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Research article

A study of a prey-predator model with disease in predator including gestation delay, treatment and linear harvesting of predator species

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Abstract: In this work, we investigated a predator-prey model based on ecodemiology in which the predator population was infected with a transmissible illness, and the time delay equaled the predator's gestation period. Predators can be divided into two separate groups: The ones that are infected and the ones that are susceptible to infection. Assume that the infected predator's sickness is thought to be treatable. The explanations for the solutions' positive invariance and the reason there are equilibria are stated in the suggested system. We showed the boundedness of the system solutions. Additionally, the local stability of every possible equilibrium point was investigated with respect to delayed as well as non-delayed systems. Furthermore, time delay has been demonstrated to be essential for controlling the dynamics of the system, and the periodic solutions were known to exist through Hopf bifurcation in connection to gestation delay. Also, our finding showed that treatment for infected predators have an influence on the dynamics of the system. Lastly, a computational simulation is used to verify the conceptual inquiry's conclusion.

Keywords: prey-predator; linear harvesting; gestation time delay; disease; treatment; numerical simulations
Mathematics Subject Classification: 92D25, 92D40, 34D20, 37G15

1. Introduction

Predator-prey dynamic relationships are fundamental to both mathematics and biological ecology. Due to the fundamental work of A. J. Lotka [1] and V. Volterra [2], the first predator-prey model was developed and examined. Since then, a tremendous amount of effort has been completed, and it continues to increase yearly. Many academics have been developed and studied mathematical models

that consider the myriad complex interactions that take place between interacting species in recent years [3–7].

Diseases are prevalent in nature, and most of diseases are spread by contact between members of the same species or distinct species who are diseased and healthy. As a result, from an ecological and mathematical perspective, comprehending the eco-epidemiological model is essential. One of the most crucial aspects of epidemiology is the mathematical representation of disease transmission. In the majority of epidemic models, the assumption is that disease transmission proceeds according to a density-dependent rule. Several noteworthy investigations have been carried out about the influence of infectious diseases on the dynamics of the population of prey and predators, in which the illness infects the prey as well as in which the illness infects the predator (see e.g., [8–13].) In [11, 12], nonlinear infection rate has been studied using Holling type III functional responses. Notable studies on illness in populations of prey and predators at the same time have also been conducted in recent years [14–17]. It has been well acknowledged that one way to lessen and completely eradicate illness in a population is through treatments. Multiple studies have found different treatments for different infectious illnesses. Managing the transmission of disease infection among both prey and predator species by therapy has been studied in a prey-predator system in the past few years [18–20].

Considering lags in time happen in almost all biological circumstances, time-delayed models are significantly more realistic. It makes more sense to assume that the time required for the predator to complete the gestation period will cause the reproduction process to be delayed rather than occurring immediately after its prey is consumed. By introducing temporal delays, the models display more intricate responses and adopt a more pragmatic perspective on understanding the relationships among prey and predator. Delay differential equations typically exhibit far more complex dynamics in contrast to standard differential equations. Consequently, throughout time, investigations have been conducted on more realistic models of interacting populations by introducing time delays into biological models [21–25]. Many researchers created and studied delayed eco-epidemic models, in which the illness infects the prey and as well as disease infection in the predator [26–30].

Our knowledge indicates that Hopf bifurcation is crucial to comprehending the system's dynamic character. Hopf bifurcation finds the parameter value at which the stability of a system suddenly shifts and a periodic solution appears [31–34]. One of the main goals of the study is to determine if the interior equilibrium is asymptotically stable and this condition reflects the possible equilibrium to which the system will surely develop from any beginning condition.

The confluence of a transmissible sickness that is affecting predator populations with the temporal lag is brought on by the predator's gestation of Holling type III functional responses and also by treatment of infected predator, which we are concerned about. The prey population is represented by $x(t)$, the susceptible predator population denoted by $y(t)$, the infected predator population denoted by $z(t)$, and the treatable predator population is denoted by $w(t)$ at a given time t. These presumptions are made in order to create the intended model:

(A1) Assume that the prey species increases logistically with a natural growth rate r and an environmental carrying capacity K in a lack of diseases and predators. Predator that is susceptible or infected can eat prey and come up with a Holling type-II functional response in the process. In this case, the half saturation constants for the susceptible predator and the infected predator, respectively, are a_1 and a_2. The prey-predation coefficients in this case are m and n, respectively, with regard to the

susceptible predator and the infected predator. As a result, the prey population's differential equation is as

$$\frac{dx}{dt} = rx\left(1-\frac{x}{K}\right)-\frac{mxy}{a_1+x^2}-\frac{nxz}{a_2+x^2}. \tag{1.1}$$

(A2) A straightforward mass action rule αyz that follows the spread of illness between the predator groups. As determined by e_1 and e_2, respectively, the prey population's conversion factors into susceptible and infected predator populations. For susceptible and infected predator populations, we assume proportional harvesting, with $H_1 = q_1E_1$ and $H_2 = q_2E_2$. Here, q_1, q_2 represent the catchability coefficients for susceptible and infected predator species, and E_1, E_2 represent the harvesting efforts for the respective susceptible and infected predator species. We also assume that τ be the gestation delay of the susceptible and infected predator. As a result, the susceptible as well as infected predator population's differential equation is as

$$\frac{dy}{dt} = \frac{e_1mx(t-\tau)y(t-\tau)}{a_1+x^2(t-\tau)}-\alpha yz-H_1y+bw, \tag{1.2}$$

$$\frac{dz}{dt} = \frac{e_2nx(t-\tau)z(t-\tau)}{a_2+x^2(t-\tau)}+\alpha yz-H_2z-\beta zw. \tag{1.3}$$

(A3) Treatment rate of infected predator is β, and b is the rate of infected predator that can only recover through treatment. Also, c is the death rate of infected predator under treatment. The proposed system demonstrated clearly in the schematic diagram (cf. Figure 1). As a result, the treatment population's differential equation is as

