



**University of
Nottingham**
UK | CHINA | MALAYSIA

A detailed study of the eco-epidemic model with incubation delay in predator-prey model

Juntao Li, Tianqi Li, Fusheng Luo, Yabing Yu, Zhichao Zhou, Ziyue Zheng

April 12, 2024

Abstract

In this paper, we investigated a delay differential mathematical model to represent the predator-prey dynamics with the Allee effect. We analyzed the basic mathematical characteristics of the proposed model such as permanence, positiveness and boundedness. In addition, we discussed the local stability within a no time-delay system and the global stability and hopf bifurcation analysis of the special point $E4$. Stability and bifurcation analysis were also conducted in models with time-delay. Moreover, we perform forward bifurcation analysis on the model based on the basic reproduction number. We conduct various numerical simulations to verify the effectiveness of the theoretical results. In the end, we added white noise to improve the reality of this model through considering random effects.

Keywords: Predator-prey; Intra species competition; Stability; Bifurcation; White noise; Colour noise.

Contents

1	Introduction	3
2	Development of the Model	5
2.1	Notation	5
2.2	Assumption	5
2.3	Construction process	5
3	Mathematical Analysis	8
3.1	Equilibria	8
3.2	Preliminary and paradox of enrichment	8
3.2.1	Positiveness of the solution of the delayed system	9
3.2.2	Boundedness	9
3.2.3	Existence analysis of predators	10
3.2.4	Permanence of the model	11
3.3	Basic reproduction number of the mathematical model system	12
3.4	The system with no time delay	13
3.4.1	Local stability analysis	13
3.4.2	Bifurcation Analysis for E_4 without delay	17
3.4.3	Global Stability Analysis for E_4	18
3.5	The system with time delay	20
3.5.1	Local stability and bifurcation analysis	20
4	Numerical analysis	24
4.1	Forward bifurcation analysis	24
4.2	The system with no delay	25
4.3	The system with delay	27
4.3.1	The point E_4	27
4.3.2	The point E_6^*	29
4.3.3	Two parameters analysis around E_6^*	30
5	Optimization of the model	30
6	Conclusion	32
A	Appendix	37
A.1	Stability analysis of the system with delay	37
A.2	Variable Gradient Method	39
A.3	Figures of the system with no delay	40
A.4	Figure of the system with delay	41

1 Introduction

The characteristic of an ecosystem is the interaction among different species in a fluctuating and complex natural environment.[10] The study of mathematical models helps to understand the interactions between predators and prey in these systems. According to the impact of environmental factors on different species, there are different relationships between predators and prey, including predation competition and mutualism. Among them, the relationship between predation is particularly important.

The predator-prey model has long been used in theoretical ecology. The first authoritative theory of the population principle that emerged came from Thomas Malthus[1], who believed that although the population grew logarithmically when the demand for resources eventually exceeded the attack, the population growth relying on resource supply would stop. Therefore, the first mathematical model logistic equation was formed. After several years, since the initial Lotka Volterra system was formed by Lotka [8] and Volterra [22], the model equations have largely evolved to more realistically describe the processes of predation, reproduction, and death. At the same time, the studies of predator-prey models over the past half century have shown differences in their functional responses. We have also constructed the basic architecture of our predator model based on this model.

American ecologist jailer Clyde Ali proposed this question [6]: What is the minimum number of species needed to survive in nature? He discussed the evidence of the impact of crowding on demographic and demographic life history characteristics. Therefore, the growth rate is not always positive for small densities, and it may not decrease as in the logical model. Generally speaking, if the growth rate of a population is average, then that population is called the "Allee effect", where per capita growth increases with increasing population density [21]. This effect arises from positive density dependence, meaning that the reproductive success or survival of individuals is enhanced by higher population densities[7]. At its core, the Allee effect is a manifestation of the interplay between social behaviors, ecological interactions, and demographic processes within populations [3]. The Allee effect also could be classified into strong and weak Allee effect [20, 23] which has been shown to fundamentally alter the dynamics of predator-prey interactions and strongly impact species persistence, and previous research has mainly focused on Allee effect scenarios in prey populations [18]. We chose to apply it in this scenario as well.

Moreover, the delayed process is also commonly present in biological systems and these systems are closely related to dynamics based on the time scale of the unit [12]. The delay differential equation has been explored by researchers in a long history, especially in the modeling of predator-prey systems[19], where it is used to represent and consider the response time, pregnancy period, feeding time, etc. required for predator-prey systems [5]. Not considering time delay in any predator dynamics model will have a significant impact on the model's authenticity [24]. By accounting for these delays, delayed predator-prey models provide a more realistic representation of ecological dynamics and offer insights into the mechanisms underlying population fluctuations and stability[9]. One of the key features of delayed predator-prey systems is their ability to exhibit complex dynamical behavior, including oscillations, chaos, and even stability switches [17]. Time delays introduce

feedback mechanisms into the system, allowing past states to influence present dynamics. This feedback can lead to the emergence of delayed feedback loops, which can amplify or dampen population fluctuations depending on the magnitude and sign of the delay[14]. Introducing time delay in biological models has more complex dynamics based on the above[2].

Furthermore, delayed predator-prey systems can exhibit phenomena such as Hopf bifurcations, where stable equilibrium states give way to periodic oscillations as a parameter crosses a critical threshold [15]. We also analyzed these phenomena in the third part of the article. These oscillations represent sustained cycles in predator and prey populations, driven by delayed feedback mechanisms within the system. Understanding the conditions under which these bifurcations occur can provide valuable insights into the stability and resilience of ecological communities in the face of environmental perturbations [11].

In this context, this study aims to investigate the dynamics of delayed predator-prey systems and explore how time delays influence the stability, persistence, and oscillatory behavior of ecological communities. We explore a predator-prey model that exhibits the Allee effect in ecologically important predator populations, which occurs in the numerical response of predators without affecting their functional response. By developing and analyzing mathematical models of delayed predator-prey interactions, we seek to deepen our understanding of the complex dynamics that govern natural ecosystems and inform conservation efforts aimed at preserving biodiversity and ecosystem function. This paper is organized as follows: In Section 2, we construct a mathematical model for the prey-predator system with the Allee effect and time delay. In Section 3, we conduct a mathematical analysis of the paradox of enrichment, stability, and bifurcation analysis. In section 4, we describe the numerical results with and without delay.

2 Development of the Model

2.1 Notation

The following table 1 describes the notations with explanations.

Parameter	Symbol	Default value
intrinsic growth rate	r	(0.04,1.5)
carrying capacity of environment	k	(8,200)
consumption rate of prey	m	(0.005,0.5)
conversion rate	e	(0.02,1)
predator death rate 1	μ	(-0.04,0.5)
predator death rate 2	d	(0.1,0.7)
saturation constant	b	(5,20)
Allee constant	n	(0,10)
transmission rate	α	(0.01,0.1)
infected predator capture coefficient	p	(0,1)

Table 1: Notations

2.2 Assumption

The assumptions of the models below can be summarised as follow:

- i) The construction of the **continuous** deterministic models(including the full model and our model, so we can have non-integer population) and their quantitative analysis is based on the famous **logistic model**, the natural birth/death rates, carrying capacities and interactive parameters are fixed with respect to time.
- ii) The activity domain of x,y and z is limited, and there is no invasion(leaving) of x,y and z from(for) other domain so that there is no external force to alter the population of x,y,z .
- iii) The only predators of prey x are predators y and z , and x can run out.
- iv) The predators y and z have other food sources.
- v) The disease cannot be transmitted to the prey, and there exists time delay of the disease incubation.
- vi) The derivation of the iterative terms in the full model is based on the established classic Lotka-Volterra equation.

2.3 Construction process

This model is mainly composed of prey and predator. Firstly, we analyzed the part of prey. Based on the assumption, the prey grows according to the Logistic model which has a positive effect on the prey density. Additionally, some prey is affected by predators which

has a harmful effect, so the prey population reduced. Secondly, we analyzed the predator part. The growth of predator population mainly depends on the conversion of affected prey, so there is a positive impact on the number of predators. Then, the mortality of predators leads to a fall in the number of predators.

$$\frac{dx}{dt} = rx \left(1 - \frac{x}{k}\right) - mxy, \quad \frac{dy}{dt} = emxy - qy;$$

What makes our model different from most models is the introduction of Allee affect. The Allee effect refers to the side effects on the growth of a population when the center is too sparse or crowded. In this model, the introduction of Allee effect will lead to a part of the negative effect in the process of increasing the number of prey, resulting in a decrease in growth. The saturation constant in the model represents the most appropriate population density of prey quantity in the model. We start with a demographic model as below:

$$\frac{dx}{dt} = rx \left(1 - \frac{x}{k} - \frac{n}{x+b}\right) - mxy, \quad \frac{dy}{dt} = emxy - qy;$$

where $x(t)$, and $y(t)$ stand for the prey and predator density, respectively, at time t ; and r, k, m, e, q, b and n are positive constants that stand for intrinsic growth rate, carrying capacity of the environment, consumption rate of prey, conversion rate, predator death rate, a saturation constant, and Allee constant, respectively.

Improvement: Moreover, the model we need to build has an infectious disease in predator, hence we should introduce another infected variable z .

$$\begin{aligned} \frac{dx}{dt} &= rx \left(1 - \frac{x}{k} - \frac{n}{x+b}\right) - mxy - pmxz, & \frac{dy}{dt} &= emxy - qy, \\ \frac{dz}{dt} &= emzpx - qz, \end{aligned}$$

where m and p are different infection rates to prey x . What we are considering is more inclined towards theory. In fact, infectious diseases are prone to occur in the ecological environment. There are diseases that spread between predators, such as what diseases, in a certain article (Murray et al., 2002). In nature, the process of interaction can lead to diseases. In addition to the initially established demographic models, there are also some ecosystems that combine diseases, which are named ecological surface dynamics models. Then, considering the transmitted disease, the disease can be transmitted (with a transmission rate α) by contact among the latter. We could get the given form:

$$\begin{aligned} \frac{dx}{dt} &= rx \left(1 - \frac{x}{k} - \frac{n}{x+b}\right) - mxy - pmxz, & \frac{dy}{dt} &= emxy - qy - \alpha zy, \\ \frac{dz}{dt} &= emzpx + \alpha zy - qz, \end{aligned}$$

In addition, we consider that there may be a time delay during infection and it cannot be transmitted immediately. There are various factors similar to incubation period and some

investigators have mentioned this issue. On that basis, the model has been modified to:

$$\begin{aligned}\frac{dx}{dt} &= rx \left(1 - \frac{x}{k} - \frac{n}{x+b}\right) - mxy - pmxz, & \frac{dy}{dt} &= emxy - qy - \alpha zy, \\ \frac{dz}{dt} &= emzpx + \alpha y(t-\tau)z(t-\tau) - qz,\end{aligned}$$

Additionally, the predator may have other food resources, which is represented by the parameter β . At the same time, we should also consider the disease-induced mortality (the rate expressed by δ) in z . Then, the model was modified by:

$$\begin{aligned}\frac{dx}{dt} &= rx \left(1 - \frac{x}{k} - \frac{n}{x+b}\right) - mxy - pmxz, & \frac{dy}{dt} &= emxy - (q - \beta)y - \alpha zy, \\ \frac{dz}{dt} &= emzpx + \alpha y(t-\tau)z(t-\tau) - (q + \delta)z,\end{aligned}$$

Therefore, dynamic models with time delays are closer to reality than those without time delays. Given this fact, in our current work, we have considered a Lotka Volterra predator-prey ecological epidemic model. We assume that this communicable disease will affect predators, and in this species, we also consider the delay in newborn pregnancy. This is the first attempt to consider the impact of delay in predator disease ecological models.

Compared with ordinary differential equations, it can disrupt equilibrium points and generate stable limit cycles. Moreover, considering the transmitted disease, we create an eco-epidemic model:

$$\begin{aligned}\frac{dx}{dt} &= rx \left(1 - \frac{x}{k} - \frac{n}{x+b}\right) - mxy - pmxz = F_1(x, y, z); \\ \frac{dy}{dt} &= emxy - \alpha zy - \mu y = F_2(x, y, z); \\ \frac{dz}{dt} &= \alpha y(t-\tau)z(t-\tau) + emzpx - dz = F_3(x, y, z);\end{aligned}\tag{2.1}$$

where α is the transmission rate.

Under this circumstance, the prey x reproduce logically, the number of which is affected by both sound y and infected z predators, through a different rates expressed by parameters m and p .

3 Mathematical Analysis

3.1 Equilibria

In order to determine the equilibria of this model, we set the derivatives equal to zero and we get:

$$\begin{aligned}\dot{x} &= 0 \text{ when } x = 0 \text{ or } y = \frac{r}{m} \left(1 - \frac{x}{k} - \frac{n}{x+b} \right) - pz \\ \dot{y} &= 0 \text{ when } y = 0 \text{ or } z = \frac{emx - \mu}{\alpha} \\ \dot{z} &= 0 \text{ when } z = 0 \text{ or } y = \frac{d - empx}{\alpha}\end{aligned}$$

Hence, we can find eight equilibrium points (x^*, y^*, z^*) equal to

1. $E_0(0, 0, 0)$
2. $E_1(x_1, 0, 0)$ where $x_1 = \frac{\sqrt{(k+b)^2 - 4kn} + (k-b)}{2}$
3. $E_2(x_2, 0, 0)$ where $x_2 = \frac{-\sqrt{(k+b)^2 - 4kn} + (k-b)}{2}$
4. $E_3\left(\frac{d}{emp}, 0, \frac{r}{mp} \left(1 - \frac{d}{kemp} - \frac{n}{\frac{d}{emp} + b}\right)\right)$
5. $E_4\left(\frac{\mu}{em}, \frac{r}{m} \left(1 - \frac{\mu}{kem} - \frac{n}{\frac{\mu}{em} + b}\right), 0\right)$, where $\mu > 0$
6. $E_5\left(0, \frac{d}{\alpha}, \frac{-\mu}{\alpha}\right)$, where $\mu < 0$
7. $E_6(x_6^*, y_6^*, z_6^*)$ where $y_6^* = \frac{d - empx_6^*}{\alpha}$, $z_6^* = \frac{mex_6^* - \mu}{\alpha}$
8. $E_7(x_7^*, y_7^*, z_7^*)$ where $y_7^* = \frac{d - empx_7^*}{\alpha}$, $z_7^* = \frac{mex_7^* - \mu}{\alpha}$

Particularly, $x_{6,7}^*$ are the roots of the equation $\frac{-\mu}{\alpha} + \frac{d}{\alpha p} = \frac{r}{mp} \left(1 - \frac{x}{k} - \frac{n}{x+b}\right)$

3.2 Preliminary and paradox of enrichment

The preliminary results, including the positiveness, existence and boundedness of solutions of the system can be easily shown below.

3.2.1 Positiveness of the solution of the delayed system

One can write the first equation of the delay system (2.1) as

$$\frac{dx}{x} = \left[r \left(1 - \frac{x}{k} - \frac{n}{x+b} \right) - my - pmz \right] dt$$

Integrating between the limits 0 and t, we have

$$x(t) = x(0) \exp \left\{ \int_0^t \left[r \left(1 - \frac{x}{k} - \frac{n}{x+b} \right) - my - pmz \right] ds \right\}$$

Similarly from the second and the third equation of the delay system, we have

$$y(t) = y(0) \exp \left\{ \int_0^t [emx - \alpha z - \mu] ds \right\}$$

and

$$z(t) = z(0) \exp \left\{ \int_0^t \left[\frac{\alpha}{z} y(t-\tau) z(t-\tau) + empx - d \right] ds \right\}$$

where $x(0) = x_0 > 0, y(0) = y_0 > 0$ and $z(0) = z_0 > 0$, Therefore, $x(t) > 0, y(t) > 0$ and $z(t) > 0$.

