0) < \delta, \quad P(0) = 0$$

obeys

$$\lim_{t \rightarrow \infty} (S(t), I(t), P(t)) = (0, 0, 0).$$

Proof. Choose

$$0 < \delta < \min \left\{ \frac{L}{2}, \frac{K}{4}, \frac{a_1}{2e_0} \right\}.$$

Suppose that

$$0 < S(0) < \delta, \quad 0 \leq I(0) < \delta, \quad P(0) = 0.$$

By Proposition 1,

$$P(t) = 0, \quad \text{for all } t \geq 0.$$

Assume that

$$0 < S(t) \leq \delta, \quad 0 \leq I(t) \leq \delta.$$

Then

$$S(t) + I(t) \leq 2\delta < \frac{K}{2},$$

and hence

$$1 - \frac{S + I}{K} \geq \frac{1}{2}.$$

Moreover,

$$S - L \leq -\frac{L}{2}.$$

It follows that

$$\begin{aligned} \frac{dS}{dt} &= b_0 S \left(1 - \frac{S + I}{K} \right) (S - L) - e_0 S I \\ &\leq -\frac{b_0 L}{4} S. \end{aligned}$$

Thus $S(t)$ is decreasing and cannot cross the boundary $S = \delta$.

Similarly,

$$\frac{dI}{dt} = I(e_0 S - a_1).$$

Because

$$S(t) \leq \delta < \frac{a_1}{2e_0},$$

we obtain

$$\frac{dI}{dt} \leq -\frac{a_1}{2} I.$$

Therefore $I(t)$ is also decreasing and cannot cross the boundary $I = \delta$. Consequently,

$$(0, \delta] \times [0, \delta] \times \{0\}$$

is positively invariant.

Applying the comparison theorem gives

$$S(t) \leq S(0)e^{-\frac{b_0 L}{4}t}$$

and

$$I(t) \leq I(0)e^{-\frac{a_1}{2}t}.$$

Hence,

$$S(t) \rightarrow 0, \quad I(t) \rightarrow 0, \quad P(t) = 0$$

as $t \rightarrow \infty$. Therefore, E_0 is locally asymptotically stable relative to the predator-free face when $L > 0$. $\square$

Remark 4.2. The preceding results reveal a direction-dependent structure near the extinction equilibrium. If predators are initially present, sufficiently small susceptible prey populations may reach zero in finite time because the sublinear predation term $S^r P$ dominates the linear demographic terms near $S = 0$. If predators are absent, finite-time extinction is impossible, and the asymptotic behavior is controlled by the Allee threshold: E_0 is unstable for $L \leq 0$, but locally asymptotically stable relative to the predator-free face for $L > 0$. These features cannot be detected through standard Jacobian linearization and arise directly from the non-Lipschitz structure of the model.

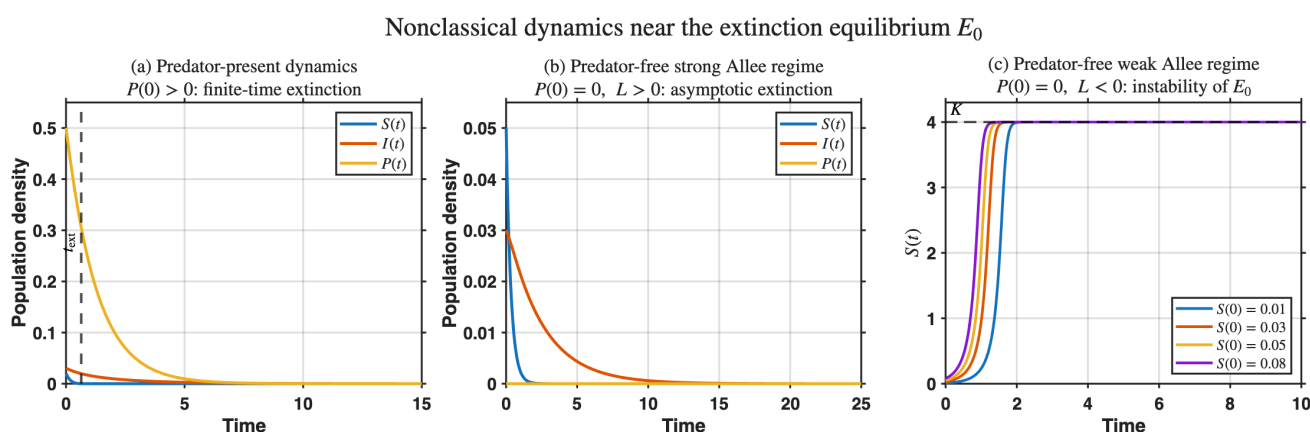


Figure 2. Numerical illustration of the nonclassical dynamics near the extinction equilibrium $E_0 = (0, 0, 0)$. Panel (a) shows finite-time extinction of the susceptible prey population when predators are initially present, using $L = 1$ and $(S(0), I(0), P(0)) = (0.02, 0.03, 0.50)$. The vertical dashed line denotes the numerical extinction time t_{ext} ; following this event, the infected prey and predator populations decay exponentially. Panel (b) illustrates the predator-free strong Allee regime with $L = 1$ and $(S(0), I(0), P(0)) = (0.05, 0.03, 0)$. In this case, both prey classes converge asymptotically to zero, while $P(t) \equiv 0$, demonstrating that finite-time extinction does not occur in the absence of predators. Panel (c) shows the predator-free weak Allee regime with $L = -1$ and several small susceptible-prey initial densities on the invariant axis $I = P = 0$. The trajectories move away from E_0 and approach the carrying capacity K , confirming instability of the extinction equilibrium for $L < 0$. All other parameter values are given in Table 1.

Example 4.1. For the trajectory shown in Figure 2 and the baseline parameter values listed in Table 1, the susceptible prey remains below the strong Allee threshold and the total prey population remains below carrying capacity before extinction:

$$0 < S(t) < L, \quad S(t) + I(t) < K, \quad 0 \leq t < t_{\text{ext}}^{\text{num}}.$$

Hence,

$$\frac{b_0}{1 + k_1 P(t)} \left(1 - \frac{S(t) + I(t)}{K} \right) (S(t) - L) - \frac{e_0 I(t)}{1 + k_2 P(t)} \leq 0$$

throughout the pre-extinction interval. Therefore, the estimate in Theorem 4.1 may be applied with

$$\alpha = 0,$$

so that

$$\gamma = (1 - r)\alpha + a_2 = a_2 = 0.8.$$

We first verify the sufficient condition

$$S_0^{1-r} < \frac{(1-r)d_0 P_0}{\gamma}.$$

Indeed,

$$S_0^{1-r} = (0.02)^{1/2} = 0.141421,$$

whereas

$$\frac{(1-r)d_0 P_0}{\gamma} = \frac{(0.5)(0.7)(0.50)}{0.8} = 0.21875.$$

Thus,

$$0.141421 < 0.21875$$

and the finite-time extinction criterion in Theorem 4.1 is satisfied.