$$\frac{dw}{dt} = \beta zw-bw-cw. \tag{1.4}$$

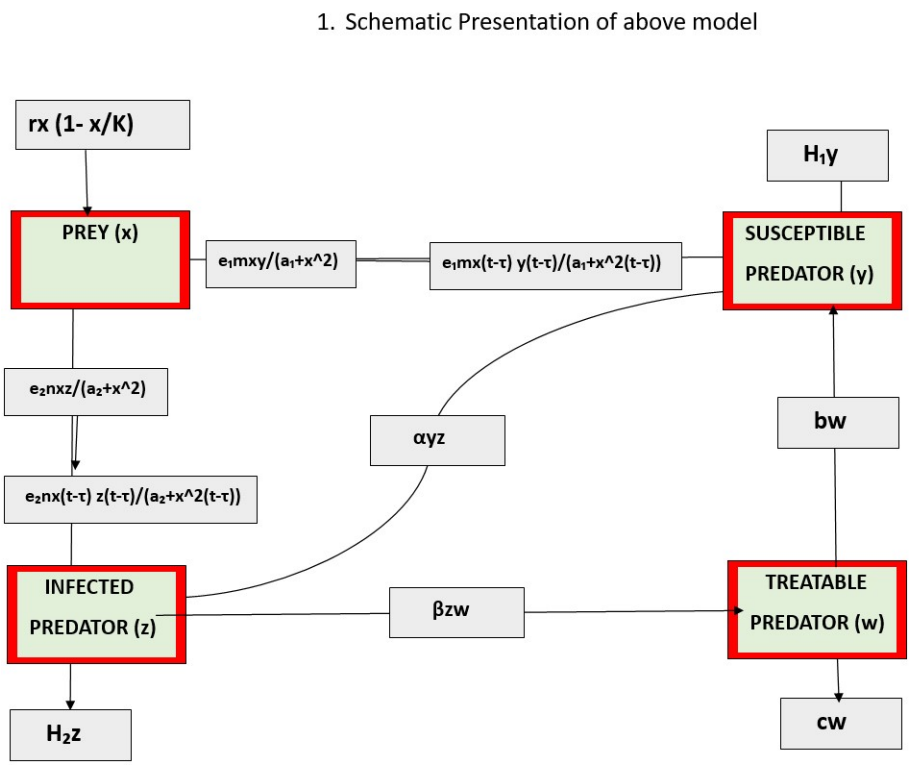


Figure 1. Schematic representation of the system (1.5).

We have the following mathematical delayed model based on the previously stated assumptions:

$$\begin{aligned}
\frac{dx}{dt} &= rx\left(1-\frac{x}{K}\right)-\frac{mxy}{a_1+x^2}-\frac{nxz}{a_2+x^2},\\
\frac{dy}{dt} &= \frac{e_1mx(t-\tau)y(t-\tau)}{a_1+x^2(t-\tau)}-\alpha yz-H_1y+bw,\\
\frac{dz}{dt} &= \frac{e_2nx(t-\tau)z(t-\tau)}{a_2+x^2(t-\tau)}+\alpha yz-H_2z-\beta zw,\\
\frac{dw}{dt} &= \beta zw-bw-cw,
\end{aligned} \tag{1.5}$$

with inital conditions:

$$x(t)=\phi_1(t)\geq 0,\ y(t)=\phi_2(t)\geq 0,\ z(t)=\phi_3(t)\geq 0,\ w(t)=\phi_4(t)\geq 0\quad t\in[-\tau,0]. \tag{1.6}$$

The current investigation is set up as follows: An eco-epidemic model has been built and explained with introduction in Section 1. The positive invariance of the model's solutions, as well as the viability and validity of the equilibria, are covered in Section 2. The Hopf bifurcation's existence and local stability are covered in Section 3. The stability direction of the Hopf bifurcation analysis is examined in Section 4. In Section 5, we present some numerical simulations to illustrate our hypothesis. Lastly, the findings are given in Section 6.

2. Basic properties of the model

In addition to the positivity invariance and boundedness of the solutions, this section describes the equilibria of the delayed system (1.5).

2.1. Positive invariance

Proposition 1. *Here is an a positive solution to the system* (1.5) *for the a positive starting condition* $x(t) = \phi_1(t) \geq 0,\ y(t) = \phi_2(t) \geq 0,\ z(t) = \phi_3(t) \geq 0,\ w(t) = \phi_4(t) \geq 0 \quad t \in [-\tau, 0]$.

Proof. We are aware that the effectiveness of the system's solutions determines whether populations survive. The system (1.5) can be written as

$$\frac{dY}{dt} = S\ (Y, t, \tau)\ \text{ where, }\ Y(t) = (y_1(t), y_2(t), y_3(t), y_4(t))^T = (x(t), y(t), z(t), w(t))^T \in R^4$$

and $S\ (Y, t, \tau)$ is given by

$$S\ (Y, t, \tau) = \begin{bmatrix} S_1 \\ S_2 \\ S_3 \\ S_4 \end{bmatrix} = \begin{bmatrix} rx\left(1 - \frac{x}{K}\right) - \frac{mxy}{a_1 + x^2} - \frac{nxz}{a_2 + x^2} \\ \frac{e_1 mx(t-\tau)y(t-\tau)}{a_1 + x^2(t-\tau)} - \alpha xy - h_1 y + bw, \\ \frac{e_2 nx(t-\tau)z(t-\tau)}{a_2 + x^2(t-\tau)} + \alpha yz - h_1 z - \beta zw \\ \beta zw - bw - cw. \end{bmatrix}.$$

It is easy to confirm using system (1.5) that if $Y(t) \in R_+^4$ is selected so that $x = 0, y = 0, z = 0$, and $w = 0$, then

$$S_i(Y)|_{y_i = 0, Y \in R_+^4} = S_i(0) \geq 0 \quad \text{for } i = 1, 2, 3, 4.$$

Each possible solution of the system (1.5) with starting values $x(t) = \phi_1(t) \geq 0,\ y(t) = \phi_2(t) \geq 0),\ z(t) = \phi_3(t) \geq 0,\ w(t) = \phi_4(t) \geq 0, t \in [-\tau, 0], \phi_i(t) \in R_+^4$ by using the lemma (cf. Kuang [35]), say $Y(t) = Y[t; U(0)]\ \ \forall\ t > 0$, meaning that it stays positive over the whole region $R_+^4,\ \forall\ t > 0$.

2.2. Boundedness

Proposition 2. *The total number of predators, prey, and treatable populations in the system* (1.5) *are all still restricted from above.*

Proof. Let $X = (e_1 + e_2)x(t - \tau) + y + z + w$. For any $\eta > 0$, we have

$$\begin{aligned} \frac{dX}{dt} + \eta X &= (e_1 + e_2) rx(t-\tau)\left(1 - \frac{x(t-\tau)}{K}\right) - \frac{e_2 mx(t-\tau)y(t-\tau)}{a_1 + x^2(t-\tau)} - \frac{e_1 nx(t-\tau)z(t-\tau)}{a_2 + x^2(t-\tau)} \\ &\quad - H_1 y - H_2 z - cw + \eta\left[(e_1 + e_2)x(t-\tau) + y + z\right], \\ &\leq (e_1 + e_2) rx(t-\tau)\left(1 + \eta - \frac{x(t-\tau)}{K}\right) + (\eta - H_1)y + (\eta - H_2)z + (\eta - c)\, w, \\ &\leq \frac{K(\eta + 1)(e_1 + e_2)}{4} + (\eta - H_1)y + (\eta - H_2)z + (\eta - c)\, w. \end{aligned}$$

Choosing sufficiently small η such that $\eta < H_1,\ \eta < H_2,$ and $\eta < c$ we get

$$\frac{dX}{dt} + \eta X \leq M\left(= \frac{K(\eta+1)(e_1+e_2)}{4}\right).$$

By use of Gronwall's inequality [36] application we get

$$0 \leq X(t) \leq \frac{M}{\eta}\left(1 - e^{-\eta t}\right) + X(0)e^{-\eta t}.$$

Consequently, $t \to \infty \implies 0 < X(t) < \frac{M}{\eta}$. This suggests that any solutions for the system represented by (1.5) are bounded.

