Dynamics of a predator-prey system with prey subject to Allee effects and disease

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Abstract

In this article, we propose a general predator-prey system with prey subject to Allee effects and disease with the following unique features: (i) Allee effects built in the reproduction process of prey where infected prey (I-class) has no contribution; (ii) Consuming infected prey would contribute less or negatively to the growth rate of predator (P-class) in comparison to the consumption of susceptible prey (S-class). We provide basic dynamical properties for this general model and perform the detailed analysis on a concrete model (SIP-Allee Model) as well as its corresponding model in the absence of Allee effects (SIP-no-Allee Model); we obtain the complete dynamics of both models: (a) SIP-Allee Model may have only one attractor (extinction of all species), two attractors (bi-stability either induced by small values of reproduction number of both disease and predator or induced by competition exclusion), or three attractors (tri-stability); (b) SIP-no-Allee Model may have either one attractor (only S-class survives or the persistence of S and I-class or the persistence of S and P-class) or two attractors (bi-stability with the persistence of S and I-class or the persistence of S and P-class). One of the most interesting findings is that neither models can support the coexistence of all three S, I, P-class. This is caused by the assumption (ii), whose biological implications are that I and P-class are at exploitative competition for S-class whereas I-class cannot be superior and P-class cannot gain significantly from its consumption of I-class. In addition, the comparison study between the dynamics of SIP-Allee Model and SIP-no-Allee Model lead to the following conclusions: 1) In the presence of Allee effects, species are prone to extinction and initial condition plays an important role on the surviving of prey as well as its corresponding predator; 2) In the presence of Allee effects, disease may be able to save prey from the predation-driven extinction and leads to the coexistence of S and I-class while predator can not save the disease-driven extinction. All these findings may have potential applications in conservation biology.

Key words: Allee Effect, Functional Responses, Disease/Predation-Driven Extinction, Bi-stability, Tri-stability, Eco-epidemiological System

1. Introduction

Allee effects, referred to a biological phenomenon characterized by a positive correlation between the population of a species' size or density and its per capita growth rate at its low population sizes/densities [1, 56, 49], have great impacts in species' establishment, persistence, invasion [3, 76, 24, 70, 62, 9, 49, 44] and evolutionary traits [22]. Empirical evidence of Allee effect has been reported in many natural populations including plants [25, 29], insects [52], marine invertebrates [67], birds and mammals [21]. Various mechanisms at low population sizes/densities, such as the need of a minimal group size necessary to successfully raise offspring, produce seeds, forage, and/or sustain predator attacks, have been proposed as potential sources of Allee effects [42, 53, 60, 19, 65, 66, 61]. Recently, many researchers have studied the impact of Allee effects on population interactions [e.g., see [61, 80, 43, 51, 75, 45, 46, 44] as well as the interplay of Allee effects and disease on species's establishment and persistence [38, 79, 37, 71, 47, 48]. All these research suggest the profound effects of Allee effects in population dynamics, especially when it couples with disease.

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Eco-epidemiology is comparatively a new branch in mathematical biology which simultaneously considers the ecological and epidemiological processes [5]. Hadeler and Freedman [33] first introduced a eco-epidemiological model regarding predator-prey interactions with both prey and predator subject to disease. Since the work of Hadeler and Freedman (1989), the research on eco-epidemiology as well as its biological importance has gained great attention [26, 7, 8, 32, 72, 13, 15, 78, 14, 73, 35, 39, 5, 69, 68]. Many species suffer from Allee effects, disease and predation. For instance, the combined impact of disease and Allee effect has been observed in the African wild dog *Lycaon pictus* [12, 20] and the island fox *Urocyon littoralis* [17, 4]. Both the African wild dog and island fox should have their enemies in the wild. Thus, understanding the combined impact of Allee effects and disease on population dynamics of predator-prey interactions can help us have better insights on species' abundance as well as the outbreak of disease. Therefore, we can make better policies to regulate the population and disease. Thus, for the first time, we propose a general predator-prey model with Allee effects and disease in prey to investigate how the interplay of Allee effects and disease in prey affect the population dynamics of both prey and predator. More specifically, we would like to explore the following ecological questions:

1. How do Allee effects affect the population dynamics of both prey and predator?
2. Which conditions allow healthy prey, infected prey and predator to coexist?
3. In the presence of Allee effects, can disease save the population from predation-driven extinction?
4. In the presence of Allee effects, can predation save the population from disease-driven extinction?

We will try to answer the questions above by 1) obtaining a complete global picture of the population dynamics of the proposed susceptible prey-infected prey-predator interaction model (SIP-Allee Model) as well as its corresponding model without Allee effects (SIP-no-Allee Model); 2) comparing the dynamics of the model with Allee effects to the one without Allee effects.

The rest of the paper is organized as follows: In Section 2, we provide the detailed formulation of a general prey-predator system with prey subject to Allee effects and disease; and we show the basic dynamical properties of such general model. In Section 3, we obtain the complete dynamics of a concrete model when it is disease free and/or predation free (i.e., the submodels of SIP-Allee Model); and we compare the dynamics to their corresponding models in the absence of Allee effects. In Section 4, we provide detailed analysis and its related numerical simulations to obtain the complete dynamical feature of this SIP-Allee model. Our results include sufficient conditions on its global attractors as well as its corresponding basins of attractions in different scenarios. In Section 5, we perform analysis of SIP-no-Allee Model under the same assumptions. In addition, we provide the biological implications on the impacts of Allee effects, disease and predation. In the last section, we conclude our findings and provide a potential future study.

2. Development of the model

We start from the assumption that prey is facing an infectious disease that can be captured by an SI (Susceptible-Infected) framework where predator (P-class) feeds on both susceptible prey (S-class) and infected prey (I-class). Let S be the normalized susceptible prey population; I, P denote the infected prey population and the predator population, respectively, both of which are relative to the susceptible prey population; and $N = S + I$ denotes the total population of prey.

In the absence of disease and predation, we assume that the population dynamic of prey can be described by the following generic single species population model with an Allee effect:

$$\frac{dS}{dt} = rS(S-\theta)(1-S) \tag{1}$$

where S denotes the normalized health prey population; the parameter r denotes the maximum birth-rate of species, which can be scaled to be 1 by altering the time scale; the parameter $0 < \theta < 1$ denotes

the Allee threshold (normalized susceptible population). The population of (1) converges to 0 if initial conditions are below θ while it converges to 1 if initial conditions are above θ.

We assume that a) disease does not have vertical transmission but it is untreatable and causes an additional death rate; b) I-class does not contribute to the reproduction of newborns; and c) the net reproduction rate of newborns is modified by the disease (e.g, infectivies compete for resource but do not contribute to reproduction). Then in the presence of disease (i.e., $I > 0$) and the absence of predation (i.e., $P = 0$), the formulation of susceptible prey population dynamics can be described by the following (2):

$$\frac{dS}{dt} = \underbrace{rS(S-\theta)(1-S-I)}_{\text{the net reproduction modified by disease}} - \underbrace{\phi(N)\frac{I}{N}S}_{\text{new infections}} \tag{2}$$

where $\phi(N)$ is the disease transmission function that can be either density-dependent (i.e., $\phi(N) = \beta N$ which is also referred to the law of mass action) or frequency-dependent (i.e., $\phi(N) = \beta$). Thus, the formulation of infective population can be described by the following (3),

$$\frac{dI}{dt} = \phi(N)\frac{I}{N}S - \underbrace{\mu I}_{\text{the natural mortality plus an additional mortality due to disease}}. \tag{3}$$

In the presence of disease but in the absence of predation $P = 0$, a general SI model subject to Allee effects in prey can be represented as follows:

$$\begin{aligned} \frac{dS}{dt} &= rS(S-\theta)(1-S-I) - \phi(N)\frac{I}{N}S \\ \frac{dI}{dt} &= \phi(N)\frac{I}{N}S - \mu I \end{aligned} \tag{4}$$

where the parameter μ denotes the death rate of I-class, which includes an additional disease-induced death rate. The SI model (4) is a special case of an SI model studied by Kang and Castillo-Chavez [48] where $\phi(N) = \beta N, \rho = 0, \alpha_1 = 0$ and $\alpha_2 = 1$. This modeling approach is similar to the work by Boukal and Berec [11], Deredec and Courchamp [23], Courchamp et al. [18] and Hilker et al. [37] regarding the effects of Allee effects and disease (our detailed approach of the host population without disease and predation is represented in Appendix). There are many literatures using this phenomenological model (4) to study the disease dynamics as well as invasion of pest (e.g., see [54, 30, 2, 57, 36, 62, 27]).

In the presence of predation, we assume that predator consumes S and I-class at the rate of $h(S, N)$ and $h(I, N)$, respectively, where I-class has less or negative contribution to the growth rate of predator in comparison to S-class. The functional responses $h(S, N), h(I, N)$ can take the form of Holling-Type I or II or III, i.e.,

$$\begin{aligned} Holling - Type\, I: &\qquad h(S,N) = aS;\ h(I,N) = aI \\ Holling - Type\, II: &\qquad h(S,N) = \tfrac{aS}{k+S+I};\ h(I,N) = \tfrac{aI}{k+S+I} \\ Holling - Type\, III: &\qquad h(S,N) = \tfrac{aS^2}{k^2+(S+I)^2};\ h(I,N) = \tfrac{aI^2}{k^2+(S+I)^2} \end{aligned}.$$

Therefore, a general predator-prey model where prey is subject to Allee effects and disease, is given by the following set of nonlinear differential equations:

$$\begin{aligned} \frac{dS}{dt} &= rS(S-\theta)(1-S-I) - \phi(N)\frac{I}{N}S - h(S,N)P, \\ \frac{dI}{dt} &= \phi(N)\frac{I}{N}S - h(I,N)P - \mu I, \\ \frac{dP}{dt} &= P\left[ch(S,N) + \gamma h(I,N) - d\right]. \end{aligned} \tag{5}$$

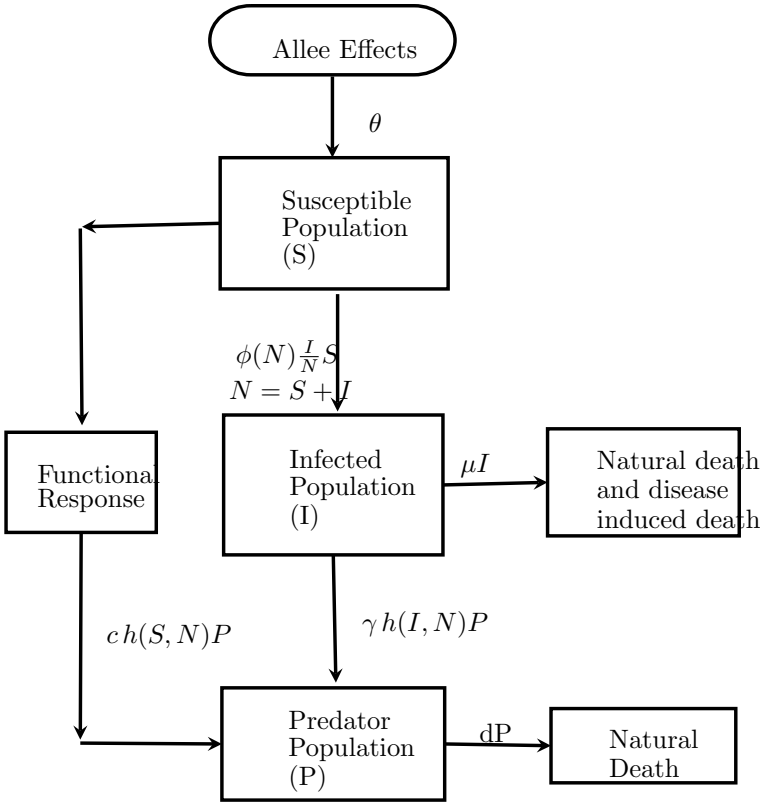


Figure 1: Schematic diagram of a general prey-predator model with prey subject to Allee effects and disease (see the presentation of the model in (5)).

where all parameters except γ are nonnegative. The parameter d represents the natural death rate of predator; the parameter $c \in (0, 1]$ is the conversion rate of susceptible prey biomass into predator biomass; and γ indicates that the effects of the consumption of infected prey on predator which could be positive or negative. More specifically, we assume that $-\infty < \gamma < c$; $\gamma < 0$ indicates the consumption of infected prey increases the death rate of the predator (see [16]), while $\gamma > 0$ indicates the consumption of susceptible prey increases the growth rate of the predator. The biological significance of all parameters in Model (5) is provided in Table 1. The conceptual schematic diagram of this general model is presented in the schematic diagram 1.

In summary, the formulation of a general SIP model (5) subject to Allee effects in prey is based on the following three assumptions: **(a)** Disease does not have vertical transmission but it is untreatable and causes an additional death rate; **(b)** Allee effects are built in the reproduction process of S-class which I-class does not contribute to; **(c)** Predator consumes S and I-class at the rate of $h(S, N)$ and $h(I, N)$, respectively, whose growth rate is benefit less or even getting harm from I-class. Our modeling assumptions are supported by many ecological situations. For example, in Salton Sea (California), predatory birds get additional mortality though eating fish species that are infected by a vibrio class of bacteria and could also be subject to Allee effects (see more discussions in [16, 5]). In nature, it is also possible that predator captures infected prey who is given up by predator due to its unpleasant taste or malnutrition from infections. We would like to point out that the assumption **(c)** is critical to the dynamical outcomes of (5) as we should see from our analysis in the next few sections.

To continue our study, let us define the state space of (5) as $X = \{(S, I, P) \in \mathbb{R}^3_+\}$ whose interior is defined as $\mathring{X} = \{(S, I, P) \in \mathbb{R}^3_+ : SIP > 0\}$. In the case that $\phi(N) = \beta$, we define the state space as

$X = \{(S, I, P) \in \mathbb{R}^3_+ : S + I > 0\}$. Notice that $h(x, N)$ is chosen from Holling Type I or II or III and $\phi(N) = \beta N$ or β, then the basic dynamical property of (5) can be summarized as the following theorem:

Theorem 2.1 (Basic dynamical features)**.** *Assume that*

$$c \in (0, 1],\ d > 0,\ \theta \in (0, 1),\ -\infty < \gamma < c,\ \mu > r\theta.$$

Then System (5) is positively invariant and uniformly ultimately bounded in X with the following property

$$\limsup_{t\to\infty} S(t) + I(t) \le 1.$$

In addition, we have the following:

1. *If $\frac{\phi(N)}{N} \le \mu$, for all $N > 0$, then $\limsup_{t\to\infty} I(t) = 0$.*
2. *If $S(0) < \theta$, then $\lim_{t\to\infty} \max\{S(t), I(t), P(t)\} = 0$.*

Proof. For any $S \ge 0, I \ge 0, P \ge 0$, we have

$$\left.\frac{dS}{dt}\right|_{S=0} = 0,\ \left.\frac{dI}{dt}\right|_{I=0} = 0 \text{ and } \left.\frac{dP}{dt}\right|_{P=0} = 0$$

which implies that $S = 0$, $I = 0$ and $P = 0$ are invariant manifolds, respectively. Due to the continuity of the system, we can easily conclude that System (5) is positively invariant in $\mathbb{R}^3_+$.

Choose any point $(S, I, P) \in X$ such that $S > 1$, then due to the positive invariant property of (5), we have

$$\left.\frac{dS}{dt}\right|_{S>1} = rS(S-\theta)(1-S-I) - \phi(N)\frac{I}{N}S - h(S,N)P < 0.$$

In addition, since we have $\left.\frac{dS}{dt}\right|_{S=1,I=0,P=0} = 0$ and $\left.\frac{dS}{dt}\right|_{S=1,I+P>0} < 0$, thus we can conclude that

$$\limsup_{t\to\infty} S(t) \le 1.$$

Now we define the following two functions as $N(t) = S + I$ and $Z(t) = S + I + P$, then we have

$$\frac{dN(t)}{dt} = rS(S-\theta)(1-N) - \mu I - P\left[h(S,N) + h(I,N)\right] \le rS(S-\theta)(1-N) - \mu I \tag{6}$$

$$\frac{dZ(t)}{dt} = rS(S-\theta)(1-N) - \mu I - dP - P\left[h(S,N) + h(I,N) - ch(S,N) - \gamma h(I,N)\right]. \tag{7}$$

Since $\mu > r\theta > \frac{r\theta^2}{4}$ and $\limsup_{t\to\infty} S(t) \le 1$, then for any $\epsilon > 0$, there is a T large enough such that for any $t > T$, we have

$$\frac{dN(t)}{dt} \le rS(S-\theta+\mu/r) - \left[rS(S-\theta)+\mu\right]N \le r(1+\epsilon)(1+\epsilon-\theta+\mu/r) - \left[-\frac{r\theta^2}{4} + \mu\right]N.$$

By applying the theory of differential inequality [10] (or Gronwalls inequality) and letting $\epsilon \to 0$, we obtain

$$\limsup_{t\to\infty} N(t) = \limsup_{t\to\infty} S(t) + I(t) \le \frac{r - r\theta + \mu}{\mu - \frac{r\theta^2}{4}}.$$

This implies that both $N(t)$ and $I(t)$ are uniformly ultimately bounded. Similarly, since $c \in (0, 1]$ and $-\infty < \gamma < c$, then we have for any $\epsilon > 0$, there is a T large enough such that for any $t > T$,

$$\begin{aligned}\frac{dZ(t)}{dt} &= rS(S-\theta)(1-N) - \mu I - dP - P\left[h(S,N) + h(I,N) - ch(S,N) - \gamma h(I,N)\right] \\ &\le rS(S-\theta)(1-N) - \mu I - dP = L_\epsilon - \min\{\mu, d\}Z\end{aligned}$$

where

$$L_\epsilon = \max_{\{0\le S\le 1+\epsilon, 0\le N\le \frac{r-r\theta+\mu}{\mu-\frac{r\theta^2}{4}}+\epsilon\}} \left\{rS(S-\theta)(1-N)+\min\{\mu,d\}S\right\}.$$

This implies that $\limsup_{t\to\infty} Z(t) = \limsup_{t\to\infty} S(t)+I(t)+P(t) \le \frac{L}{\min\{\mu,d\}}$ where

$$L_\epsilon = \max_{\{0\le S\le 1, 0\le N\le \frac{r-r\theta+\mu}{\mu-\frac{r\theta^2}{4}}\}} \left\{rS(S-\theta)(1-N)+\min\{\mu,d\}\right\}.$$

Thus $P(t)$ is also uniformly ultimately bounded. Therefore, System (5) is positively invariant and uniformly ultimately bounded in X.