The theorem therefore gives

$$T_{\text{ext}} \leq -\frac{1}{\gamma} \ln \left(1 - \frac{\gamma S_0^{1-r}}{(1-r)d_0 P_0} \right).$$

Substituting the parameter values yields

$$\begin{aligned} T_{\text{ext}} &\leq -\frac{1}{0.8} \ln \left(1 - \frac{0.8(0.02)^{1/2}}{(0.5)(0.7)(0.50)} \right) \\ &\approx 1.2998. \end{aligned}$$

In the numerical simulation, extinction is detected when the susceptible prey density first reaches the prescribed numerical tolerance

$$\varepsilon_{\text{ext}} = 10^{-8}.$$

Accordingly, we define

$$t_{\text{ext}}^{\text{num}} = \inf \{ t > 0 : S(t) \leq \varepsilon_{\text{ext}} \}.$$

The MATLAB simulation gives

$$t_{\text{ext}}^{\text{num}} = 0.64030724.$$

Consequently,

$$t_{\text{ext}}^{\text{num}} = 0.6403 < 1.2998,$$

which confirms the analytical upper estimate obtained from Theorem 4.1.

The numerical extinction occurs earlier because the proof uses only the conservative predator lower bound

$$P(t) \geq P_0 e^{-a_2 t}$$

and neglects the additional negative contributions of the strong Allee term and disease transmission in the susceptible-prey equation. Following the finite-time loss of the susceptible prey population, the infected prey and predator populations decay exponentially, in agreement with the cascade-collapse conclusion of Theorem 4.1.

5. Bifurcation dynamics

In this section, we explore and analyze the occurrence of possible local co-dimension one and two bifurcations. These bifurcations provide valuable insight into the qualitative changes in the dynamics of model (2.1) that arise when key parameters of interest—namely, the level of fear k_1 , Allee effect L , and prey aggregation exponent r are varied.

5.1. Co-dimension one bifurcation

Co-dimension one bifurcations are typically associated with the creation and collision of steady states or period orbits when single parameters of interest are varied. We establish the conditions for the occurrence of a saddle-node bifurcation, Hopf bifurcation, and transcritical bifurcation via the following theorems:

Theorem 5.1. *Model (2.1) experiences a saddle-node bifurcation around E_5 at $L = L^*$ when $\text{tr}(J_{E_5}) < 0$ and $\det(J_{E_5}) = 0$.*

Proof. Suppose $\text{tr}(J_{E_5}) < 0$ and $\det(J_{E_5}) = 0$. Then model (2.1) has a simple zero eigenvalue. Let $v = (v_1, v_2, v_3)^T \neq 0$ and $w = (w_1, w_2, w_3)^T \neq 0$ be eigenvectors corresponding to the zero eigenvalue of J_{E_5} and $J_{E_5}^T$, respectively. Let $Z = (z_1, z_2, z_3)^T$, where

$$\begin{aligned} z_1 &= \frac{b_0 S}{1 + k_1 P} \left(1 - \frac{S + I}{K} \right) (S - L) - d_0 S^r P - \frac{e_0 S I}{1 + k_2 P}, \\ z_2 &= \frac{e_0 S I}{1 + k_2 P} - d_1 I P - a_1 I, \\ z_3 &= d_2 S^r P + d_3 I P - a_2 P. \end{aligned}$$

By ensuring $w_1 \neq 0$ and $S^* + I^* \neq K$,

$$w^T Z_L(E_2, L^*) = w^T \left(-\frac{b_0 S^*}{1 + k_1 P^*} \left(1 - \frac{S^* + I^*}{K} \right), 0, 0 \right)^T$$

$$= -\frac{w_1 b_0 S^*}{1 + k_1 P^*} \left(1 - \frac{S^* + I^*}{K} \right) \neq 0.$$

Therefore the second transversality condition is validated. Furthermore,

$$w^T \left[D^2 Z(E_5, L^*) (v, v) \right] \neq 0,$$

where $D^2 Z = (\nabla^2 z_1, \nabla^2 z_2, \nabla^2 z_3)^2$. By Sotomayor's theorem [29], model (2.1) undergoes a saddle-node bifurcation around the coexistence equilibrium E_5 at $L = L^*$. $\square$

Theorem 5.2. *Model (2.1) experiences a Hopf bifurcation around E_5 at $r = r^*$ provided the following conditions are satisfied:*

$$\sigma_1(r_H) > 0, \sigma_3(r_H) > 0, \sigma_1(r_H)\sigma_2(r_H) - \sigma_3(r_H) = 0, \quad (5.1)$$

and

$$[\sigma_1(r)\sigma_2(r)]'_{r=r_H} - \sigma_3'(r_H) \neq 0. \quad (5.2)$$

Proof. We rewrite the characteristic equation (3.17) in the form

$$[\lambda^2(r_H) + \sigma_2(r_H)][\lambda(r_H) + \sigma_1(r_H)] = 0, \quad (5.3)$$

so that a Hopf bifurcation can occur with roots $\lambda_1(r_H) = i\sqrt{\sigma_2(r_H)}$, $\lambda_2(r_H) = -i\sqrt{\sigma_2(r_H)}$, $\lambda_3(r_H) = -\sigma_1(r_H) < 0$. It is easy to see that $\sigma_3(r_H) = \sigma_1(r_H)\sigma_2(r_H)$. Next, we establish the transversality condition

$$\left. \frac{d(\operatorname{Re} \lambda_k(r))}{dr} \right|_{r=r_H} \neq 0, k = 1, 2, \quad (5.4)$$

to enable the verification of the existence of periodic solutions bifurcating around E_5^* at $r = r_H$. We substitute $\lambda_k(r) = \Gamma(r) + i\Lambda(r)$ into (5.3) and compute the derivative to obtain

$$\varpi_1(c)\Gamma'(c) - \varpi_2(c)\Lambda'(c) + \varpi_4(c) = 0, \quad (5.5)$$

$$\varpi_2(c)\Gamma'(c) + \varpi_1(c)\Lambda'(c) + \varpi_3(c) = 0, \quad (5.6)$$

where

$$\begin{aligned} \varpi_1(r) &= 3\Gamma^2(r) - 3\Lambda^2(r) + \sigma_2(r) + 2\sigma_1(r)\Gamma(r), \\ \varpi_2(r) &= 6\Gamma(r)\Lambda(r) + 2\sigma_1(r)\Lambda(r), \\ \varpi_3(r) &= 2\Gamma(r)\Lambda(r)\sigma_1'(r) + \sigma_2'(r)\Lambda(r), \\ \varpi_4(r) &= \sigma_2'(r)\Gamma(r) + \Gamma^2(r)\sigma_1'(r) - \Lambda^2(r)\sigma_1'(r) + \sigma_3'(r). \end{aligned}$$

By using Cramer's rule, we solve for $\Gamma'(r_H)$ from the linear systems in (5.5) and (5.6). It is worth noting that, at $r = r_H$, $\Gamma(r_H) = 0$ and $\Lambda(r_H) = \sqrt{\sigma_2(r_H)}$, resulting in

$$\varpi_1(r_H) = -2\sigma_2(r_H),$$

$$\begin{aligned}\varpi_2(r_H) &= 2\sigma_1(r_H)\sqrt{\sigma_2(r_H)}, \\ \varpi_3(r_H) &= \sigma'_2(r_H)\sqrt{\sigma_2(r_H)}, \\ \varpi_4(r_H) &= \sigma'_3(r_H) - \sigma_2(r_H)\sigma'_1(r_H).\end{aligned}$$

We now have

$$\begin{aligned}\left.\frac{d\operatorname{Re}(\lambda_k(r))}{dr}\right|_{r=r_H} &= \Gamma'(r_H), \\ &= -\frac{\varpi_3(r_H)\varpi_2(r_H) + \varpi_4(r_H)\varpi_1(r_H)}{\varpi_1^2(r_H) + \varpi_2^2(r_H)}, \\ &= \frac{\sigma'_3(r_H) - \sigma_1(r_H)\sigma'_2(r_H) - \sigma_2(r_H)\sigma'_1(r_H)}{2(\sigma_2(r_H) + \sigma_1^2(r_H))} \neq 0,\end{aligned}$$

on the condition that $[\sigma_1(r)\sigma_2(r)]'_{r=r_H} - \sigma'_3(r_H) \neq 0$.