2.3. Equilibria

The prey-predator model (1.5) has at least eight feasible and two infeasible equilibrium points.

(1) Trivial equilibrium point $E_0(0,0,0,0)$ and equilibrium point $E_1(K,0,0,0)$ to the system (1.5) are always exist.

(2) Disease-free and treatment-free equilibrium point $E_2^{\pm}\left(x_2^{\pm}, y_2^{\pm}, 0, 0\right)$ with $x_2^{\pm}$ are the positive roots of $H_1x^2 - e_1mx + H_1a_1 = 0$ and $y_2^{\pm} = \frac{re_1(K-x_2^{\pm})}{KH_1}$. This is possible to reach the equilibrium point E_2 if $H_1 < \frac{e_1m}{\sqrt{2a_1}}$ $x_2 < K$. Here, without treatment disease does not exist between the predator.

(3) Healhy predator-free and treatment-free equilibrium point $E_3^{\pm}\left(x_3^{\pm}, 0, z_3^{\pm}, 0\right)$ with $x_3^{\pm}$ are the positive roots of $H_2x^2 - e_2nx + H_2a_2 = 0$ and $z_3^{\pm} = \frac{re_2(K-x_3^{\pm})}{KH_2}$. E_3 is feasible if $H_2 < \frac{e_2n}{\sqrt{2a_2}}$ and $x_3^{\pm} < K$. Only infected predator species coexisted with prey species without treatment in this instance.

(4) There exists an infeasible boundary equilibrium point, namely $E_4(x_4, y_4, z_4, w_4)$, where $x_4 = 0$, $y_4 = \frac{H_2}{\alpha}$, $z_4 = -\frac{H_1}{\alpha}$, $w_4 = 0$.

(5) Treatment-free equilibrium point $E_5\,(x_5, y_5, z_5, 0)$ where $y_5 = \frac{H_2x^2 - e_2nx + H_2a_2}{\alpha(a_2+x^2)}$, $z_5 = \frac{-H_1x^2 + e_1mx - H_1a_1}{\alpha(a_1+x^2)}$, and where x_5 is the positive root of the equation

$$l_{55}x^5 + l_{54}x^4 + l_{53}x^3 + l_{52}x^2 + l_{51}x + l_{50} = 0, \tag{2.1}$$

where

$l_{55} = r\alpha, l_{44} = -Kr\alpha,\ l_{54} = r(a_{1+a_2})\alpha,\ l_{52} = K(mH_2 - nH_1) - rK\alpha(a_1 + a_2),$

$l_{51} = ra_1a_2(e_1 - e_2),\ l_{50} = K(mH_2a_2 - nH_1a_1) - ra_1a_2K\alpha.$

The equilibrium point E_5 is feasible if (2.1) has at least one positive root with $e_2n > 2H_2\sqrt{a_2}$ and $e_1m > 2H_1\sqrt{a_1}$. Maximum numbers of positive roots of the (2.1) in x_5 are given in the following Table 1.

Table 1. Possibilities of multiple positive roots of the Eq (2.1).

Max. No. of +ve roots	sign of l_{55}	sign of l_{54}	sign of l_{53}	sign of l_{52}	sign of l_{51}	sign of l_{50}
4	+	−	+	−	+	−
3	+	−	+	−	+	+
3	+	−	+	+	+	−
3	+	−	+	+	+	+

(6) There exists another infeasible boundary equilibrium point, namely, $E_6(x_6, y_6, z_6, w_6)$, where $x_6 = 0$, $y_6 = -\frac{H_2 b}{H_1\beta+\alpha c}$, $z_6 = \frac{b+c}{\beta}$, $w_6 = -\frac{H_2(H_1\beta+\alpha b+\alpha c)}{\beta(H_1\beta+\alpha c)}$.

(7) The interior equilibrium point $E^*(x^*, y^*, z^*, w^*)$ with

$$y^* = \frac{1}{\alpha}\left(H_2 + \frac{\alpha(b+c)+\beta}{b} - \frac{e_2 n x^*}{a_2 + x^{*2}} - \frac{e_1 m\beta x^*}{b(a_1 + x^{*2})}\right),$$
$$z^* = \frac{b+c}{\beta},$$
$$w^* = \frac{1}{b}\left(H_1 + \frac{\alpha(b+c)}{b} - \frac{e_1 m x^*}{a_1 + x^{*2}}\right)$$

and, here, x^* is the positive solution of this equation:

$$L(x) = l_{77}x^7 + l_{76}x^6 + l_{75}x^5 + l_{74}x^4 + l_{73}x^3 + l_{72}x^2 + l_{71}x + l_{70} = 0, \tag{2.2}$$
$$\text{with } l_{77} = rb\alpha\beta,\ l_{76} = -rbK\alpha\beta,\ l_{75} = rb\alpha\beta(2a_1 + a_2),$$
$$l_{74} = K\beta(mH_2 b + mb\alpha + mc\alpha + m\beta H_1 - rb\alpha(2a_1 + a_2)),$$
$$l_{73} = \beta\left(rba_1\alpha(1 + 2a_2) - \beta m^2 K e_1 - be_2 mn\right),$$
$$l_{72} = Kbn\alpha(b + c) + mK\beta(a_1 + a_2)(\beta H_1 + \beta H_2 + \alpha b + \alpha c) - rb\alpha\beta a_1 K(1 - 2a_2),$$
$$l_{71} = ra_1^2 a_2 b\alpha\beta - Ke_1 a_2 m^2\beta^2 - b\beta e_2 mna_1,$$
$$l_{70} = a_1 a_2 mK\beta(b\alpha + c\alpha + bH_1) + a_1 a_2 mKH_2 b\beta + a_1 b\alpha nK(b + c) - Ka_1^2 a_2.$$

Here, E^* is feasible if at least one positive root exists in $L(x)$ above, y^* and w^* are also positive numbers. Finding the precise condition under which E^* becomes viable in a system without an explicit expression and with additional complexity for the equilibrium points is difficult. However, a numerical integration of system (2) offers important information. Now if we chose parameters as follows: $(r, k, m, n, e_1, e_2, a_1, a_2, b, c, H_1, H_2, \alpha, \beta) = (5, 1, 2.5, 1.2, 2.1, 1, 0.8, 1.1, 0.2, 0.1, 1.5, 1.4, 0.5, 0.1)$, then the system (1.5) has a positive equilibrium point $E^*(x^*, y^*, z^*, w^*) = (3.36, 6.97, 8.85, 1.01)$.