The fact that

$$\begin{aligned}
\frac{dS}{dt} &= rS(S-\theta)(1-S-I)-\phi(N)\tfrac{I}{N}S-h(S,N)P \le rS(1-S-I)(S-\theta)-\phi(N)\tfrac{I}{N}S \\
\frac{dI}{dt} &= \phi(N)\tfrac{I}{N}S-h(I,N)P-\mu I \le \phi(N)\tfrac{I}{N}S-\mu I \le I\left(\tfrac{\phi(N)}{N}S-\mu\right)
\end{aligned}$$

implies that the dynamics of the SI model (4) can govern the dynamics of S, I-class in Model (5). If $\frac{\phi(N)}{N}\le\mu$, then the SI model (4) has no interior equilibrium since $\limsup_{t\to\infty} S(t)\le 1$. Then according to Poincaré-Bendixson Theorem [31], any trajectory of (4) converges to either a locally asymptotically stable equilibrium or a limit cycle. However, no interior equilibrium and no equilibrium on I-axis indicates that any trajectory converges to a boundary equilibrium located on S-axis. Thus, we have

$$\limsup_{t\to\infty} I(t) = 0 \text{ if } \frac{\phi(N)}{N}\le\mu.$$

Assume that the initial susceptible prey population is less than θ and the initial infective population is large enough, the susceptible prey population can increase at the beginning due to the possibility of

$$\frac{dS}{dt}\Big|_{t=0} = rS(0)\left[(S(0)-\theta)(1-S(0)-I(0))-\frac{\phi(N(0))}{N(0)}I(0)\right] > 0.$$

However, the susceptible prey population can never increase to θ since

$$\frac{dS}{dt}\Big|_{S=\theta} = rS\left[(S-\theta)(1-S-I)-\frac{\phi(N)}{N}I\right]\Big|_{S=\theta} = -\frac{\phi(N)}{N}SI\Big|_{S=\theta} < 0.$$

This implies that

$$S(t)<\theta \text{ whenever } S(0)<\theta, \text{ for all } t>0.$$

Since $\frac{\phi(N)}{N}\le\mu$ implies that $\limsup_{t\to\infty} I(t)=0$, thus the limiting dynamics is

$$\frac{dS}{dt} = rS(S-\theta)(1-S) \text{ with } S(t)<\theta.$$

This indicates the susceptible prey population will eventually converge to 0. Therefore, we have

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

Now assume that $\frac{\phi(N)}{N}>\mu$, for all $N>0$. Since $\mu>r\theta$ and $\limsup_{t\to\infty} S(t)\le\theta$, then we have

$$\begin{aligned}
\tfrac{dS}{dt} &\le S\left[r(S-\theta)(1-S-I)-\tfrac{\phi(N)}{N}I\right] \\
&< rS\left[(S-\theta)(1-S)-(S-\theta+\mu/r)I\right] \\
&\le rS\left[(S-\theta)(1-S)-(-\theta+\mu/r)I\right] < rS(S-\theta)(1-S)
\end{aligned}$$

This implies that $\lim_{t\to\infty} S(t) = 0$. Therefore, we have

$$\lim_{t\to\infty} \max\{S(t), I(t), P(t)\} = 0 \text{ whenever } S(0) < \theta.$$

In the case that $S(0) = \theta$, then we have $S(t) < \theta$ if $I(0) + P(0) > 0$ or $S(t) = \theta$ if $I(0) + P(0) = 0$.

Without loss of generality, let us assume $S(0) + I(0) > 1$ and $I(0) > 0$. Then according to the argument above, we have

$$\lim_{t\to\infty} \max\{S(t), I(t), P(t)\} = 0 \Rightarrow \limsup_{t\to\infty} S(t) + I(t) \leq 1 \text{ whenever there exists a } T \text{ such that } S(T) \leq \theta.$$

Now assume that $S(t) > \theta$, for all $t \geq 0$, then we have

$$\frac{dN(t)}{dt} = rS(S-\theta)(1-N) - \mu I - P[h(S,N) + h(I,N)] \leq rS(S-\theta)(1-N) - \mu I < 0 \text{ whenever } N(0) > 1.$$

Therefore, we have

$$\limsup_{t\to\infty} N(t) = \limsup_{t\to\infty} S(t) + I(t) \leq 1.$$

□

Notes: The assumption of $\mu > r\theta$ follows from the fact that the natural mortality rate of the susceptible prey is $r\theta$ (see the derivation of this assumption in the Appendix A). Theorem 2.1 indicates that our general prey-predator model with Allee effects and disease in prey has a compact global attractor living in the set

$$\left\{(S,I,P) \in X : 0 \leq S + I \leq 1, 0 \leq S + I + P \leq \frac{\max_{\{0\leq N\leq 1\}} \{rS(S-\theta)(1-N) + \min\{\mu, d\}\}}{\min\{\mu, d\}}\right\}.$$

In addition, Theorem 2.1 implies that initial population of susceptible prey plays an important role in the persistence of S, or I or P due to Allee effects in prey. One direct application of Theorem 2.1 is presented as the following corollary:

Corollary 2.1. *[Range of susceptible and infective population] Assume that*

$$c \in (0,1],\ d > 0,\ \theta \in (0,1),\ -\infty < \gamma < c, \mu > r\theta.$$

Then a necessary condition for the endemicity of the disease of System (5) is as follows:

$$\liminf_{t\to\infty} S(t) > \theta \text{ and } \limsup_{t\to\infty} I(t) < 1 - \theta.$$

Theorem 2.1 and its corollary 2.1 provide the basic dynamical features of the general prey-predator model (5). In order to explore more complete dynamics of (5), we will focus on the case when $\phi(N) = \beta N$ and $h(x,N) = ax$. Then, in the presence of both disease and predator, depending on whether infectives have a positive or negative impact on the growth rate of predator (i.e., the sign of γ being positive or negative), the predator-prey model subject to Allee effects (e.g., induced by mating limitations) and disease (5) can be written as the following if we scale away r (i.e., $r = 1$) :

$$\begin{aligned}
\tfrac{dS}{dt} &= S(S-\theta)(1-S-I) - \beta SI - aSP = S[(S-\theta)(1-S-I) - \beta I - aP] = Sf_1(S,I,P),\\
\tfrac{dI}{dt} &= \beta SI - aIP - \mu I = I[\beta S - aP - \mu] = If_2(S,I,P),\\
\tfrac{dP}{dt} &= a(cS + \gamma I)P - dP = P[bS + \alpha I - d] = Pf_3(S,I,P)
\end{aligned} \tag{8}$$

where the parameter a indicates the attack rate of predator. For convenience, we let $b = ac \in (0, a]$ and $\alpha = a\gamma \in (-\infty, ac]$. Variables and parameters used in Model (8) (SIP-model) are presented in Table 1.

Variables/Parameters	Biological meaning
S	Density of susceptible prey
I	Density of infected prey
P	Density of predator
θ	Allee threshold
β	Rate of infection
a	Attack rate of predator
b	The total effect to predator by consuming susceptible prey
μ	Death rate of infected prey
c	Conversion efficiency on susceptible prey
γ	Conversion efficiency on infected prey
α	The total effect to predator by consuming infected prey
d	Natural death rate of predator

Table 1: Variables and parameters used Model (8)

Notes: The term $S(S-\theta)(1-S-I)$ of $\frac{dS}{dt}$ in (8) models the net reproduction rate of newborns, a term that accounts for Allee effects due to mating limitations as well as reductions in fitness due to the competition for resource from infectives. Our model normalizes the susceptible population to be 1 in a disease-free environment; and defines the infected prey population as well as the predator population *relative to this normalization*. Our modeling approach (see the Appendix A) and assumptions **(a), (b), (c)** require that the parameters of (8) are subject to the following condition:

H: $0 < \theta < 1$, $\mu > \theta$, $0 < b = ac \leq a$ and $-\infty < \alpha < b$.

The features outline above include factors not routinely considered in infectious-disease models. Allee effects are found in the epidemiological literature (e.g., see [35, 71, 37]) as well as in the predator-prey interaction models [9, 75]. The rest of our article is focus on studying the dynamics of this simple SIP model (8) that incorporates Allee effects in its reproduction process, disease-induced additional death, and disease-induced effects on predation.

3. Dynamics of submodels

In order to understand the full dynamics of (8), we should have a complete picture of the dynamics of the following two submodels:

1. The predator-prey model in the absence of the disease in (8) is represented as

$$\begin{array}{rcl} \frac{dS}{dt} & = & S\left[(S-\theta)(1-S)-aP\right] = Sf_1(S,0,P), \\ \frac{dP}{dt} & = & P\left[bS-d\right] = Pf_3(S,0,P). \end{array} \tag{9}$$

The submodel (9) has been introduced by other researchers (e.g., [9, 74, 75]). For convenience, we introduce a disease-free demographic reproduction number for predator

$$R_0^P = \frac{b}{d}$$

which gives the expected number of offspring b of an average individual predator in its lifetime $\frac{1}{d}$. The reproduction number R_0^P is based upon the assumptions that the susceptible prey is at unit density (i.e. $S = 1$) and the disease is absent (i.e. $I = 0$). The value of $R_0^P < 1$ indicates that the predator cannot invade while the value of $R_0^P > 1$ indicates that the predator may invade.

2. The SI model in the absence of predation in (8) is represented as

$$\begin{array}{rcl} \frac{dS}{dt} & = & S\left[(S-\theta)\left(1-S-I\right)-\beta I\right] = Sf_1(S,I,0), \\ \frac{dI}{dt} & = & I\left[\beta S-\mu\right] = If_2(S,I,0). \end{array} \tag{10}$$

Kang and Castillo-Chavez [48] have studied a simple SI model with strong Allee effects (where they consider a susceptible-infectious model with the possibility that susceptible and infected individuals reproduce with the S-class being the best fit, and also infected individuals loose some ability to compete for resources at the cost imposed by the disease. The submodel (10) is a special case of the SI model studied by them where $\rho = 0, \alpha_1 = 0$ and $\alpha_2 = 1$. We adopt the notations in Kang and Castillo-Chavez [48] and introduce the basic reproductive ratio

$$R_0^I = \frac{\beta}{\mu}$$

whose numerator denotes the number of secondary infections $\beta S^* = \beta$ per unit of time (at the locally asymptotically stable equilibrium $S^* = 1$) and denominator denotes the inverse of the average infectious period μ. The value of $R_0^I < 1$ indicates that the infection cannot invade while $R_0^I > 1$ indicates that the disease can invade.

A direct application of Theorem 2.1 to the submodels (9) and (10) gives the following corollary:

Corollary 3.1 (Positiveness and boundedness of submodels)**.** *Assume that both* (9) *and* (10) *are subject to Condition* ***H****. Then both submodels are positively invariant and uniformly ultimately bounded in* $\mathbb{R}^2_+$*. In addition, the submodel* (10) *has the following property:*

$$\limsup_{t\to\infty} S(t) + I(t) \leq 1.$$

In the next two subsections, we explore the detailed dynamics of both submodels (9) and (10).

3.1. Equilibria and local stability

It is easy to check that both submodels (9) and (10) have $(0,0), (\theta,0)$ and $(1,0)$ as their boundary equilibria. For convenience, for Model (9), we denote

$$E_0^P = (0,0),\ E_\theta^P = (\theta,0),\ E_1^P = (1,0) \text{ and } E_i^P = \left(\frac{1}{R_0^P}, \frac{1}{a}\left(\frac{1}{R_0^P}-\theta\right)\left(1-\frac{1}{R_0^P}\right)\right)$$

while for Model (10), we denote

$$E_0^I = (0,0),\ E_\theta^I = (\theta,0),\ E_1^I = (1,0) \text{ and } E_i^I = \left(\frac{1}{R_0^I}, \frac{\left(\frac{1}{R_0^I}-\theta\right)\left(1-\frac{1}{R_0^I}\right)}{\frac{1}{R_0^I}-\theta+\beta}\right)$$

where E_i^P, E_i^I are interior equilibria for the submodel (9) and (10), respectively, provided their existence. The local stability of equilibria of both submodels (9) and (10) can be summarized in the following proposition:

Proposition 3.1. *[Local stability of equilibria for submodels* (9) *and* (10)*] The local stability of boundary equilibria of both submodels* (9) *and* (10) *is summarized in Table 2 while the local stability of interior equilibrium of both submodels* (9) *and* (10) *is summarized in Table 3. Moreover, the equilibria* E_i^P *of the submodel* (9) *undergoes a supercritical Hopf-bifurcation at* $R_0^P = \frac{2}{\theta+1}$ *and the equilibria* E_i^I *of the submodel* (10) *undergoes a supercritical Hopf-bifurcation at* $R_0^I = \frac{\beta-\theta+\sqrt{\beta^2-\beta\theta+\beta}}{\beta+\beta\theta-\theta^2}$.

Boundary Equilibria	Stability Condition
E_0^P and E_0^I	Always locally asymptotically stable
E_θ^P	Saddle if $R_0^P < \frac{1}{\theta}$; Source if $R_0^P > \frac{1}{\theta}$
E_θ^I	Saddle if $R_0^I < \frac{1}{\theta}$; Source if $R_0^I > \frac{1}{\theta}$
E_1^P	Locally asymptotically stable if $R_0^P < 1$; Saddle if $R_0^P > 1$
E_1^I	Locally asymptotically stable if $R_0^I < 1$; Saddle if $R_0^I > 1$

Table 2: The local stability of boundary equilibria for both submodels (9) and (10)

Interior Equilibrium	Condition for existence	Condition for local asymptotic stability
E_i^P	$1 < R_0^P < \frac{1}{\theta}$	$1 < R_0^P < \frac{2}{\theta+1}$
E_i^I	$1 < R_0^I < \frac{1}{\theta}$	$1 < R_0^I < \frac{\beta-\theta+\sqrt{\beta^2-\beta\theta+\beta}}{\beta+\beta\theta-\theta^2}$.