3.2.2 Boundedness

Theorem 1.

All the solutions of the system which initiate on \mathbb{R}^3 are uniformly bounded when predators' natural mortality exceeds saturation effect coefficient which means μ is positive.

Proof.

Under the assumption of $q - \beta < 0$ we could prove the boundedness of the total number of three species, otherwise the unbounded case could be happen. Then, we define the function $\omega(x, y, z) = x + y + z$, this equation is obviously positive definite. Thus, we construct a function as below and let $\eta \in \mathbb{R}_{++}^1$

$$\begin{aligned} \eta\omega + \frac{d\omega}{dt} &= rx \left(1 - \frac{x}{k} - \frac{n}{x+b} \right) - mxy - pmxz + emxy - \mu y + emzpx - dz \\ &\leq \eta(x + y + z) + rx \left(1 - \frac{x}{k} - \frac{n}{x+b} \right) - dz - \mu y, \quad \text{since } 0 < e < 1 \\ &< (\eta - d)z + (\eta - \mu)y + r \left(1 + \frac{\eta}{r} - \frac{x}{k} \right) \\ &< rx \left(-\frac{x}{k} + 1 + \frac{\eta}{r} \right), \quad \text{take } \eta < \min(d, \mu) \end{aligned}$$

This negative quadratic function is bounded above.

$$\begin{aligned}\omega + \eta \frac{d\omega}{dt} &= rx \left(-\frac{x}{k} + 1 + \frac{\eta}{r} \right) \\ &\leq \frac{rk}{4} \left(1 + \frac{\eta}{r} \right)^2 \\ &= \Theta\end{aligned}$$

The differential inequality theory could refer that,

$$0 < \omega \leq \frac{\Theta}{\eta} (1 - e^{-\eta t}) + \omega(0)e^{-\eta t} \leq \max \left(\frac{\Theta}{\eta}, \omega(0) \right)$$

When $t \rightarrow \infty$, which is bounded independent of the initial condition. For all $(x, y, z) \in \mathbb{R}^3$ and $\exists \epsilon > 0$, such that $0 < \omega < \frac{\Theta}{\eta} + \epsilon$.

3.2.3 Existence analysis of predators

Under certain conditions, the postiveness of $y + z$ could be proved, which implies the existence of predators.

$$\begin{aligned}\frac{dy}{dt} + \frac{dz}{dt} &= emxy - \mu y - empxz - dz \\ &= (emx - \mu)y + (empx - d)z\end{aligned}$$

This identity could be analysed through observing the range of x first.

$$\begin{aligned}\frac{dx}{dt} + \beta x &= rx \left(1 - \frac{x}{k} - \frac{n}{x+b} \right) + \beta x \\ &\leq rx \left(1 + \frac{\beta}{r} - \frac{x}{k} - \frac{n}{b} \right) \\ &\leq \frac{kr}{4}\end{aligned}$$

Take $\beta > \frac{rn}{b}$. Then we can use the conclusion in the previous section to conclude that $0 < x < R = \frac{kr}{4}$ as $t \rightarrow \infty$, where $R < \infty$.

$$\begin{aligned}\frac{dy}{dt} + \frac{dz}{dt} &< emxy - \mu y - empxz - dz \\ &\leq \theta(y + z)\end{aligned}$$

So when the parameter θ satisfies $\theta = \max \{emR - \mu, empR - d\}$. In this case, if $\theta < 0$ then we can conclude that $y + z = 0$ as $t \rightarrow \infty$. Hence, θ greater than 0 is the survival condition for predators.

3.2.4 Permanence of the model

Theorem 2.

System is permanent provided where $x_1 > \frac{\mu + \alpha\epsilon_1}{em}$ where x_1 is defined before.

Proof.

In this section, we need to show that the boundary planes of \mathbb{R}_+^3 repel the positive solutions of system (3.13) uniformly. Let us define

$$\begin{aligned} C_1 &= \{(\varpi_1, \varpi_2, \varpi_3) \in C([-\tau, 0], \mathbb{R}_+^3) : \varpi_1(\phi) = 0, \phi \in [-\tau, 0]\} \\ C_2 &= \{(\varpi_1, \varpi_2, \varpi_3) \in C([-\tau, 0], \mathbb{R}_+^3) : \varpi_2(\phi) = 0, \varpi_1(\phi) \neq 0, \phi \in [-\tau, 0]\} \\ C_3 &= \{(\varpi_1, \varpi_2, \varpi_3) \in C([-\tau, 0], \mathbb{R}_+^3) : \varpi_3(\phi) = 0, \varpi_1(\phi)\varpi_2(\phi) \neq 0, \phi \in [-\tau, 0]\} \end{aligned}$$

where $C([\tau, 0], \mathbb{R}_+^3)$ denote the space of continuous function mapping $[\tau, 0]$ into \mathbb{R}_+^3 . If $C_0 = C_1 \cup C_2 \cup C_3$ and $C^0 = \text{int}C([\tau, 0], \mathbb{R}_+^3)$, it suffices to show that there exists an $\epsilon_0 > 0$ such that for any solution u_t of the system (2.1) initiating from C^0 , $\lim_{t \rightarrow \infty} \inf d(u_t, C_0) \geq \epsilon_0$.

There are five constant solutions E_0, E_1, E_2 and E_4, E_5 in C_0 corresponding to $(x(t) = 0, y(t) = 0, z(t) = 0)$, $(x(t) = x_1, y(t) = 0, z(t) = 0)$, $(x(t) = x_2, y(t) = 0, z(t) = 0)$ and $(x(t) = x_4, y(t) = y_4, z(t) = 0)$, $(x(t) = x_5, y(t) = 0, z(t) = 0)$ respectively, i.e. if $(x(t), y(t), z(t))$ refers to any solution originating from C_1 in the system (2.1) and $x(t) \rightarrow 0, y(t) \rightarrow 0, z(t) \rightarrow 0$ as $t \rightarrow \infty$. Since E_1 has the similar characteristic with E_2 , so does E_4 and E_5 , we just investigate three points without loss of generality. If $(x(t), y(t), z(t))$ is a solution of the system (2.1) originating from C_2 with $\varpi_1(0) > 0$, it follows that $x(t) \rightarrow x_1, y(t) \rightarrow 0, z(t) \rightarrow 0$ as $t \rightarrow \infty$. If $(x(t), y(t), z(t))$ is a solution of the system (2.1) originating from C_3 with $\varpi_1(0)\varpi_2(0) > 0$, it follows that $x(t) \rightarrow x_4, y(t) \rightarrow y_4, z(t) \rightarrow 0$ as $t \rightarrow \infty$

.

The proof of the first equation is simple so we just ignore it. For the proof of the second equation, we assume contradiction i.e. $W^s(E_1) \cap C^0 \neq \Phi$, then there exists a positive solution $(x(t), y(t), z(t))$ of the system (2.1) such that $(x(t), y(t), z(t)) \rightarrow (x_1, 0, 0)$ as $t \rightarrow \infty$. Let us choose $\epsilon_1 > 0$ small enough such that

$$x_1 > \frac{\mu + \alpha\epsilon_1}{em} \quad \text{and} \quad 0 < z(t) < \epsilon_1 \tag{3.1}$$

For some large $t > t_1$, where t_1 be sufficiently large. Then from first and second equation of the system (2.1) we have, for $t > t_1$

$$\frac{dx(t)}{dt} \geq rx \left(1 - \frac{x}{k} - \frac{n}{x+b} - my\right) \tag{3.2}$$

$$\frac{dy(t)}{dt} \geq y(emx - \mu - \alpha\epsilon_1) \tag{3.3}$$

Now let us consider

$$\frac{dx_1(t)}{dt} \geq rx_1 \left(1 - \frac{x_1}{k} - \frac{n}{x_1 + b} - mx_2 \right) \quad (3.4)$$

$$\frac{dx_2(t)}{dt} \geq x_2 (emx_1 - \mu - \alpha\epsilon_1) \quad (3.5)$$

Let $v = (v_1, v_2)$ and $\varphi > 0$ be small enough such that $\varphi v_1 < x(t_1)$, $\varphi v_2 < y(t_1)$. If $(x_1(t), x_2(t))$ is a solution of system (3.5) satisfying $x_i(t_1) = \varphi v_i$, $i = 1, 2$. We know from comparison theorem $x(t) > x_1(t)$, $y(t) > x_2(t)$ for all $t > t_1$. It is easy to see that system has a unique positive equilibrium

$$(x_1^*, x_2^*) = \left(\frac{\mu + \alpha\epsilon_1}{em}, \frac{r}{m} \left(1 - \frac{\mu + \alpha\epsilon_1}{kem} - \frac{enm}{bem + \mu + \alpha\epsilon_1} \right) \right) \quad (3.6)$$

Now $x(t) > x_1(t)$, $y(t) > x_2(t)$ for all $t > t_1$ and $\lim_{t \rightarrow \infty} x_2(t) = x_2^*$. This is a contradiction. Hence $W^s(E_1) \cap C^0 = \Phi$.

Let $W^s(E_4) \cap C^0 \neq \Phi$. Then there exists a positive solution $(x(t), y(t), z(t))$ of the system such that as $t \rightarrow \infty$, $(x(t), y(t), z(t)) \rightarrow (x_4, y_4, 0)$. Then we choose $\epsilon_2 > 0$ small enough such that $x_4 - \epsilon_2 < x_4 + \epsilon_2$ for $t > t_2 - \tau$. Then from third equation of the system (2.1) we have, for $t > t_2 - \tau$

$$\frac{dz(t)}{dt} \geq z \left(\frac{\alpha(y_4 - \epsilon_2)z(t - \tau)}{z} - empx_4 - d \right) \quad (3.7)$$

As $z(t - \tau) \geq z(t) e^{-m_1\tau}$

$$\frac{dz(t)}{dt} \geq z \left(\frac{\alpha(y_4 - \epsilon_2)z(t)e^{-m_1\tau}}{z} - empx_4 - d \right) \quad (3.8)$$

Let u_1 and $w > 0$ be small enough so that $wu_1 < z(t_2)$. If z_4 is a solution of system (3.8) satisfying $z_4(t_2) = wu_1$, we know from comparison theorem, $z(t) \geq z_4(t)$ for all $t > t_2 - \tau$. We also find that the solution z_1 of Equation (3.8) satisfies, $\lim_{t \rightarrow \infty} z_4(t) > 0$.

Since $z(t) \geq z_4(t)$ for all $t > t_2$ and so $\lim_{t \rightarrow \infty} z(t) \not\rightarrow 0$, this is a contradiction. Therefore $W^s(E_2) \cap C^0 \neq \Phi$. Hence the system (2.1) is permanent. This proves the theorem.

3.3 Basic reproduction number of the mathematical model system

We will use the basic reproduction number of diseases to analyze the community structure of model system (1.1), which can be fully explained by following threshold quantity. The basic reproduction number can be obtained through the definition of the reproduction number, and the expression for the basic reproduction number R_0 is derived as,

$$R_0 = \frac{empx + \alpha y}{d}$$

Here, the $\frac{1}{d}$ is the mean lifespan of the predator and the $empx + \alpha y$ is the infection rate of the prey. Subsequently, this product describes the mean number of the susceptible predators,

which is also the meaning of the ecological basic reproduction number. Hence, when the reproduction number is less than 1, the disease may die out. When the reproduction number is more than 1, the disease break out and it finally remains an endemic equilibrium.

3.4 The system with no time delay

For the following section, we try to analyse the eight different equilibrium points separately regarding their existence and stability conditions, along with the Jacobian matrix. Additionally, for the disease free equilibrium point E_4 Hopf Bifurcation analysis was implemented to find periodic solutions combined with limit cycle.

3.4.1 Local stability analysis

We first study the system (2.1) with no time lag. The system (2.1) without delay can be written as

$$\begin{aligned} \frac{dx}{dt} &= rx \left(1 - \frac{x}{k} - \frac{n}{x+b} \right) - mxy - pmxz; \\ \frac{dy}{dt} &= emxy - \alpha zy - \mu y; \\ \frac{dz}{dt} &= \alpha yz + emzpx - dz; \end{aligned} \tag{3.9}$$

Theorem 3.

1) The equilibrium E_0 is stable if and only if

$$i) n > b, \quad ii) \mu > 0.$$

2) The equilibrium E_1 , if feasible, is stable if and only if

$$i) (x_1 + b)^2 > nk, \quad ii) x_1 < \frac{\mu}{em}, \quad iii) x_1 < \frac{d}{emp}.$$

3) The equilibrium E_2 , if feasible, is unconditionally unstable.

4) The equilibrium E_3 , if feasible, is stable if and only if

$$i) \frac{d}{p} - z_3\alpha - \mu < 0, \quad ii) (b + \frac{d}{emp})^2 > nk, \quad iii) 1 - \frac{x_3}{k} - \frac{n}{x_3 + b} > 0$$

5) The equilibrium E_4 , if feasible, is stable if and only if

$$\begin{array}{ll} i) y_4\alpha - \mu - d < 0, & ii) (x_4 + b)^2 > nk, \\ iii) 1 - \frac{x_4}{k} - \frac{n}{x_4 + b} > 0, & iv) \mu > 0 \end{array}$$

6) The equilibrium E_5 , if feasible, is unconditionally unstable.

Proof.

Generally, the Jacobian matrix is denoted by $J(x, y, z) \equiv DF(x, y, z) = \pi_{ij} \in \mathbb{R}^{3 \times 3}$, with

$$J(x, y, z) = \begin{pmatrix} r \cdot \left(1 - \frac{2x}{k} - \frac{nb}{(x+b)^2}\right) - my - pmz & -mx & -pmx \\ emy & emx - z\alpha - \mu & -\alpha y \\ emzp & \alpha z & \alpha y + empz - d \end{pmatrix}$$

As a shorthand, for $i = 1, 2, 3$, $j = 1, 2, 3$, $k = 0, 1, 2, 3, 4, 5, 6, 7$, we express $J_k = J(E_k)$, $\pi_{ij}^k = \pi_{ij}(E_k)$, and λ_i are eigenvalues of J_k .

1. Point $E_0(0, 0, 0)$ The corresponding Jacobian matrix to this point is

$$J_1 = \begin{pmatrix} r \left(1 - \frac{n}{b}\right) & 0 & 0 \\ 0 & -\mu & 0 \\ 0 & 0 & -d \end{pmatrix}.$$

Note that the related characteristic equation is

$$\Phi(\lambda) = \left(\lambda - r\left(1 - \frac{n}{b}\right)\right) \cdot (\lambda + \mu) \cdot (\lambda + d)$$

Given d, r, n, b are positive, E_0 is stable if and only if $n > b$ and $\mu > 0$ which implies that all the eigenvalues (i.e. λ) would be negative or at least having negative real parts.