Therefore, the transversality condition is established, which leads to the conclusion that the model experiences a Hopf bifurcation around E_5^* at $r = r_H$. $\square$

Theorem 5.3. *Model (2.1) experiences a transcritical bifurcation around E_2 at $r = r^* = \frac{\ln\left(\frac{a_2}{d_2}\right)}{\ln L}$.*

Proof. Simple computations show that at the critical susceptible prey aggregation exponent $r = r^* = \frac{\ln\left(\frac{a_2}{d_2}\right)}{\ln L}$ around E_2 ,

$$J_{E_2} = \begin{pmatrix} \frac{b_0 L(K-L)}{K} & -Le_0 & -\frac{d_0 a_2}{d_2} \\ 0 & Le_0 - a_1 & 0 \\ 0 & 0 & 0 \end{pmatrix}. \quad (5.7)$$

The associated eigenvalues for the matrix in (5.7) are $\lambda_1 = 0$, $\lambda_2 = \frac{b_0 L(K-L)}{K}$, and $\lambda_3 = Le_0 - a_1$. We let $G = (g_1, g_2, g_3)^T$ and $N = (n_1, n_2, n_3)^T$ represent the eigenvectors corresponding to the zero eigenvalue of J_{E_2} and $J_{E_2}^T$, respectively. Computations yield $G = \left(\frac{KL^{r-1}d_0}{(K-L)b_0}, 0, 1\right)^T$ and $N = (0, 0, 1)^T$. We proceed to validate the transversality conditions using Sotomayor's theorem [29].

$$N^T Z_r(E_2, r^*) = (0, 0, 1)(0, 0, 0)^T = 0.$$

Also,

$$\begin{aligned}N^T [DZ_r(E_2, r^*)G] &= \begin{pmatrix} 0 & 0 & 1 \end{pmatrix} \begin{pmatrix} 0 & 0 & -d_0 r L^{r-1} \\ 0 & 0 & 0 \\ 0 & 0 & d_2 r L^{r-1} \end{pmatrix} \begin{pmatrix} \frac{KL^{r-1}d_0}{(K-L)b_0} \\ 0 \\ 1 \end{pmatrix} \\ &= d_2 r L^{r-1} \neq 0\end{aligned}$$

and

$$N^T [D^2 Z(E_2, r^*)(G, G)] \neq 0.$$

Hence model (2.1) experiences a transcritical bifurcation at E_2 when $r = r^* = \frac{\ln\left(\frac{a_2}{d_2}\right)}{\ln L}$. $\square$

5.2. Numerical validation

This subsection focuses on providing numerical experiments to validate our theoretical findings. We use MATLAB version R2026a and MATCONT [30] software in performing the numerical experiments. We show the possible occurrence of co-dimension one bifurcations using bifurcation diagrams, in particular, for the parameters k_1 , r , and L when all other parameters are fixed. We shall use parameter values from Table 1 for our bifurcation experiments.

5.2.1. Impact of Allee effect parameter L

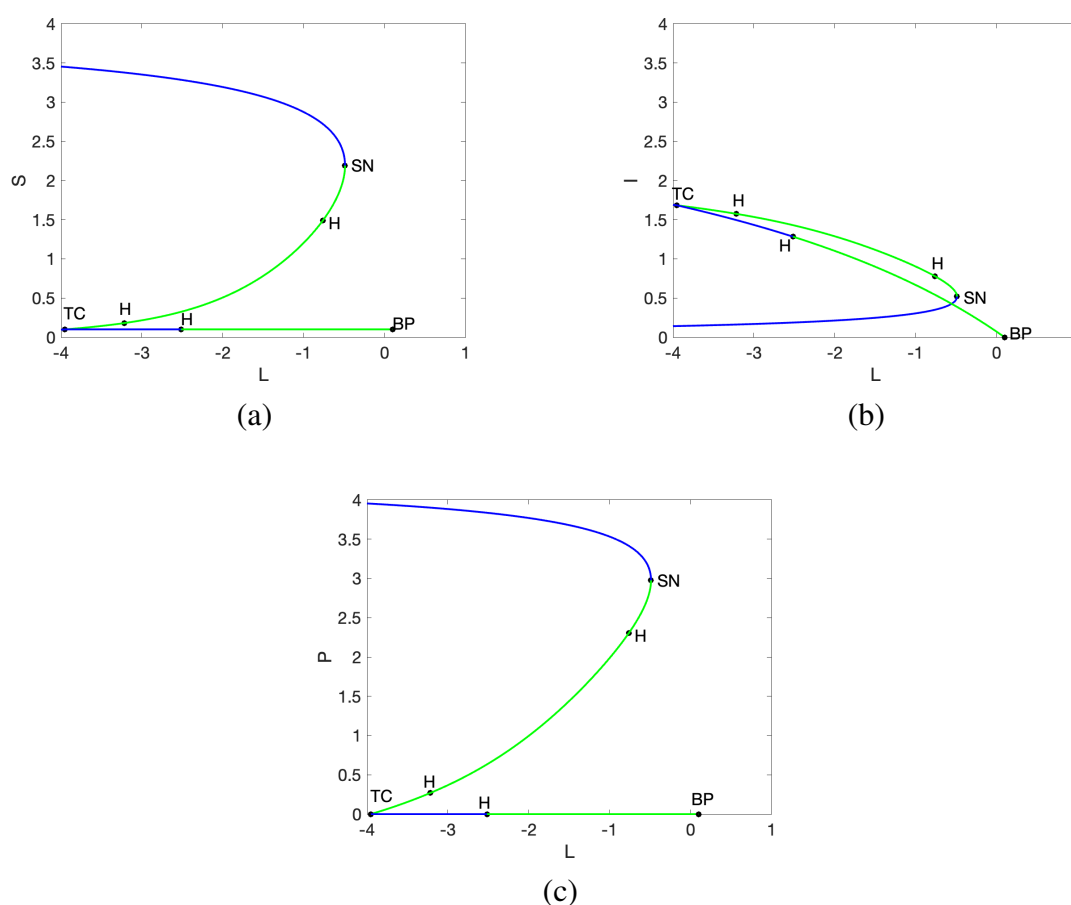


Figure 3. Bifurcation diagram for L for model (2.1). The blue and green lines denote stable and unstable branches, respectively. Note: SN = Saddle-node point, H = Hopf point, TC = Transcritical point, and BP = Branch point.