Table 3: The local stability of interior equilibrium for both submodels (9) and (10)

Proof. The Jacobian matrix of the submodel (9) at its equilibrium (S^*, P^*) is presented as follows

$$J^P|_{(S^*,P^*)} = \begin{bmatrix} (S^*-\theta)(1-S^*) - aP^* + S^*(1-2S^*+\theta) & -aS^* \\ bP^* & bS^* - d \end{bmatrix} \tag{11}$$

while the Jacobian matrix of the submodel (10) at its equilibrium (S^*, I^*) is presented as follows

$$J^I|_{(S^*,I^*)} = \begin{bmatrix} (S^*-\theta)(1-S^*-I^*) - \beta I^* + S^*(1-2S^*-I^*+\theta) & S^*(-S^*+\theta-\beta) \\ \beta I^* & \beta S^* - \mu \end{bmatrix}. \tag{12}$$

After substituting $(S^*, P^*) = E_u^P, u = 0, \theta, 1, i$ into (11), we obtain the eigenvalues for each equilibrium:

1. $E_0^P = (0,0)$ is always locally asymptotically stable since both eigenvalues associated with (11) at E_0^P are negative, i.e.
$$\lambda_1 = -\theta \text{ and } \lambda_2 = -d.$$

2. $E_\theta^P = (\theta, 0)$ is a saddle if $R_0^P < \frac{1}{\theta}$ and is a source if $R_0^P > \frac{1}{\theta}$ since both eigenvalues associated with (11) at E_θ^P can be represented as follows:
$$\begin{aligned} \lambda_1 &= \theta(1-\theta)(>0) \\ \lambda_2 &= d\theta\left(R_0^P - \frac{1}{\theta}\right)\begin{cases} <0 \ if \ R_0^P < \frac{1}{\theta} \\ >0 \ if \ R_0^P > \frac{1}{\theta}. \end{cases} \end{aligned}$$

3. $E_1^P = (1,0)$ is locally asymptotically stable if $R_0^P < 1$ and is a saddle if $R_0^P > 1$ since both eigenvalues associated with (11) at E_1^P can be represented as follows:
$$\begin{aligned} \lambda_1 &= (\theta-1)(<0) \\ \lambda_2 &= d\left(R_0^P - 1\right)\begin{cases} <0 \ if \ R_0^P < 1 \\ >0 \ if \ R_0^P > 1. \end{cases} \end{aligned}$$

4. The unique interior equilibrium $E_i^P = (S^*, P^*) = \left(\frac{1}{R_0^P}, \frac{1}{a}\left(\frac{1}{R_0^P} - \theta\right)\left(1 - \frac{1}{R_0^P}\right)\right)$ exists only if $1 < R_0^P < \frac{1}{\theta}$. The Jacobian matrix evaluated at E_i^P is given by
$$J^P\Big|_{E_i^P} = \begin{bmatrix} A & -B \\ C & 0 \end{bmatrix} = \begin{bmatrix} \frac{1}{R_0^P}\left(1+\theta-\frac{2}{R_0^P}\right) & -\frac{a}{R_0^P} \\ \frac{b}{a}\left(\frac{1}{R_0^P} - \theta\right)\left(1-\frac{1}{R_0^P}\right) & 0 \end{bmatrix}$$

whose characteristic equation is given by

$$\lambda^2 - A\lambda + BC = 0$$

where $BC > 0$ and

$$A > 0 \text{ if } R_0^P > \frac{2}{1+\theta} \text{ while } A < 0 \text{ if } R_0^P < \frac{2}{1+\theta}.$$

This indicates that the eigenvalues of $J^P\Big|_{E_i^P}$ are

$$\lambda_1 = \frac{A - \sqrt{A^2 - 4BC}}{2} \text{ and } \lambda_2 = \frac{A + \sqrt{A^2 - 4BC}}{2} \text{ when } A^2 > 4BC$$

or

$$\lambda_1 = \frac{A - i\sqrt{4BC - A^2}}{2} \text{ and } \lambda_2 = \frac{A + i\sqrt{4BC - A^2}}{2} \text{ when } A^2 < 4BC.$$

Therefore, E_i^P exists and is locally asymptotically stable if

$$1 < R_0^P < \min\{\frac{1}{\theta}, \frac{2}{1+\theta}\} = \frac{2}{1+\theta}.$$

Notice that $A = 0$ when $R_0^P = \frac{2}{1+\theta}$, and

$$\frac{dA}{d\left(R_0^P\right)} = \frac{(\theta+1)\,R_0^P - 2\left(\theta R_0^P + R_0^P - 2\right)}{\left(R_0^P\right)^3} \text{ with } \frac{dA}{d\left(R_0^P\right)}\Big|_{R_0^P = \frac{2}{1+\theta}} = \frac{(\theta+1)^3}{4} > 0,$$

thus according to Theorem 3.1.3 in Wiggins [77], we know that the submodel (9) undergoes a Hopf-bifurcation at $R_0^P = \frac{2}{\theta+1}$. Then apply Theorem 3.1 from Wang et al. [75], we can conclude that the Hopf-bifurcation is supercritical.

Similarly, after substituting $(S^*, I^*) = E_u^I, u = 0, \theta, 1, i$ into (12), we obtain the eigenvalues for each equilibrium:

1. $E_0^I = (0,0)$ is always locally asymptotically stable since both eigenvalues associated with (12) at E_0^I are negative, i.e.
$$\lambda_1 = -\theta \text{ and } \lambda_2 = -\mu.$$

2. $E_\theta^I = (\theta, 0)$ is a saddle if $R_0^I < \frac{1}{\theta}$ and is a source if $R_0^I > \frac{1}{\theta}$ since both eigenvalues associated with (12) at E_θ^I can be represented as follows:
$$\begin{aligned} \lambda_1 &= \theta\,(1-\theta)\,(>0) \\ \lambda_2 &= \mu\theta\left(R_0^I - \tfrac{1}{\theta}\right) \begin{cases} <0 \ if \ R_0^I < \frac{1}{\theta} \\ >0 \ if \ R_0^I > \frac{1}{\theta}. \end{cases} \end{aligned}$$

3. $E_1^I = (1,0)$ is locally asymptotically stable if $R_0^I < 1$ and is a saddle if $R_0^I > 1$ since both eigenvalues associated with (12) at E_1^I can be represented as follows:
$$\begin{aligned} \lambda_1 &= (\theta - 1)\,(<0) \\ \lambda_2 &= \mu\left(R_0^I - 1\right) \begin{cases} <0 \ if \ R_0^I < 1 \\ >0 \ if \ R_0^I > 1. \end{cases} \end{aligned}$$

4. The unique interior equilibrium $E_i^I = (S^*, I^*) = \left(\frac{1}{R_0^I}, \frac{\left(\frac{1}{R_0^I} - \theta\right)\left(1 - \frac{1}{R_0^I}\right)}{\frac{1}{R_0^I} + \beta - \theta} \right)$ exists only if $1 < R_0^I < \frac{1}{\theta}$ since from Condition **H**, we have

$$\frac{1}{R_0^I} + \beta - \theta = \frac{\mu}{\beta} + \beta - \theta > \frac{\theta^2}{4\beta} + \beta - \theta = \frac{\theta^2 + 4\beta^2 - 4\theta\beta}{4\beta} = \frac{(\theta - 2\beta)^2}{4\beta} \geq 0.$$

178 The Jacobian matrix evaluated at E_i^I is given by

$$J^I\Big|_{E_i^I} = \begin{bmatrix} A & -B \\ C & 0 \end{bmatrix} = \begin{bmatrix} \frac{1}{R_0^I}\left(1 - \frac{2}{R_0^I} - \frac{\left(\frac{1}{R_0^I} - \theta\right)\left(1 - \frac{1}{R_0^I}\right)}{\frac{1}{R_0^I} + \beta - \theta} + \theta\right) & -\frac{1}{R_0^I}\left(\frac{1}{R_0^I} + \beta - \theta\right) \\ \frac{\beta\left(\frac{1}{R_0^I} - \theta\right)\left(1 - \frac{1}{R_0^I}\right)}{\frac{1}{R_0^I} + \beta - \theta} & 0 \end{bmatrix}$$

whose characteristic equation is given by

$$\lambda^2 - A\lambda + BC = 0$$

where $BC > 0$ and

$$A = \frac{1}{R_0^I}\left(1 - \frac{2}{R_0^I} - \frac{\left(\frac{1}{R_0^I} - \theta\right)\left(1 - \frac{1}{R_0^I}\right)}{\frac{1}{R_0^I} + \beta - \theta} + \theta\right) = \frac{(\beta + \beta\theta - \theta^2)(R_0^I)^2 - 2R_0^I(\beta - \theta) - 1}{(R_0^I)^2\left(1 + R_0^I(\beta - \theta)\right)},$$

$$= \frac{1}{\left(R_0^I\right)^2\left(1 + R_0^I\left(\beta - \theta\right)\right)}\left(R_0^I - \frac{(\beta - \theta) + \sqrt{\beta^2 - \beta\theta + \beta}}{\beta + \beta\theta - \theta^2}\right)\left(R_0^I - \frac{(\beta - \theta) - \sqrt{\beta^2 - \beta\theta + \beta}}{\beta + \beta\theta - \theta^2}\right).$$

Thus, we have

$$A > 0 \text{ if } R_0^I > \frac{\beta - \theta + \sqrt{\beta^2 - \beta\theta + \beta}}{\beta + \beta\theta - \theta^2} \text{ while } A < 0 \text{ if } R_0^I < \frac{\beta - \theta + \sqrt{\beta^2 - \beta\theta + \beta}}{\beta + \beta\theta - \theta^2}.$$

This indicates that the eigenvalues of $J^I\Big|_{E_i^I}$ are

$$\lambda_1 = \frac{A - \sqrt{A^2 - 4BC}}{2} \text{ and } \lambda_2 = \frac{A + \sqrt{A^2 - 4BC}}{2} \text{ when } A^2 > 4BC$$

or

$$\lambda_1 = \frac{A - i\sqrt{4BC - A^2}}{2} \text{ and } \lambda_2 = \frac{A + i\sqrt{4BC - A^2}}{2} \text{ when } A^2 < 4BC.$$

Therefore, E_i^I exists and is locally asymptotically stable if

$$1 < R_0^I < \min\{\frac{1}{\theta}, \frac{\beta - \theta + \sqrt{\beta^2 - \beta\theta + \beta}}{\beta + \beta\theta - \theta^2}\} = \frac{\beta - \theta + \sqrt{\beta^2 - \beta\theta + \beta}}{\beta + \beta\theta - \theta^2}.$$

Notice that $A = 0$ when $R_0^I = \frac{\beta - \theta + \sqrt{\beta^2 - \beta\theta + \beta}}{\beta + \beta\theta - \theta^2}$ and

$$\frac{dA}{d\left(R_0^I\right)}\Big|_{R_0^I = \frac{\beta - \theta + \sqrt{\beta^2 - \beta\theta + \beta}}{\beta + \beta\theta - \theta^2}} = \frac{2\sqrt{\beta(\beta - \theta) + \beta}}{\left(\beta + \theta\left(\beta - \theta\right)\right)\left(R_0^I\right)^2\left(1 + R_0^I\left(\beta - \theta\right)\right)^2} > 0.$$

179 Thus according to Theorem 3.1.3 in Wiggins [77] and Theorem 3.1 in Wang et al. [75] again,
180 we can conclude that the submodel (10) undergoes a supercritical Hopf-bifurcation at $R_0^I =$
181 $\frac{\beta - \theta + \sqrt{\beta^2 - \beta\theta + \beta}}{\beta + \beta\theta - \theta^2}$.

□

Notes: Local analysis results provided in Proposition 3.1 and Table 3 suggest that the coexistence of prey and predation at the equilibrium E_i^P in the subsystem (9) is determined by the **Allee threshold** θ since E_i^P is locally asymptotically stable if

$$1 < R_0^P < \frac{2}{\theta+1} \text{ since } \frac{2}{\theta+1} < \frac{1}{\theta}.$$

And the coexistence of health prey and infected prey at the equilibrium E_i^I in the subsystem (10) is determined by both the **Allee threshold** θ and the disease transmission rate β since E_i^I is locally asymptotically stable if

$$1 < R_0^I < \frac{\beta-\theta+\sqrt{\beta^2-\beta\theta+\beta}}{\beta+\beta\theta-\theta^2} \text{ since } \frac{\beta-\theta+\sqrt{\beta^2-\beta\theta+\beta}}{\beta+\beta\theta-\theta^2} < \frac{1}{\theta}.$$

3.2. Disease/predation-driven extinctions and global features of submodels

In this subsection, we focus on the disease/predation-driven extinctions as well as the features of global dynamics of both submodels. First, we have the following theorem regarding the extinction of one or both species:

Theorem 3.1. *[Extinction] Assume that both submodels (9) and (10) subject to Condition **H**. Then*

1. *If $R_0^P \leq 1$, then the population of predator in the submodel (9) goes extinction for any initial condition taken in $\mathbb{R}^2_+$ [see Figure 2(a)]. Similarly, if $R_0^I \leq 1$, then the population of infectives in the submodel (10) goes extinction for any initial condition taken in $\mathbb{R}^2_+$ [see Figure 2(c)].*
2. *If $R_0^P \geq \frac{1}{\theta}$, then System (9) converges to $(0,0)$ for any initial condition taken in the interior of $\mathbb{R}^2_+$, which is predation-driven extinction [see Figure 2(b)]. Similarly, if $R_0^I \geq \frac{1}{\theta}$, then System (10) converges to $(0,0)$ for any initial condition taken in the interior of $\mathbb{R}^2_+$, which is disease-driven extinction [see Figure 2(d)].*
3. *If $S(0) < \theta$, then all species in both submodels (9) and (10) converge to $(0,0)$.*

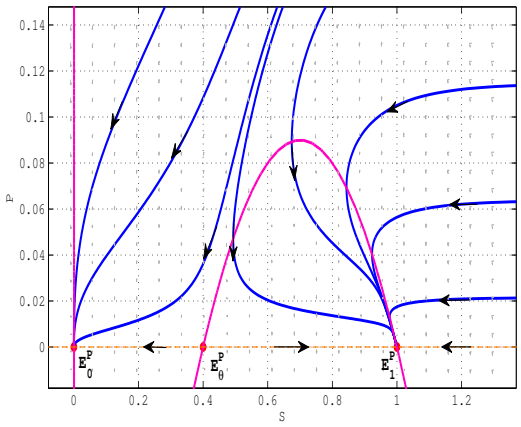
Proof. The detailed proof for the submodel (9) is similar to the proof for the submodel (10), thus we only focus on the submodel (10).

According to Proposition 3.1, if $R_0^I \leq 1$ or $R_0^I \geq \frac{1}{\theta}$, then the submodel (10) only has three boundary equilibria $E_u^I, u = 0, \theta, 1$ where E_θ^I is a saddle and E_1^I is locally asymptotically stable when $R_0^I < 1$ while E_θ^I is a source and E_1^I is a saddle when $R_0^I > 1$. For $R_0^I = 1$, E_1^I is nonhyperbolic with one zero eigenvalue and the other negative while E_θ^I remains saddle. For $R_0^I = \frac{1}{\theta}$, E_θ^I is nonhyperbolic with one zero eigenvalue and the other positive while E_1^I remains saddle.

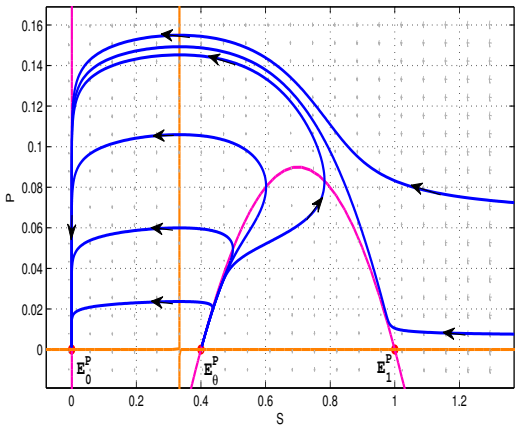
According to Theorem 2.1, the submodel (10) has a compact global attractor. Thus, from an application of the Poincaré-Bendixson theorem [31] we conclude that the trajectory starting at any initial condition living in the interior of $\mathbb{R}^2_+$ converges to one of three boundary equilibria $E_u^I, u = 0, \theta, 1$ when (10) has no interior equilibrium. This implies that $\limsup_{t\to\infty} I(t) = 0$ when $R_0^I \leq 1$ or $R_0^I \geq \frac{1}{\theta}$. Since E_0^I is the only locally asymptotically stable boundary equilibrium when $R_0^I \geq \frac{1}{\theta}$, therefore, System (10) converges to $(0,0)$ for any initial condition taken in the interior of $\mathbb{R}^2_+$.

The third part of Theorem 3.1 can be a direct application of results from Theorem 2.1. Therefore, the statement holds.

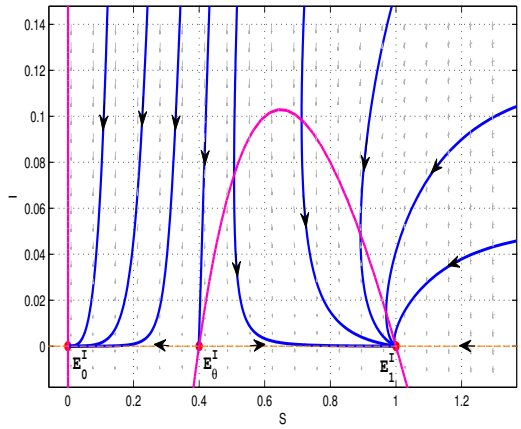
□



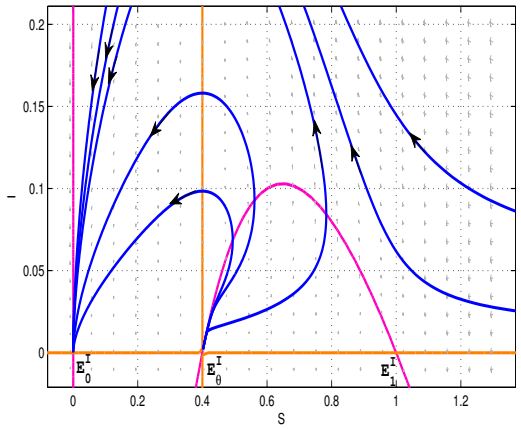
(a) Predation free for the submodel (9)



(b) Predation-driven extinction for the submodel (9)



(c) Disease free for the submodel (10)



(d) Disease-driven extinction for the submodel (10)

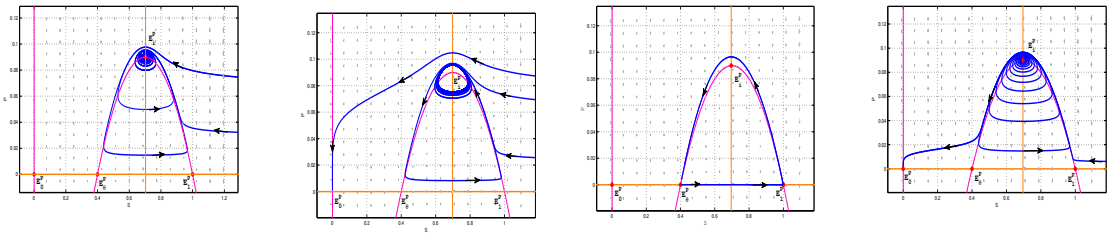
Figure 2: Phase portraits of submodel (9) (first row) and (10) (second row) when $\beta = 0.6, \theta = 0.4, a = 1$ and $b = 0.1$. (a)-$E_0^P \cup E_1^P$ is the global attractor when $R_0^P = 0.6$ for the submodel (9); (b)-E_0^P is the global attractor when $R_0^P = 3$; (c)-$E_0^I \cup E_1^I$ is the global attractor when $R_0^I = 0.5$ for the submodel (10); (d)-E_0^I is the global attractor when $R_0^I = 2.6$ for the submodel (10). Notice that $R_0^P = \frac{b}{d}$ and $R_0^I = \frac{\beta}{\mu}$.