2. Points $E_1(x_1, 0, 0)$ and $E_2(x_2, 0, 0)$ where $x_{1,2} = \frac{\pm\sqrt{(k+b)^2 - 4kn} + (k-b)}{2}$, which is derived from the equation:

$$x^2 + (b-k)x + (n-b) \cdot k = 0$$

We continue our deduction by conforming to the Jacobian matrix

$$J_{1,2} = \begin{pmatrix} r \left(1 - \frac{2x_{1,2}}{k} - \frac{nb}{(x_{1,2}+b)^2}\right) & -mx_{1,2} & -pmx_{1,2} \\ 0 & emx_{1,2} - \mu & 0 \\ 0 & 0 & empz_{1,2} - d \end{pmatrix}.$$

This is an upper triangle matrix, and its eigenvalues are

$$\lambda_1 = emx_{1,2} - \mu, \quad \lambda_2 = empz_{1,2} - d, \quad \lambda_3 = r \left(1 - \frac{2x_{1,2}}{k} - \frac{nb}{(x_{1,2}+b)^2}\right).$$

Note that $r \left(1 - \frac{x_{1,2}}{k} - \frac{n}{x_{1,2} + b} \right) = 0$, thus

$$r \left(1 - \frac{2x_{1,2}}{k} - \frac{nb}{(x_{1,2} + b)^2} \right) = rx \left(\frac{n}{(x_{1,2} + b)^2} - \frac{1}{k} \right).$$

The stability of both equilibrium points needs all related eigenvalues to be negative or at least to have negative real parts. Thus, the following needs to be satisfied:

$$\text{i) } (x_{1,2} + b)^2 > nk, \quad \text{ii) } x_{1,2} < \frac{\mu}{em}, \quad \text{iii) } x_{1,2} < \frac{d}{emp}$$

Otherwise, they are unstable points. Combining with the existence conditions, we can conclude that E_1 is stable if and only if ii) and iii) satisfied. However, E_2 is always unstable since there is a contradiction between i) and $x_2 > 0$

$$3. \text{ Point } E_3 \left(\frac{d}{emp}, 0, \frac{r}{mp} \left(1 - \frac{x_3}{k} - \frac{n}{x_3 + b} \right) \right) \text{ where } x_3 = \frac{d}{emp}.$$

Firstly, we examine the existence of biological significance of this fixed point, and find that if $1 - \frac{x_3}{k} - \frac{n}{x_3 + b} > 0$, that is to say, the point implied a equilibrium of state that only contains preys and infected predators. This implied that the disease in predators prolongs forever and the prey-predator eco-epidemiological system still prevails when the disease is introduced.

Secondly, the correlated jacobian matrix is

$$J_3 = \begin{pmatrix} r \frac{d}{emp} \left(\frac{n}{(\frac{d}{emp} + b)^2} - \frac{1}{k} \right) & -m \frac{d}{emp} & -pm \frac{d}{emp} \\ 0 & \frac{d}{p} - z_3\alpha - \mu & 0 \\ empz_3 & z_3\alpha & 0 \end{pmatrix}$$

The characteristic equations of the matrix for eigenvalues λ is

$$(\lambda - \pi_{22}^{[3]}) \cdot (\lambda^2 - \pi_{11}^{[3]}\lambda - \pi_{13}^{[3]}\pi_{31}^{[3]}) = 0$$

The solution to this, $\lambda_1 = \pi_{22}^{[3]} = \frac{d}{p} - z_3\alpha - \mu$, $\lambda_{2,3}$ satisfy the equations

$$\lambda^2 - r \frac{d}{emp} \left(\frac{n}{(\frac{d}{emp} + b)^2} - \frac{1}{k} \right) \cdot \lambda + em^2 p^2 \frac{d}{emp} z_3 = 0$$

Hence, combining with existence conditions, the system obtains locally asymptotically stability around E_3 if and only if

$$\text{i) } \frac{d}{p} - z_3\alpha - \mu < 0, \quad \text{ii) } (b + \frac{d}{emp})^2 > nk, \quad \text{iii) } 1 - \frac{x_3}{k} - \frac{n}{x_3 + b} > 0$$

4. Point $E_4(\frac{\mu}{em}, \frac{r}{m}(1 - \frac{x_4}{k} - \frac{n}{x_4 + b}), 0)$, where $x_4 = \frac{\mu}{em}$

To make this point have biological significance, we can obtain that $\frac{r}{m}(1 - \frac{x_4}{k} - \frac{n}{x_4 + b}) > 0$ and $\mu > 0$.

The jacobian is

$$J_4 = \begin{pmatrix} rx_4(\frac{n}{(x_4+b)^2} - \frac{1}{k}) & -mx_4 & -pmx_4 \\ emy_4 & 0 & -y_4\alpha \\ 0 & 0 & y_4\alpha - emx_4 - d \end{pmatrix}.$$

Note that $\pi_{33}^{[4]} = y_4\alpha - \mu - d$ and the characteristic equation of λ is

$$(\lambda - \pi_{33}^{[4]}) \cdot (\lambda^2 - \pi_{11}^{[4]}\lambda - \pi_{12}^{[4]}\pi_{21}^{[4]}) = 0$$

Similar to E_3 , the system obtains locally asymptotical stability around E_4 if and only if

- i) $y_4\alpha - \mu - d < 0$,
- ii) $(x_4 + b)^2 > nk$,
- iii) $1 - \frac{x_4}{k} - \frac{n}{x_4 + b} > 0$,
- iv) $\mu > 0$

5. Point $E_5(0, \frac{d}{\alpha}, -\frac{\mu}{\alpha})$

This point have biological significance, if and only if $\mu < 0$. And the jacobian matrix is

$$J_5 = \begin{pmatrix} r(1 - \frac{n}{b} + \frac{pm\mu-md}{\alpha}) & 0 & 0 \\ \frac{emd}{\alpha} & 0 & -d \\ -\frac{em\mu}{\alpha} & -\mu & 0 \end{pmatrix}.$$

The determinant characteristic polynomial with respect to λ is

$$(\lambda - \pi_{11}^{[5]}) \cdot (\lambda^2 - d\mu) = 0$$

This characteristic equation gives 3 different eigenvalues $\lambda_{1,2,3}$, where $\lambda_{2,3} = \pm\sqrt{-d\mu}i$, are a pair of pure imaginary roots (μ is negative). In this case, as the real parts of all there eigenvalues cannot be negative at the same time, this point of equilibrium is unstable and enter into hopf bifurcation directly.

Theorem 4.

- 1) The system without delay is locally asymptotically stable around the positive interior equilibrium E_6^* if and only if

i) $\sqrt{\Delta_2} = (V - \frac{rb}{k}) - \frac{4r^2n}{k} > 0$

- ii) $-(V + \frac{rb}{k}) + \sqrt{\Delta_2} > 0$
- iii) $\underline{x} < x_6^* < \bar{x}$ where $\underline{x} = \max\{0, \frac{\mu}{em}\}$ and $\bar{x} = \frac{d}{emp}$
where $V = \frac{(d-p\mu)m-r\alpha}{\alpha}$ ($V < 0$).

2) The system without delay is unconditionally unstable around the positive interior equilibrium E_7^*

Proof

For point $E_{6,7}(x_{6,7}^*, y_{6,7}^*, z_{6,7}^*)$, the relations between $x_{6,7}, y_{6,7}$, and $z_{6,7}$ follows the below equations,

$$\begin{aligned} x_{6,7}^* &= \frac{k(-V - \frac{rb}{k}) \pm \sqrt{\Delta_2}}{2r} \\ z_{6,7}^* &= \frac{emx_{6,7}^* - \mu}{\alpha}, \\ y_{6,7}^* &= \frac{d - emp x_{6,7}^*}{\alpha} \end{aligned}$$

where $V = \frac{(d-p\mu)m-r\alpha}{\alpha}$ ($V < 0$) , and $\Delta_2 = (V + \frac{rb}{k})^2 - \frac{4r}{k}(Vb + rn)$. To make this point have biological significance, we can obtain that

$$\text{i)} \Delta_2 > 0, \quad \text{ii)} -(V + \frac{rb}{k}) \pm \sqrt{\Delta_2} > 0$$

For the detailed proof of stability, see the Appendix [A.1](#).

3.4.2 Bifurcation Analysis for E_4 without delay

The point E_4 is of biological significance as this point represents that the disease finally dies out, and the system contains only preys and innocent predators. Hence, the works to analyse the stability of the point, and distinguish any underlying bifurcation are vital.

Theorem 5.

If $y_4\alpha - \mu - d < 0$, $1 - \frac{x_4}{k} - \frac{n}{x_4 + b} > 0$, $\mu > 0$, system [2.1](#) enters into Hopf Bifurcation around E_4 when k crosses the value $k^* = \frac{(\mu + bem)^2}{ne^2m^2}$.

Proof.

To find the bifurcation in ODE system at E_4 , we need analyse the behavior of the characteristic equations. Since $z = 0$ at this circumstance, the system [2.1](#) becomes,

$$\begin{aligned} \frac{dx}{dt} &= rx \left(1 - \frac{x}{k} - \frac{n}{x+b}\right) - mxy; \\ \frac{dy}{dt} &= emxy - \mu y; \end{aligned} \tag{3.10}$$

The jacobian matrix can be simplified,

$$J'_4 = \begin{pmatrix} rx\left(\frac{n}{(x+b)^2} - \frac{1}{k}\right) & -mx \\ emy & 0 \end{pmatrix}.$$

And the characteristic equations becomes,

$$\lambda^2 - A\lambda + B = 0 \quad (3.11)$$

$$\text{where } A = \frac{r\mu}{em} \left(\frac{ne^2m^2}{(\mu+bem)^2} - \frac{1}{k} \right), \quad B = em^2xy$$

From theorem 3, we know that initially, A should be negative to be at a stage of stability. We assume A is a function of k with varying value of k . If this equilibrium point attain Hopf Bifurcation, the following conditions should be satisfied:

- (i) There exists complex eigenvalues $\lambda(k) = \mu(k) \pm \theta(k)i$ for equation 3.11.
- (ii) There exists $k = k^*$ such that $\mu(k^*) = 0$ and $\left. \frac{\partial \mu}{\partial k} \right|_{k=k^*} \neq 0$.

To obtain the derivative of A with respect to k , we have

$$\frac{dA}{dk} = \frac{r\mu}{k^2em}$$

Note that this value is constantly positive as μ needs to be positive to have biological interpretation of the state. That is to say the value of A is increasing as k increases, with an initial negative value. And there must exist a value k^* , such that $A = 0$, where $k^* = \frac{(\mu + bem)^2}{ne^2m^2}$. At k^* , we would have pure imaginary eigenvalues, and after this A becomes positive (with increasing value of k), we then have unstable equilibrium points. This point of transition is called bifurcation value k^* . Besides, this implies the existence of Hopf's bifurcation at the point E_4 .

3.4.3 Global Stability Analysis for E_4

After analyzing the local stability of the point E_4 , we would like to extend our analysis to the global stability. Since the point E_4 is of great ecological significance, it represents a state that the system would return to its original steady state even though we have introduced a disease to the predators. That means the disease in the predators would eventually die out without a huge impact on the coexistence and permanence of the system. However, the way to prove global stability is arcane and takes a lot of effort, even requires knowledge at the postgraduate level. That's why we only provide the proof of the point E_4 here. Before we reach our theorem, we need to briefly introduce the Lyapunov global stability theorem.

Theorem 6.

If a function $V(x)$ is positively definite and radially unbounded, and its time derivative is

globally negative

$$V'(x) < 0 \quad \text{for all } x \neq x^*$$

then the equilibrium x^* is globally stable. A function $V(x)$ that satisfies the conditions of the former theorem is called a Lyapunov function.

Lemma 1. Consider the following delay differential equation:

$$\dot{x}(t) = Ax(t - \tau) - Bx(t)$$

where $A, B, \tau > 0$; $x(t) > 0$, for $-\tau \leq t \leq 0$. Then we have, if $A < B$, then $\lim_{t \rightarrow \infty} = 0$.

Theorem 7.

If $\mu \geq r$ and $y \geq ek$, then the equilibrium point $E_4(\frac{\mu}{em}, \frac{r}{m}(1 - \frac{x_4}{k} - \frac{n}{x_4 + b}), 0)$ is globally asymptotically stable, where $y = \frac{r}{m}(1 - \frac{x_4}{k} - \frac{n}{x_4 + b})$, and $x_4 = \frac{\mu}{em}$

proof

Through the gradient variable method (See Appendix), we have constructed a Lyapunov function as

$$V(x, y) = \frac{1}{2}x^2 + \frac{1}{e}xy + \frac{1}{2e^2}y^2$$

This function is radially unbounded as

$$\text{for } \|(x, y)\| \rightarrow \infty, \quad \text{the function tends to } \infty.$$

As the coefficient of the highest power (i.e. 2) are positive for both x^2 and y^2 . This function is positive definite for the same reason. The derivative of the function is strictly negative definite.

$$\begin{aligned} V' &= \frac{\partial V}{\partial x} \cdot \frac{dx}{dt} + \frac{\partial V}{\partial y} \cdot \frac{dy}{dt} \\ &= (x + \frac{1}{e}y)(rx(1 - \frac{x}{k} - \frac{n}{x+b} - mxy) + (\frac{1}{e}x + \frac{1}{e^2}y)(emxy - \mu y)) \\ &= rx^2(1 - \frac{x}{k} - \frac{n}{x+b}) - mx^2y + \frac{1}{e}rxy(1 - \frac{x}{k} - \frac{n}{x+b}) - \frac{1}{e}mxy^2 \\ &\quad + myx^2 - \frac{1}{e}\mu xy + \frac{1}{e}mxy^2 - \frac{\mu}{e^2}y^2 \\ &= (1 - \frac{y}{ek})rx^2 + \frac{1}{e}(r - \mu)xy - \frac{rx^3}{k} - \frac{rx^2n}{x+b} - \frac{rxyn}{e(x+b)} - \frac{\mu y^2}{e^2} \end{aligned}$$

$V'(x, y)$ is negative definite if $1 - \frac{y}{ek} \leq 0$ and $r - \mu \leq 0$.

Now, choose $\varepsilon > 0$ such that $\varepsilon < \frac{a}{\alpha}$. For $t > T + \tau$ we get,

$$\frac{dz}{dt} \leq -dz(t) + \alpha\varepsilon z(t - \tau)$$

Therefore, $\limsup_{t \rightarrow \infty} z(t) = 0$. Hence, $\lim_{t \rightarrow \infty} z(t) = 0$. When combined with the Ly-

punov stability theorem above, this implies global asymptotic stability of E_4 .

3.5 The system with time delay

In this section, we will mainly focus on local stability of the interior equilibrium E_i^* of the delay-induced system 2.1, where $i = 6, 7$.