To understand the role of Allee effects in system (2.1), we fix all other parameters and vary L . See Figure 3. The system experiences a transcritical (TC) bifurcation at $L^{[TC]} = -3.95198$ around the predator-free state $E_4^{[TC]} = (0.1, 1.6838, 0)$. The occurrence of a TC bifurcation identifies a critical threshold for predator invasion and persistence. Ecologically, this threshold determines whether an introduced predator population can successfully establish itself or ultimately go extinct. In the context of bio-control, particularly when predators are introduced to suppress a disease-carrying prey population, the TC bifurcation provides conditions necessary for successful predator establishment and

sustained regulation of the target population. For a weak Allee threshold $L \in (-3.95198, -2.5164)$, the system experiences bistability phenomena between the predator-free state E_4 and the coexistence state E_5 . Therefore depending on the choice of initial data, solutions will either be attracted to E_4 or E_5 . Within this threshold interval, a Hopf bifurcation is noted at the critical value $L_1^{[H]} = -3.2192$ around $E_5^{[H]} = (0.1805, 1.5752, 0.2683)$. The first Lyapunov coefficient is computed as $\chi_{L_1} = 1.5666 \times 10^1$ and hence the bifurcation is subcritical. An increase away from the weak Allee threshold interval leads to the occurrence of another Hopf bifurcation at $L_2^{[H]} = -2.5164$ around $E_4^{[H]} = (0.1, 1.2836, 0)$ with its first Lyapunov coefficient calculated as $\chi_{L_2} = -7.7542 \times 10^{-2}$ and hence the bifurcation is a supercritical Hopf. A further increase of the weak Allee threshold causes the existence of a third Hopf bifurcation at $L_3^{[H]} = -0.7626$ around $E_5^{[H]} = (1.4889, 0.7798, 2.3043)$. A similar computation of the first Lyapunov coefficient yields $\chi_{L_3} = -2.0029 \times 10^0$, which means this bifurcation is also a supercritical Hopf. From our simulation, as the Allee threshold becomes less negative, the susceptible prey becomes vulnerable to demographic pressure or limitations, which can lead to a (de)stabilization of the coexistence equilibrium and drive the system toward persistent population cycles. A final increment of the weak Allee threshold causes the occurrence of a saddle-node bifurcation at $L_4^{[SN]} = -0.4882$ around $E_5^{[SN]} = (2.1913, 0.5197, 2.9760)$. Ecologically, the saddle-node bifurcation represents a catastrophic tipping point with direct conservation implications. Beyond $L_4^{[SN]}$, it will be difficult for management interventions to restore species persistence.

5.2.2. Impact of prey aggregation r

From the bifurcation diagram in Figure 4, if the strength of prey aggregation r is the interval $(0, 0.313944]$, the populations do not persist. This is due to a saddle-node bifurcation occurring at $r^{[SN]} = 0.313944$ around $E_5^{[SN]} = (2.350352, 0.692280, 3.112553)$. This shows that aggregation strength must exceed a threshold for all populations to coexist. A small increase in r results in the occurrence of a Hopf bifurcation at $r_1^{[H]} = 0.319942$ around $E_5^{[H]} = (2.252766, 0.703272, 3.029404)$ with a first Lyapunov coefficient computed as $\chi_{r_1} = -3.198244 \times 10^1$ and hence is supercritical. A further increase leads to a transcritical critical bifurcation at $r^{[TC]} = 0.544980$ around the infected prey-free equilibrium $E_3^{[TC]} = (3.567540, 0, 4.032677)$. The transcritical bifurcation provides an aggregation threshold where predators can indirectly eliminate disease from an ecosystem by depleting the susceptible prey population. This reduces the pool of hosts available for disease transmission. As a result, the infected class cannot replenish itself through new infections and ultimately goes extinct. Finally, we observe a second Hopf bifurcation occurring at $r_2^{[H]} = 0.719768$ around $E_5^{[H]} = (2.619583, 0, 5.424286)$ with a computed Lyapunov coefficient $\chi_{r_2} = -1.024881 \times 10^0$ classifying the Hopf bifurcation as supercritical.

5.2.3. Impact of fear levels k_1

We explore how predator-induced fear interacts with the Allee effect and aggregation dynamics to influence the persistence of susceptible prey in Figure 5. When we set $L = 1$, the Allee threshold is a strong one. For low fear levels $k_1 \in (0, 0.364829)$, two coexistence equilibria exist due to the density of the susceptible prey population not going below the strong Allee threshold of $L = 1$. At $k_1 = 0.364829$, the system experiences a Hopf bifurcation around $E_5^{[H]} = (2.451314, 0.347466, 2.96307)$ with its first Lyapunov coefficient calculated as $\chi_{k_1} = -2.542146$ and hence the bifurcation is supercritical. A slight further increase in fear levels results in the two coexistence equilibria colliding and annihilating each

other via a saddle-node bifurcation at $k_1 = 0.3677359$ around $E_5^{[SN]} = (2.546129, 0.32347, 3.03465)$. Similar dynamics are seen when $L = 0$. However, when $L = -1$, the Allee effect becomes a weak one. The population density of the susceptible prey is negatively impacted for fear levels k_1 in the range $(0, 1.417511)$. When $k_1^{[SN]} = 1.417511$, the system experiences a saddle-node bifurcation around $E_5^{[SN]} = (0.216553, 1.227718, 0.347142)$. A slight further increase results in the occurrence of a Hopf bifurcation at $k_1^{[H]} = 1.656706$ around $E_5^{[H]} = (1.736233, 0.545871, 2.371381)$. The computed first Lyapunov coefficient is $\chi_{k_1} = 2.4282446$ and hence the bifurcation is a subcritical Hopf. A final increase in fear levels results again in a saddle-node bifurcation at $k_1^{[SN]} = 1.718081$ around $E_5^{[SN]} = (1.272222, 0.697658, 1.919051)$. We observe that the densities of each population reduce as k_1 increases. For intermediate fear levels k_1 , the occurrence of a Hopf bifurcation shows that when fear crosses a critical threshold, the populations experience sustained population oscillations. Ecosystem managers monitoring species in disease- and fear-affected environments should consider the onset of cyclic population patterns as a signal to explore intervention strategies to prevent the populations from being ecologically vulnerable to extinction via a saddle-node bifurcation.