Notes: The second item in the statement of Theorem 3.1 is disease/predation-driven extinctions due to Allee effects of the susceptible population. The predation-driven extinction is also called "overexploitation" where both prey and predator go extinct dramatically due to large predator invasion [74, 75], i.e., predator reproduces fast enough to drive the prey population below its Allee threshold, thus lead to the extinction of both species. The biological explanation of disease-driven extinction is credited to the large disease transmission rate (i.e., the basic reproduction number R_0^I is large) while the reproduction of the susceptible population is not fast enough to sustain its own population. Thus, the susceptible population drops below its Allee threshold and decreases to zero, which eventually drives the infected population extinct eventually. The third item in the statement of Theorem 3.1 does not always hold if Condition **H** does not hold. For example, if we drop the assumption $\mu > \theta$, then the condition $S(0) < \theta$ does not always lead to the extinction of both susceptible and infective population in the submodel (10).

3.2.1. Global features of submodels (9) and (10)

The dynamics of global features of submodels (9) and (10) are similar. Fix $\beta = 0.6, \theta = 0.4, a = 1, b = 0.1$, and vary the basic reproduction numbers R_0^P, R_0^I for the submodel (9), the submodel (10), respectively:

1. For the submodel (9):
   (a) $0 < R_0^P \leq 1$**:** This leads to the predation free dynamics with $E_0^P \cup E_1^P$ as attractors according to Theorem 3.1 [see Figure 2(a)].
   (b) $1 < R_0^P < 1.428571 = \frac{2}{1+\theta}$**:** There is a transcriptical bifurcation at $R_0^P = 1$. When increasing the value of R_0^P from 1, E_1^P becomes unstable and the unique and locally asymptotically stable interior equilibrium E_i^P occurs is locally asymptotically stable [see Proposition 3.1 and Figure 3(b)].
   (c) $1.428571 = \frac{2}{1+\theta} < R_0^P < 1.437398001$**:** There is a supercritical Hopf-bifurcation at $R_0^P = 1.428571 = \frac{2}{1+\theta}$ which leads to the unique stable limit cycle [see Proposition 3.1 and Figure 3(b)]. Wang et al. [75] has provided the proof of the uniqueness of the limit cycle.
   (d) **At** $R_0^P = 1.437398001$**:** There is a heteroclinic bifurcation at $R_0^P = 1.437398001$ [see Figure 3(c)], i.e., there is a heteroclinic orbit connecting E_1^P to E_θ^P. The disappearance of the unique stable limit cycle is associated with the occurrence of heteroclinic connections: Outside the heteroclinic cycle the trajectory goes asymptotically to extinction equilibrium E_0^P, while for initial conditions inside the heteroclinic cycle the trajectory converges towards the heteroclinic cycle. Sieber and Hilker [63] and Wang et al. [75] have provided the proof of the existence of the heteroclinic orbit.
   (e) $1.437398001 < R_0^P < \frac{1}{\theta} = 2.5$**:** The predation-driven extinction occurs: the heteroclinic orbit is broken and all trajectories in the interior of $\mathbb{R}^2_+$ converge to E_0^P: For initial condition inside the curve bounded by the stable manifold of E_1^P, the orbit oscillates before finally converging slowly to E_0^P while all orbits above the unstable manifold of E_1^P converge towards E_0^P [see Figure 3(d)].
   (f) $R_0^P \geq \frac{1}{\theta} = 2.5$**:** The predation-driven extinction occurs and the system has no interior equilibrium any more. All trajectories in the interior of $\mathbb{R}^2_+$ converge to E_0^P [see Figure 2(b)].
2. For the submodel (10):
   (a) The submodel (10) exhibits exactly the same dynamics feature as the submodel (9) when we increase the value of R_0^I from 0: A transcritical bifurcation occurs at $R_0^I = 1$, For $1 < R_0^I < \frac{\beta-\theta+\sqrt{\beta^2-\beta\theta+\beta}}{\beta+\beta\theta-\theta^2} = 1.5420$, the unique interior equilibrium E_i^I is locally asymptotically stable [see Figure 3(e)]. At $R_0^I = \frac{\beta-\theta+\sqrt{\beta^2-\beta\theta+\beta}}{\beta+\beta\theta-\theta^2} = 1.5420$, a supercritical Hopf-bifurcation occurs

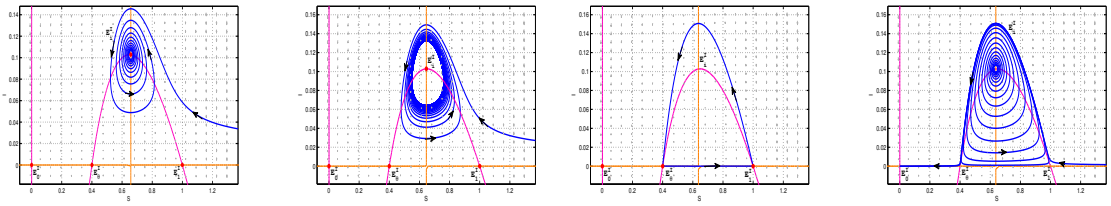


(a) Stable interior equilibrium for the submodel (9) when $R_0^P = 1.42222$

(b) Stable limit cycle for the submodel (9) when $R_0^P = 1.431$

(c) Heteroclinic orbit for the submodel (9) when $R_0^P = 1.437398$

(d) Predation-driven extinction for the submodel (9) when $R_0^P = 1.4373981$



(e) Stable interior equilibrium for the submodel (10) when $R_0^I = 1.52$

(f) Stable limit cycle for the submodel (10) when $R_0^I = 1.548$

(g) Heteroclinic orbit for the submodel (10) when $R_0^I = 1.569462683$

(h) Disease-driven extinction for the submodel (10) when $R_0^I = 1.569462684$

Figure 3: Phase portraits of submodels (9) (the first row) and (10) (the second row) when $\beta = 0.6$, $\theta = 0.4$, $a = 1$ and $b = 0.1$. Notice that $R_0^P = \frac{b}{d}$ and $R_0^I = \frac{\beta}{\mu}$.

which leads to a unique stable limit cycle for $1.5420 < R_0^I < 1.569462683$ [see Figure 3(f)]. The heteroclinic bifurcation occurs at $R_0^I = 1.569462683$ [see Figure 3(g)] and disease-driven extinction occurs when $R_0^I > 1.569462683$ [see Figure 3(h) and 2(d)].

The impact of Allee effects: Without Allee effects, the submodels (9) and (10) can be represented as the following two models:

$$\begin{array}{ll} \frac{dS}{dt} = S\left[1 - S - aP\right], & \frac{dS}{dt} = S\left[1 - S - I - \beta I\right] \\ \frac{dP}{dt} = P\left[bS - d\right], & \frac{dI}{dt} = I\left[\beta S - \mu\right] \end{array} . \tag{13}$$

The two models above have the same dynamics as the traditional Lotka-Volterra Pedator-Prey model: If $R_0^k \leq 1, k = P, I$, then both models of (13) has global stability at $(1, 0)$; while if $R_0^k > 1, k = P, I$, then both models of (13) has global stability at its unique interior equilibrium. Compare this simple dynamics to the dynamics of submodels (9) and (10), we can conclude that the effects of Allee effects:

1. **Importance of initial conditions:** Allee effects in the susceptible population, requires its initial condition being above the Allee threshold to persist.

2. **Destabilizer:** The nonlinearity induced by Allee effects destablizes the system which lead to fluctuated populations (e.g., stable limit cycle).

3. **Disease/predation-driven extinction:** This occurs when the basic reproduction number of disease or predation is large enough to drive the susceptible population below its Allee threshold, thus all species go extinct.

4. Dynamics of the full S-I-P model

After obtaining a complete dynamics of disease/predation free dynamics of the full SIP model (8) in the previous section, we continue to study the dynamics of the full model. We start with the boundary equilibria and their stability of (8). It is easy to check that System (8) has the following boundary equilibria:

$$E_0 = (0,0,0),\ E_\theta = (\theta,0,0), E_1 = (1,0,0),\ E_P^i = \left(\frac{1}{R_0^P}, 0, \frac{1}{a}\left(\frac{1}{R_0^P} - \theta\right)\left(1 - \frac{1}{R_0^P}\right)\right)$$

and

$$E_I^i = \left(\frac{1}{R_0^I}, \frac{\left(\frac{1}{R_0^I} - \theta\right)\left(1 - \frac{1}{R_0^I}\right)}{\frac{1}{R_0^I} + \beta - \theta}, 0\right).$$

The existence of E_P^i requires $1 < R_0^P < \frac{1}{\theta}$ while the existence of E_I^i requires $1 < R_0^I < \frac{1}{\theta}$.

Proposition 4.1. *[Boundary equilibrium and stability] Sufficient conditions for the existence and the local stability of boundary equilibria for System (8) are summarized in Table 4.*

Boundary Equilibria	Stability Condition
E_0	Always locally asymptotically stable
E_θ	Source if $R_0^P > \frac{1}{\theta}$ and $R_0^I > \frac{1}{\theta}$; otherwise is saddle
E_1	Locally asymptotically stable if $R_0^P < 1$ and $R_0^I < 1$
E_P^i	Locally asymptotically stable if $1 < R_0^P < \frac{2}{1+\theta}$ and $\frac{R_0^I}{R_0^P} < 1 + \frac{\left(\frac{1}{R_0^P} - \theta\right)\left(1 - \frac{1}{R_0^P}\right)}{\mu}$
E_I^i	Locally asymptotically stable if $1 < R_0^I < \frac{\beta - \theta + \sqrt{\beta^2 - \beta\theta + \beta}}{\beta + \beta\theta - \theta^2}$ and $\frac{R_0^P}{R_0^I} < 1 - \frac{\alpha\left(\frac{1}{R_0^I} - \theta\right)\left(1 - \frac{1}{R_0^I}\right)}{d\left(\frac{1}{R_0^I} + \beta - \theta\right)}$

Table 4: Sufficient conditions for the existence and local stability of boundary equilibria for System (8)

Proof. The local stability of equilibrium can be determined by the eigenvalues $\lambda_i, i = 1,2,3$ of the Jacobian matrix of System (8) evaluated at the equilibrium. By simple calculations, we have follows:

1. The equilibrium $E_0 = (0,0,0)$ is always locally asymptotically stable since its eigenvalues are

$$\lambda_1 = -\theta\,(<0)\,,\ \lambda_2 = -\mu\,(<0)\,,\ \lambda_3 = -d\,(<0)$$

2. The equilibrium $E_\theta = (\theta,0,0)$ is always unstable since its eigenvalues are

$$\lambda_1 = \theta\,(1-\theta)\,(>0)\,,\ \lambda_2 = \mu\theta\left(R_0^1 - \frac{1}{\theta}\right)\begin{cases}<0 \ if\ R_0^I < \frac{1}{\theta}\\ >0 \ if\ R_0^I > \frac{1}{\theta}\end{cases},\ \lambda_3 = d\theta\left(R_0^P - \frac{1}{\theta}\right)\begin{cases}<0 \ if\ R_0^P < \frac{1}{\theta}\\ >0 \ if\ R_0^P > \frac{1}{\theta}\end{cases}$$

3. The equilibrium $E_1 = (1,0,0)$ is locally asymptotically stable if $R_0^I < 1$ and $R_0^P < 1$ since its eigenvalues are

$$\lambda_1 = (\theta - 1)\,(<0)\,,\ \lambda_2 = \mu\left(R_0^I - 1\right)\begin{cases}<0 \ if\ R_0^I < 1\\ >0 \ if\ R_0^I > 1\end{cases},\ \lambda_3 = d\left(R_0^P - 1\right)\begin{cases}<0 \ if\ R_0^P < 1\\ >0 \ if\ R_0^P > 1\end{cases}$$

where the sign of λ_i indicates its eigenvector pointing toward (< 0) or away from (> 0) the equilibrium in S-axis ($i = 1$), I-axis ($i = 2$) and P-axis ($i = 3$), respectively.

According to Proposition 3.1, the equilibrium $E_P^i = \left(\frac{1}{R_0^P}, 0, \frac{1}{a}\left(\frac{1}{R_0^P} - \theta\right)\left(1 - \frac{1}{R_0^P}\right)\right)$ is locally asymptotically stable if it is locally asymptotically stable in the submodel (9) and

$$\frac{dI}{Idt}\Big|_{E_P^i} = \frac{\beta}{R_0^P} - \left(\frac{1}{R_0^P} - \theta\right)\left(1 - \frac{1}{R_0^P}\right) - \mu < 0 \Leftrightarrow \frac{R_0^I}{R_0^P} < 1 + \frac{\left(\frac{1}{R_0^P} - \theta\right)\left(1 - \frac{1}{R_0^P}\right)}{\mu}$$

which indicates that disease is not able to invade at E_P^i.

Similarly, the equilibrium $E_I^i = \left(\frac{1}{R_0^I}, \frac{\left(\frac{1}{R_0^I} - \theta\right)\left(1 - \frac{1}{R_0^I}\right)}{\frac{1}{R_0^I} + \beta - \theta}, 0\right)$ is locally asymptotically stable if it is locally asymptotically stable in the submodel (10) and

$$\frac{dP}{Pdt}\Big|_{E_I^i} = bS + \alpha I - d = \frac{b}{R_0^I} + \frac{\alpha\left(\frac{1}{R_0^I} - \theta\right)\left(1 - \frac{1}{R_0^I}\right)}{\frac{1}{R_0^I} + \beta - \theta} - d < 0 \Leftrightarrow \frac{R_0^P}{R_0^I} < 1 - \frac{\alpha\left(\frac{1}{R_0^I} - \theta\right)\left(1 - \frac{1}{R_0^I}\right)}{d(\frac{1}{R_0^I} + \beta - \theta)}$$

which indicates that predator is not able to invade at E_I^i.

Therefore, we can conclude that E_P^i is locally asymptotically stable if

$$1 < R_0^P < \frac{2}{1+\theta} \text{ and } \frac{R_0^I}{R_0^P} < 1 + \frac{\left(\frac{1}{R_0^P} - \theta\right)\left(1 - \frac{1}{R_0^P}\right)}{\mu}$$

and E_I^i is locally asymptotically stable if

$$1 < R_0^I < \frac{\beta - \theta + \sqrt{\beta^2 - \beta\theta + \beta}}{\beta + \beta\theta - \theta^2} \text{ and } \frac{R_0^P}{R_0^I} < 1 - \frac{\alpha\left(\frac{1}{R_0^I} - \theta\right)\left(1 - \frac{1}{R_0^I}\right)}{d(\frac{1}{R_0^I} + \beta - \theta)}.$$

□

Notes: Notice that

$$\frac{2}{1+\theta} < \frac{\beta - \theta + \sqrt{\beta^2 - \beta\theta + \beta}}{\beta + \beta\theta - \theta^2} < \frac{1}{\theta},$$

thus according to Proposition 4.1, both E_P^i and E_I^i can be locally asymptotically stable if

$$1 < R_0^P < \frac{2}{1+\theta}, \; 1 < R_0^I < \frac{\beta - \theta + \sqrt{\beta^2 - \beta\theta + \beta}}{\beta + \beta\theta - \theta^2}$$

and

$$\frac{\frac{d}{R_0^I}(\frac{1}{R_0^I} + \beta - \theta)}{d(\frac{1}{R_0^I} + \beta - \theta) - \alpha\left(\frac{1}{R_0^I} - \theta\right)\left(1 - \frac{1}{R_0^I}\right)} < \frac{1}{R_0^P} < \frac{1}{R_0^I}\left(1 + \frac{\left(\frac{1}{R_0^P} - \theta\right)\left(1 - \frac{1}{R_0^P}\right)}{\mu}\right).$$

For convenience, let $d = \mu = 1, \beta = 1.5, \theta = 0.2$, then, according to Condition **H**, we have

$$1 < R_0^I = \frac{\beta}{\mu} = 1.5 < \frac{\beta - \theta + \sqrt{\beta^2 - \beta\theta + \beta}}{\beta + \beta\theta - \theta^2} \approx 1.794, \; R_0^P = \frac{b}{d} = b \geq \alpha, \; \frac{2}{1+\theta} = \frac{5}{3} \approx 1.667,$$

and

$$\begin{aligned}\frac{\frac{d}{R_0^I}\left(\frac{1}{R_0^I}+\beta-\theta\right)}{d\left(\frac{1}{R_0^I}+\beta-\theta\right)-\alpha\left(\frac{1}{R_0^I}-\theta\right)\left(1-\frac{1}{R_0^I}\right)} &= \frac{1.967}{2.95-0.233\alpha},\\ \frac{1}{R_0^I}\left(1+\frac{\left(\frac{1}{R_0^P}-\theta\right)\left(1-\frac{1}{R_0^P}\right)}{\mu}\right) &= \frac{2\left(1+\left(\frac{1}{R_0^P}-0.2\right)\left(1-\frac{1}{R_0^P}\right)\right)}{3}.\end{aligned}$$

Thus, according to Proposition 4.1, we have the following statement when $d=\mu=1,\beta=1.5,\theta=0.2$:

1. Both E_P^i and E_I^i are locally asymptotically stable if the following inequalities hold [see the blue region of Figure 4(a)]

$$1<b=R_0^P<1.67,\ \frac{3b^2}{2b^2+2\left(1-0.2b\right)\left(b-1\right)}<b<\frac{2.95-0.233\alpha}{1.967},\ -\infty<\alpha\leq b.$$

2. E_P^i is locally asymptotically stable and E_I^i is locally asymptotically stable in the SI-plane but is unstable in $\mathbb{R}_+^3$ if the following inequalities hold [see the green region of Figure 4(a)]

$$1<b=R_0^P<1.67,\ b>\max\left\{\frac{2.95-0.233\alpha}{1.967},\frac{3b^2}{2b^2+2\left(1-0.2b\right)\left(b-1\right)}\right\},\ -\infty<\alpha\leq b.$$

3. E_I^i is locally asymptotically stable and E_P^i is locally asymptotically stable in the SP-plane but is unstable in $\mathbb{R}_+^3$ if the following inequalities hold [see the yellow region of Figure 4(a)]

$$1<b=R_0^P<1.67,\ b<\min\left\{\frac{2.95-0.233\alpha}{1.967},\frac{3b^2}{2b^2+2\left(1-0.2b\right)\left(b-1\right)}\right\},\ -\infty<\alpha\leq b.$$

According to Proposition 4.1, sufficient conditions for E_P^i and E_I^i being locally asymptotically stable in the SP-plane, SI-plane, respectively, but being unstable in $\mathbb{R}_+^3$ are as follows:

$$1<R_0^P<\frac{2}{1+\theta},\ 1<R_0^I<\frac{\beta-\theta+\sqrt{\beta^2-\beta\theta+\beta}}{\beta+\beta\theta-\theta^2}$$

and

$$1<1+\frac{\left(\frac{1}{R_0^P}-\theta\right)\left(1-\frac{1}{R_0^P}\right)}{\mu}<\frac{R_0^I}{R_0^P}<\frac{d(\frac{1}{R_0^I}+\beta-\theta)}{d(\frac{1}{R_0^I}+\beta-\theta)-\alpha\left(\frac{1}{R_0^I}-\theta\right)\left(1-\frac{1}{R_0^I}\right)}$$

which is impossible when $\alpha\leq 0$ since

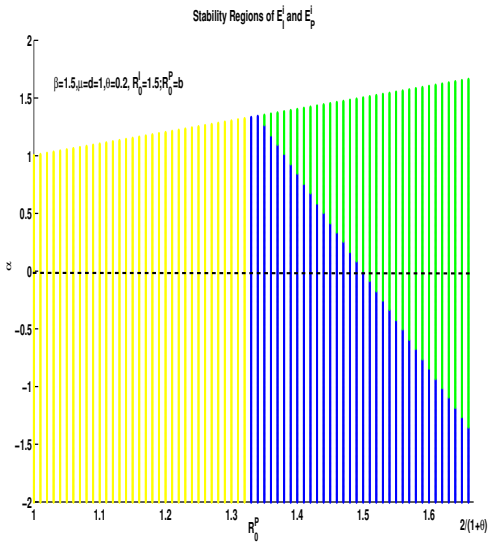
$$\frac{d(\frac{1}{R_0^I}+\beta-\theta)}{d(\frac{1}{R_0^I}+\beta-\theta)-\alpha\left(\frac{1}{R_0^I}-\theta\right)\left(1-\frac{1}{R_0^I}\right)}\leq 1 \text{ when } \alpha\leq 0.$$

In addition, numerical simulations suggest that even if $\alpha\geq 0$, E_P^i and E_I^i cannot be locally asymptotically stable in the SP-plane, SI-plane, respectively, but being unstable in $\mathbb{R}_+^3$.

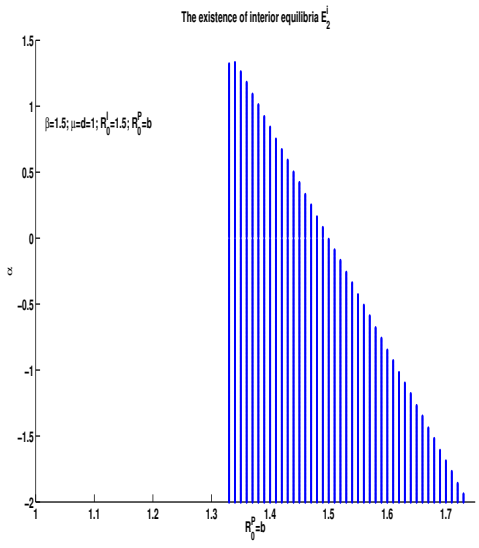
4.1. Global features

In this subsection, we first explore sufficient conditions that lead to the extinction of at least one species of S, I, P. Our study gives the following theorem:

Theorem 4.1. *[Basic global features]Assume that System (8) is subject to Condition **H**. Then*



(a) Stability regions of the boundary equilibria E_P^i and E_I^i



(b) Regions of the existence of E_2^i

Figure 4: Fix $\beta = 1.5; \mu = d = 1; \theta = 0.2$ and $a = 3$. The left graph indicates the stability regions of the boundary equilibria E_P^i and E_I^i: i) In the blue region, both equilibria are locally asymptotically stable; ii) In the green region, E_P^i is locally asymptotically stable while E_I^i is unstable; iii) In the yellow region, E_P^i is locally asymptotically stable while E_I^i is unstable. The blue region in the right graph is the region when System (8) has a unique interior equilibrium which is a saddle; while the white region in the right graph indicates no interior equilibrium.

1. *If* $R_0^I \leq 1$, *then* $\lim_{t\to\infty} I(t) = 0$. *If, in addition,* $R_0^P \leq 1$, *then*

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

While if $R_0^I \leq 1$ *and* $R_0^P > \frac{1}{\theta}$, *then* $\lim_{t\to\infty} (S(t), I(t), P(t)) = E_0$.

2. *If* $\alpha < 0$ *and* $R_0^P \leq 1$, *then* $\lim_{t\to\infty} P(t) = 0$. *If, in addition,* $R_0^I \leq 1$, *then*

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

If $\alpha < 0, R_0^P \leq 1$ *and* $R_0^I > \frac{1}{\theta}$, *then for any initial condition taken in the interior of* $\mathbb{R}^3_+$, *we have*

$$\lim_{t\to\infty} (S(t), I(t), P(t)) = E_0.$$

While if $\alpha > 0$ *and* $R_0^P + \frac{\alpha(1-\theta)}{d} \leq 1$, *then* $\lim_{t\to\infty} P(t) = 0$. *If, in addition,* $R_0^I \leq 1$, *then*

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

If $\alpha > 0, R_0^P + \frac{\alpha(1-\theta)}{d} \leq 1$ *and* $R_0^I > \frac{1}{\theta}$, *then for any initial condition taken in the interior of* $\mathbb{R}^3_+$, *we have*

$$\lim_{t\to\infty} (S(t), I(t), P(t)) = E_0.$$

3. *All trajectories of System* (8) *converge to* E_0 *if* $S(0) < \theta$.

Proof. If $R_0^I \leq 1$, then $\beta = \frac{\phi(N)}{N} \leq \mu$. According to Theorem 2.1, we have $\lim_{t\to\infty} I(t) = 0$, i.e., the limiting dynamics of System (8) is the submodel (9) which has only boundary equilibrium $(0,0), (\theta,0)$ and $(1,0)$ when $R_0^P \leq 1$. Then Poincaré-Bendixson Theorem [31] to (9), we can conclude that $\lim_{t\to\infty} P(t) = 0$. Therefore, we have

$$\lim_{t\to\infty} \max\{I(t), P(t)\} = 0 \text{ if } R_0^I \leq 1 \,\&\, R_0^P \leq 1.$$

While if, in addition, we have $R_0^P > \frac{1}{\theta}$ instead, then from Theorem 3.1 we can conclude that the omega limit set of SP-plane is $E_0 \cup E_\theta \cup E_1$. Since $R_0^I \leq 1$ indicates that, for any $\epsilon > 0$, all trajectories enter into the compact set $[0, B] \times [0, \epsilon] \times [0, B]$ when time large enough, therefore, the condition $R_0^I \leq 1$ and $R_0^P > \frac{1}{\theta}$ indicates that, for any $\epsilon > 0$, all trajectories enter into the compact set $M = [0, 1] \times [0, \epsilon] \times [0, \epsilon]$ when time large enough. Choose ϵ small enough, then the omega limit set of the interior of M is E_0 since E_0 is locally asymptotically stable and E_θ, E_1 is unstable according to Proposition 3.1. Therefore, the condition $R_0^I \leq 1$ and $R_0^P > \frac{1}{\theta}$ indicates that $\lim_{t\to\infty} (S(t), I(t), P(t)) = E_0$.

If $\alpha > 0$, then from the proof of Theorem 3.1 and Corollary 2.1, we can conclude that $\limsup_{t\to\infty} I(t) < 1 - \theta$. This indicates that for any $\epsilon > 0$, there exists a time T such that

$$\frac{dP}{Pdt} < b(1+\epsilon) + \alpha(1-\theta+\epsilon) - d = d\left(R_0^P + \frac{\alpha(1-\theta)}{d} + \frac{\epsilon(b+\alpha)}{d} - 1\right) \text{ for all } t > T$$

which implies that $\lim_{t\to\infty} P(t) = 0$ if $R_0^P + \frac{\alpha(1-\theta)}{d} < 1$. If $R_0^P + \frac{\alpha(1-\theta)}{d} = 1$, then we can apply Poincaré-Bendixson Theorem [31] to (10) to obtain that $\lim_{t\to\infty} P(t) = 0$. The rest of the second item of Theorem 4.1 can be shown by applying the similar arguments of the proof for the first item in Theorem 4.1.

The third item of Theorem 4.1 can be shown by a direct application of Theorem 2.1, i.e., all trajectories converge to E_0 whenever $S(0) < \theta$. □

Notes: A direct implication of Theorem 4.1 is that the coexistence of S, I, P population in System 8 requires $R_0^I > 1$ and

$$R_0^P > 1 \text{ when } \alpha < 0;\ R_0^P > 1 - \frac{\alpha(1-\theta)}{d} \text{ when } \alpha > 0.$$

One interesting question is that if $\alpha > 0$ but $1 - \frac{\alpha(1-\theta)}{d} < R_0^P \leq 1$, then what happens to the dynamics of System 8, e.g., can predator be able to persist under certain conditions? This has been partially answered by Theorem 4.2: System 8 has no interior equilibrium as long as $R_0^P \leq 1$. In fact, predator is not able to survive in this case.

4.2. The interior equilibrium

If System (8) has a locally stable interior equilibrium, then we can say that S, I, P-class can coexist under certain conditions. Thus, in this subsection, we explore sufficient conditions for the existence of the interior equilibrium and its stability for System (8). For convenience, let

$$B = -(\beta - \theta) + \frac{\frac{d}{\alpha} - 1}{\frac{b}{\alpha} - 1},\ C = \frac{\mu - \theta - \frac{d(\beta-\theta)}{\alpha}}{\frac{b}{\alpha} - 1} \text{ and } E_k^i = (S_k^*, I_k^*, P_k^*), k = 1, 2$$

where

$$S_1^* = \frac{B - \sqrt{B^2 - 4C}}{2},\ S_2^* = \frac{B + \sqrt{B^2 - 4C}}{2},\ P_k^* = \frac{\beta}{a}\left(S_k^* - \frac{1}{R_0^I}\right),\ I_k^* = \frac{b}{\alpha}\left(\frac{1}{R_0^P} - S_k^*\right), k = 1, 2.$$

If $\beta > \mu$, i.e., $R_0^I > 1$, then we have follows

$$\begin{aligned} B^2 - 4C &= \left(\tfrac{d-\alpha}{b-\alpha} - (\beta-\theta)\right)^2 - \tfrac{4\alpha(\mu-\theta) - 4d(\beta-\theta)}{b-\alpha} \\ &= (\beta-\theta)^2 + \tfrac{(d-\alpha)^2}{(b-\alpha)^2} + \tfrac{4d(\beta-\theta) - 4\alpha(\mu-\theta) - 2(d-\alpha)(\beta-\theta)}{b-\alpha} \\ &\begin{cases} < (\beta-\theta)^2 + \frac{(d-\alpha)^2}{(b-\alpha)^2} + \frac{4d(\beta-\theta) - 4\alpha(\beta-\theta) - 2(d-\alpha)(\beta-\theta)}{b-\alpha} = \left(\frac{d-\alpha}{b-\alpha} - (\beta-\theta)\right)^2 & \text{if } \alpha < 0 \\ > (\beta-\theta)^2 + \frac{(d-\alpha)^2}{(b-\alpha)^2} + \frac{4d(\beta-\theta) - 4\alpha(\beta-\theta) - 2(d-\alpha)(\beta-\theta)}{b-\alpha} = \left(\frac{d-\alpha}{b-\alpha} - (\beta-\theta)\right)^2 & \text{if } \alpha > 0 \end{cases}. \end{aligned}$$

Therefore, we can conclude that when $R_0^I > 1$, we have

$$S_2^* < \frac{d-\alpha}{b-\alpha} \text{ if } \alpha < 0 \qquad S_2^* > \frac{d-\alpha}{b-\alpha} \text{ if } \alpha > 0. \tag{14}$$

In the case that $\mu = \beta$ (i.e., $R_0^I = 1$), we have $S_2^* = \frac{d-\alpha}{b-\alpha}$ and $S_1^* < 0$. Now we have the following theorem regarding the number of interior equilibrium and its local stability:

Theorem 4.2. *[Interior equilibrium]Assume that Condition **H** holds for System (8).*

1. *System (8) has no interior equilibrium if one of the following conditions is satisfied:*
   - *(a)* $\left\{R_0^I \leq 1\right\}$ *or* $\left\{R_0^P \leq 1, \alpha < b\right\}$ *or*
   - *(b)* $\left\{\alpha > 0, R_0^P \geq \frac{1}{\theta}\right\}$ *or* $\left\{\alpha > 0, R_0^P > R_0^I\right\}$ *or*
   - *(c)* $\left\{\alpha < 0,\ R_0^P = \frac{d}{b} < \frac{d-\alpha}{b-\alpha} < \max\left\{\theta, \frac{1}{R_0^I}\right\}\right\}$ *or*
   - *(d)* $\left\{\alpha > \frac{d}{\frac{\mu-\theta}{\beta-\theta}}\right\}$ *or* $\left\{\mu < \theta + \frac{(b-\alpha)\left(\left(\theta-\beta+\frac{d-\alpha}{b-\alpha}\right)^2 + \frac{4d(\beta-\theta)}{b-\alpha}\right)}{4\alpha}\right\}$.

   In the case that $\alpha > 0, R_0^P > \frac{1}{\theta}$ and $R_0^I > \frac{1}{\theta}$, every trajectory of System (8) with an initial condition taking in the interior of $\mathbb{R}_+^3$ converges to E_0, i.e.,

$$\lim_{t\to\infty} (S(t), I(t), P(t)) = E_0.$$

2. *System (8) has at most one interior equilibrium $E_2^i = (S_2^*, I_2^*, P_2^*)$. The existence of E_2^i requires*

$$\alpha < \frac{d(\beta-\theta)}{\mu-\theta} = \frac{d}{\frac{\mu-\theta}{\beta-\theta}}$$

provided that

$$\max\{\theta, \frac{1}{R_0^I}\} < S_2^* < \min\{1, \frac{1}{R_0^P}\} \text{ when } \alpha > 0, \text{ or }, 1 > S_2^* > \max\left\{\theta, \frac{1}{R_0^I}, \frac{1}{R_0^P}\right\} \text{ when } \alpha < 0.$$

In addition, the real parts of all eigenvalues of the Jacobian Matrix evaluated at E_2^i can never be all negative.

Proof. Direct applications of Theorem 4.1 imply that System (8) has no interior equilibrium if

$$\mathbf{R_0^I < 1} \text{ or } (\alpha < \mathbf{0}, \mathbf{R_0^P < 1}) \text{ or } (\alpha > \mathbf{0}, \mathbf{R_0^P} + \frac{\alpha(\mathbf{1}-\theta)}{\mathbf{d}} < \mathbf{1}).$$

Thus, we omit the detailed proof for these cases.