3.5.1 Local stability and bifurcation analysis

To study the local stability of these two interior equilibrium points, let us define

$$P(t) = x(t) - x_i^*, \quad S(t) = y(t) - y_i^*, \quad I(t) = z(t) - z_i^*$$

The system 2.1 then can be expressed in matrix form as follows

$$\frac{d}{dt} \begin{pmatrix} P(t) \\ S(t) \\ I(t) \end{pmatrix} = A'_1 \begin{pmatrix} P(t) \\ S(t) \\ I(t) \end{pmatrix} + A'_2 \begin{pmatrix} P(t - \tau) \\ S(t - \tau) \\ I(t - \tau) \end{pmatrix} \quad (3.12)$$

Where A'_1 and A'_2 are 3×3 matrices given by

$$A'_1 = \begin{pmatrix} rx_i^* \left(\frac{n}{(x_i^* + b)^2} - \frac{1}{k} \right) & -mx_i^* & -pmx_i^* \\ emy_i^* & 0 & -\alpha y_i^* \\ empz_i^* & 0 & emp x_i^* - d \end{pmatrix}, \quad A'_2 = \begin{pmatrix} 0 & 0 & 0 \\ 0 & 0 & 0 \\ 0 & \alpha z_i^* & \alpha y_i^* \end{pmatrix}$$

The characteristic equation of the system is given by

$$|A'_1 + A'_2 e^{-\lambda\tau} - \lambda I| = 0$$

That is,

$$\phi(\lambda, \tau) = \lambda^3 + [A + Be^{-\lambda\tau}] \lambda^2 + [C + De^{-\lambda\tau}] \lambda + E + Fe^{-\lambda\tau} = 0 \quad (3.13)$$

Where

$$\begin{aligned}
A &= - \left[rx_i^* \left(\frac{n}{(x_i^* + b)^2} - \frac{1}{k} \right) + empx_i^* - d \right] \\
B &= -\alpha y_i^* \\
C &= em^2 x_i^* y_i^* + em^2 p^2 x_i^* z_i^* + rx_i^* \left(\frac{n}{(x_i^* + b)^2} - \frac{1}{k} \right) (empx_i^* - d) \\
D &= \alpha^2 y_i^* z_i^* + \alpha r x_i^* y_i^* \left(\frac{n}{(x_i^* + b)^2} - \frac{1}{k} \right) \\
E &= -\alpha em^2 p x_i^* y_i^* z_i^* - em^2 x_i^* y_i^* (empx_i^* - d) \\
F &= -\alpha^2 r x_i^* y_i^* z_i^* \left(\frac{n}{(x_i^* + b)^2} - \frac{1}{k} \right) - \alpha em^2 x_i^* y_i^{*2} + \alpha em^2 p x_i^* y_i^* z_i^*
\end{aligned}$$

It is clear that for the delay-induced system 2.1, E_i^* will be asymptotically stable if all the roots of the corresponding characteristic equation 3.13 have negative real parts. However, compared with the ODE characteristic equation that we have discussed in section 3.4.1, equation 3.13 cannot be solved by the classical Routh-Hurwitz criterion. Since it is a transcendental equation with infinitely many eigenvalues. To guarantee the steady state of E_i^* , the following conditions should be satisfied:

- (i) E_i^* is stable in case of ODE (non-delayed) system.
- (ii) The transcendental equation 3.13 does not exist purely imaginary roots, by Rouche's theorem [13](Dieudonne', 1960) (Theorem 9.17.4) and the continuity in τ .

To be more precise, we take

$$\lambda(\tau) = \eta(\tau) + iw(\tau) \quad (3.14)$$

Where η and ω are real. We have known that $\eta(0) < 0$, since E_6^* is stable in ODE system. By continuity, if $\tau > 0$ is sufficiently small, we still have $\eta(\tau) < 0$, therefore, E_6^* is still stable. However, if there exists a certain value $\tau^* > 0$ such that $\eta(\tau^*) = 0$ and $\omega \neq 0$, the steady state of system 2.1 will gradually lose stability and ultimately becomes unstable with $\eta(\tau)$ becoming positive.

Since E_7^* is always unstable in the ODE system, which we have discussed in section 3.4.1. E_6^* is the only interior equilibrium point able to satisfy the above conditions, maintaining the steady state when $\tau > 0$. Therefore, the following mathematical analysis will focus on E_6^* .

First, we assume that $\lambda(\tau_0) = i\bar{w}$ is a root of 3.13 if and only if

$$-i\bar{w}^3 - \bar{w}^2 [A + Be^{-i\bar{w}\tau}] + i\bar{w} [C + De^{-i\bar{w}\tau}] + E + Fe^{-i\bar{w}\tau} = 0 \quad (3.15)$$

Equating the real and imaginary parts of both sides, we get

$$(B\bar{w}^2 - F) \sin \bar{w}\tau + D\bar{w} \cos \bar{w}\tau = \bar{w}^3 - c\bar{w} \quad (3.16)$$

$$(B\bar{w}^2 - F) \cos \bar{w}\tau - D\bar{w} \sin \bar{w}\tau = -A\bar{w}^2 + E \quad (3.17)$$

To eliminate τ by squaring and adding 3.16 and 3.17, we could get the equation:

$$\bar{w}^6 + (A^2 - B^2 - 2C)\bar{w}^4 + (C^2 - D^2 + 2BF - 2AE)\bar{w}^2 + E^2 - F^2 = 0 \quad (3.18)$$

if we assume

$$\bar{w}^2 = P, M_1 = A^2 - B^2 - 2C, M_2 = C^2 - D^2 + 2BF - 2AE, M_3 = E^2 - F^2$$

then we obtain a cubic equation from 3.18

$$T(P) = P^3 + M_1P^2 + M_2P + M_3 = 0 \quad (3.19)$$

To determine the sign of roots for equation 3.19, we calculate the derivative of $T(P)$

$$\frac{dT(P)}{dP} = 3P^2 + 2M_1P + M_2 \quad (3.20)$$

Set $\frac{dT(P)}{dP} = 0$. The roots of 3.20 are given by

$$P_{1,2} = \frac{1}{3} \left[-M_1 \pm \sqrt{M_1^2 - 3M_2} \right] \quad (3.21)$$

Theorem 8.

Suppose that E_6^* exists and is locally asymptotically stable with $\tau = 0$. If

$$(i) M_1 > 0, M_2 > 0, M_3 > 0 \text{ or } M_1^2 - 3M_2 < 0$$

then, for $\forall \tau > 0$, all roots of characteristic equation 3.13 have negative real parts, and the system with any delays is locally asymptotically stable around the positive interior equilibrium E_6^* .

Proof

If $M_1 > 0$, $M_2 > 0$ and $M_3 > 0$, then we could get that P_1 and P_2 are both negative. Thus there does not exist positive roots for equation 3.20. Since

$$\phi(0) = M_3 > 0, \quad \frac{dT(P)}{dP} > 0 \text{ for } \forall P > 0, \quad \text{and } \lim_{P \rightarrow \infty} \phi(P) \rightarrow \infty$$

Thus equation 3.19 $T(P)$ does have any positive roots, then this is no \bar{w} such that $i\bar{w}$ is an eigenvalue of characteristic equation 3.13. Therefore, all roots of characteristic equation 3.13 have negative real parts for all $\tau > 0$.

Theorem 9.

Suppose that E_6^* exists and is locally asymptotically stable with $\tau = 0$.

- (i) If $M_1 \cdot M_2 < 0$, and $M_3 < 0$, there exists $\tau = \tau^*$ such that E_6^* of the delay system 2.1 is asymptotically stable when $0 \leq \tau < \tau^*$ and unstable for $\tau > \tau^*$
- (ii) Additionally, the system will undergo a Hopf bifurcation at E_6^* when $\tau = \tau^*$, provided by $N_4 L_1 \neq N_3 L_2$.

Proof

If $M_3 < 0$, $M_1 > 0$ and $M_2 < 0$, or ($M_1 < 0$ and $M_2 > 0$), then we could get one of roots $P_{1,2}$ is positive. Thus there exist a pair of purely imaginary roots $\pm i\bar{w}$ such that $\eta(\tau^*) = 0$ and $w(\tau^*) = \bar{w}$. Solving τ^* from 3.16 and 3.17, we have

$$\tau_j^* = \frac{1}{\bar{w}} \arccos \left[\frac{(D - AB)\bar{w}^4 + (AF + BE - DC)\bar{w}^2 - EF}{(B\bar{w}^2 - F) + D^2\bar{w}^2} \right] + \frac{2j\pi}{\bar{w}}, \text{ where } j = 0, 1, 2, \dots$$

Also, we can verify the following transversality condition:

$$Re \left[\left(\frac{d\lambda}{d\tau} \right) \right]_{\tau=\tau^*} \neq 0.$$

Differentiating equation 3.13 with respect to τ , we obtain

$$\frac{d\lambda}{d\tau} [e^{\lambda\tau} (3\lambda^2 + 2A\lambda + C) - \tau (B\lambda^2 + D\lambda + F) + 2B\lambda + D] = B\lambda^3 + D\lambda^2 + F\lambda$$

At $\tau = \tau_0 = \tau^*$ with $\lambda = i\bar{w}$, we obtain

$$\begin{aligned} \left(\frac{d\lambda}{d\tau} \right)^{-1} &= \frac{e^{\lambda\tau} (3\lambda^2 + 2A\lambda + C) + 2B\lambda + D}{\lambda (B\lambda^2 + D\lambda + F)} - \frac{\tau}{\lambda} \\ &= \frac{(-3\bar{w}^2 + 2A\bar{w}i + C)(\cos \bar{w}\tau + i \sin \bar{w}\tau) + 2B\bar{w}i + D}{\bar{w}[i(F - B\bar{w}^2) - D\bar{w}]} - \frac{\tau}{\lambda} \end{aligned}$$

Solving the above system, we could obtain the real part of $(\frac{d\lambda}{d\tau})^{-1}$, given by

$$\begin{aligned} Re \left[\left(\frac{d\lambda}{d\tau} \right)^{-1} \right] &= \frac{N_4(N_1 \cos \bar{w}\tau - N_2 \sin \bar{w}\tau + D) - N_3(N_2 \cos \bar{w}\tau - N_1 \sin \bar{w}\tau + 2B\bar{w})}{-\bar{w}(N_4^2 + N_3^2)} \\ &= \frac{N_4 L_1 - N_3 L_2}{-\bar{w}(N_4^2 + N_3^2)} \end{aligned}$$

Where

$$N_1 = -3\bar{w}^2 + C, \quad N_2 = 2\bar{w}A, \quad N_3 = F - B\bar{w}^2, \quad N_4 = -D\bar{w}$$

$$L_1 = N_1 \cos \bar{w}\tau - N_2 \sin \bar{w}\tau + D, \quad L_2 = N_2 \cos \bar{w}\tau - N_1 \sin \bar{w}\tau + 2B\bar{w}$$

Now we have

$$\operatorname{Re} \left[\left(\frac{d\lambda}{d\tau} \right)^{-1} \right]_{\tau=\tau^*} \neq 0, \quad \text{if } N_4 L_1 \neq N_3 L_2$$

This completes the proof of the theorem.

4 Numerical analysis

In the last performance, we used Matlab to show the system with different parameters and observe the change in the dynamic behavior of the system. According to the previous analysis, we will divide this section into several parts, about the bifurcation and the improvement of the system, but the most important thing is the bifurcation.

4.1 Forward bifurcation analysis

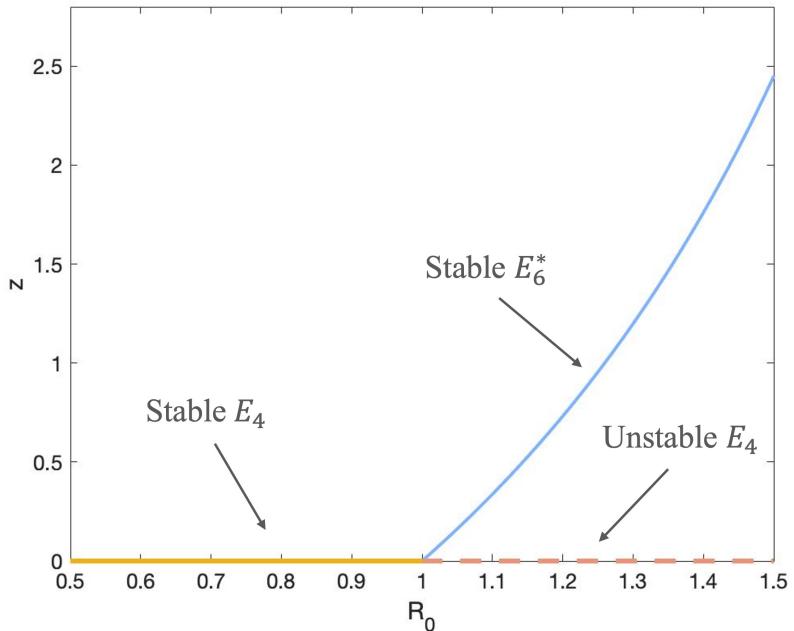


Figure 1: Figure shows forward bifurcation of system(1.1) when $R_0 > 1$.Here, blue solid line indicates stable endemic equilibrium E_6^* , yellow solid line represents stable equilibrium E_4 , orange dashed line stands for the unstable disease-free equilibrium E_4

We could take R_0 as a forward bifurcation number and plot R_0 along the x-axis and the equilibrium density of infected predator z along the y-axis. Figure 1 reveals that disease does not exist in the system when $R_0 < 1$.By the definition of the forward bifurcation, the stable equilibrium point loses stability with the change of parameters and generates two new equilibrium points, one stable and the other unstable. It can be shown in the Figure 1. Therefore, there is a case of the forward bifurcation phenomenon. This phenomenon also means that small changes in parameters may cause the system to transition from one equilibrium point to another when $R_0 > 1$. Epidemiologically, forward bifurcation indicates

that the initial population size is vital for disease control, and reducing R_0 below unity can eliminate diseases to some extent.

4.2 The system with no delay

In the beginning, we will show the dynamic behavior of the system without delay, and in these equilibria, only E_4 and E_6^* will be indicated carefully (Others will be shown in the Appendix.) because these two points are the most meaningful point. E_4 means that the disease disappears and at E_6^* , all the three kinds of study objects are alive together.

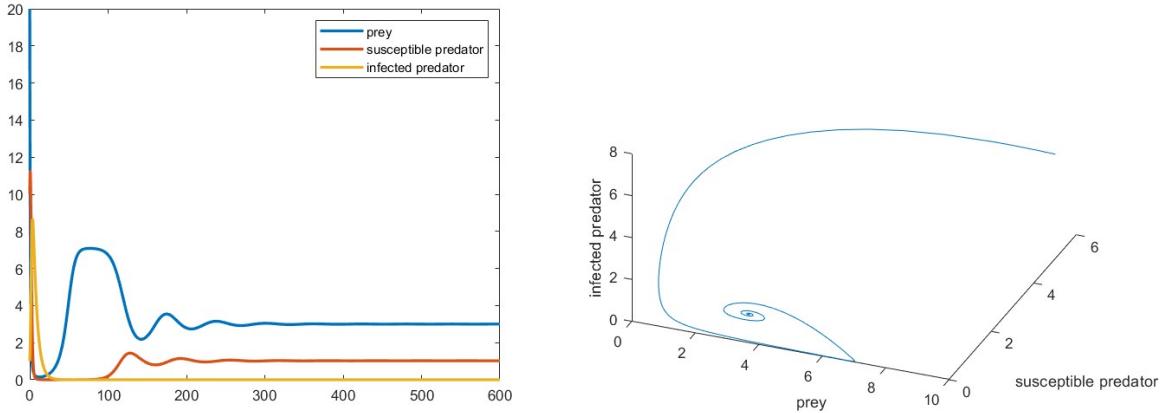


Figure 2: Behavior of the system at E_4 with $k = 15, \alpha = 0.1, m = 0.1, \mu = 0.1, d = 0.2, r = 0.95, n = 9, b = 10, p = 0.5, e = 0.333333$

Figure 2 shows a spiral of the equilibrium E_4 . With these values of parameters, the prey and the susceptible predator will live together and become a steady state, also, the infected predator will extinct, which shows the disease will disappear through long time evolution.