3. Analysis of local stability and bifurcations

At this portion, the local stability of each of the system's seven potential equilibrium points—both with and without delay—is investigated. Moreover, see the effect of the parameter τ on the

system (1.5). First, we compute system (1.5)'s Jacobian matrix, which is provided by

$$J_E = \begin{bmatrix} a_{11} & a_{12} & a_{13} & 0 \\ b_{21}e^{-\lambda\tau} & a_{22} + b_{22}e^{-\lambda\tau} & -\alpha y & b \\ b_{31}e^{-\lambda\tau} & \alpha z & a_{33} + b_{33}e^{-\lambda\tau} & -\beta z \\ 0 & 0 & \beta w & a_{44} \end{bmatrix},$$

where

$$a_{11} = r\left(1 - \frac{2x}{K}\right) - \frac{my\left(a_1 - x^2\right)}{\left(a_1 + x^2\right)^2} - \frac{nz\left(a_2 - x^2\right)}{\left(a_2 + x^2\right)^2},\ a_{12} = -\frac{mx}{a_1 + x^2},$$
$$a_{13} = -\frac{nx}{a_2 + x^2},\ b_{21} = \frac{e_1 my(a_1 - x^2)}{(a_1 + x)^2},\ a_{22} = -H_1 - \alpha z,\ \ b_{22} = \frac{e_1 mx}{a_1 + x^2},$$
$$b_{31} = \frac{e_2 nz\left(a_2 - x^2\right)}{\left(a_2 + x^2\right)^2},\ a_{33} = \alpha y - H_2,\ b_{33} = \frac{e_2 nx}{a_2 + x^2},\ a_{44} = \beta z - b - c$$

and where λ is the characteristic value obtained from the delayed system's linearization.

Theorem 1. *E_0 to the system is always unstable.*

Proof. At E_0, the eigenvalues are $\lambda_{01} = r > 0,\ \lambda_{02} = -h_1 < 0,\ \lambda_{03} = -h_2 < 0$, and $\lambda_{04} = -b - c < 0$. Therefore, E_0 is always unstable.

Theorem 2. *For any τ, $E_1(K, 0, 0, 0)$ to the system* (1.5) *is asymptotically stable if, and only if, $\frac{e_1 mK}{a_1 + K^2} < H_1$ and $\frac{e_2 nK}{a_2 + K^2} < H_2$ are satisfied. It will be unstable otherwise.*

Proof. The eigenvalues of J_{E_1} are $\lambda_{11} = -r, \lambda_{12} = -b - c$

$$\lambda_{13} = \frac{e_1 mK}{a_1 + K^2} e^{-\lambda_{13}\tau} - H_1, \tag{3.1}$$

$$\lambda_{14} = \frac{e_2 nK}{a_2 + K^2} e^{-\lambda_{14}\tau} - H_2. \tag{3.2}$$

For $\tau = 0$, the eigenvalues of J_{E_1} are $-r,\ -b - c,\ \frac{e_1 mK}{a_1 + K^2} - H_1$, and $\frac{e_2 nK}{a_2 + K^2} - H_2$. Thus, for $\frac{e_1 mK}{a_1 + K^2} < H_1$ and $\frac{e_2 nK}{a_2 + K^2} < H_2$, E_1 to the system (1.5) is stable; if not, it will become unstable.

When $\tau \neq 0$, an eigenvalue $\lambda_{11} = -r, \lambda_{12} = -b - c$ is unable to bring about stability in the system. The locations of each of these Eqs (3.1) and (3.2)' roots determine whether this equilibrium point E_1 is stable. Write $\lambda_{13} = \mu_1 + i\sigma_1$ in Eq (3.1) and, following splitting it into its real and imaginary components, we obtain

$$\mu_1 + H_1 = \frac{e_1 mK}{a_1 + K^2} e^{-\mu_1\tau} \cos(\sigma_1\tau), \tag{3.3}$$

$$\sigma_1 = -\frac{e_1 mK}{a_1 + K^2} e^{-\mu_1\tau} \sin(\sigma_1\tau). \tag{3.4}$$

Because the left-hand portion of the real component of (3.3) remains strictly greater than the righthand side's magnitude, μ_1 must be negative if $H_1 > \frac{e_1 mK}{a_1 + K^2}$ and $\mu_1 \geq 0$. As a result, there must be a negative real component at the roots of Eq (3.1). Likewise, we can demonstrate that there is a negative real component for (3.2) if $H_2 > \frac{e_2 nK}{a_2 + K^2}$. Therefore, E_1 is stable for all τ for $H_1 > \frac{e_1 mK}{a_1 + K^2}$ and $H_2 > \frac{e_2 nK}{a_2 + K^2}$. Otherwise, it will be unstable.

Theorem 3. *Disease-free and treatment-free equilibrium point* $E_2(x_2, y_2, 0, 0)$
(i) when $\tau = 0$*, it is asymptotically stable to the system* (1.5) *for* $\frac{e_2 n x_2}{a_2 + x_2^2} < H_2 - \alpha y_2$, $A_1 + A_3 > 0$, *and* $A_2 + A_4 > 0$*;*
(ii) when $\tau \neq 0$*, the system* (1.5) *can undergo a Hopf-bifurcation if* $\frac{e_2 n x_2}{a_2 + x_2^2} > H_2 - \alpha y_2$ *and* $A_2^2 - A_4^2 < 0$.

Proof. At $E_2(x_2, y_2, 0, 0)$, the eigenvalues of J_{E_2} are $\lambda_{21} = -b - c$,

$$\lambda_{22} = \frac{e_2 n x_2}{a_2 + x_2^2} e^{-\lambda_{22}\tau} - H_2 + \alpha y_2, \tag{3.5}$$

and the other the two eigenvalues λ_{23} and λ_{24} consider the roots of this equation

$$\lambda^2 + A_1\lambda + A_2 + (A_3\lambda + A_4)\, e^{-\lambda\tau} = 0, \tag{3.6}$$

where

$$A_1 = H_1 - r\left(1 - \frac{2x_2}{K}\right) + \frac{m y_2 \left(a_1 - x_2^2\right)}{\left(a_1 + x_2^2\right)^2},\ A_2 = -rH_1\left(1 - \frac{2x_2}{K}\right) + \frac{mH_1 y_2 \left(a_1 - x_2^2\right)}{\left(a_1 + x_2^2\right)^2}$$
$$A_3 = -\frac{e_1 m x_2}{a_1 + x_2^2},\ A_4 = r\left(1 - \frac{2x_2}{K}\right)\frac{e_1 m x_2}{a_1 + x_2^2}.$$