If (S^*, I^*, P^*) is an interior equilibrium for System (8), then S^* is a positive root of the quadratic equation

$$(S-\theta)\left(1 - S - \frac{b}{\alpha}\left(\frac{1}{R_0^P} - S\right)\right) - \beta\frac{b}{\alpha}\left(\frac{1}{R_0^P} - S\right) - \beta\left(S - \frac{1}{R_0^I}\right) = 0 \Leftrightarrow S^2 - BS + C = 0 \quad (15)$$

provided that

$$B = -(\beta-\theta) + \frac{\frac{d}{\alpha} - 1}{\frac{b}{\alpha} - 1}, \; C = \frac{\mu - \theta - \frac{d(\beta-\theta)}{\alpha}}{\frac{b}{\alpha} - 1}$$

and

$$P^* = \frac{\beta}{a}\left(S^* - \frac{1}{R_0^I}\right) > 0, \; I^* = \frac{b}{\alpha}\left(\frac{1}{R_0^P} - S^*\right) > 0. \quad (16)$$

The equation (16) implies that a necessary condition for the existence of the interior equilibrium (S^*, I^*, P^*) is as follows:

$$\frac{1}{R_0^I} < S^* < \frac{1}{R_0^P} \text{ if } \alpha > 0; \text{ while } S^* > \max\left\{\frac{1}{R_0^I}, \frac{1}{R_0^P}\right\} \text{ if } \alpha < 0.$$

In the case that $R_0^I = \frac{\mu}{\beta} = 1$, we have $S^* = \frac{d-\alpha}{b-\alpha}$, thus the interior equilibrium (S^*, I^*, P^*) exists if

$$1 < S^* < \frac{1}{R_0^P} \text{ if } \alpha > 0; \text{ while } S^* > \max\left\{1, \frac{1}{R_0^P}\right\} \text{ if } \alpha < 0.$$

This is a contradiction to $\limsup_{t\to\infty} S(t) \leq 1$ according to Theorem 2.1. This implies that there is no interior equilibrium if $R_0^I = 1$. Notice that Theorem 4.1 indicates that one necessary condition for System (8) having an interior equilibrium is that $R_0^I \geq 1$ otherwise $\lim_{t\to\infty} I(t) = 0$, thus, there is no interior equilibrium if

$$\mathbf{R_0^I \leq 1}.$$

Recall that Theorem 2.1 and Theorem 4.1 indicate that $\theta < S^* < 1$. Therefore, the existence of an interior equilibrium (S^*, I^*, P^*) requires $R_0^I > 1$ (i.e., $\mu < \beta$) and

$$\max\{\theta, \frac{1}{R_0^I}\} < S^* < \min\{1, \frac{1}{R_0^P}\} \text{ if } \alpha > 0; \text{ while } 1 > S^* > \max\left\{\theta, \frac{1}{R_0^I}, \frac{1}{R_0^P}\right\} \text{ if } \alpha < 0.$$

This implies that there is no interior equilibrium (S^*, I^*, P^*) if

$$\max\{\theta, \frac{1}{R_0^I}\} > \min\{1, \frac{1}{R_0^P}\} \text{ when } \alpha > 0$$

or

$$\max\left\{\theta, \frac{1}{R_0^I}, \frac{1}{R_0^P}\right\} \geq 1 \text{ when } \alpha < 0.$$

Therefore, there is no interior equilibrium if

$$(\mathbf{R_0^I \leq R_0^P}, \alpha > \mathbf{0}) \text{ or } (\mathbf{R_0^P \geq \frac{1}{\theta}}, \alpha > \mathbf{0}) \text{ or } (\mathbf{R_0^P \leq 1}, \alpha < \mathbf{0}).$$

Since we assume that System (8) satisfies Condition **H**, thus we have

$$0 < \theta < 1,\ \mu > \theta,\ 0 < b \leq a \text{ and } -\infty < \alpha < b.$$

The requirement $R_0^I > 1$ implies that $\theta < \mu < \beta$. The equation (15) has only one positive root $S_2^* = \frac{B+\sqrt{B^2-4C}}{2}$ if

$$C = \frac{\mu - \theta - \frac{d(\beta-\theta)}{\alpha}}{\frac{b}{\alpha} - 1} = \frac{\alpha(\mu-\theta) - d(\beta-\theta)}{b-\alpha} < 0 \Leftrightarrow 0 < \alpha < \frac{d(\beta-\theta)}{\mu-\theta} = \frac{d}{\frac{\mu-\theta}{\beta-\theta}}.$$

Therefore, System (8) has a unique interior equilibrium $E_2^i = (S_2^*, I_2^*, P_2^*)$ where $S_2^* = \frac{B+\sqrt{B^2-4C}}{2}$ if

$$\alpha < \frac{\mathbf{d}}{\frac{\mu-\theta}{\beta-\theta}},\ \max\{\theta, \mathbf{\frac{1}{R_0^I}}\} < \mathbf{S_2^*} < \min\{\mathbf{1}, \mathbf{\frac{1}{R_0^P}}\} \text{ when } \alpha > \mathbf{0},\ \text{ or, }\ \mathbf{1 > S_2^* > \max\left\{\theta, \frac{1}{R_0^I}, \frac{1}{R_0^P}\right\}} \text{ when } \alpha < \mathbf{0}.$$

In the case that $\alpha < 0$, it is easy to check that $C < 0$ since $\theta < \mu < \beta$ implies that $\alpha < \frac{d}{\frac{\mu-\theta}{\beta-\theta}}$ holds whenever $\alpha < 0$. Thus, it is impossible that (15) has two positive roots when $\alpha < 0$. If (15) has two positive roots

$$S_1^* = \frac{B - \sqrt{B^2-4C}}{2} < S_2^* = \frac{B + \sqrt{B^2-4C}}{2},$$

then it requires that $\alpha > 0$ and

$$\begin{aligned} B &> 0 \Leftrightarrow -(\beta-\theta) + \tfrac{d-\alpha}{b-\alpha} > 0 \Leftrightarrow 0 < \beta - \theta < \tfrac{d-\alpha}{b-\alpha} \text{ s.t. } \alpha < \min\{b, d\}, \\ C &> 0 \Leftrightarrow \tfrac{\alpha(\mu-\theta)-d(\beta-\theta)}{b-\alpha} > 0 \Leftrightarrow 0 < \beta - \theta < \tfrac{\alpha(\mu-\theta)}{d}, \\ B^2 &> 4C \Leftrightarrow (\beta-\theta)^2 + \left(\tfrac{d-\alpha}{b-\alpha}\right)^2 + \tfrac{2(\beta-\theta)(d+\alpha)-4\alpha(\mu-\theta)}{b-\alpha} > 0. \end{aligned}$$

Thus, $B > 0$ and $C > 0$ require that

$$0 < \beta - \theta < \frac{d-\alpha}{b-\alpha} \text{ s.t. } 0 < \alpha < \min\{b, d\} \text{ and } 0 < \beta - \theta < \frac{\alpha(\mu-\theta)}{d} < \mu - \theta$$

which is a contradiction since $0 < \mu - \theta < \beta - \theta$ and $b > \alpha$. Therefore, System (8) has at most one interior equilibrium E_2^i and System (8) has no interior equilibrium if $C > 0$ or $B^2 < 4C$ which implies follows:

1. $\mathbf{C > 0} \Leftrightarrow \alpha > \frac{\mathbf{d}}{\frac{\mu-\theta}{\beta-\theta}}$.

2. $\mathbf{B^2 - 4C < 0} \Leftrightarrow \mu < \theta + \frac{(\mathbf{b}-\alpha)\left(\left(\theta-\beta+\frac{\mathbf{d}-\alpha}{\mathbf{b}-\alpha}\right)^{\mathbf{2}}+\frac{\mathbf{4d}(\beta-\theta)}{\mathbf{b}-\alpha}\right)}{\mathbf{4}\alpha}$

The argument above implies that System (8) has at most one interior equilibrium E_2^i with $S_2^* = \frac{B+\sqrt{B^2-4C}}{2}$. From (14), we have

$$S_2^* > \frac{d-\alpha}{b-\alpha} \text{ if } \alpha > 0;\ S_2^* < \frac{d-\alpha}{b-\alpha} \text{ if } \alpha < 0$$

which implies that

$$S_2^* > \frac{d-\alpha}{b-\alpha} \geq 1 \text{ when } b > \alpha > 0, d \geq b (i.e., R_0^P \leq 1).$$

This is a contradiction to the fact that $\limsup_{t\to\infty} S(t) \leq 1$. Therefore, System (8) has no interior equilibrium if $R_0^P \leq 1, \alpha > 0$. Combining the discussions above, we can conclude that System (8) has no interior equilibrium if

$$\mathbf{R_0^P \leq 1}, \alpha < \mathbf{b}.$$

The existence of E_2^i requires

$$\max\{\theta, \frac{1}{R_0^I}\} < S_2^* < \min\{1, \frac{1}{R_0^P}\} \text{ if } \alpha > 0;\ \text{ while } 1 > S_2^* > \max\left\{\theta, \frac{1}{R_0^I}, \frac{1}{R_0^P}\right\} \text{ if } \alpha < 0$$

which implies that System (8) has no interior equilibrium if

$$\mathbf{S_2^*} < \frac{\mathbf{d}-\alpha}{\mathbf{b}-\alpha} < \max\left\{\theta, \frac{\mathbf{1}}{\mathbf{R_0^I}}, \frac{\mathbf{1}}{\mathbf{R_0^P}}\right\} = \max\left\{\theta, \frac{\mathbf{1}}{\mathbf{R_0^I}}\right\} \text{ when } \mathbf{R_0^P > 1}, \alpha < \mathbf{0}$$

since

$$\frac{1}{R_0^P} = \frac{d}{b} < \frac{d-\alpha}{b-\alpha} \text{ when } R_0^P > 1, \alpha < 0.$$

The above argument also implies that System (8) has no interior equilibrium if

$$\alpha > 0,\ R_0^P > \frac{1}{\theta} \text{ and } R_0^I > \frac{1}{\theta}$$

which implies that, according to Proposition 3.1, the only possible boundary equilibria for System (8) are E_0, E_θ and E_1 where only E_0 is locally asymptotically stable; E_θ is a source and E_1 is a saddle with one stable manifold on S-axis. This implies that all trajectories of System (8) that are not living on the stable manifold of E_1 converge to E_0.

The local stability of the interior equilibrium $E_2^i = \left(S_2^*, \frac{b}{\alpha}\left(\frac{1}{R_0^P} - S_2^*\right), \frac{\beta}{a}\left(S_2^* - \frac{1}{R_0^I}\right)\right)$ can be determined by the eigenvalues of the Jacobian Matrix of (8) evaluated at this equilibrium, i.e., $J_{E_2^i}$:

$$J_{E_2^i} = \begin{pmatrix} S_2^*\left(1+\theta-\frac{d}{\alpha}+(\frac{b}{\alpha}-2)S_2^*\right) & S_2^*\left(\theta-\beta-S_2^*\right) & -aS_2^* \\ \frac{\beta(d-bS_2^*)}{\alpha} & 0 & -\frac{a(d-bS_2^*)}{\alpha} \\ \frac{b(\beta S_2^*-\mu)}{a} & \frac{\alpha(\beta S_2^*-\mu)}{a} & 0 \end{pmatrix} \tag{17}$$

where its characteristic equation reads as follows:

$$\begin{aligned} &-\lambda^3 + \tfrac{S_2^*[\alpha(1+\theta)-d+(b-2\alpha)S_2^*]}{\alpha}\lambda^2 - \tfrac{\beta S_2^*(S_2^*-\theta+\beta)(d-bS_2^*)+\alpha d(\beta S_2^*-\mu)}{\alpha}\lambda \\ &+\tfrac{S_2^*(d-bS_2^*)(\beta S_2^*-\mu)[\alpha-d+(\beta-\theta+2S_2^*)(b-\alpha)]}{\alpha} = (\lambda_1-\lambda)(\lambda_2-\lambda)(\lambda_3-\lambda) = 0. \end{aligned} \tag{18}$$

with $\lambda_i, i = 1, 2, 3$ being roots of (18). If all real part of $\lambda_i, i = 1, 2, 3$ are negative, then we have

$$\sum_{i=1}^3 \lambda_i = \tfrac{S_2^*[\alpha(1+\theta)-d+(b-2\alpha)S_2^*]}{\alpha} < 0 \Leftrightarrow S_2^* \begin{cases} < \frac{d-\alpha(1+\theta)}{b-2\alpha} \text{ if } 0 < \alpha < \min\{b/2, d/(1+\theta)\} \\ > \frac{\alpha(1+\theta)-d}{2\alpha-b} \text{ if } \alpha > \max\{b/2, d/(1+\theta)\} \\ > \frac{d-\alpha(1+\theta)}{b-2\alpha} \text{ if } \alpha < 0 \end{cases}$$

$$\sum_{i,j=1,i\neq j}^{3}\lambda_i\lambda_j = \frac{\beta S_2^*(S_2^*-\theta+\beta)(d-bS_2^*)+\alpha d(\beta S_2^*-\mu)}{\alpha} = \beta S_2^*(S_2^*-\theta+\beta)\frac{(d-bS_2^*)}{\alpha}+d(\beta S_2^*-\mu)>0$$

$$\prod_{i=1}^{3}\lambda_i = \frac{d-bS_2^*}{\alpha}S_2^*(\beta S_2^*-\mu)\left[\alpha-d+(\beta-\theta+2S_2^*)(b-\alpha)\right]<0 \Leftrightarrow S_2^* < \frac{\frac{d-\alpha}{b-\alpha}-(\beta-\theta)}{2} = B/2$$

Notice that the existence of E_2^i requires $C<0$ (since it is impossible for (15) having two positive roots), thus, we have

$$S_2^* = \frac{B+\sqrt{B^2-4C}}{2} > B/2$$

which is a contradiction to the fact that all real part of $\lambda_i, i=1,2,3$ being negative requires $S_2^*<B/2$. Therefore, the real parts of eigenvalues of $J_{E_2^i}$ can never be all negative. □

Notes: Theorem 4.2 suggests that System (8) has at most one interior equilibrium which is always unstable (see Figure 4(b) as an example), thus the coexistence of S, I, P seems impossible. In fact, the existence of the unique interior equilibrium indicates the tri-stability of the system, i.e., any trajectory starting from the interior of $\mathbb{R}^3_+$ either converges to E_0 or SP-plane or SI-plane. For example, let $\beta=1.5;\mu=d=1;\theta=0.2;a=2;\alpha=0.5;b=1.35$, then we can obtain follows regarding System (8):

1. The locally asymptotically stable boundary equilibria:

$$E_0=(0,0,0),\ E_I^i=(0.66667,0.079096,0) \text{ and } E_P^i=(0.74074,0,0.070096).$$

2. The unique interior equilibrium $E_2^i=(0.7329,0.0211,0.0497)$ where the eigenvalues of $J_{E_2^i}$ are

$$\lambda_1=-0.1146+0.3713i,\ \lambda_2=-0.1146-0.3713i \text{ and } \lambda_3=0.018896>0.$$

3. System (8) has only three attractors E_0, E_I^i and E_P^i where their basins of attractions are presented in Figure 5(b): The white regions are the basins of attraction of E_0; the blue regions are the basins of attraction of E_I^i; and the green regions are the basins of attraction of E_P^i.

In addition, the second part of Theorem 4.2 implies that the full SIP system has only one attractor E_0 when its subsystem (9) has predation-driven extinction and its subsystem (10) has disease-driven extinction in the case that $\alpha>0$, i.e., $R_0^k>\frac{1}{\theta}, k=P,I$.

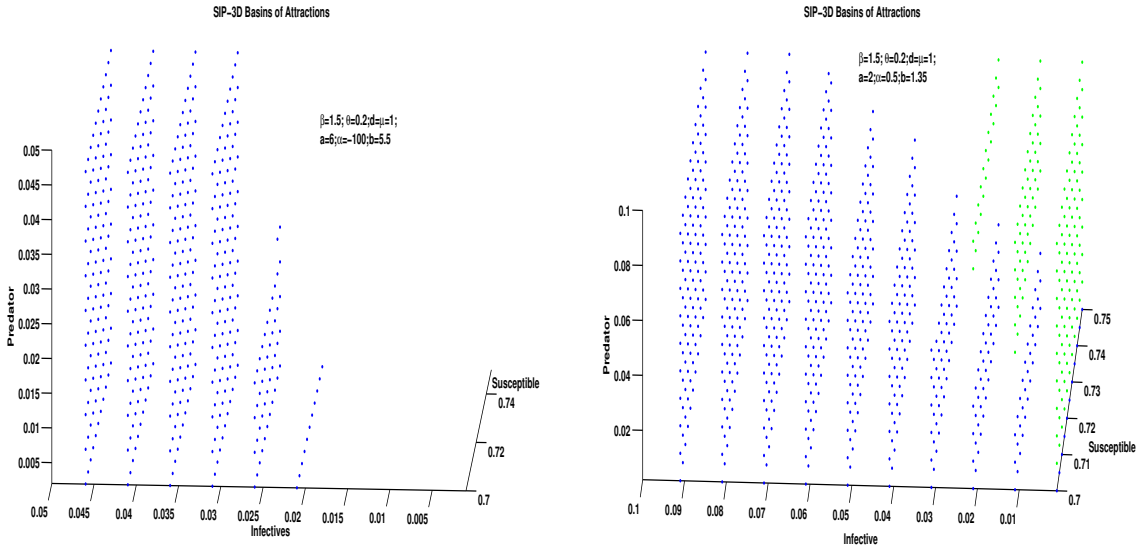
Based on our analysis and numerical simulations, the predator-prey system (8) with prey subject to Allee effects and disease can have one (i.e., extinction of all species), two (i.e., competition exclusion or bi-stability) or three (i.e., tri-stability) attractors but can never have the coexistence of S, I, P-populations. We summarize the **global dynamical features** of System (8) as follows (also see Table 5):

1. **The importance of initial conditions**: From Theorem 4.1, we know that if $S(0)<\theta$, then the trajectory converges to E_0, i.e., the extinction of S, I, P occurs. In addition, when System (8) exhibits bi-stability or tri-stability (see below), different initial conditions may lead to different attractors.

2. **The extinction state** E_0 is always an attractor due to Allee effects in prey according to Proposition 4.1. In addition, Theorem 4.1 and Theorem 4.2 implies that E_0 is a global attractor if $(R_0^I\leq 1, R_0^P>1/\theta)$ or $(\alpha<0, R_0^P\leq 1, R_0^I>1/\theta)$ or $(\alpha>0, R_0^P+\frac{\alpha(1-\theta)}{d}\leq 1, R_0^I>1/\theta)$ or $(\alpha>0, R_0^P>1/\theta, R_0^I>1/\theta)$.