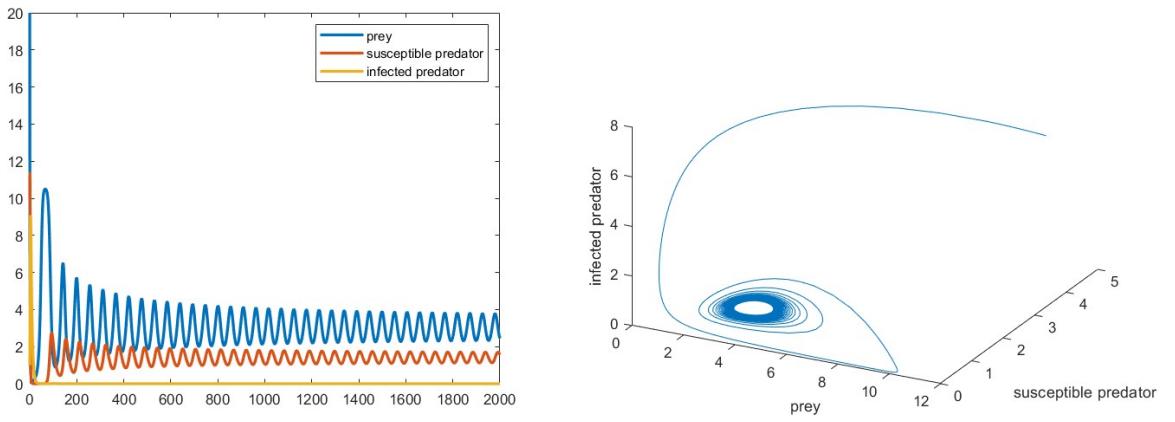


Figure 3: Behavior of the system at E_4 with $k = 18.79, \alpha = 0.1, m = 0.1, \mu = 0.1, d = 0.2, r = 0.95, n = 9, b = 10, p = 0.5, e = 0.333333$

However, if we take the larger value of k , the stability of this point will change. (Figure 3) A limit cycle will appear around the point, which means the point enters a Hopf bifurcation.

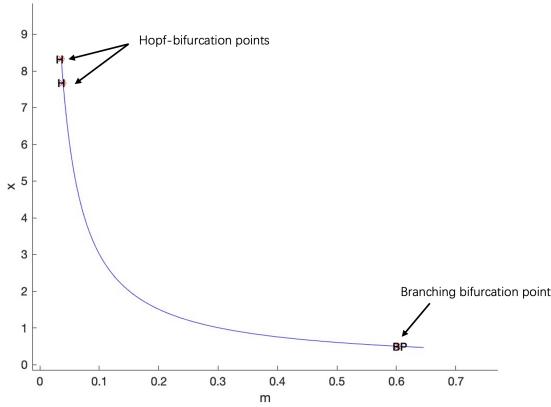


Figure 4: Bifurcation diagram around the point E_4 in $m - x$ plane.

Figure 4 illustrates two Hopf-bifurcation points and one branching bifurcation point about variable m when we investigate the stability of the system around E_4 , mainly investigating the relationship between the number of infected prey and the consumption rate of the prey. In the ecological context of this system, the presence of Hopf-bifurcation points may indicate that under certain predation rates, the infected prey population no longer remains stable but begins to exhibit periodic fluctuations. This may have significant implications for the stability and dynamics of the ecosystem, potentially affecting the overall balance and stability of the food chain. A Branching bifurcation point indicates the possibility of two different stable states in the system. In this scenario, there may exist two distinct stable states, where the infected prey population can stabilize at two different levels depending on the consumption rate of prey. This bistability may lead to several stable states in the ecosystem, potentially resulting in different ecosystem structures and functions, thereby impacting the stability and biodiversity of the ecosystem.

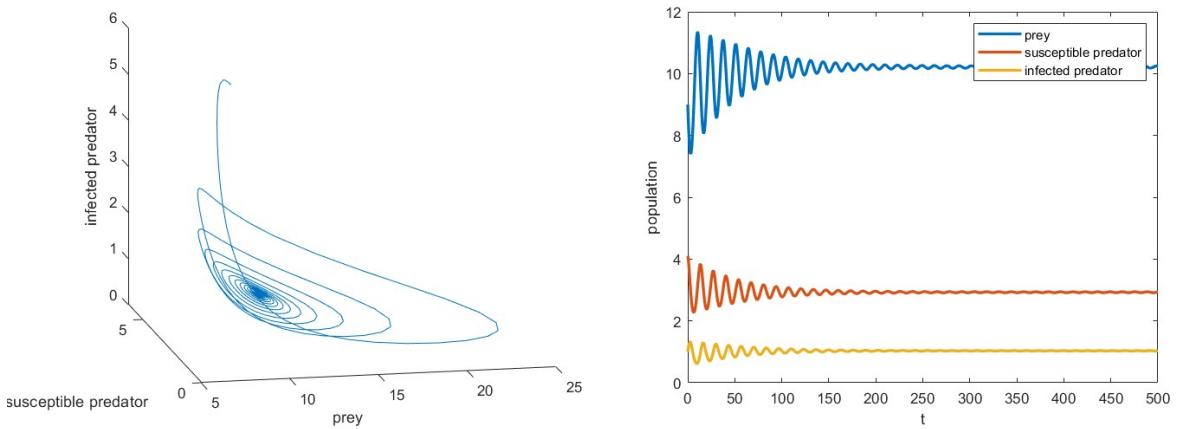


Figure 5: Behavior of the system at E_6 with $k = 50, \alpha = 0.2, m = 0.1, \mu = 0.1, d = 0.8, r = 0.5, n = 1, b = 5, p = 0.7, e = 0.3$

Figure 5 shows that when we choose the fixed parameters values: $k = 50, \alpha = 0.2, m = 0.1, \mu = 0.1, d = 0.8, r = 0.5, n = 1, b = 5, p = 0.7, e = 0.3$, we can get a stable equilibrium with prey, susceptible predator and infected predator alive.

4.3 The system with delay

Then we will put the τ in our system and choose different parameter values to illustrate some meaningful equilibria using figures, especially E_4 and E_6^* , and explain them biologically. These figures will concentrate on the behavior of the system and the bifurcation of these two points. Otherwise, we will incorporate white noise to improve this model, which can add randomness to the model.

To summarize the stability of E_4 and E_6^* , we compute the values of R_0 and τ in Table 2. From section 1, we know that when R_0 and τ are low, the system 2.1 is asymptotically stable around E_4 . But if we increase the value of delay: τ , keeping other parameters including R_0 unaltered, the system 2.1 will become unstable with a neutral saddle. Similarly, when we take $R_0 > 1$, the system 2.1 is asymptotically stable around E_6^* for small delay. Consequently, the system 2.1 remains stable for $\tau < \tau^*$ ($= 0.3$ approx.) and undergoes a Hopf bifurcation at $\tau = \tau^*$.

Equilibrium	Range of R_0	Default R_0	Values of τ	LAS	Figure No.
E_4	$R_0 < 1$	0.6284	0.00099999985	stable	Figure 6
E_4	$R_0 < 1$	0.6284	1.4293429	unstable	Figure 7
E_6^*	$R_0 > 1$	2.5875	0.001	stable	Figure 8
E_6^*	$R_0 > 1$	2.5875	1	unstable (Hopf)	Figure 9

Table 2: The stability of E_4 and E_6^* with the value of R_0 and τ

4.3.1 The point E_4

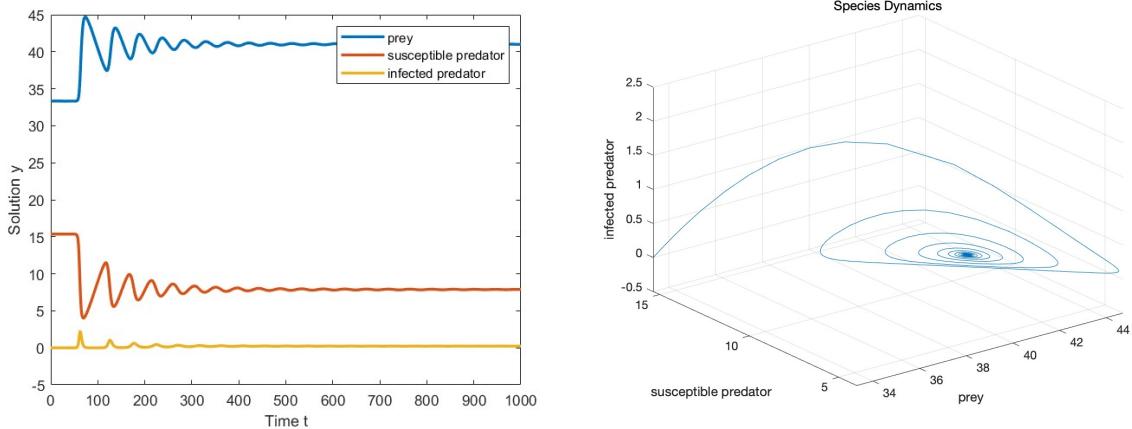


Figure 6: Behavior of the system at E_4 with $k = 50$, $\alpha = 0.1$, $m = 0.01$, $\mu = 0.1$, $d = 0.8$, $r = 0.5$, $n = 1$, $b = 5$, $p = 0.1$, $e = 0.3$, where $\tau = 0.00099999985$

Figure 6 and 7 clearly show the dynamics of the system transferring from stability to a bifurcation. Before the branching bifurcation point occurs, the stable state of the system is characterized by the absence of infected predators (with a quantity of 0), while the populations of prey and susceptible predators are both greater than 0.

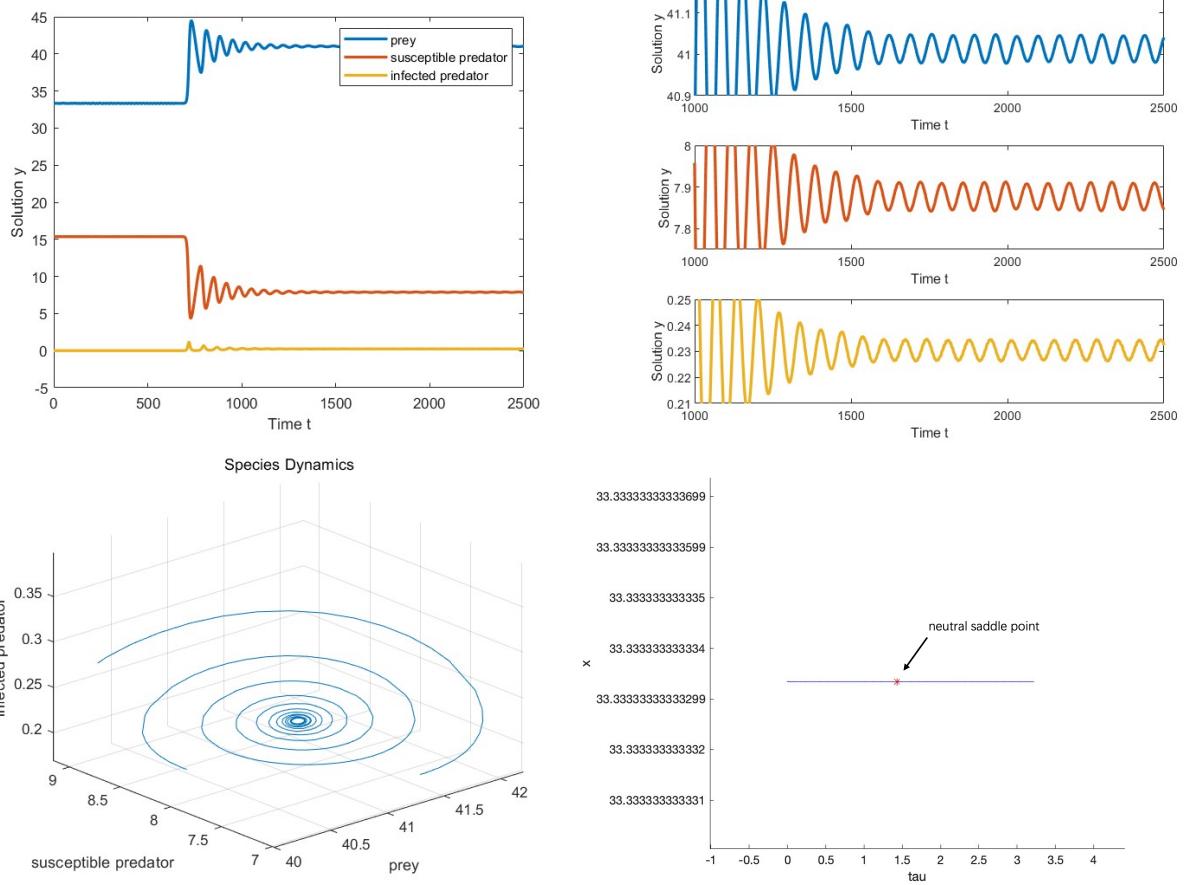


Figure 7: Behavior of the system at E_4 with $k = 50$, $\alpha = 0.1$, $m = 0.01$, $\mu = 0.1$, $d = 0.8$, $r = 0.5$, $n = 1$, $b = 5$, $p = 0.1$, $e = 0.3$, where $\tau = 1.4293429$ and Bifurcation diagram of the infected prey population with respect to the delay τ around E_4 , in $x - \tau$ plane.

Biologically, the branching bifurcation point signifies a critical transition in the ecosystem dynamics. It suggests that the system has entered a regime where periodic oscillations in the populations of the species occur. This could be due to the emergence of feedback mechanisms, predator-prey interactions, or other ecological factors that drive cyclic behavior in the populations of the involved species. The branching bifurcation point marks a pivotal moment where the system's stability undergoes a qualitative change, leading to the emergence of periodic dynamics in the population sizes of prey, susceptible predators, and infected predators.

We also construct a bifurcation diagram (Figure 7) to investigate the relationship between bifurcation and the value of τ in detail. It is clear from the bifurcation diagram that when the delay τ exceeds the critical value $\check{\tau}$ (= 1.5 days approx.), the system around E_4 bifurcates from steady state to unstable state. From Matcont, we know that this critical value point is the neutral saddle. To be more precise, the system will shift from E_4 to E_6^* through simple bifurcation, which is shown in Figure 6 and Figure 7. Notably, in this case, E_6^* is also not an equilibrium value, which enters to Hopf bifurcation at $\check{\tau} = 1.5$ days approx.

4.3.2 The point E_6^*

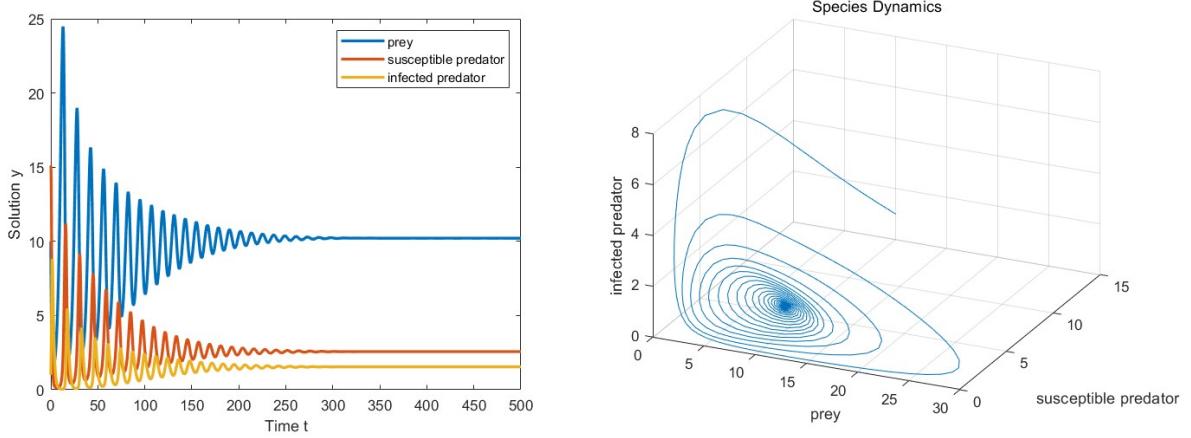


Figure 8: Behavior of the system at E_6^* with $k = 50$, $m = 0.1$, $\alpha = 0.2$, $\mu = 0.1$, $d = 0.8$, $r = 0.5$, $n = 1$, $b = 5$, $p = 0.7$, $e = 0.4$ where $\tau = 0.001$.