When $\tau = 0$, the eigenvalues of J_{E_3} become $\lambda_{21} = -b - c$, $\lambda_{22} = \frac{e_2 n x_2}{a_2 + x_2^2} - H_2 + \alpha y_2$, and λ_{23} and λ_{24} are the roots of the equation $\lambda^2 + (A_1 + A_3)\lambda + A_2 + A_4 = 0$. Therefore, E_2 will be asymptotically stable if $A_1 + A_3 > 0, A_2 + A_4 > 0$ and $\frac{e_2 n x_2}{a_2 + x_2^2} < H_2 - \alpha y_2$; otherwise, it is unstable.
Take $\tau \neq 0$. In order to ascertain the effect of the time delay τ on stability, we therefore study τ as the bifurcation factor. In (3.5), we begin by looking for a purely imaginary root $i\sigma_2$, $\sigma_2 \in R$. After splitting each of its real and imaginary components and removing τ, we obtain

$$\sigma_2^2 = \left(\frac{e_2 n x_2}{a_2 + x_2^2}\right)^2 - (H_2 - \alpha y_2)^2. \tag{3.7}$$

Then, from (3.7) $\sigma_2 \in R$ if $\frac{e_2 n x_2}{a_2 + x_2^2} > H_2 - \alpha y_2$. Once more, searching in (3.6) for a purely imaginary root $i\sigma_3$, $\sigma_3 \in R$, we have divided the expression into its real and imaginary components, removing τ. Then,

$$\sigma_3^4 + (A_1^2 - A_3^2 - 2A_2)\sigma_3^2 + A_2^2 - A_4^2 = 0, \tag{3.8}$$
$$\text{where, } A_1^2 - A_3^2 - 2A_2 = H_1^2 + \left[r\left(1 - \frac{2x_2}{K}\right) - \frac{m y_2 \left(a_1 - x_2^2\right)}{\left(a_1 + x_2^2\right)^2}\right]^2 - \frac{m^2 e_1^2 x_2^2}{\left(a_1 + x_2^2\right)^2},$$
$$A_2^2 - A_4^2 = \left[\frac{mH_1 y_2 \left(a_1 - x_2^2\right)}{\left(a_1 + x_2^2\right)^2} - rh_1\left(1 - \frac{2x_2}{K}\right)\right]^2 - \left[r\left(1 - \frac{2x}{K}\right)\frac{e_1 m x_2}{a_1 + x_2^2}\right]^2.$$

We can see that if $A_2^2 - A_4^2 < 0$, then there is a positive real root of (3.8). Consequently, adding a temporal delay to the model may cause a Hopf-bifurcation, which we shall demonstrate using MATLAB in the numerical simulation section.

Theorem 4. *Treatment and healthy predator-free equilibrium point* $E_3(x_3, 0, z_3, 0)$
(i) when $\tau = 0$*, it is asymptotically stable to the system* (1.5) *for* $\frac{e_1 m x_3}{a_1 + x_3^2} < H_1 + \alpha z_3$, $B_1 + B_3 > 0$, *and* $B_2 + B_4 > 0$*;*
(ii) when $\tau \neq 0$*, the system* (1.5) *can undergo a Hopf-bifurcation if* $\frac{e_1 m x_3}{a_1 + x_3^2} > H_1 + \alpha z_3$ *and* $B_2^2 - B_4^2 < 0$.

Proof. At $E_2(x_3, 0, z_3, 0)$, the eigenvalues of J_{E_3} are $\lambda_{31} = -b - c$,

$$\lambda_{32} = \frac{e_1 m x_3}{a_1 + x_3^2} e^{-\lambda_{22}\tau} - H_1 - \alpha z_3, \tag{3.9}$$

and other the two eigenvalues, λ_{33} and λ_{34}, are the roots of the following equation

$$\lambda^2 + B_1\lambda + B_2 + (B_3\lambda + B_4)\, e^{-\lambda\tau} = 0, \tag{3.10}$$

where

$$B_1 = H_2 - r\left(1 - \frac{2x_3}{K}\right) + \frac{n z_3 \left(a_2 - x_3^2\right)}{\left(a_2 + x_3^2\right)^2},\ B_2 = -rH_2\left(1 - \frac{2x_3}{K}\right) + \frac{nH_2 z_3 \left(a_2 - x_3^2\right)}{\left(a_2 + x_3^2\right)^2}$$
$$B_3 = -\frac{e_2 n x_3}{a_2 + x_3^2},\ B_4 = r\left(1 - \frac{2x_2}{K}\right)\frac{e_2 n x_3}{a_2 + x_3^2}.$$

When $\tau = 0$, the eigenvalues of J_{E_3} become $\lambda_{31} = -b - c$, $\lambda_{32} = \frac{e_1 m x_3}{a_1 + x_3^2} - H_1 - \alpha z_3$, and λ_{33} and λ_{34} are the roots of the equation $\lambda^2 + (B_1 + B_3)\,\lambda + B_2 + B_4 = 0$. E_3 gets asymptotic stability when $B_1 + B_3 > 0, B_2 + B_4 > 0$, and $\frac{e_1 m x_3}{a_1 + x_3^2} < H_1 + \alpha z_3$; otherwise it is, unstable.

Take $\tau \neq 0$. We now investigate τ as the bifurcation parameter in an attempt to determine the impact of the time delay τ on stability. In (3.9), we begin by looking for a purely imaginary root $i\sigma_4$, $\sigma_4 \in R$. After splitting it into its real and imaginary components and removing τ, we obtain

$$\sigma_4^2 = \left(\frac{e_1 m x_3}{a_1 + x_3^2}\right)^2 - (H_1 + \alpha z_3)^2. \tag{3.11}$$

Then, from (3.11), $\sigma_4 \in R$ if $\frac{e_1 m x_3}{a_1 + x_3^2} > H_1 + \alpha z_3$. Once more, searching in (3.10) for a purely imaginary root $i\sigma_5$, $\sigma_5 \in R$, we obtain it after dividing it into its real and imaginary components and removing τ,

$$\sigma_5^4 + (B_1^2 - B_3^2 - 2B_2)\sigma_5^2 + B_2^2 - B_4^2 = 0. \tag{3.12}$$
$$\text{with } B_1^2 - B_3^2 - 2B_2 = H_1^2 + \left[r\left(1 - \frac{2x_3}{K}\right) - \frac{n z_3 \left(a_2 - x_3^2\right)}{\left(a_2 + x_3^2\right)^2}\right]^2 - \frac{n^2 e_2^2 x_3^2}{\left(a_2 + x_3^2\right)^2}$$
$$B_2^2 - B_4^2 = \left[\frac{nH_2 z_3 \left(a_2 - x_3^2\right)}{\left(a_2 + x_3^2\right)^2} - rh_2\left(1 - \frac{2x_2}{K}\right)\right]^2 - \left[r\left(1 - \frac{2x_3}{K}\right)\frac{e_2 n x_3}{a_2 + x_3^2}\right]^2.$$

We can see that if $B_2^2 - B_4^2 < 0$, consequently, there is a real, positive root of (3.12). Consequently, adding a temporal delay to the model may cause a Hopf-bifurcation, which we shall demonstrate using MATLAB numerical simulation.