3. The **bi-stability** occurs in the absence of an interior equilibrium in the following two cases:

   (a) **Only susceptible prey is able to survive:** According to Theorem 4.1, this occurs when both the reproduction number of disease and predator are small, i.e., both $R_0^I\leq 1$ and $R_0^P\leq 1$.



(a) Basins of Attractions of E_0 and E_I^i

(b) Basins of Attractions of E_0, E_P^i and E_I^i

Figure 5: The left graph is presenting the basins of attractions of E_0 (white regions) and E_I^i (blue regions) when $\beta = 1.5; \theta = 0.2; d = \mu = 1; \alpha = -100; b = 5.5 < a = 6$ ($R_0^P > \frac{1}{\theta}$, $R_0^I = 1.5$) and $S(0) \in [0.70, 0.75], I(0) = [0.001, 0.05], P(0) = [0.002, 0.05]$; The right graph is presenting the basins of attractions of E_0 (white regions),E_P^i (green regions) and E_I^i (blue regions) when $\beta = 1.5; \theta = 0.2; d = \mu = 1; a = 2; \alpha = 0.5; b = 1.35$ ($R_0^P = 1.35$, $R_0^I = 1.5$) and $S(0) \in [0.70, 0.75], I(0) = [0.001, 0.1], P(0) = [0.002, 0.1]$.

(b) **Competition exclusion**: In this case System (8) has two attractors: one is E_0 and the other one is either in SP-plane or in SI-plane which can be a locally asymptotically stable boundary equilibrium E_I^i (or E_P^i if in SP-plane) or the unique stable limit cycle around E_I^i (or around E_P^i if in SP-plane). See Figure 5(a) as an example.

4. The **tri-stability** in the presence of the unique interior equilibrium: Theorem 4.2 indicates that System (8) can have at most one interior equilibrium which is always unstable; thus (8) has no coexistence of S, I, P-populations. In this case, (8) has three attractors: one is E_0, the second one is a locally asymptotically stable boundary equilibrium E_I^i or the unique stable limit cycle around E_I^i that locates in SI-plane and the third one is a locally asymptotically stable boundary equilibrium E_P^i or the unique stable limit cycle around E_P^i that locates in SP-plane (see Figure 5(b) as an example).

5. The **effects of disease & predation-driven extinction**: Theorem 4.1 and Theorem 4.2 indicate that all populations go extinction if $(R_0^I \leq 1, R_0^P > 1/\theta)$ or $(\alpha < 0, R_0^P \leq 1, R_0^I > 1/\theta)$ or $(\alpha > 0, R_0^P + \frac{\alpha(1-\theta)}{d} \leq 1, R_0^I > 1/\theta)$ or $\alpha > 0, R_0^P > 1/\theta, R_0^I > 1/\theta)$. In addition, there is no interior equilibrium if $(\alpha > 0, R_0^P > 1/\theta)$ or $(\alpha < 0, \frac{d-\alpha}{b-\alpha} < \max\{\theta, \frac{1}{R_0^I}\})$. The interesting question is that what population dynamics of System (8) in the following two cases:

   (a) $\alpha < \mathbf{0}, \mathbf{R_0^P} > \frac{\mathbf{1}}{\theta}$ and $\mathbf{1} < \mathbf{R_0^I} < \frac{\mathbf{1}}{\theta}$: In this case, competition exclusion occurs, i.e., only S and I-class are able to coexist while P-class goes extinction. In fact, E_I^i can be locally asymptotically stable if $\alpha < 0$ and $|\alpha|$ large enough such that the following condition satisfied (from Proposition 4.1)

$$1 < R_0^I < \frac{\beta - \theta + \sqrt{\beta^2 - \beta\theta + \beta}}{\beta + \beta\theta - \theta^2} \text{ and } \frac{1}{\theta} < R_0^P < R_0^I \left(1 - \frac{\alpha\left(\frac{1}{R_0^I} - \theta\right)\left(1 - \frac{1}{R_0^I}\right)}{d(\frac{1}{R_0^I} + \beta - \theta)}\right).$$

   For example, let $\beta = 1.5; \mu = d = 1; \theta = 0.2; \alpha = -100; b = 5.5 < a = 6$, then we can obtain follows regarding System (8):

   i. The locally asymptotically stable nontrivial boundary equilibria: $E_I^i = (0.66667, 0.079096, 0)$

   ii. The unique interior equilibrium $E_2^i = (0.7231, 0.0298, 0.014104)$ where the eigenvalues of $J_{E_2^i}$ are

$$\lambda_1 = -0.38780 + 0.66810i,\ \lambda_2 = -0.38780 - 0.66810i,\ \text{and } \lambda_3 = 0.57608.$$

   iii. System (8) has only two attractors E_0 and E_I^i where their basins of attractions are presented in Figure 5(a): The white regions are the basins of attraction of E_0 and the blue regions are the basins of attraction of E_I^i.

   (b) $\mathbf{1} < \mathbf{R_0^P} < \frac{\mathbf{1}}{\theta} < \mathbf{R_0^I}$: According to Proposition 4.1, E_P^i cannot be locally asymptotically stable since it requires

$$R_0^I < R_0^P \left(1 + \frac{\left(\frac{1}{R_0^P} - \theta\right)\left(1 - \frac{1}{R_0^P}\right)}{\mu}\right).$$

   Let $F(R_0^P) = R_0^P \left(1 + \frac{\left(\frac{1}{R_0^P} - \theta\right)\left(1 - \frac{1}{R_0^P}\right)}{\mu}\right), 1 < R_0^P < \frac{1}{\theta}$, then we have

$$\max_{1 \leq R_0^P \leq \frac{1}{\theta}} \{F(R_0^P)\} = F(\frac{1}{\theta}) = \frac{1}{\theta} \text{ since } F'(R_0^P) = \frac{1 + (R_0^P)^2(\mu - \theta)}{\mu(R_0^P)^2} > 0.$$

However, we have $\frac{1}{\theta} < R_0^I$, thus it is impossible that $R_0^I < F(R_0^P)$ holds. Numerical simulations suggest that System (8) has global stability at E_0 and there is a orbit connecting E_P^i to E_0.

6. The parameter a does not affect the existence and local stability of E_P^i, E_I^i and the unique interior equilibrium E_2^i.

Attractor(s)	Sufficient Condition	Biological Implications
E_0	From Theorem 4.1: **1.** $R_0^I \leq 1$, $R_0^P > \frac{1}{\theta}$; **2.** $\alpha < 0, R_0^P \leq 1$, $R_0^I > \frac{1}{\theta}$; **3.** $0 < \alpha < b, R_0^P + \frac{\alpha(1-\theta)}{d} \leq 1$, $R_0^I > \frac{1}{\theta}$; From Theorem 4.2: **4.** $\alpha > 0, R_0^P > \frac{1}{\theta}$, $R_0^I > \frac{1}{\theta}$; From Simulations: **5.** $1 < R_0^P < \frac{1}{\theta} < R_0^I$.	No interior equilibrium & No E_P^i, E_I^i; Predation/Disease-Driven extinction combined with the low reproduction value leads to the extinction of all species.
$E_0 \cup E_1$	From Theorem 4.1: $R_0^P \leq 1$ and $R_0^I \leq 1$	No interior equilibrium & No E_P^i, E_I^i; Low reproduction values of disease and predation makes susceptible prey be the only possible survivor.
$E_0 \cup E_P^i$	From Proposition 4.1 and Theorem 4.1 combined with simulations: $1 < R_0^P < \frac{2}{1+\theta}, \frac{R_0^I}{R_0^P} < 1 + \frac{\left(\frac{1}{R_0^P}-\theta\right)\left(1-\frac{1}{R_0^P}\right)}{\mu}$	**1. Competition exclusion**: No interior equilibrium; E_I^i exists; Predator wins and disease free; **2. Predation can not save prey from disease-induced extinction**: No interior equilibrium; Predator is the inferior competitor
$E_0 \cup E_I^i$	From Proposition 4.1 and Theorem 4.1-4.2 combined with simulations: $1 < R_0^I < \frac{\beta-\theta+\sqrt{\beta^2-\beta\theta+\beta}}{\beta+\beta\theta-\theta^2}$, $\frac{R_0^P}{R_0^I} < 1 - \frac{\alpha\left(\frac{1}{R_0^I}-\theta\right)\left(1-\frac{1}{R_0^I}\right)}{d(\frac{1}{R_0^I}+\beta-\theta)}$ and { **1.** $R_0^P \leq 1$; or **2.** $R_0^P > \frac{1}{\theta}$}	**1. Competition exclusion**: No interior equilibrium; E_P^i exists; Predator wins and disease free; **2. Disease can save prey from predation-induced extinction**: the unique interior equilibrium exists, no E_P^i, disease is the superior competitor.
$E_0 \cup E_I^i \cup E_i^P$	From Proposition 4.1: $1 < R_0^P < \frac{2}{1+\theta}, \frac{R_0^I}{R_0^P} < 1 + \frac{\left(\frac{1}{R_0^P}-\theta\right)\left(1-\frac{1}{R_0^P}\right)}{\mu}$ and $1 < R_0^I < \frac{\beta-\theta+\sqrt{\beta^2-\beta\theta+\beta}}{\beta+\beta\theta-\theta^2}$, $\frac{R_0^P}{R_0^I} < 1 - \frac{\alpha\left(\frac{1}{R_0^I}-\theta\right)\left(1-\frac{1}{R_0^I}\right)}{d(\frac{1}{R_0^I}+\beta-\theta)}$	**Tri-stability:** Unique **unstable** interior equilibrium; Has both E_I^i and E_I^i; Different initial conditions lead to predator wins or disease wins

Table 5: From the analysis of the stability of equilibria and numerical simulations, sufficient condition for the global attractors for System (8) as well as its corresponding biological implications

5. The impact of Allee effects, disease and predation

First, we would like to explore the impact of Allee effects by comparing the dynamics of (8) to the following model without Allee effects in prey:

$$\begin{array}{rcl} \frac{dS}{dt} & = & S\left(1-S-I\right)-\beta SI-aSP=S\left[1-S-(1+\beta)I-aP\right]=Sg_1(S,I,P), \\ \frac{dI}{dt} & = & \beta SI-aIP-\mu I=I\left[\beta S-aP-\mu\right]=If_2(S,I,P), \\ \frac{dP}{dt} & = & a\left(cS+\gamma I\right)P-dP=P\left[bS+\alpha I-d\right]=Pf_3(S,I,P) \end{array} \tag{19}$$

where the biological meaning of all parameters are listed in Table 1. The SIP-model without Allee effects (19) has the following boundary equilibria: $E_0^{na}=(0,0,0)$, $E_1^{na}=(1,0,0)$ and

$$\begin{array}{rcl} (E_P^i)^{na} & = & \left(\frac{1}{R_0^P},0,\frac{R_0^P-1}{aR_0^P}\right)=\left(\frac{d}{b},0,\frac{b-d}{ab}\right), \\ (E_I^i)^{na} & = & \left(\frac{1}{R_0^I},\frac{R_0^I-1}{(1+\beta)R_0^I},0\right)=\left(\frac{\mu}{\beta},\frac{\beta-\mu}{\beta(1+\beta)},0\right) \end{array} \tag{20}$$

as well as the unique interior equilibrium

$$\begin{array}{rcl} (E_i)^{na} & = & \left(\frac{bR_0^I(1+\beta)-\alpha R_0^P(R_0^I+\beta)}{R_0^PR_0^I(1+\beta)(b-\alpha)},\frac{b\left[R_0^P(\beta+R_0^I)-R_0^I(1+\beta)\right]}{R_0^PR_0^I(1+\beta)(b-\alpha)},\frac{\beta\left[b(1+\beta)(R_0^I-R_0^P)-\alpha R_0^P(R_0^I-1)\right]}{aR_0^PR_0^I(1+\beta)(b-\alpha)}\right) \\ & = & \left(\frac{d(1+\beta)-\alpha(1+\mu)}{(1+\beta)(b-\alpha)},\frac{b(1+\mu)-d(1+\beta)}{(1+\beta)(b-\alpha)},\frac{(1+\beta)(d\beta-b\mu)-\alpha(\beta-\mu)}{a(1+\beta)(b-\alpha)}\right)=(S^*,I^*,P^*). \end{array} \tag{21}$$

Therefore, $(E_P^i)^{na}$ exists if and only if $R_0^P>1$ while $(E_I^i)^{na}$ exists if and only if $R_0^I>1$. In addition, we can conclude that $(E_i)^{na}$ exists if and only if the following inequalities hold

$$\begin{array}{rcl} \frac{d(1+\beta)-\alpha(1+\mu)}{(1+\beta)(b-\alpha)} & > & 0\Leftrightarrow d(1+\beta)-\alpha(1+\mu)>0\Leftrightarrow\frac{1+\beta}{1+\mu}>\frac{\alpha}{d}, \\ \frac{b(1+\mu)-d(1+\beta)}{(1+\beta)(b-\alpha)} & > & 0\Leftrightarrow b(1+\mu)-d(1+\beta)>0\Leftrightarrow\frac{1+\beta}{1+\mu}<\frac{b}{d}\Leftrightarrow d\beta-b\mu<b-d, \\ \frac{(1+\beta)(d\beta-b\mu)-\alpha(\beta-\mu)}{a(1+\beta)(b-\alpha)} & > & 0\Leftrightarrow(1+\beta)(d\beta-b\mu)-\alpha(\beta-\mu)>0\Leftrightarrow d\beta-b\mu>\frac{\alpha(\beta-\mu)}{1+\beta}. \end{array} \tag{22}$$

since we assume that $b>\alpha$ holds for (19) (e.g., predator hunts less infective prey than healthy prey and may even be harmed by infective prey due to the disease). Now we summarize main global dynamics of Model (19) as the following theorem:

Theorem 5.1. *[Dynamics of SIP-model without Allee effects]Assume that $a>b>\alpha$. Then the following statements hold:*

1. *Model (19) is positively invariant in X and bounded by $[0,1]\times[0,1]\times[0,\frac{b}{a\min\{1,d\}}]$with the following property*
$$\limsup_{t\to\infty}S(t)+I(t)\leq 1.$$

2. *If $R_0^I\leq 1$, then the infective population of Model (19) goes extinct. In addition, if $R_0^P\leq 1$, then Model (19) has global stability at E_1^{na}; while if $R_0^P>1$ instead, then Model (19) has global stability at $(E_P^i)^{na}$. Similarly, the prey population of Model (19) goes extinct if*
$$(R_0^P\leq 1,\alpha\leq 0)\ \text{or}\ (R_0^P\leq 1-\frac{\alpha}{d},\alpha>0).$$
In addition, Model (19) has global stability at $(E_I^i)^{na}$ if
$$(\alpha>0,\ R_0^P\leq 1-\frac{\alpha}{d},\ R_0^I>1)\ \text{or}\ (\alpha\leq 0,\ R_0^P\leq 1,\ R_0^I>1).$$

3. *The existence of the unique interior equilibrium $(E_i)^{na}$ requires $R_0^I>1, R_0^P>1$ and both nontrivial boundary equilibria $(E_P^i)^{na}$ and $(E_I^i)^{na}$ are locally asymptotically stable. In addition, the interior equilibrium $(E_i)^{na}$ is always unstable.*

4. *If* $(E_P^i)^{na}$ *is unstable, then* $(E_I^i)^{na}$ *exists and is stable; while if* $(E_I^i)^{na}$ *is unstable, then* $(E_P^i)^{na}$ *exists and is stable.*

Proof. It is easy to check that (19) is positively invariant in $\mathbb{R}^3_+$ since $S = 0, P = 0, I = 0$ are invariant manifolds, respectively. For any initial conditions taken in $\mathbb{R}^3_+$, we have

$$\frac{dS}{dt} \leq S(1-S) \Rightarrow \limsup_{t\to\infty} S(t) \leq 1.$$

Thus, for any $\epsilon > 0$, then there exists some time T large enough such that

$$\frac{dS}{dt} + \frac{dI}{dt} \leq (1+\epsilon)(1-S-I) \text{ for all } t > T \Rightarrow \limsup_{t\to\infty} S(t) + I(t) \leq 1.$$

Now define $V = \frac{b(S+I)}{a} + P$, then we have

$$\frac{dV}{dt} \leq \frac{bS(1-S-I)}{a} - bIP - \frac{b\mu}{a}I + \alpha PI - dP \leq b/a - b/a(S+I) - dP \Rightarrow \limsup_{t\to\infty} V(t) \leq \frac{b}{a\min\{1,d\}}.$$

This indicates that $\limsup_{t\to\infty} V(t) \leq \frac{b}{a\min\{1,d\}}$. Thus, the first statement of Theorem 5.1 holds.