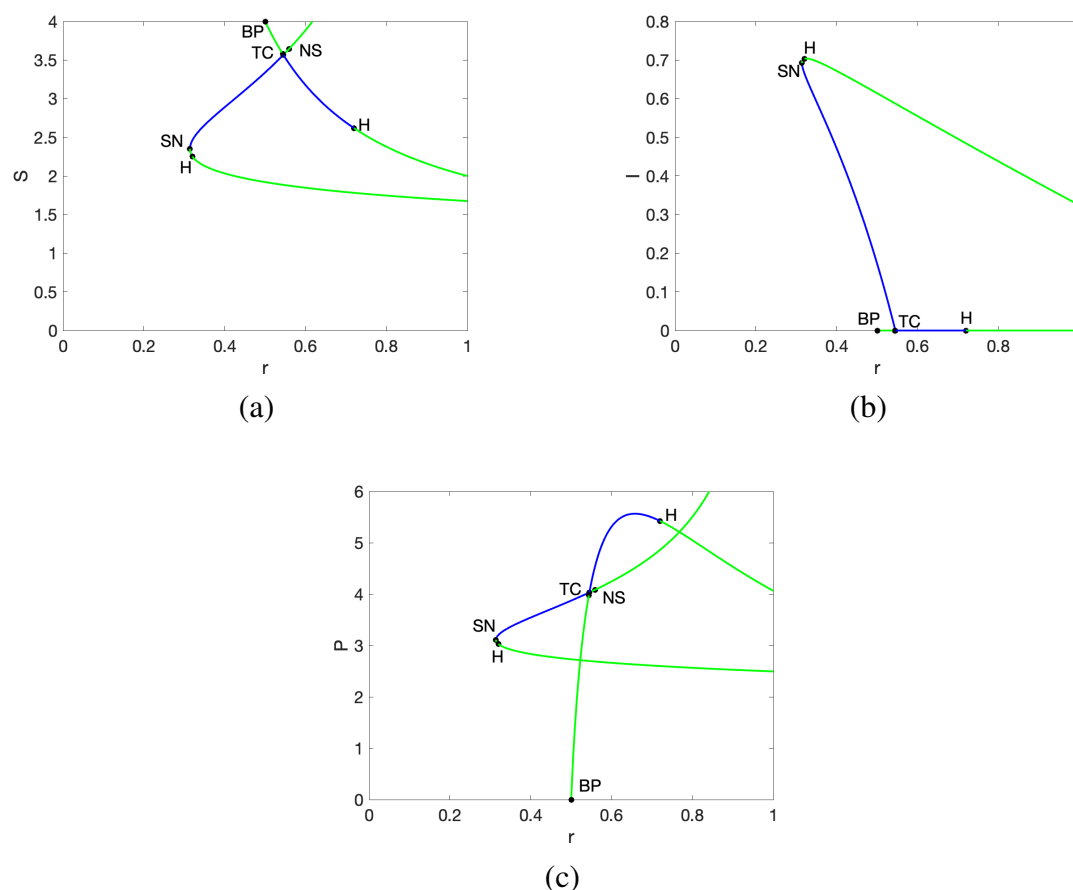


Figure 4. Bifurcation diagram for the aggregation exponent r on susceptible prey for model (2.1). The blue and green lines denote stable and unstable branches, respectively. In addition, we choose $b_0 = 8$ and $L = 1$. Note: SN = Saddle-node point, H = Hopf point, BP = Branch point, and NS = Neutral Saddle.

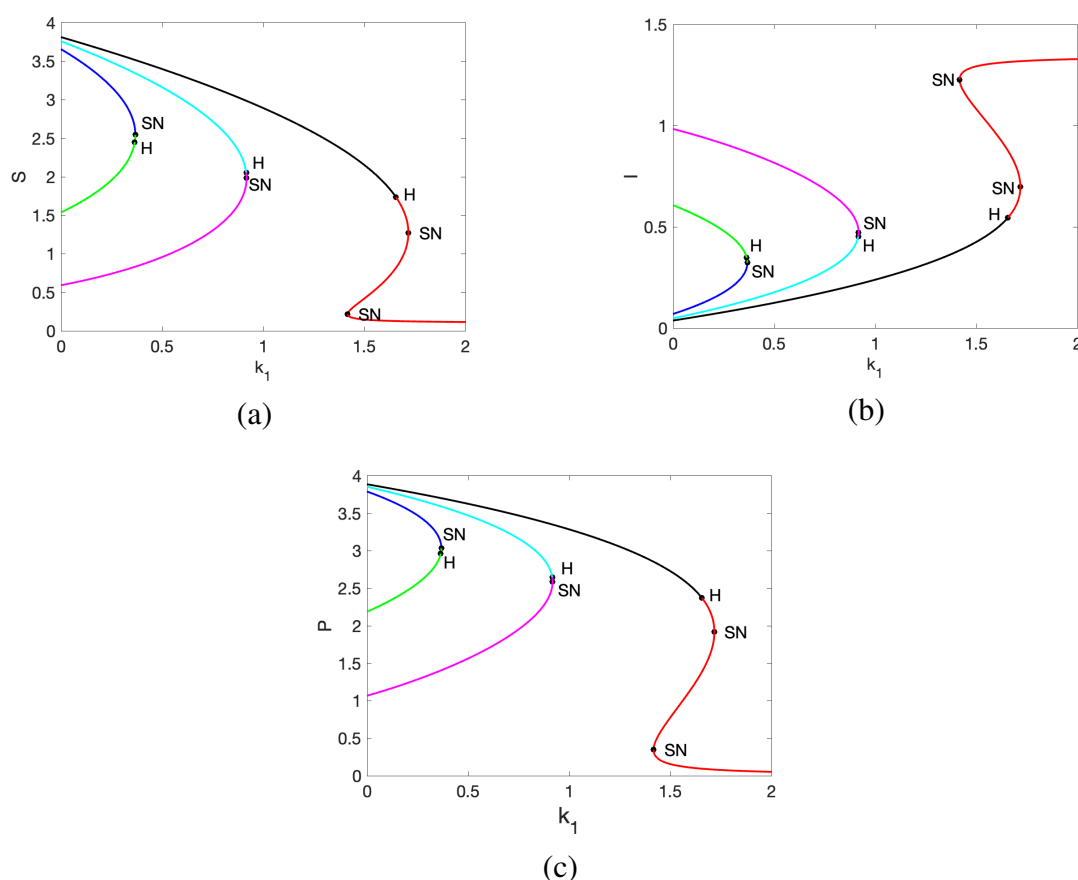


Figure 5. Bifurcation diagram for model (2.1) showing the impact of predator-induced fear k_1 on susceptible prey across different Allee thresholds L . Fixed parameter values are set at $b_0 = 8$, $k_2 = 1$, and $d_3 = 0.5$. Stable branches are indicated by blue ($L = 1$), cyan ($L = 0$), and black ($L = -1$) lines, whereas unstable branches are represented by green ($L = 1$), magenta ($L = 0$), and red ($L = -1$) lines. Note: SN = Saddle-node point, and H = Hopf point.

5.3. Two-parameter bifurcation

We explore a two-parameter bifurcation in the (k_1, L) and (k_1, k_2) planes respectively to deepen our understanding of how the interplay of fear-driven behavioral responses and Allee effects shape the long-term dynamics of the SIP system. We vary these pairs to reveal regions within the parameter space that support stable coexistence, oscillatory coexistence, predator-free state, infected prey-free state and finite-time extinction dynamics.