Theorem 5. *It is assumed that the conditions* $(D_1), (D_2), (D_3)$ *hold for system* (1.5). *Treatment-free equilibrium point* $E_5(x_5, y_5, z_5)$ *is locally asymptotically stable when* $\tau \in [0, \tau_0)$ *and a Hopf-bifurcation takes place when* $\tau = \tau_0$.

Proof. Our current objective is to look at how time delays affect the system in the absence of treatment. The system's characteristic equation at E_5 may be written as

$$\lambda^3 + d_1\lambda^2 + d_2\lambda + d_3 + \left(d_4\lambda^2 + d_5\lambda + d_6\right)e^{-\lambda\tau} + (d_7\lambda + d_8)\,e^{-2\lambda\tau} = 0, \tag{3.13}$$

$$\text{where } d_1 = H_1 + H_2 + \alpha(z_5 - y_5) - r\left(1 - \frac{2x_5}{K}\right) + \frac{my_5\left(a_1 - x_5^2\right)}{(a_1 + x_5^2)^2} + \frac{nz_5\left(a_2 - (x^*)^2\right)}{\left(a_2 + x_5^2\right)^2},$$

$$d_2 = \left[r\left(1 - \frac{2x_5}{K}\right) - \frac{m\left(a_1 - x_5^2\right)y_5}{(a_1 + x_5^2)^2} - \frac{nz_5\left(a_2 - x_5^2\right)}{\left(a_2 + x_5^2\right)^2}\right][\alpha(y_5 - z_5) - (H_1 + H_2)]$$
$$+ H_1H_2 + \alpha z_5(H_2z_5 - H_1y_5),$$

$$d_3 = \left[r\left(1 - \frac{2x_5}{K}\right) - \frac{my_5\left(a_1 - x_5^2\right)}{(a_1 + x_2^2)^2} - \frac{nz_5\left(a_2 - x_5^2\right)}{\left(a_5 + x_5^2\right)^2}\right][\beta(H_1y_5 - H_2z_5) - H_1H_2],$$

$$d_4 = -\frac{e_1mx_5}{a_1 + x_5^2} - \frac{e_2nx_5}{a + x_5^2},$$

$$d_5 = r\left(1 - \frac{2x_5}{K}\right)\left[\frac{e_1mx_5}{a_1 + x_5^2} + \frac{e_2nx_5}{a_2 + x_5^2}\right] + \frac{e_1m(\alpha y_5 - H_2)x_5}{a_1 + x_5^2} - \frac{e_2n(H_1 + \alpha z_5)x_5}{a_2 + x_5^2}$$
$$- \left[\frac{mnx_5}{(a_2 + x_5^2)(a_1 + x_5^2)}\right]\left[\frac{e_2y^*\left(a_1 - x_5^2\right)}{a_1 + x_5^2} + \frac{e_1z_5\left(a_2 - x_5^2\right)}{a_2 + x_5^2}\right],$$

$$d_6 = \left[\frac{mnx_5}{(a_2 + x_5^2)(a_1 + x^2)}\right]\left[\frac{((e_1 - e_2)\alpha z_5 - e_2H_1)\,\alpha y^*}{\alpha + x^*} + \frac{[(e_1 - e_2)\beta y^* - e_1H_2]\,z^*}{a + bx^* + c(x^*)^2}\right.$$
$$\left.\times\left(a - c(x_5)^2\right)\right] + r\left(1 - \frac{2x_5}{K}\right)\left[\frac{e_2n(H_1 + \beta z_5)x_5}{a + bx^* + cx_5^2} - \frac{e_1m(\beta y_5 - H_2)x_5}{\alpha + x_5}\right],$$

$$d_7 = \frac{e_1e_2mnx_5^2}{(a_1 + x_5^2)(a_2 + x_5^2)},\ d_8 = -\frac{re_1e_2mnx_5^2z_5\,(K - 2x_5)}{K(a_1 + x_5^2)\left(a_2 + x_5^2\right)}.$$

Case 1: When $\tau = 0$, then characteristic equation changes to

$$\lambda^3 + (d_1 + d_4)\lambda^2 + (d_2 + d_5 + d_7)\lambda + d_3 + d_6 + d_8 = 0. \tag{3.14}$$

E_5 is asymptotically stable when using the Routh-Hurwitz criteria on (3.14), provided that the following is true $(D_1) : d_1 + d_4 > 0, d_3 + d_6 + d_8 > 0$, and $(d_1 + d_4)(d_2 + d_5 + d_7) - (d_3 + d_6 + d_8) > 0$. It follows that these formulas cannot be used to infer the explicit parametric conditions required for the asymptotic stability of the treatment-free equilibrium point E_5. We shall demonstrate these results using numerical simulations.

Case 2: Take $\tau > 0$. Currently, we are attempting to identify a periodic solution with biological

significance. We replace $\lambda = i\omega(\omega > 0)$ in (3.13) to obtain the periodic solution of (1.5). By comparing both the real and imaginary portions, we obtain these equations:

$$(d_6 - d_4\omega^2)\cos(\omega\tau) + d_5\omega\sin(\omega\tau) + d_8\cos(2\omega\tau) + d_7\omega\sin(2\omega\tau) = d_1\omega^2 - d_3$$
$$(d_6 - d_4\omega^2)\sin(\omega\tau) - d_5\omega\cos(\omega\tau) + d_8\sin(2\omega\tau) - d_7\omega\cos(2\omega\tau) = d_2\omega - \omega^3$$

which gives

$$\sin(\omega\tau) = \frac{\omega\left[d_4\omega^4 + \{d_1d_5 - d_6 - d_4(d_7 + d_2)\}\omega^2 + d_6(d_7 + d_2) - d_5(d_3 + d_8)\right]}{\omega^6 + (d_1^2 - 2d_2)\omega^4 + (d_2^2 - 2d_1d_3 - d_7^2)\omega^2 + d_3^2 - d_8^2}. \tag{3.15}$$

$$\cos(\omega\tau) = \frac{(d_5 - d_1d_4)\omega^4 + \{d_5(d_7 - d_2) - d_4(d_8 - d_3) + d_1d_6\}\omega^2 + d_6(d_8 - d_3)}{\omega^6 + (d_1^2 - 2d_2)\omega^4 + (d_2^2 - 2d_1d_3 - d_7^2)\omega^2 + d_3^2 - d_8^2}. \tag{3.16}$$