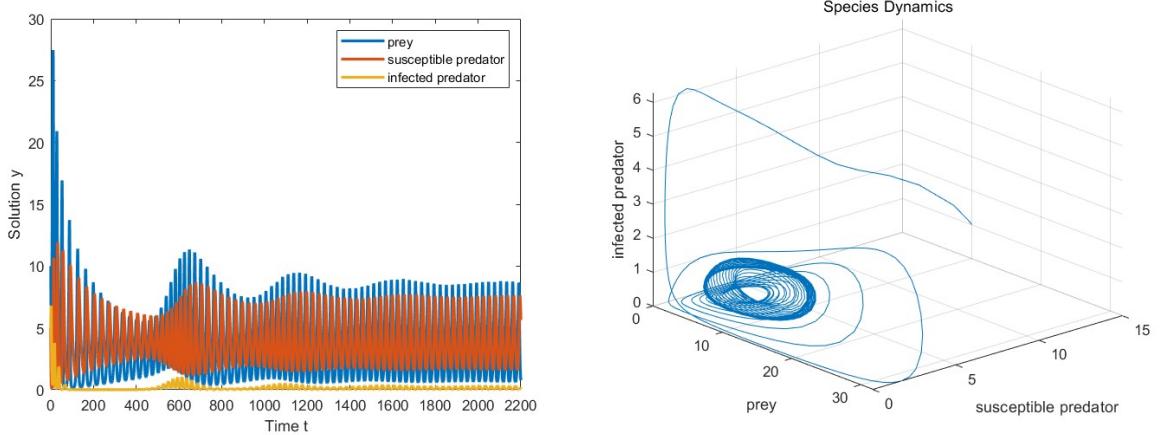


Figure 9: Behavior of the system at E_6^* with $k = 50$, $\alpha = 0.2$, $m = 0.1$, $\mu = 0.1$, $d = 0.8$, $r = 0.5$, $n = 1$, $b = 5$, $p = 0.7$, $e = 0.4$ and $\tau = 1$

In Figure 10, we investigate the dynamics of the system when τ varies, especially focused on the second hopf bifurcation point which is around 12. It illustrates successive numbers of the infected prey population. The figure clearly shows that when the delay τ exceeds its critical value $\check{\tau} = 12.145$, the system bifurcates and the amplitude of oscillation increases with increasing τ .

At the equilibrium point E_6^* , the left picture of Figure 8 shows a stable state with prey, susceptible predator, and infected predator surviving together. Choosing these parameters: $k = 50$, $m = 0.1$, $\alpha = 0.2$, $\mu = 0.1$, $d = 0.8$, $r = 0.5$, $n = 1$, $b = 5$, $p = 0.7$, $e = 0.4$ and $\tau = 0.001$, the condition of Theorem 9 is satisfied, consequently, the system (2.1) remains stable for $\tau < \tau^*$ ($=0.3$ approx.) and unstable when τ exceeds τ^* . At $\tau = \tau^*$, a Hopf bifurcation will occur. To observe the dynamics of the system, we also construct a bifurcation figure of E_7 . It is clear that when the delay τ exceeds the τ^* , the system (2.1) bifurcates from the stable state to a limit cycle, showing in Figure 9.

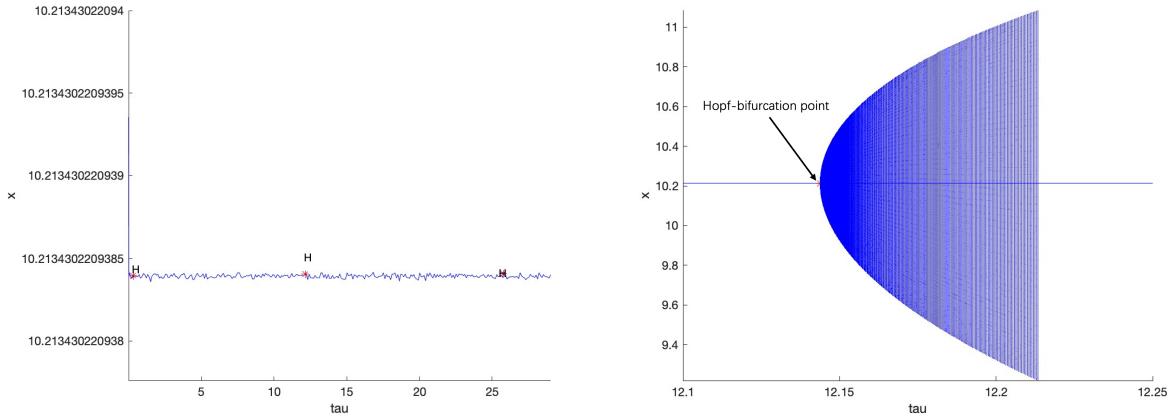


Figure 10: Bifurcation diagram of the infected prey population with respect to the delay τ around E_6^* in $x - \tau$ plane

In this case, as τ is very small, the system is almost the same as the ODE system (Figure 5), because we can ignore τ approximately. However, if we increase the value of τ , the condition will change, and the large τ influence the spread of disease seriously. When the predators infect the disease, they will not transform directly, but have a period to incubate, then at some time, a group of infected predators will manifest, which will cause the number of susceptible predators to decrease and the number of prey to increase. After that, due to the high death rate of infected predators, the effect from the number of them can only exist periodically, so the system (2.1) will begin to oscillate.

4.3.3 Two parameters analysis around E_6^*

Through Matlab, we compute the Jacobian matrix of the system around E_6^* to investigate the stability regions of two parameters: μ and e . In this Figure 11, we construct a two-dimensional grid to represent a pair of parameter values: μ and e . For each pair of μ and e , the blue points represent stable cases, whereas, the yellow points represent unstable cases. To compute this Figure, we begin with stable cases for appropriate μ and e , where all of their eigenvalues of the Jacobian matrix obtain negative real parts. After changing the vector $\langle \mu, e \rangle$, we get different Jacobian matrices with different eigenvalues. Once real parts of one of the eigenvalues become positive, the system turns to be unstable from the steady state. We update these results in Figure 11 and we can know that when the vector $\langle \mu, e \rangle$ falls within the region in the top right corner, the system is stable around E_6^* . To be more precise, a high amount of death rate μ demonstrates the stable nature of this equilibrium for a relatively low convention rate e .

5 Optimization of the model

Biological systems are often subject to various sources of randomness, such as environmental fluctuations, genetic variability, and demographic stochasticity. Incorporating white noise helps make the model more realistic by accounting for these random effects. White

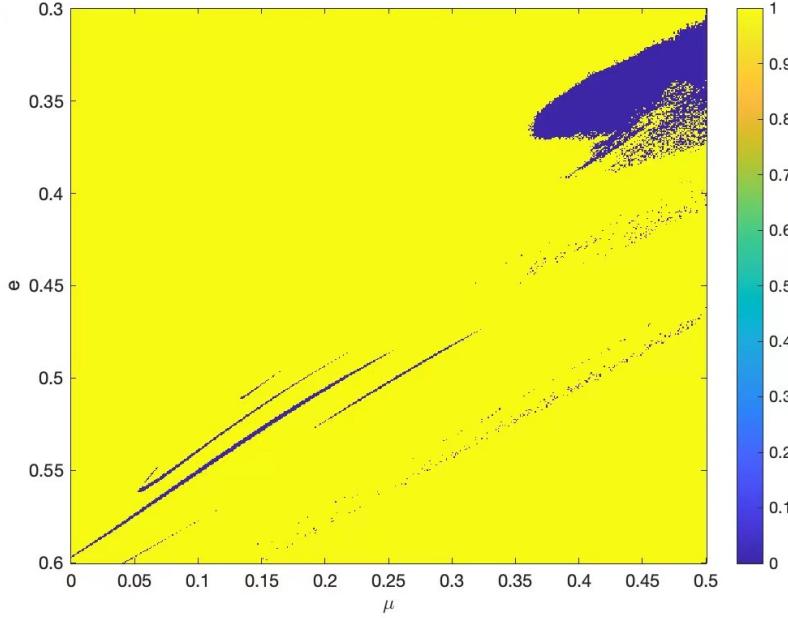


Figure 11: Stability region of the interior point E_6^* in $\mu - e$ parameter plane, where $\tau = 10$

noise introduces randomness into the model dynamics, which can lead to more accurate predictions by accounting for unpredictable events or perturbations that may occur in the system. This can be particularly important when modeling complex biological systems where deterministic models may fail to capture all sources of variability[4].

We analyze the Delay system around E_6 . Thus, we reconstructed our model as follows.

$$\begin{aligned} dx &= F_1(x, y, z) dt + \sigma_x(x - x_7) d\eta_t^{(1)}; \\ dy &= F_2(x, y, z) dt + \sigma_y(y - y_7) d\eta_t^{(2)}; \\ dz &= F_3(x, y, z) dt + \sigma_z(z - z_7) d\eta_t^{(3)}; \end{aligned} \quad (5.1)$$

where σ_i 's are real constants and are known as the intensities of environmental fluctuations; η denotes noise (also known as increments of Brownian motion), which obeys $\langle \eta_t^{(i)} \rangle = 0$ and standard Wiener processes independent of each other (standard Brownian motion) [10]. Then We use the Euler method to simulate the process.

We first introduce a proper variable to transform our delay items into an ordinary differential equation.

$$\frac{dz(t)}{dt} = F_3(x, y(t - \tau), z(t - \tau)) \quad (5.2)$$

Then we define two new variable, namely Y and Z , where $Y(t) = y(t - \tau)$ and $Z(t) =$

$z(t - \tau)$. And now,

$$\frac{dZ(t)}{dt} = \frac{dz(t)}{dt} = F_3(x, Y, Z) \quad (5.3)$$

Then we can get a new system to implement Euler method.

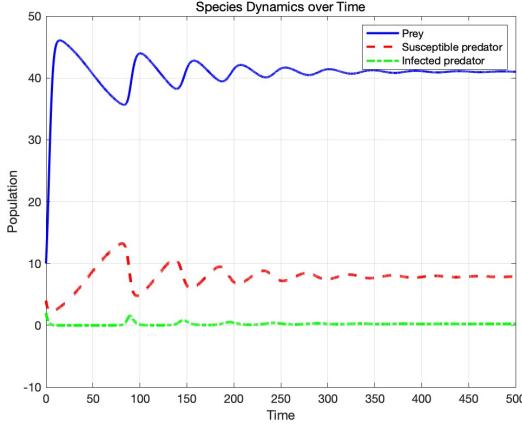


Figure 12: Stochastic process using Euler method around E_4

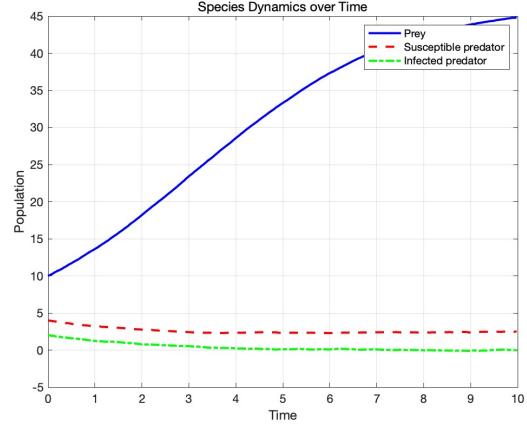


Figure 13: Stochastic process using Euler Maruyama method

However, when dealing with smaller time steps and appropriately selected random term coefficients, the Euler-Maruyama method could be a better choice. Where the general process is modified to

$$X_{n+1} = X_n + \delta t \cdot f(t_n, X_n) + \sqrt{\delta t} \cdot g(t_n, X_n) \eta_t^{(i)} \quad (5.4)$$

As is indicated in figure 14, in some cases, the stability of the interior point transfers its property to E_4 , where infected predators die out.

This method allows us to simulate the impact of random terms at each time step and update the system state based on its impact[16]. Combining this method into our program, Figure 14 illustrates the dynamics of the system, while it only works in a relevant short period. We also implement the same process for 50 times, the introduction of random terms in stochastic process simulation represents the uncertainties and random fluctuations in the environment. By repeating the simulation, we can consider and analyze the impact of this randomness on population sizes. Each repetition may yield different outcomes, allowing us to identify and understand the role of stochastic factors. Also, we can check the rationality of the simulation method by observing the range of variation.

6 Conclusion

In the table 3 below, we summarize all the situations for the equilibria of ODEs systems without the existence of delay.

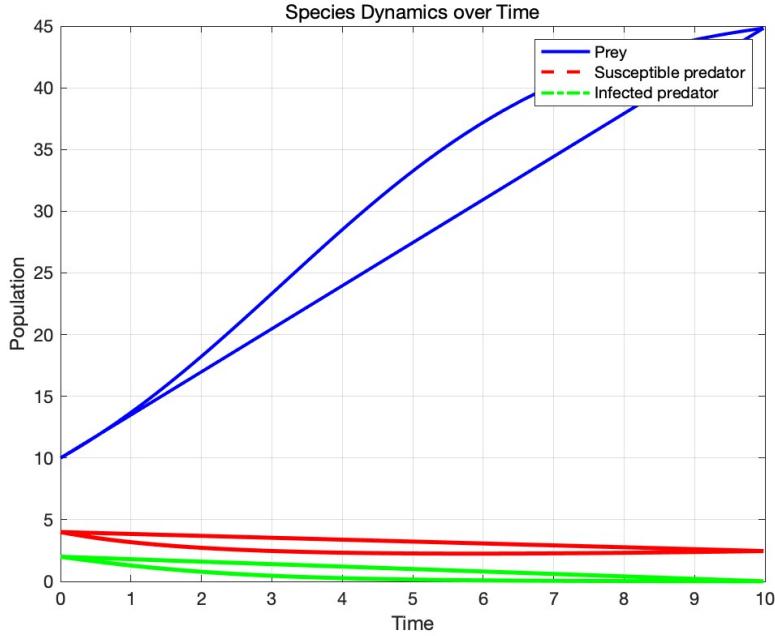


Figure 14: Implementing Euler-Maruyama method 50 times to see the range of variation

It is not hard to find that E_0 is always an equilibrium for the system (1.1), and it is always feasible. In the Proof 1., we conclude that E_0 is locally asymptotically stable if $n > b$ and $\mu > 0$ (i.e. there are additional food sources for the predators outside the system). The fact that E_1 and E_2 share some similarities is due to their common originality from the equation: $x^2 + (b - k)x + (n - b)k = 0$. x_1 is stable if the LAS conditions are satisfied, but E_2 is always unstable since $x_2 < 0$. Similarly, we can obtain E_6 and E_7 , and E_7 is always unstable for the same reason. The proof of LAS conditions of the stability can be checked at theorem 4. Among all other points, we have additionally prove the global stability of the point E_4 at theorem 7 with the help of theorem 6 and lemma 1.