5.3.1. Combined effect of k_1 and L

The two-parameter bifurcation plot in the (k_1, L) plane seen in Figure 6 reveals interesting ecological dynamics. When the Allee threshold is sufficiently negative, the susceptible prey population maintains a large buffer of its density where the system settles into a stable coexistence (SC) state among all three populations. As the fear parameter k_1 increases, the growth rate of susceptible prey is suppressed enough to cause a destabilization of the SC state. The SC region is then displaced by the finite-time

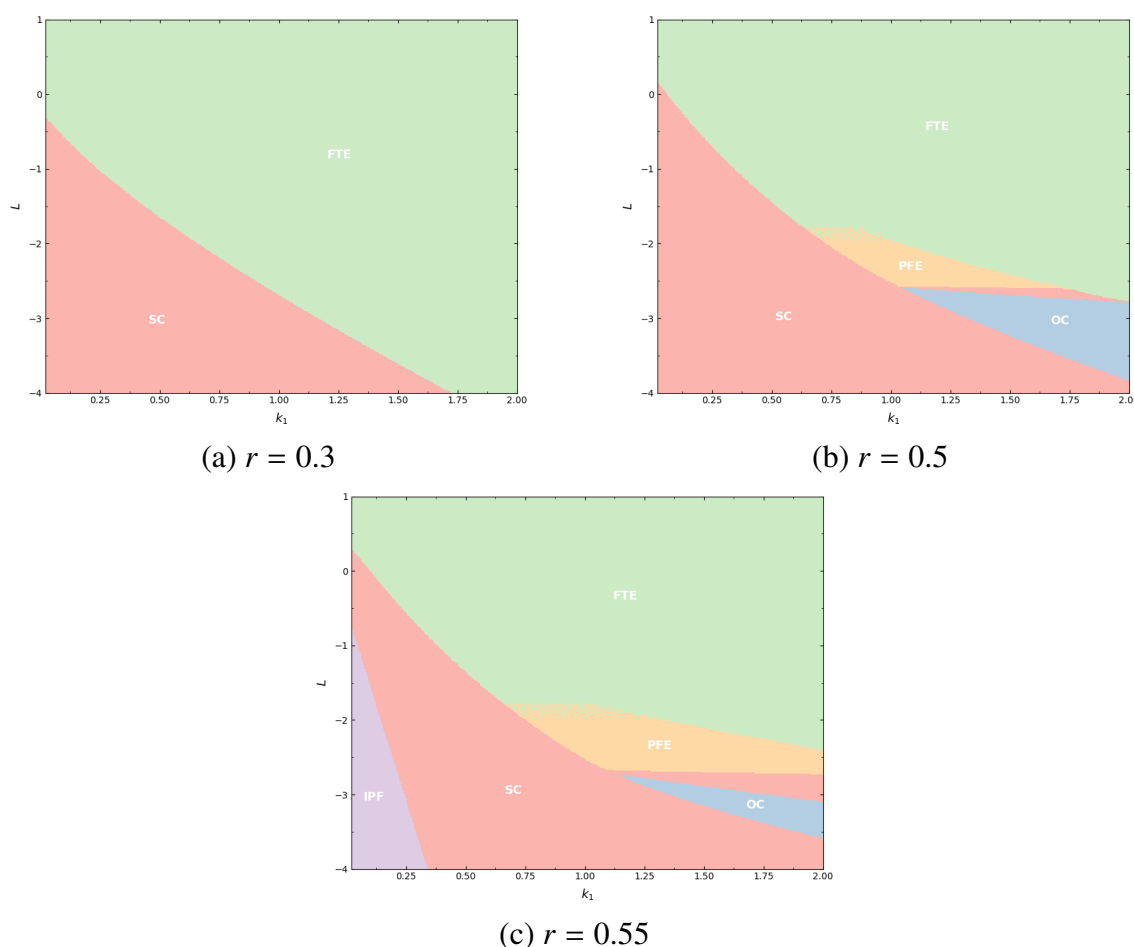


Figure 6. Plots showing dynamic, color-coded regions of a two-parameter bifurcation in the (k_1, L) space representing stable coexistence (SC), oscillatory coexistence (OC), finite-time extinction (FTE), predator-free equilibrium (PFE), and infectious prey-free (IPF) states. Here, we choose $b_0 = 2$ and $e_0 = 2$. All other parameters chosen are seen in Table 1.

extinction (FTE), predator-free equilibrium (PFE), and the oscillatory coexistence (OC) states. When the system enters the FTE regime, it collapses the susceptible prey population to zero in finite time with a subsequent cascading effect on the extinction of infected prey and predator populations, respectively. We also observed that, as L increases toward zero, moderate fear levels suppressing reproduction is sufficient to trigger the collapse of the system. The emergence of the PFE state indicates the critical role played by the prey aggregation exponent r for intermediate fear levels k_1 . By increasing r , the attack on susceptible prey is efficient and consequently reduces its population density. This reduction suppresses disease transmission between susceptible and infected individuals, thereby reducing the population density of the infected class. The predator gains energy from both prey classes and so if the combined energy intake from both sources is very low, the predator population can no longer sustain itself and will go extinct. The appearance of the infected prey-free (IPF) state at $r = 0.55$ for low fear levels k_1 and a strongly negative L indicates that predators become more effective in attacking susceptible prey, thereby reducing their population density. This in turn leads to a reduction of the rate at which new infections are produced and will eventually lead to the extinction of the infected class. The IPF region disappears

at $r = 0.5$ and $r = 0.3$ because predatory attacks are weaker and the susceptible prey population remain at higher densities, which will lead to increased contact with the infected class. In all, (k_1, L) shows that fear and Allee effect combined can act as extinction and destabilizing drivers.