After squaring and adding (3.15) and (3.16), we obtain

$$\omega^{12} + t_5\omega^{10} + t_4\omega^8 + t_3\omega^6 + t_2\omega^4 + t_1\omega^2 + t_0 = 0, \tag{3.17}$$

$$t_5 = 2d_1^2 - 4d_2 - d_4^2,$$

$$t_4 = \left(d_1^2 - 2d_2\right)^2 - 2\left(d_2^2 - 2d_1d_3 - d_7^2\right) - (d_5 - d_1d_4)^2 + 2d_4\left(d_6 + d_4(d_7 + d_2) - d_1d_5\right),$$

$$t_3 = 2\left(d_3^2 - d_8^2\right) + 2\left(d_1^2 - 2d_2\right)\left(d_2^2 - 2d_1d_3 - d_7^2\right) - [d_6 + d_4(d_7 + d_2) - d_1d_5]^2$$
$$- 2d_4\left[d_6(d_7 + d_2) - d_5(d_3 + d_8) - 2(d_5 - d_1d_4)\right]\left[d_5(d_7 - d_2) + d_4(d_3 - d_8) + d_1d_6\right],$$

$$t_2 = \left\{(d_2^2 - 2d_1d_3 - d_7^2)\right\}^2 + 2\left(d_1^2 - 2d_2\right)\left(d_3^2 - d_8^3\right) - \{d_5(d_7 - d_2) + d_4(d_3 - d_8) + d_1d_6\}^2$$
$$- 2\{d_1d_5 - d_6 - d_4(d_7 + d_2)\}\{d_6(d_7 + d_2) - d_5(d_3 + d_8)\} - 2d_6(d_5 - d_1d_4)(d_8 - d_3),$$

$$t_1 = 2\left(d_2^2 - d_7^2 - 2d_1d_3\right)\left(d_3^2 - d_8^2\right) - 2d_6(d_8 - d_3)\{d_5(d_7 - d_2) + d_4(d_3 - d_8) + d_1d_6\},$$
$$- \{d_6(d_7 + d_2) - d_5(d_3 + d_8)\}^2,$$

$$t_0 = d_3^4 + d_8^4 - 2d_3^2d_8^2 - d_6^2(d_3 - d_8)^2.$$

(D_2) : ω_0 is taken to be a positive root of Eq (3.17). Then from (3.15) and (3.16), we have

$$\tau_k = \frac{1}{\omega_0}\arctan\left[\frac{\omega_0\left[d_4\omega_0^4 + \{d_1d_5 - d_6 - d_4(d_7 + d_2)\}\omega_0^2 + d_6(d_7 + d_2) - d_5(d_3 + d_8)\right]}{(d_5 - d_1d_4)\omega_0^4 + \{d_5(d_7 - d_2) - d_4(d_8 - d_3) + d_1d_6\}\omega_0^2 + d_6(d_8 - d_3)}\right] + \frac{2k\pi}{\omega_0},$$
$$k = 0, 1, 2, 3...$$

We will now investigate the transversality condition of the Hopf bifurcation. When (3.13) is differentiated with regard to τ, it yields

$$\frac{d\lambda}{d\tau}\left[2d_4\lambda + d_5 + (3\lambda^2 + 2d_1\lambda + d_2)e^{\lambda\tau} - d_7e^{-\lambda\tau} + \tau\left\{(\lambda^3 + d_1\lambda + d_2\lambda^2 + d_3)e^{\lambda\tau} - (d_7\lambda + d_8)e^{-\lambda\tau}\right\}\right]$$
$$= \lambda\left\{(d_7\lambda + d_8)e^{-\lambda\tau} - (\lambda^3 + d_1\lambda^2 + d_2\lambda + d_3)e^{\lambda\tau}\right\}$$
$$= \lambda\left\{2(d_7\lambda + d_8)e^{-\lambda\tau} - (d_4\lambda^2 + d_5\lambda + d_6)e^{\lambda\tau}\right\}$$

which leads to $\left(\frac{d\lambda}{d\tau}\right)^{-1} = \frac{2d_4\lambda + d_5 + (3\lambda^2 + 2d_1\lambda + d_2)e^{\lambda\tau} + d_7e^{-\lambda\tau}}{\lambda\{2(d_7\lambda + d_8)e^{-\lambda\tau} - (d_4\lambda^2 + d_5\lambda + d_6)e^{\lambda\tau}\}} - \frac{\tau}{\lambda}.$

Following basic yet typical computations, we obtain

$$\Re\left[\frac{d\lambda}{d\tau}\right]^{-1}_{\lambda=i\omega_0,\tau=\tau_0} = \frac{C_1C_3 + C_2C_4}{C_3^2 + C_4^2}, \text{ where}$$
$$C_1 = 2p_1\sigma_0\cos\omega_0\tau_0 - (3\omega_0^2 - p_2 - p_7)\sin\omega_0\tau_0 + 2p_4\omega_0,$$
$$C_2 = (3\omega_0^2 - p_2 - p_7)\cos\omega_0\tau_0 + 2p_1\omega_0\sin\omega_0\tau_0 - p_5,$$
$$C_3 = \omega_0\left\{2p_8\cos\omega_0\tau_0 + 2p_7\omega_0\sin\omega_0\tau_0 + p_4\omega_0^2 + p_6\right\},$$
$$C_4 = \omega_0\left\{2p_7\omega_0\cos\omega_0\tau_0 - 2p_8\sin\omega_0\tau_0 - p_5\omega_0\right\}.$$

However, the sign of $\left[\frac{d(\Re(\lambda))}{d\tau}\right]_{\lambda=i\omega_0,\tau=\tau_0}$ is the same as the sign of $\Re\left[\frac{d\lambda}{d\tau}\right]_{\lambda=i\omega_0,\tau=\tau_0}$. Thus, the transversality condition $\Re\left[\frac{d\lambda}{d\tau}\right]_{\lambda=i\omega_0,\tau=\tau_0} > 0$ holds only when $(D_3) : C_1C_3 + C_2C_4 > 0$.

Therefore, the treatment-free equilibrium point E_5 is locally asymptotically stable when $\tau \in [0, \tau_0)$, and a Hopf-bifurcation occurs at the equilibrium point E_5 when $\tau = \tau_0$ if the conditions: $(D_1), (D_2), (D_3)$ are fulfilled.

Our current objective is to examine the impact of time delay on the fluctuating behavior of system (1.5) at the interior equilibrium point $E^*(x^*, y^*, z^*, w^*)$, given the presence of prey, predator, disease, and treatment. The system's characteristic equation at E^* written as

$$\lambda^4 + p_1\lambda^3 + p_2\lambda^2 + p_3\lambda + p_4 + \left(p_5\lambda^3 + p_6\lambda^2 + p_7\lambda + p_8\right)e^{-\lambda\tau} + \left(p_9\lambda^2 + p_{10}\lambda + p_{11}\right)e^{-2\lambda\tau} = 0, \tag{3.18}$$

where

$$p_1 = H_1 + H_2 - b - c + \beta z^* + \alpha(z^* - y^*) - r\left(1 - \frac{2x^*}{K}\right) + \frac{my^*(a_1 - x^2)}{(\alpha + x^*)^2} + \frac{nz^*\left(a_2 - (x^*)^2\right)}{\left(a_2 + x^{*2}\right)^2},$$