This study employs mathematical modeling, theoretical analysis, and numerical simulation to investigate in depth the predator-prey system that includes the Allee effect and delay. Our goal is to understand the stability of delay-affected systems and how to find ways to suppress disease outbreaks.

In the mathematical modeling stage, we integrate the time delay of predator disease transmission into the classic Lotka -Volterra model. This model includes the intrinsic growth and interactions of prey and takes into account their relationships. The model is formalized as a set of delay differential equations, where τ as the delay coefficient is a key parameter affecting system stability.

In the theoretical analysis stage, we delved into the stability conditions and Hopf bifurcation analysis of the extended model to determine the conditions for the system to transition from a stable state to an unstable state under certain coefficients. We also analyzed the properties of a system such as permanence and boundedness through mathematical formulas and fundamental theorems. The local stability of the equilibrium point of the no-time-delay system was determined through the Jacobian matrix and eigenvalue analysis. However,

Equilibrium	Feasible Conditions	LAS Conditions
$E_0 \equiv (0, 0, 0)$	Always	$n > b$ and $\mu > 0$
$E_1 \equiv (x_1, 0, 0)$ $x_1 = \frac{\sqrt{(k+b)^2 - 4kn + (k-b)}}{2}$	$k > b$ $(k+b)^2 > 4kn$	$x_1 < \min\left\{\frac{\mu}{em}, \frac{d}{emp}\right\}$
$E_2 \equiv (x_2, 0, 0)$ $x_1 = \frac{-\sqrt{(k+b)^2 - 4kn + (k-b)}}{2}$	$(k+b)^2 > 4kn$ $k > b + \sqrt{(k+b)^2 - 4kn}$	always unstable
$E_3 \equiv (\frac{d}{emp}, 0, z_3)$ $x_3 = \frac{d}{emp}$ $z_3 = \frac{r}{mp}(1 - \frac{x_3}{k} - \frac{n}{x_3+b})$	$1 - \frac{x_3}{k} - \frac{n}{x_3+b} > 0$	$\frac{d}{p} - z_3\alpha - \mu < 0$ $(b + \frac{d}{emp})^2 > nk$
$E_4 \equiv (\frac{\mu}{em}, y_4, 0)$ $x_4 = \frac{\mu}{em}$ $y_4 = \frac{r}{m}(1 - \frac{x_4}{k} - \frac{n}{x_4+b})$	$\frac{\mu}{m}(1 - \frac{x_4}{k} - \frac{n}{x_4+b}) > 0$	$y_4\alpha - \mu - d < 0$ and $(x_4 + b)^2 > nk$
$E_5 \equiv (0, \frac{d}{\alpha}, -\frac{\mu}{\alpha})$	$\mu < 0$	always unstable
$E_6 \equiv (x_6^*, y_6^*, z_6^*)$	$\Delta_2 > 0$ $\Delta_2 = (V + \frac{rb}{k})^2 - \frac{4r}{k}(Vb + rn)$ $-(V + \frac{rb}{k}) + \sqrt{\Delta_2} > 0$ $V = \frac{(d-p\mu)m-r\alpha}{\alpha}$	$\frac{d}{emp} < x_6^* < \max\{0, \frac{\mu}{em}\}$
$E_7 \equiv (x_7^*, y_7^*, z_7^*)$ $x_{6,7}^* = \frac{k(-V + \frac{rb}{k})pm\sqrt{\Delta_2}}{2r}$ $y_{6,7}^* = \frac{d-empx_{6,7}^*}{\alpha}$ $z_{6,7}^* = \frac{emx_{6,7}^*\alpha - \mu}{\alpha}$	$\Delta_2 > 0$ $\Delta_2 = (V + \frac{rb}{k})^2 - \frac{4r}{k}(Vb + rn)$ $-(V + \frac{rb}{k}) - \sqrt{\Delta_2} > 0$	always unstable

Table 3: Conditions of feasibility and local asymptotic stability for all ODE equilibrium

when introducing time delay, we found that the system may lose stability and Hopf bifurcation may occur. Specifically, we derived the characteristic equation of the delay system and used Rouche's theorem and continuity conditions to determine the critical delay value τ^* for system stability. We further validated the Hopf bifurcation condition and provided an example to illustrate how the system transitions from stability to periodicity. These results provide important references for further studying the dynamic behavior of time-delay predator-prey systems.

In the numerical simulation section, we delved into the transition of two special points from stable to unstable states. In the simulation, these images reflect how predators alter the distribution of resources by altering the number of predators through time delay. We constructed a bifurcation diagram to study the relationship between bifurcation and time delay coefficient τ , including saddle bifurcation and branching bifurcation. We found that

$R_0 = 1$ is the critical value of the stability and they can be shown in Figure 6 and Figure 7. Additionally, the point E_6^* can be affected by the time delay coefficient τ which is shown in Figure 8 and Figure 9. However we found that the predictor of y is extremely prone to extinction, and we should make changes to the system model, such as adding the birth rate of predators based on the Lotka-Volterra model.

The optimization of the model through the incorporation of stochastic elements has provided valuable insights into the dynamics of biological systems. By introducing white noise and employing simulation techniques such as the Euler-Maruyama method, we have been able to capture the effects of random fluctuations in the environment on the behavior of the system. Furthermore, comparing the results obtained using the Euler method and the Euler-Maruyama method has highlighted the advantages of the latter, particularly when dealing with smaller time steps and appropriately selected random term coefficients. The Euler-Maruyama method allows for a more accurate representation of the impact of random fluctuations at each time step, resulting in a more realistic simulation of the system dynamics.

References

- [1] A. Berryman Alan. The origins and evolution of predator-prey theory. pages 1530–1535, 1992.
- [2] FathallaA.R. *Delay Differential Equations and Applications to Biology*. 2021.
- [3] F.Courchamp, L. Berec, and J.Gascoigne. Allee Effects in Ecology and Conservation. *Journal of Mammalogy*, 91:1530–532, 2008.
- [4] M. Giannakou1 and B. Waclaw. Resonant noise amplification in a predator-prey model with quasi-discrete generations. page 21, 2024.
- [5] J.A. Hebatallah, K. Soumen, and A.R. Fathalla. Delay differential model of one-predator two-prey system with Monod-Haldane and holling type II functional responses. *Applied Mathematics and Computation*, 397, 2021.
- [6] Junping S. Junjie W. Jinfeng, W. Predator-prey system with strong Allee effect in prey. *J. Math. Biol.*, 2010.
- [7] W. Jinfeng and Junjie. Bifurcation analysis of a delayed predator-prey system with strong Allee effect and diffusion. *Applicable Analysis*, 91:1219–1242, 2011.
- [8] A.J. Lotka. Elements of physical biology. 1925.
- [9] M. MacDonauld. Biological Delay Systems: Linear Stability Analysis. 1989.
- [10] H. Mainul. A detailed study of the Beddington–DeAngelis predator–prey model. *Mathematical Biosciences*, 234(7):1–16, 2011.
- [11] H. Mainul, S. Sahabuddin, and V. Simon, P. Ezio. Effect of delay in a Lotka–Volterra type predator–prey model with a transmissible disease in the predator species. *Mathematical Biosciences*, 234:47–57, 2011.
- [12] Len N. The effect of long time delays in predator-prey systems. *Theoretical Population Biology*, 27(2):201–221, 1985.
- [13] Leopoldo Nachbin. *J.DieudonnéFoundations of Modern Analysis*, volume 12. 1960.
- [14] N.Bairagi, R.R.Sarkar, and J.Chattopadhyay. Impacts of incubation delay on the dynamics of an eco-epidemiological system-a theoretical study. *Bulletin of Mathematical Biology*, 2008.
- [15] N.Macdonald. Time delay in prey-predator models. *Mathematical Biosciences*, 28:321–330, 1976.
- [16] Z. Noah and Z. David. Heterogeneous Nucleation and Growth of Sessile Chemically Active Droplets. 2024.
- [17] F. A. Rihan, A. A. Azamov, and H. J. Al-Sakaji. An Inverse problem for delay differential equations: parameter estimation, nonlinearity, sensitivity. 12:63–74, 2018.

- [18] B. Santanu, K.S. Sourav, S.B. Sudip, Md Saifuddina, J.A.K. Qamar, and C. Joydev. A delayed eco-epidemiological system with infected prey and predator subject to the weak Allee effect. *Mathematical Biosciences*, 263:198–208, 2015.
- [19] L.A. Segel and J.L. Jackson. A Delay-differential equation model of HIV infection of CD4+ T-cells. *Math Bioscience*, 165:27–39, 2003.
- [20] S.K. Sasmal and J. Chattopadhyay. An eco-epidemiological system with infected prey and predator subject to the weak Allee effect. *Math Bioscience*, 246:260–271, 2013.
- [21] Partha S.M. Udai K. Role of Allee effect on prey–predator model with component Allee effect for predator reproduction. *Mathematics and Computers in Simulation*, 193:623–665, 2022.
- [22] V. Volterra. Fluctuations in the abundance of species. *Nature*, pages 118–588, 1926.
- [23] M. Wang and M. Kot. Speeds of invasion in a model with strong or weak Allee effects. *Mathematical Biosciences*, 171:83–97, 2001.
- [24] Y. Kuang. Delay Differential Equations: With Applications in Population Dynamics. 191, 1993.

A Appendix

A.1 Stability analysis of the system with delay

To study the stability of these two interior equilibrium points, let us define the characteristic equation of the system is given by

$$\left| A'_1 + A'_2 e^{-\lambda\tau} - \lambda I \right| = 0$$

That is,

$$\phi(\lambda, \tau) = \lambda^3 + [A + Be^{-\lambda\tau}] \lambda^2 + [C + De^{-\lambda\tau}] \lambda + E + Fe^{-\lambda\tau} = 0 \quad (\text{A.1})$$

Where

$$\begin{aligned} A &= - \left[rx_i^* \left(\frac{n}{(x_i^* + b)^2} - \frac{1}{k} \right) + empx_i^* - d \right] \\ B &= -\alpha y_i^* \\ C &= em^2 x_i^* y_i^* + em^2 p^2 x_i^* z_i^* + rx_i^* \left(\frac{n}{(x_i^* + b)^2} - \frac{1}{k} \right) (empx_i^* - d) \\ D &= \alpha^2 y_i^* z_i^* + \alpha r x_i^* y_i^* \left(\frac{n}{(x_i^* + b)^2} - \frac{1}{k} \right) \\ E &= -\alpha em^2 p x_i^* y_i^* z_i^* - em^2 x_i^* y_i^* (empx_i^* - d) \\ F &= -\alpha^2 r x_i^* y_i^* z_i^* \left(\frac{n}{(x_i^* + b)^2} - \frac{1}{k} \right) - \alpha em^2 x_i^* y_i^{*2} + \alpha em^2 p x_i^* y_i^* z_i^* \end{aligned}$$

If there is no delay, i.e. $\tau = 0$, then the equation (3.2) becomes

$$\phi(\lambda, \tau) = \lambda^3 + [A + B]\lambda^2 + [C + D]\lambda + E + F = 0 \quad (\text{A.2})$$

Following the Routh-Hurwitz criteria, we need to calculate $X = A + B$, $Y = C + D$, $Z = E + F$ and the sign of the determinant $\begin{bmatrix} X & Z \\ 1 & Y \end{bmatrix}$.

$$X = rx^*(\frac{1}{k} - \frac{n}{(b+x^*)^2})$$

Note that X is only positive when $nk < (x^* + b)^2$.

$$Y = \alpha^2 y^* z^* + em^2 p^2 x^* z^* + em^2 x^* y^*$$

Note that Y is always positive as our initial assumption for all parameters to be positive except for μ .

$$\begin{aligned} Z &= -(\alpha^2)rx^*y^*z^*(\frac{n}{(b+x^*)^2} - \frac{1}{k}) - \alpha em^2 x^*(y^*)^2 - em^2 x^* y^*(-d + emp x^*) \\ &= -\alpha^2 rx^*y^*z^*(\frac{n}{(b+x^*)^2} - \frac{1}{k}) \end{aligned}$$

Z is positive if and only if $nk < (x^* + b)^2$.

$$\begin{aligned} XY - Z &= (rx^*(\frac{1}{k} - \frac{n}{(b+x^*)^2}) \cdot (\alpha^2 y^* z^* + em^2 p^2 x^* z^* + em^2 x^* y^*)) \\ &\quad - (-(\alpha^2)rx^*y^*z^*(\frac{n}{(b+x^*)^2} - \frac{1}{k}) - \alpha em^2 x^*(y^*)^2 - em^2 x^* y^*(-d + emp x^*)) \\ &= -rx^*(\frac{n}{(b+x^*)^2} - \frac{1}{k})(em^2 x^* y^* + em^2 p^2 x^* y^* + \alpha^2 y^* z^*) \\ &\quad - (-rx^*(\frac{n}{(b+x^*)^2} - \frac{1}{k})\alpha^2 y^* z^*) \\ &= -rx^*(\frac{n}{(b+x^*)^2} - \frac{1}{k})(em^2 x^* y^* + em^2 p^2 x^* y^*) \end{aligned} \quad (\text{A.3})$$

As all the parameters except μ are positive, then the sign of $XY - Z$ depends completely on the term $-rx^*(\frac{n}{(b+x^*)^2} - \frac{1}{k})$. Hence, if and only if $\frac{1}{k} - \frac{n}{(x^*+b)^2} > 0$, i.e. $k < \frac{b^2}{n}$, $XY - Z$ would be positive. To summarize, if $nk < (x^* + b)^2$, then $X, Y, Z, XY - Z$ are positive. Combined with Routh-Hurwitz criteria, all the roots of the polynomial functions ?? are negative or have negative real parts. This implies the stability of the equilibrium if the above condition is satisfied.

However, for the point $E_7(x_7^*, y_7^*, z_7^*)$, $\frac{1}{k} - \frac{n}{(x^*+b)^2}$ is strictly less than zero, as $(x^* + b)^2 - kn = \frac{2k^2\sqrt{\Delta_2}\cdot(\sqrt{\Delta_2}-(\frac{rb}{k}-V))}{4r^2}$ is smaller than naught. We here reach a conclusion that the point E_7

is unstable.

For Point $E_7(x_6^*, y_6^*, z_6^*)$ $E_7(x_7^*, y_7^*, z_7^*)$, $\frac{1}{k} - \frac{n}{(x^*+b)^2}$ is strictly greater than zero, as $(x^* + b)^2 - kn = \frac{2k^2\sqrt{\Delta_2} \cdot (\sqrt{\Delta_2} - (\frac{rb}{k} - V))}{4r^2}$ is greater than naught. Therefore, the system without delay is locally asymptotically stable around the positive interior equilibrium E_6

However, for the point $E_7(x_7^*, y_7^*, z_7^*)$, $\frac{1}{k} - \frac{n}{(x^*+b)^2}$ is strictly less than zero (similar to E_6). We here reach a conclusion that the point E_7 is unstable.