5.3.2. Combined effect of k_1 and k_2

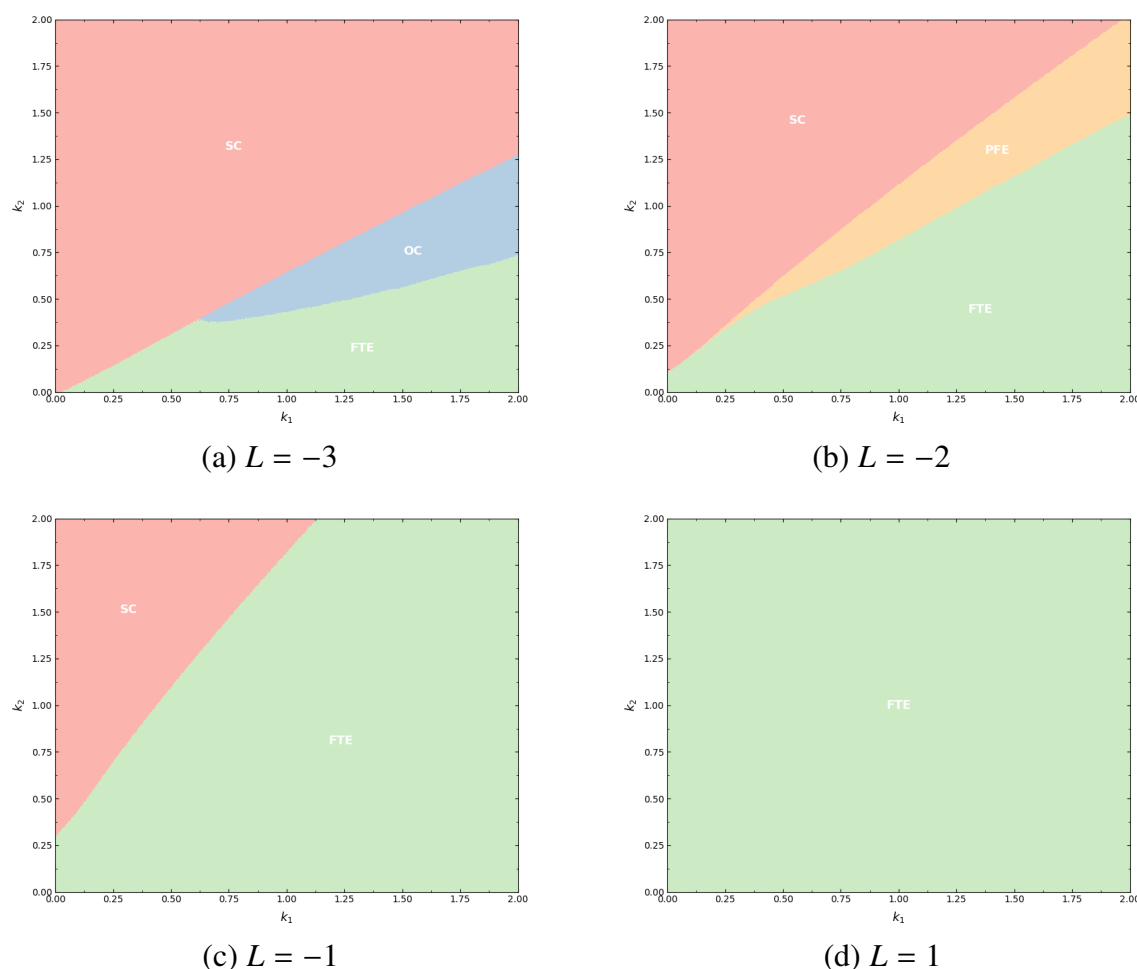


Figure 7. Plots showing dynamic, color-coded regions of a two-parameter bifurcation in the (k_1, k_2) space representing stable coexistence (SC), oscillatory coexistence (OC), finite-time extinction (FTE), and predator-free equilibrium (PFE) states. Here, we choose $b_0 = 2$ and $e_0 = 2$. All other parameters chosen are seen in Table 1.

The (k_1, k_2) plane in Figure 7 highlights the interplay between two distinct fear-driven suppression pathways. For a strongly negative Allee threshold $L = -3$, low fear levels for k_1 and k_2 enable all populations to persist. Increasing k_1 to suppress reproduction eventually drives the system to an FTE state as the susceptible prey population collapses. For high fear levels k_1 and moderate fear levels k_2 , the system transitions to an oscillatory coexistence (OC) regime as the stable coexistence (SC) state is destabilized via a Hopf bifurcation. At higher k_2 , disease transmission is reduced. This reduces predator access to energetically profitable infected prey. With moderate prey aggregation and moderate predation, all populations can coexist. As L increases toward -1 , we observe that the SC region contracts and

the FTE region expands. This demonstrates the key role of Allee effects on governing the ecological resilience and persistence of interacting populations. At $L = 1$, the Allee threshold is positive and the entire (k_1, k_2) plane is characterized by species extinction. This shows that, when the Allee effect is strong, the collapse of the susceptible prey population is inevitable regardless of the two distinct fear levels for a chosen parameter regime.

6. Path dependence and ecological tipping points

The saddle-node bifurcation identified in Figure 3 gives rise to parameter intervals in which two locally stable attractors coexist. Such alternative stable states have important ecological consequences because they imply that ecosystem recovery depends not only on current environmental conditions but also on initial population densities and the history of environmental change. This path dependence is the mathematical signature of hysteresis and is a defining feature of ecological tipping points.

Ecological systems often exhibit abrupt transitions between qualitatively distinct dynamical regimes when critical thresholds are crossed. Such transitions are commonly referred to as ecological tipping points. In general, a tipping point occurs when a small change in environmental conditions, demographic structure, or population densities produces a disproportionately large change in the long-term behavior of the ecosystem.

Theorem 6.1. *Consider model (2.1) with the Allee threshold L as the bifurcation parameter. Suppose that*

- 1) *the model undergoes a generic saddle-node bifurcation of coexistence equilibria at $L = L^{[SN]}$;*
- 2) *there exists an interval*

$$L \in (L^{[SN]}, L^{[SN]} + \delta_1), \quad \delta_1 > 0,$$

over which the predator-free equilibrium $E_4(L)$ and the coexistence equilibrium $E_5(L)$ are both locally asymptotically stable.

Then the model exhibits path-dependent dynamics throughout

$$L \in (L^{[SN]}, L^{[SN]} + \delta_1).$$

More precisely,

- (i) *there exist two disjoint basins of attraction, $\mathcal{B}_4(L)$ and $\mathcal{B}_5(L)$, such that solutions initiated from different regions of the state space converge to different long-term ecological states under identical parameter values;*
- (ii) *consequently, transient perturbations capable of moving the system across the basin boundary may induce an abrupt transition between predator persistence and predator exclusion, even though the environmental conditions remain unchanged;*
- (iii) *the coexistence of these alternative stable states provides the dynamical mechanism through which hysteretic responses may arise under gradual variation of the Allee threshold.*

Proof. By assumption, the saddle-node bifurcation creates a parameter interval

$$L \in (L^{[SN]}, L^{[SN]} + \delta_1)$$

for which both the predator-free equilibrium $E_4(L)$ and the coexistence equilibrium $E_5(L)$ are locally asymptotically stable.

Local asymptotic stability guarantees the existence of open neighborhoods $U_4(L)$ and $U_5(L)$ of $E_4(L)$ and $E_5(L)$, respectively, whose trajectories converge to the corresponding equilibria. Since

$$E_4(L) \neq E_5(L),$$

these neighborhoods may be chosen disjointly and are contained in distinct basins of attraction, $\mathcal{B}_4(L)$ and $\mathcal{B}_5(L)$.

Consequently, trajectories evolving under identical parameter values but originating from different initial conditions converge to different asymptotic ecological states. The long-term outcome therefore depends on the path taken through the state space, establishing path dependence.

Ecologically, this implies that temporary perturbations capable of moving the system across the basin boundary may trigger abrupt shifts between predator persistence and predator exclusion without any change in the underlying environmental conditions.

Finally, because the two attracting states originate through a saddle-node bifurcation, gradual variation of the Allee threshold may lead trajectories to switch between alternative attracting branches after a critical threshold is crossed. Consequently, the alternative stable-state structure established above provides the dynamical mechanism through which hysteretic responses may occur.

□

To illustrate the path-dependent dynamics predicted by Theorem 6.1, we consider parameter values within the interval

$$L \in (-3.95198, -2.5164),$$

for which both the predator-free equilibrium E_4 and the coexistence equilibrium E_5 are locally asymptotically stable. Within this bistable region, Theorem 6.1 predicts that trajectories initiated in different basins of attraction converge to different long-term ecological states despite identical parameter values.

Figure 8 illustrates the dependence of the long-term dynamics on the initial population densities by comparing two trajectories generated under identical parameter values but different initial conditions. For the initial condition

$$(S(0), I(0), P(0)) = (3.00, 0.50, 1.50),$$

the solution converges to the coexistence equilibrium E_5 , indicating the long-term persistence of susceptible prey, infected prey, and predators. In contrast, the initial condition

$$(S(0), I(0), P(0)) = (0.25, 1.60, 0.05)$$

converges to the predator-free equilibrium E_4 , leading to predator exclusion despite identical environmental conditions and parameter values.