From the positive invariant property of (19), we have follows

$$\begin{array}{rcl} \frac{dS}{dt} & = & S\left(1-S-I\right) - \beta SI - aSP \leq S\left[1-S-(1+\beta)I\right], \\ \frac{dI}{dt} & = & \beta SI - aIP - \mu I \leq \left[\beta S - \mu\right]. \end{array}$$

Thus the infective population of (19) is always less or equal to (if $P = 0$) the infective population of the following dynamics:

$$\begin{array}{rcl} \frac{dS}{dt} & = & S\left[1-S-(1+\beta)I\right] \\ \frac{dI}{dt} & = & \left[\beta S - \mu\right] \end{array}$$

which is the well-known Lotka-Volterra prey predator system that has $\lim_{t\to\infty} I(t) = 0$ if $R_0^I \leq 1$ (see the detailed proof in Kang and Wedekin [50]). Therefore, the infective population of (19) goes extinct if $R_0^I \leq 1$. This implies that the limiting system of (19) is the well-known Lotka-Volterra prey predator system again:

$$\begin{array}{rcl} \frac{dS}{dt} & = & S\left[1-S-aP\right] \\ \frac{dP}{dt} & = & P\left[bS-d\right] \end{array}$$

which has global stability at $(1,0)$ when $R_0^P \leq 1$ and has global stability at $\left(\frac{1}{R_0^P}, \frac{R_0^P-1}{aR_0^P}\right)$ when $R_0^P > 1$ by using the local stability of boundary equilibria, Poincaré-Bendixson Theorem and Dulac's criterion [31]. The detailed proof can be found in Kang and Wedekin [50]. Similarly, we can prove the dynamical properties of (19) when

$$(R_0^P \leq 1, \alpha \leq 0) \text{ or } (R_0^P \leq 1 - \frac{\alpha}{d}, \alpha > 0).$$

Thus, the second part of of Theorem 5.1 holds.

The argument above indicates that one necessary condition for (19) having an interior equilibrium $(E_i)^{na} = (S^*, I^*, P^*)$ (see the detailed expression of $(E_i)^{na}$ in (21)) is that $R_0^I > 1$, i.e., $\beta > \mu$. Thus from (22), we can conclude that $(E_i)^{na}$ exists if and only if

$$d\beta - b\mu > \frac{\alpha\left(\beta-\mu\right)}{1+\beta} \text{ and } \max\{1, \frac{\alpha}{d}\} < \frac{1+\beta}{1+\mu} < \frac{b}{d} \text{ since } \beta > \mu, b > \alpha.$$

Now if $R_0^P \leq 1$, i.e., $d \leq b$, then we have $1 < \frac{1+\beta}{1+\mu} < \frac{b}{d} \leq 1$ which is impossible. Thus, the existence of $(E_i)^{na}$ requires

$$R_0^I > 1 \text{ and } R_0^P > 1$$

which indicates the existence of $(E_I^i)^{na}$ and $(E_P^i)^{na}$.

Notice that $(E_I^i)^{na}$ is globally stable in the SI-plane (i.e., $P = 0$) and $(E_P^i)^{na}$ is globally stable in the SP-plane (i.e., $I = 0$), therefore, the locally stability of $(E_I^i)^{na}$ and $(E_P^i)^{na}$ is determined by the signs of $\frac{dP}{dt}\big|_{(E_I^i)^{na}}, \frac{dI}{dt}\big|_{(E_P^i)^{na}}$, respectively, i.e.,

$$\frac{dP}{dt}\Big|_{(E_I^i)^{na}} = -\frac{aP^*(1+\beta)(b-\alpha)}{\beta}, \qquad \frac{dI}{dt}\Big|_{(E_P^i)^{na}} = -\frac{I^*(1+\beta)(b-\alpha)}{b}. \tag{23}$$

This implies that if we have $(E_i)^{na} = (S^*, I^*, P^*) \in int\mathbb{R}^3_+$, then $\frac{dP}{dt}\big|_{(E_I^i)^{na}} < 0$ and $\frac{dI}{dt}\big|_{(E_P^i)^{na}} < 0$, thus, both $(E_I^i)^{na}$ and $(E_P^i)^{na}$ are locally asymptotically stable whenever $(E_P^i)^{na}$ exists. Therefore, the existence of $(E_i)^{na}$ requires $R_0^I > 1, R_0^P > 1$ and both $(E_I^i)^{na}$ and $(E_P^i)^{na}$ being locally asymptotically stable. If one of $(E_I^i)^{na}$ and $(E_P^i)^{na}$ is unstable, then there is no interior equilibrium, thus, (19) can never be permanent due to Schauder fixed point theorem [see Theorem 6.3 by Hutson and Schmitt [41]] when $b > \alpha$.

Let $J_{(E_i)^{na}}$ be the Jacobian matrix of System (19) evaluated at $(E_i)^{na} = (S^*, I^*, P^*)$, then by simple calculations, we can obtain that

$$det(J_{(E_i)^{na}}) = aS^*I^*P^*\,(1+\beta)\,(b-\alpha) > 0.$$

Therefore, $(E_i)^{na}$ is always unstable whenever it exists.

Assume that $(E_P^i)^{na}$ is unstable, then from (21), (22) and (23), we have the following inequalities hold

$$b > d \text{ and } d\beta - b\mu = d\mu\left(R_0^I - R_0^P\right) \geq b - d > 0.$$

Therefore, we have $R_0^I > R_0^P > 1$, i.e., $\beta > \mu$, thus $(E_I^i)^{na}$ exists. Then $(E_I^i)^{na}$ has to be stable, otherwise, (19) is permanent which is impossible. Therefore, if $(E_P^i)^{na}$ is unstable, then $(E_I^i)^{na}$ exists and is stable.

Assume that $(E_I^i)^{na}$ is unstable, then from (21), (22) and (23), we have the following inequalities hold

$$\beta > \mu \text{ and } d\beta - b\mu = d\mu\left(R_0^I - R_0^P\right) \leq \frac{\alpha(\beta-\mu)}{1+\beta}.$$

If $d \geq b$, i.e., $R_0^P \leq 1$, then we have

$$\alpha < b \leq d \Rightarrow d\beta - b\mu \geq b(\beta-\mu) > \frac{\alpha(\beta-\mu)}{1+\beta}$$

which is impossible. Thus, we have $R_0^P > 1$ which implies that $(E_P^i)^{na}$ exists and is stable.

□

Notes: Theorem 5.1 suggests that it may be impossible for System (19) to have the coexistence of S, I, P-population under the assumption that $b > \mu$ since the permanence of (19) may occur only if

$$\alpha > b > 0 \text{ and } R_0^I > R_0^P.$$

In addition, Theorem 5.1 and numerical simulations suggests that the dynamics of System (19) with $b > \alpha$ can be classified into the following three cases:

1. **Only S-population persists:** This occurs only if both $R_0^P \leq 1$ and $R_0^I \leq 1$.
2. **Competition exclusion:** either S and P persist or S and I persist. This occurs if

$$R_0^P > 1,\ R_0^I > 1,\ \text{and } d\mu\left(R_0^I - R_0^P\right) > b - d > 0 \Rightarrow \text{ S and P persist}$$

or

$$R_0^P > 1,\ R_0^I > 1,\ \text{and } d\mu\left(R_0^I - R_0^P\right) < \frac{\alpha(\beta-\mu)}{1+\beta} \Rightarrow \text{ S and I persist.}$$

3. **Bi-stability:** This occurs when both $(E_I^i)^{na}$ and $(E_P^i)^{na}$ are locally asymptotically stable, i.e.,

$$R_0^P > 1,\ R_0^I > 1,\ \text{and}\ \frac{\alpha(\beta-\mu)}{1+\beta} < d\mu\left(R_0^I - R_0^P\right) < b - d.$$

Depending on initial conditions, the trajectories may converge to $(E_I^i)^{na}$ or $(E_P^i)^{na}$.

By comparing the population dynamics of System (8) (with Allee effects in prey) to the population dynamics of (19) (no Allee effects in prey), we are able to obtain the following conclusion:

1. **The impacts of Allee effects in the full SIP model:** Not surprisingly, *Allee effects* make the system prone to extinction and initial conditions playing an extreme important role in the surviving of S health prey, or the surviving of I, P when System (8) has tri-stability. In addition, System (8) has more complicated disease-free or predator-free dynamics (e.g., limit cycle, heteroclinic orbit, disease/predation-driven extinction) than (19) does due to the nonlinearity introduced by Allee effects.

2. **The impacts of disease and predation:** Notice that both System (8) (with Allee effects in prey) and System (19) (no Allee effects in prey) can not have the coexistence of S, I, P-population. This interesting phenomenon is due to the assumption $b > \alpha$, i.e., predator cannot distinguish the infected and healthy prey but the consumption of the infected prey has less or even harm the growth of predator. The proofs of our analytical results imply that the coexistence of all S, I, P-population is possible only if $b < \alpha$, i.e., predator can have more benefits in the capture and consumption of the infected prey than the healthy prey. In fact, if $b > \alpha$, then under certain values of parameters, both System (8) and (19) can exhibit the locally asymptotically interior equilibrium or stable interior limit cycle (see the coexistence condition and its related numerical simulations in Hethcote et al. [35], Singh et al. [64]).

3. **The impacts of Allee effects, disease and predation:** In the presence of Allee effects and predation-driven extinction (i.e., $R_0^P > \frac{1}{\theta}$) in the subsystem (9) of System (8), disease may be able to save the predation-driven extinction and have the coexistence of both S and I. However, predation can not save the disease-driven extinction (i.e., $R_0^I > \frac{1}{\theta}$). This suggests that disease may be the superior competitor and predator is the inferior competitor.

6. Discussion

Mathematical modeling has been a great tool for understanding species' interactions as well as the disease dynamics, which allow us to obtain useful biological insights and enable us to make correct policies to maintain the diversity in nature. Many mathematical models have been used to understand the impacts of Allee effects on species' abundance and persistence [24, 70, 9, 49, 51] especially in the presence of disease [38, 79, 37, 71, 47]. Recently, there is significant research on eco-epidemiological models [26, 13, 15, 35, 37, 5, 68] that incorporate both the interactions of species and disease since the first work introduced by Hadeler and Freedman [33]. For example, recently Bairagi et al. [5] studied the role of infection on the stability of predator-prey systems with different response functions. In this article, we propose a general predator-prey model with prey subject to Allee effects and disease. There are three unique features of our assumptions: (a) Disease has no vertical transmission but it is untreatable and causes additional mortality in infected prey; (b) Allee effects built in the reproduction of health prey while infected prey has no reproduction; (c) Predator captures health and infected prey at the same rate but the consumption of infected prey has less benefits or even causes harm to predator. These assumptions contribute great impacts on the dynamical outcomes of the proposed model. To explore how interplay among Allee effects, disease and predation affect species' abundance and persistence, we focus on a concrete system with additional two assumptions: (d) disease transmission follows the law of mass action; (e) prey and predator have Holling-Type 1 functional responses. In a nutshell, we summarize our main findings as well as their related biological implications as follows:

1. Based on assumptions **(a), (b), (c)**, we propose a general model described by nonlinear equations (5) whose schematic presentation is showned in Figure 1. Model (5) is general enough to cover all common scenarios: i) prey and predator can have Holling-Type I or II or III; ii) the disease transmission can be density-dependent or frequency-dependent, whose basic dynamical properties have been given in Theorem 2.1. Theorem 2.1 and its corollary 2.1 indicate that Allee effects in prey make initial conditions being extremely important for the persistence of prey as well predator, which partially answers the first question listed in the introduction regarding the impact of Allee effects.

2. Proposition 3.1 and Theorem 3.1 combined with numerical simulations [see Figure 2, 3] provide us a full picture on the dynamics of of the concrete model (8) when it's disease-free or predation-free: these subsystems have very complicated features due to the nonlinearity introduced by Allee effects. By comparing to their corresponding models without Allee effects, we can conclude that Allee effects can destablize systems and make the system prone to extinction through disease/predation-driven extinction or small initial conditions. These results not only provide us an access to investigate the full system but also partially answer the first question listed in the introduction.

3. Proposition 4.1 and Theorem 4.1 combined with numerical simulations [see Figure 4] indicates that the full system can have the extinction of all species (caused by the combinations of the low reproduction number, disease and predation-driven extinctions), bistability (caused by the low reproduction numbers of both disease and predator, competition exclusions or disease/predation-driven extinctions) and tri-stability. One of our most interesting findings is that disease may be able to save prey from predation-driven extinction and leads to the coexistence of S and I-class while predation cannot save the disease-driven extinction. These answer the last two questions listed in the introduction regarding how the interplay among Allee effects, disease and predation may promote species' persistence. In addition, Theorem4.2 and numerical simulation [see Figure 5] suggests that there is no coexistence of health prey, infected prey and predator. This answers the second question listed in the introduction regarding the possibility of coexistence.

4. Theorem 5.1 gives us the global picture of the dynamics of the SIP model without Allee effects. The comparison study between the concrete SIP model with its corresponding model without Allee effects implies that **no coexistence of S, I, P-population** is not caused by Allee effects but it is caused by our assumption **(c)**: predation on infected prey has less or negative contribution to the growth rate of predator, i.e., $b > \alpha > -\infty$. The biological explanation for this is that I and P-class are at exploitative competition for S-class whereas I-class cannot be superior and P-class cannot gain significantly from its consumption of I-class. Further more, our analysis and simulations show that the coexistence of S, I, P-class occurs only if $b < \alpha$ and the interior attractors can be very complicated, e.g., limit cycles. This result complement the previous study on SIP systems without Allee effects but with assumption that predator may gain more benefits from hunting weak/sick prey, which may promote prey surviving and avoid the disease-driven extinctions [see more discussions in [35]].

6.1. Potential future work

Transmission of disease is influenced by aggregation patterns in the host population as well as its social organization. Two different types of incidence rate (new infections per unit time) are usually distinguished [6, 34, 55, 58]: density-dependent transmission (also called mass action transmission) is the case when contact rate between susceptible and infective individuals increases linearly with population size; while frequency-dependent transmission (also called standard incidence or proportionate mixing) is the case when number of contacts is independent of population size. We focused on a concrete example when disease has density-dependent transmission in this article. It will be interesting to explore how frequency-dependent incidence rate may generate different dynamics in the presence of Allee effects and predation in the future.

Holling-Type I functional response in predator-prey interaction occurs when predator's handling time can be ignored, which has the form $h(N) = aN$ with a being the attack rate of predator and N being the prey density. This functional responses implies that there is no upper limit to the prey consumption rate and satiation of the predator. While Holling-Type II or III functional response has predator satiation at the high density of prey [40]: Holling-Type II represents an asymptotic curve that decelerates constantly as prey number increases, e.g., $h(N) = \frac{aN}{k+N}$ with k being the half-saturation constant, while Holling-Type III functional response is sigmoidal, rising slowly when prey are rare, accelerating when more abundant and last reaching a saturated upper limit, e.g., $h(N) = \frac{aN^2}{k^2+N^2}$, which is suitable to describe predation when switching prey and learning ability are more common to predator [59]. The predation satiation property of both Holling-Type II or III functional responses can be mechanisms of generating Allee effects in prey [28]. It will be interesting to explore how double Allee effects may arise from predation satiation and Allee effects built in the reproduction of prey, and thus, may produce different dynamical outcomes.

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Appendix A- an approach of Model (4)

In the absence of disease and predation, we assume that the population dynamic of prey can be described by the following generic single species population model with an Allee effect:

$$\begin{aligned}\frac{dS}{dt} &= \underbrace{r(1+\theta)S^2\left(1-\frac{S}{1+\theta}\right)}_{\text{combined reproduction effort due to mating limitations (i.e., Allee effects) and limited resource}} - \underbrace{r\theta S}_{\text{natural mortality}} \\ &= rS(S-\theta)(1-S)\end{aligned} \tag{24}$$

where S denotes the normalized susceptible prey population; the parameter r denotes the maximum birth-rate of species, which can be scaled to be 1 by altering the time scale; the parameter $0 < \theta < 1$ denotes the Allee threshold (normalized susceptible population). This approach assumes that the susceptible prey population has a constant mortality rate $r\theta$. This property is inherited by the infected prey I-class, thus in the presence of disease, I-class has a constant mortality $\mu_d + r\theta$ which is a sum of the natural mortality and the additional mortality due to disease.

We assume that a) disease does not have vertical transmission but it is untreatable and causes an additional death rate; b) I-class does not contribute to the reproduction of newborns; and c) the net reproduction rate of newborns is modified by the disease (e.g, infectivies compete for resource but do not contribute to reproduction). In the presence of disease (i.e., $I > 0$) and the absence of predation (i.e., $P = 0$), the formulation of susceptible prey population dynamics can be described by the following (25):

$$\frac{dS}{dt} = \underbrace{rS(S-\theta)(1-S-I)}_{\text{the net reproduction modified by disease due to the competition for resource}} - \underbrace{\phi(N)\frac{I}{N}S}_{\text{new infections}} \tag{25}$$

where $\phi(N)$ is the disease transmission function that can be either density-dependent (i.e., $\phi(N) = \beta N$ which is also referred to the law of mass action) or frequency-dependent (i.e., $\phi(N) = \beta$). Thus, the formulation of infective population can be described by the following (26),

$$\begin{aligned} \tfrac{dI}{dt} &= \underbrace{\phi(N)\frac{I}{N}S}_{\text{Infected population per unit time}} - \underbrace{r\theta I}_{\text{natural mortality}} - \underbrace{\mu_d I}_{\text{additional mortality due to disease}} \\ &= \phi(N)\tfrac{I}{N}S - (r\theta + \mu_d)\, I \\ &= \phi(N)\tfrac{I}{N}S - \underbrace{\mu I}_{\text{the natural mortality plus an additional mortality due to disease}} . \end{aligned} \tag{26}$$

where the parameter $\mu > r\theta$ denotes the death rate of I-class, which includes an additional disease-induced death rate. This modeling approach is similar to the work by Boukal and Berec [11], Deredec and Courchamp [23], Courchamp et al. [18] and Hilker et al. [37] as well as many others [54, 30, 2, 57, 36, 62, 27] regarding the effects of Allee effects and disease.