A.2 Variable Gradient Method

A logical and systematic method of generating Lyapunov functions for determining stability of nonlinear autonomous systems is introduced. The method is based upon the assumption of a variable gradient function from which both V and V' may be determined. The n -unknown elements of each of the n -components of the variable gradient are determined from constraints on V and $\frac{n(n-1)}{2}$ generalized curl equations.

To begin with, the equilibrium point E_4 contains no infected predators ($z = 0$), then the system (1.1) becomes:

$$\begin{aligned}\frac{dx}{dt} &= rx\left(1 - \frac{x}{k} - \frac{n}{x+b}\right) - mxy \\ \frac{dy}{dt} &= emxy - y\mu\end{aligned}$$

We assume an arbitrary

$$\text{div } V = \begin{pmatrix} a_{11}x + a_{12}y \\ a_{21}x + a_{22}y \end{pmatrix}$$

And,

$$\begin{aligned}V' &= \frac{\partial V}{\partial x} \cdot \frac{dx}{dt} + \frac{\partial V}{\partial y} \cdot \frac{dy}{dt} \\ &= (a_{11}x + a_{12}y)(rx\left(1 - \frac{x}{k} - \frac{n}{x+b}\right) - mxy) + (a_{21}x + a_{22}y)(emxy - y\mu) \\ &= ((e - \frac{\mu}{km})x + y) \cdot (rx\left(1 - \frac{x}{k} - \frac{n}{x+b}\right) - mxy) + (x + \frac{2\mu}{er}y)(emxy - y\mu) \\ &= -\frac{a_{11}r}{k}x^3 + a_{11}rx^2 - \frac{a_{11}nr}{x+b}x^2 - (a_{11}x + a_{12}y)mxy + a_{12}rxy\left(1 - \frac{x}{k} - \frac{n}{x+b}\right) \\ &\quad + (a_{21}x + a_{22}y)emxy - a_{21}\mu xy - a_{22}\mu y^2\end{aligned}$$

We assume a_{11} and a_{22} positive to make V' to be negative definite with some probabilities. Besides, we hypothesize

$$a_{21} = \frac{1}{e}a_{11}, \quad a_{22} = \frac{1}{e}a_{12}$$

to cancel out the term $a_{11}rx^2 - \frac{a_{11}nr}{x+b}x^2 - (a_{11}x + a_{12}y)mxy$ and $(a_{21}x + a_{22}y)emxy$. Then we are only required to construct some proper values of aii , for $i = 1, 2$ within the following parts,

$$\begin{aligned}\Xi(x, y) &= a_{11}rx^2 - \frac{a_{11}nr}{x+b}x^2 + a_{12}rxy - \frac{a_{12}r}{k}yx^2 - a_{12}rxy\frac{n}{x+b} - a_{21}\mu xy \\ &= (a_{11}r - \frac{a_{12}r}{k})x^2 + (a_{12}r - a_{21}\mu)xy + (-a_{11}r - a_{12}rxy)\frac{n}{x+b}x^2\end{aligned}$$

To make $\Xi(x, y)$ negative definite, we reach the following relationships,

$$\begin{aligned}a_{11}r - \frac{a_{12}r}{k}y &\leq 0 \\ a_{12}r - a_{21}\mu &\leq 0\end{aligned}$$

Following the above analysis, we then have the following results given criteria,

1. $\frac{\mu}{re} \geq \frac{1}{e}$
2. $y \geq ke$

And we need our function satisfying the curl requirements at $n = 2$,

$$\frac{\partial \Delta V_1}{\partial x_2} = \frac{\partial \Delta V_2}{\partial x_1}$$

Hence, we reach our desired Lyapunov function,

$$\text{div } V = \begin{pmatrix} x + \frac{1}{e}y \\ \frac{1}{e}x + \frac{1}{e^2}y \end{pmatrix}$$

Then, by gradient variable method, we have successfully configured the Lyapunov function.

$$V = \frac{1}{2}x^2 + \frac{1}{e}xy + \frac{1}{2e^2}y^2$$

A.3 Figures of the system with no delay

From the above proof, when $\tau = 0$, E_0 , E_1 , E_4 can be stable at their own variables value. For the E_0 , if μ is larger than 0 and n is larger than b , E_0 can be stable and all species will extinct, indicated by Figure 15.

Equilibrium E_1 and E_2 is a pair of points but due to the signal of δ , E_1 can be stable at some situation, but E_2 can not be stable at any time. At this point, the prey can be alive and all the predators whatever it is susceptible predator or infected predator will extinct (Figure 16).

At point E_3 , in this case, after many years evolution, there are only prey and infected predator alive and the susceptible predator will extinct (Figure 17).

For the Figure 18, it is special equilibrium with $x = 0$, and y, z survive. From the analysis part, it is clear to know that in this case, the dynamic system will enter the hopf bifurcation

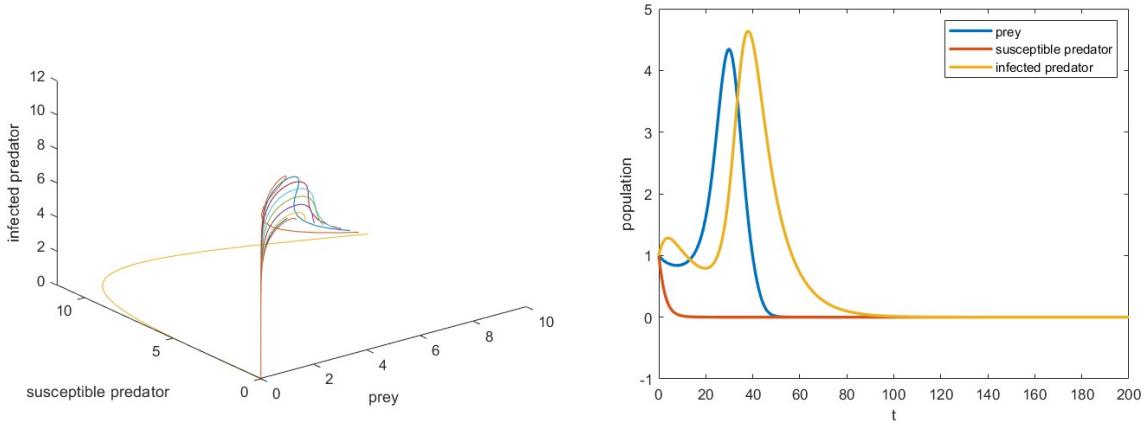


Figure 15: Behavior of the system at E_0 with $k = 10, \alpha = 0.2, m = 0.1, \mu = 0.21, d = 0.1, r = 0.5, n = 1, b = 0.6, p = 0.9, e = 0.7$

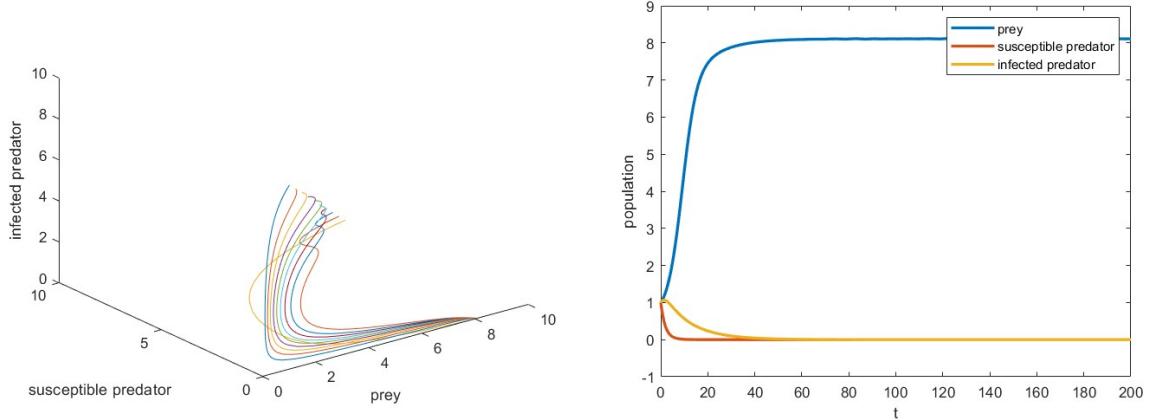


Figure 16: Behavior of the system at E_1 with $k = 9, \alpha = 0.2, m = 0.1, \mu = 0.31, d = 0.1, r = 0.5, n = 1, b = 2, p = 0.9, e = 0.02$

directly, because it has two eigenvalues with only imaginary part. Moreover, this system can only have the limit cycle with low infection rate (α), if we choose the larger α the limit cycle will disappear and the system will tend to another equilibrium (Figure 19). The reason is that the infected predators eat less prey than the susceptible predators and then prey have better survival condition. Hence, the number of prey will increase and the number of infected predator will decrease. In other words, the power of disease is smaller when α become smaller.

A.4 Figure of the system with delay

Figure 20 is a Hopf bifurcation in E_5

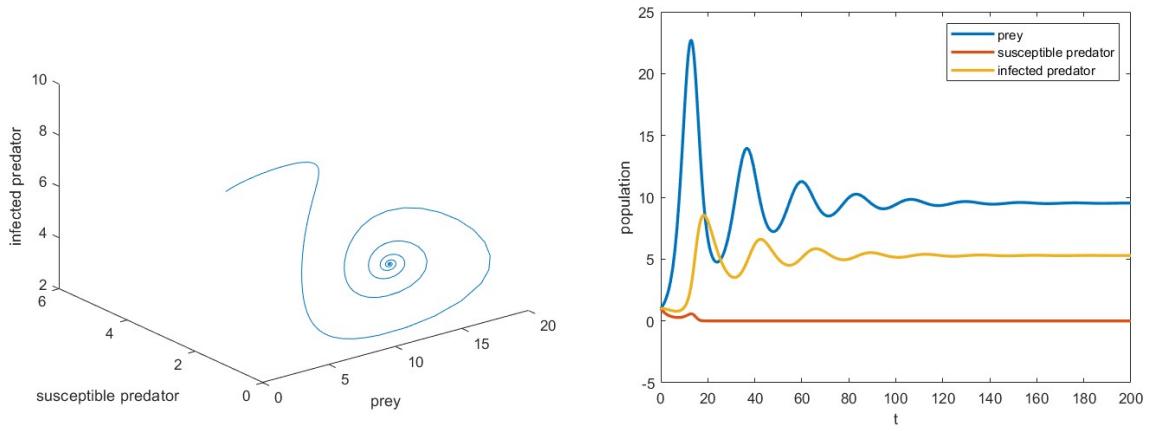


Figure 17: Behavior of the system at E_3 with $k = 50, \alpha = 0.2, m = 0.1, \mu = 0.1, d = 0.2, r = 0.5, n = 1, b = 5, p = 0.7, e = 0.3$

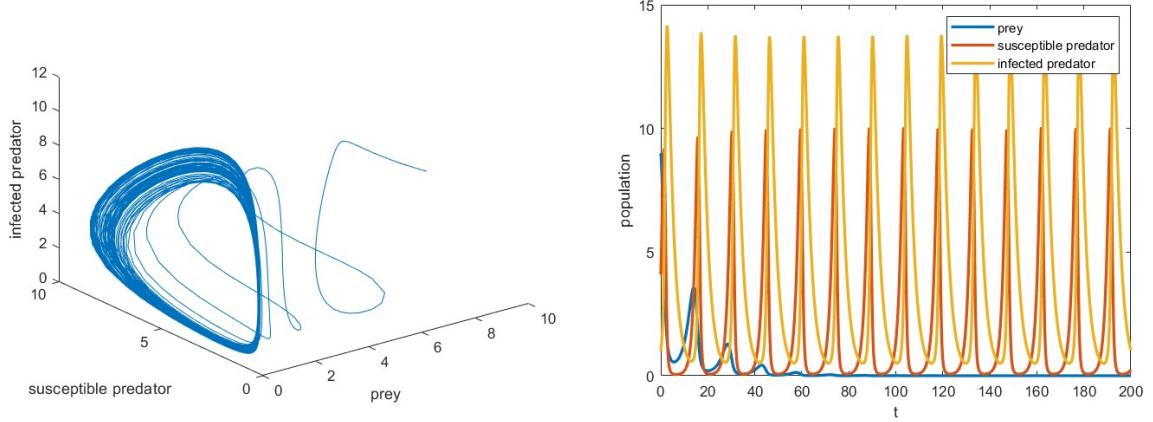


Figure 18: Behavior of the system at E_5 with $k = 50, \alpha = 0.2, m = 0.1, \mu = -0.8, d = 0.4, r = 0.5, n = 1, b = 5, p = 0.7, e = 0.3$

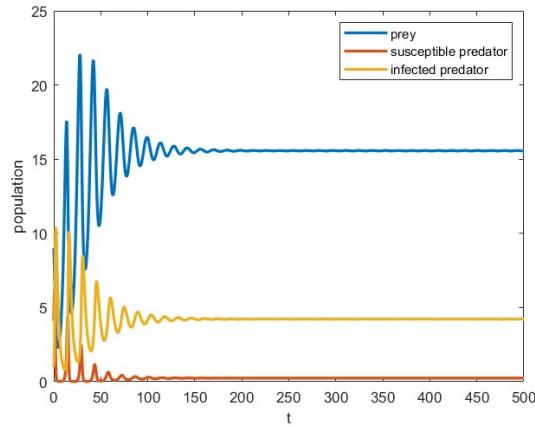


Figure 19: Behavior of the system at E_5 with $k = 50, \alpha = 0.3, m = 0.1, \mu = -0.8, d = 0.4, r = 0.5, n = 1, b = 5, p = 0.7, e = 0.3$

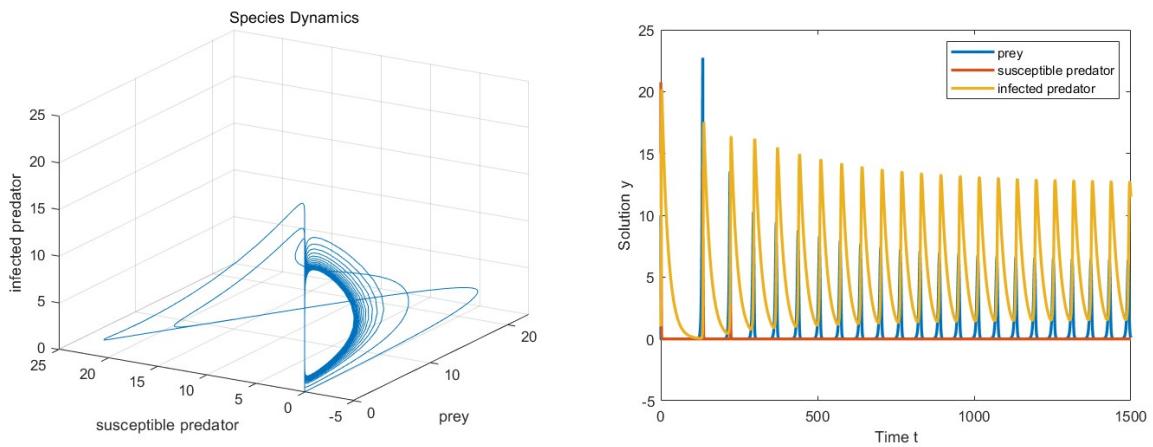


Figure 20: E_5 with $k = 50$, $\alpha = 0.131$, $m = 0.602$, $\mu = 0.05$, $d = 0.05$, $r = 0.6$, $n=1$, $b=5$, $p=0.15$, $e=0.7$ and $\tau = 0.05$