These simulations provide a numerical illustration of Theorem 6.1. Although the governing parameters are identical in both simulations, the trajectories converge to different attractors because their initial conditions belong to different basins of attraction associated with the locally stable equilibria E_4 and E_5 . Consequently, the long-term ecological outcome depends on the initial state of the system, providing direct evidence of path-dependent dynamics induced by alternative stable states.

From an ecological perspective, the coexistence of alternative stable states implies that ecosystem resilience depends not only on environmental conditions but also on the position of the population within the state space. Consequently, temporary perturbations capable of moving the system across the basin boundary separating the two basins of attraction may trigger abrupt transitions from predator persistence to predator exclusion without any change in the underlying environmental conditions. This behavior is characteristic of ecological tipping points and highlights that the interaction between predator-induced fear, prey aggregation, and Allee dynamics can substantially reduce ecosystem resilience by creating multiple long-term ecological outcomes under identical parameter regimes.

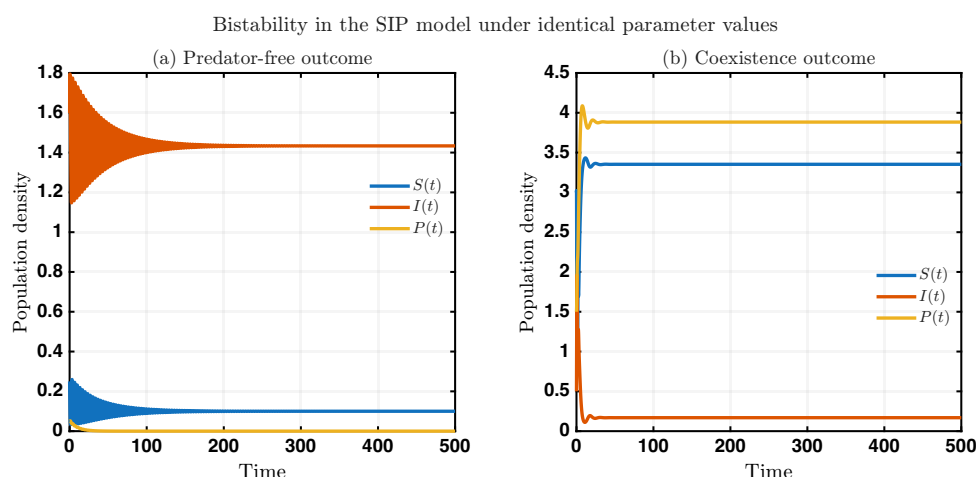


Figure 8. Time series illustrating bistability in system (1). The same parameter values are used in both panels with $L = -3$, which lies in the bistable interval identified in Figure 3. Different initial conditions lead to distinct long-term outcomes: convergence to the predator-free equilibrium (E_4) in panel (a), and convergence to the coexistence equilibrium (E_5) in panel (b). This demonstrates the presence of alternative stable states and highlights the sensitivity of the system to initial population densities.

7. Conclusions

This study demonstrates that predator-induced fear, prey aggregation, and Allee dynamics are not merely additive modifiers of predator–prey–disease interactions, but interacting mechanisms capable of generating qualitatively richer and more dangerous ecological dynamics than any pairwise combination alone. The central finding is that fear-induced aggregation can paradoxically accelerate ecosystem collapse by increasing predation efficiency when susceptible populations become rare, thereby amplifying demographic vulnerability near the Allee threshold. Importantly, the analysis distinguishes the complementary roles of these mechanisms: the non-Lipschitz aggregation process provides the mathematical driver of finite-time extinction, while the Allee effect determines the ecological conditions under which populations become susceptible to collapse. Together, these mechanisms produce abrupt regime shifts that connect the present work to the broader theory of ecological tipping points and critical transitions [23, 31–33].

The analytical results establish that the aggregation-dependent predation term $d_0 S^r P$, with $0 < r < 1$, is not merely a mathematical convenience but a mechanistically meaningful representation

of aggregation-based predation capable of generating extinction dynamics fundamentally different from those of classical smooth predator–prey systems. The explicit upper bound on extinction time quantifies how aggregation strength and predator pressure jointly determine the speed of ecological collapse, providing a practical measure of extinction risk for threatened populations. Furthermore, the cascade-collapse analysis demonstrates that finite-time extinction of the susceptible prey population inevitably eliminates both the infected prey and predator populations, revealing the structural fragility of ecological communities in which susceptible prey constitute the primary energetic foundation of disease transmission and predator persistence [26].

Beyond extinction, the model exhibits a remarkably rich dynamical structure characterized by transcritical, saddle-node, and Hopf bifurcations together with extensive regions of bistability. In particular, the coexistence of alternative stable states implies that long-term ecological outcomes depend not only on environmental conditions but also on the initial population state, thereby introducing strong path dependence into the system dynamics. Two populations subjected to identical environmental parameters may therefore evolve toward entirely different ecological states depending on their initial conditions or perturbation history. The two-parameter continuation diagrams further demonstrate that decreasing the aggregation exponent systematically enlarges the region supporting finite-time extinction while simultaneously reducing the domain of long-term coexistence, identifying aggregation strength as a qualitative determinant of ecological resilience and persistence [17, 23, 31, 34].

From a conservation perspective, these findings suggest that wildlife populations simultaneously experiencing infectious disease, predator-induced fear, and low-density demographic effects may be substantially more vulnerable than predicted by models that treat these mechanisms independently [35]. The presence of alternative stable states implies that relatively small perturbations may push populations across critical thresholds into predator-free or extinction states from which natural recovery may be impossible despite subsequent improvement in environmental conditions [31, 32]. Such hysteresis highlights the importance of proactive management before ecological thresholds are crossed rather than relying on restoration efforts after collapse has begun.

Although critical slowing down has emerged as one of the principal approaches for anticipating ecological transitions, its applicability to the present model remains uncertain. Classical early-warning indicators, including increasing variance and lag-one autocorrelation, are typically associated with transitions driven by the gradual loss of local stability through eigenvalue crossings [36, 37]. In contrast, the finite-time extinction mechanism established here originates from the non-Lipschitz aggregation term rather than from a conventional local bifurcation. Consequently, standard critical-slowness indicators may provide limited, delayed, or qualitatively different warning signals prior to aggregation-mediated collapse. Determining the reliability of these indicators through systematic analyses of variance, autocorrelation, recovery rates, and related metrics therefore represents an important avenue for future investigation.

Several directions remain open for future research. Incorporating spatial dispersal would permit investigation of whether immigration from refuge populations can mitigate aggregation-driven collapse, thereby linking the present framework to metapopulation persistence and rescue effects [38, 39]. Environmental stochasticity may further interact with the Allee threshold to influence extinction probabilities and facilitate comparisons with long-term wildlife monitoring data [40, 41]. Allowing aggregation intensity to evolve adaptively in response to perceived predation risk would introduce an additional behavioral feedback capable of either stabilizing or destabilizing population dynamics depending on

the relative timescales of behavioral adaptation and demographic change [3, 5]. Finally, developing early-warning indicators specifically tailored to aggregation-driven finite-time collapse represents both an important mathematical challenge and a practical conservation priority, with the potential to improve prediction and management of abrupt ecological transitions [32, 36].

Use of AI tools declaration

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

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Conflict of interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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