$$p_2 = \left[r\left(1 - \frac{2x}{K}\right) - \frac{my\left(a_1 - x^2\right)}{\left(a_1 + x^2\right)^2} - \frac{nz\left(a_2 - x^2\right)}{\left(a_2 + x^2\right)^2}\right][-H_1 - \alpha z^* + \alpha y^* - H_2 + \beta z^* - b - c]$$
$$- (H_1 + \alpha z)(\alpha y^* - H_2 + \beta z^* - b - c) + (\alpha y^* - H_2)(\beta z^* - b - c) + \alpha^2 y^* z^* + \beta^2 w^* z^*,$$

$$p_3 = \left[r\left(1 - \frac{2x^*}{K}\right) - \frac{my^*\left(a_1 - x^{*2}\right)}{\left(a_1 + x^{*2}\right)^2} - \frac{nz^*\left(a_2 - x^{*2}\right)}{\left(a_2 + x^{*2}\right)^2}\right]\left[H_1\alpha y^* - H_2\alpha z^* - h_1h_2 - \beta^2 w^* z^*\right.$$
$$\left. + (\beta z^* - b - c)(\alpha y^* - \alpha z^* - H_1 - H_2)\right] + (\beta z^* - b - c)(H_1\alpha y^* - H_2\alpha z^* - H_1H_2)$$
$$+ \beta z^{*2}(\beta(H_1 + \alpha z^*) - b\alpha),$$

$$p_4 = \left[r\left(1 - \frac{2x}{K}\right) - \frac{my\left(a_1 - x^2\right)}{\left(a_1 + x^2\right)^2} - \frac{nz\left(a_2 - x^2\right)}{\left(a_2 + x^2\right)^2}\right][(\beta z^* - b - c)(H_1H_2 + H_2\alpha z^* - H_1\alpha y^*)$$
$$-H_1b\beta w^* z^*]$$

$$p_5 = -\frac{e_1mx^*}{a_1 + x^{*2}} - \frac{e_2nx^*}{a_2 + x^{*2}},$$

$$p_6 = \left[\beta z^* - b - c + \alpha y^* - H_2 + r\left(1 - \frac{2x^*}{K}\right) - \frac{my^*\left(a_1 - x^{*2}\right)}{\left(a_1 + x^{*2}\right)^2} - \frac{nz^*\left(a_2 - x^{*2}\right)}{\left(a_2 + x^{*2}\right)^2}\right]\left(\frac{e_2nx^*}{a_2 + x^{*2}}\right.$$

$$+\frac{e_1mx^*}{a_1+x^{*2}}\Bigg)+\frac{e_1m^2x^*y_*(a_1-x^{*2})}{(a_1+x_{*2})^3}+\frac{e_2n^2x^*z^*\left(a_2-x^{*2}\right)}{(a_2+x^{*2})^3}$$

$$p_7=\left[r\left(1-\frac{2x}{K}\right)-\frac{my\left(a_1-x^2\right)}{\left(a_1+x^2\right)^2}-\frac{nz\left(a_2-x^2\right)}{\left(a_2+x^2\right)^2}\right]\left[\frac{e_2nx(H_1+\alpha z-\beta z+b+c)}{a_2+x^2}\right.$$

$$\left.-\frac{e_1mx(\alpha y+H_2+\beta z-b-c)}{a_1+x^2}\right]-\frac{e_1mx\left[\beta^2zw+(\alpha y-H_2)(\beta z-b-c)\right]}{a_1+x^2}$$

$$+\frac{e_2nx(H_1+\alpha z)(\beta z-b-c)}{a_2+x^2}-\frac{mx}{a_1+x^2}\left[\frac{e_1my(a_1-x^2)(\beta z-b-c+\alpha y-H_2)}{(a_1+x)^2}\right.$$

$$\left.+\frac{e_2nz\left(a_2-x^2\right)\alpha y}{\left(a_2+x^2\right)^2}\right]-\frac{nx}{a_2+x^2}\left[\frac{e_2nz\left(a_2-x^2\right)(\beta z-b-c-H_1-\alpha z)}{\left(a_2+x^2\right)^2}-\frac{e_1my(a_1-x^2)\alpha z}{(a_1+x)^2}\right]$$

$$p_8=\left[r\left(1-\frac{2x}{K}\right)-\frac{my\left(a_1-x^2\right)}{\left(a_1+x^2\right)^2}-\frac{nz\left(a_2-x^2\right)}{\left(a_2+x^2\right)^2}\right]\left[\frac{e_1mx\left[(\alpha y-H_2)(\beta z-b-c)+\beta^2zw\right]}{a_1+x^2}\right.$$

$$\left.-\frac{e_2nx(\beta z-b-c)(H_1+\alpha z)}{a_2+x^2}\right]-\left[\frac{e_1my(a_1-x^2)\alpha z}{(a_1+x)^2}+\frac{e_2nz\left(a_2-x^2\right)(H_1+\alpha z)}{\left(a_2+x^2\right)^2}\right]$$

$$\times\left(\frac{nx(\beta z-b-c)}{a_2+x^2}\right)+\frac{mx}{a_1+x^2}\left[\frac{e_1my(a_1-x^2)\left[(\alpha y-H_2)(\beta z-b-c)+\beta^2zw\right]}{(a_1+x)^2}\right.$$

$$\left.+\frac{e_2nz\left(a_2-x^2\right)[b\beta w+\alpha y(\beta z-b-c)]}{\left(a_2+x^2\right)^2}\right]$$

$$p_9=\frac{e_1e_2mnx^{*2}}{(a_1+x^{*2})(a_2+x^{*2})}$$

$$p_{10}=-\frac{e_1e_2mnx^{*2}}{(a_1+x^{*2})(a_2+x^{*2})}\left[r\left(1-\frac{2x^*}{K}\right)-\frac{my^*\left(a_1-x^{*2}\right)}{\left(a_1+x^{*2}\right)^2}-\frac{nz^*\left(a_2-x^{*2}\right)}{\left(a_2+x^{*2}\right)^2}-\beta z^*-b-c\right]$$

$$-\frac{mne_1e_2x^{*2}}{(a_1+x^{*2})(a_2+x^{*2})}\left[\frac{nz^*(a_2-x^{*2})}{a_2+x^{*2}}+\frac{my^*(a_1-x^{*2})}{a_1+x^{*2}}\right]$$

$$p_{11}=\left(\frac{e_1mx^*(\beta z^*-b-c)}{a_1+x^{*2}}\right)\left(\frac{e_2nx^*}{a_2+x^{*2}}\right)\left(r-\frac{2rx^*}{K}\right).$$

Case 1: When $\tau=0$, the characteristic equation becomes

$$\lambda^4+(p_1+p_5)\lambda^3+(p_2+p_6+p_9)\lambda^2+(p_3+p_7+p_{10})\lambda+p_4+p_8+p_{11}=0. \tag{3.19}$$

Using the Routh-Hurwitz criteria on the aforementioned Eq (3.19), E^* exhibits asymptotic stability provided that the subsequent condition is satisfied $(H(i))$: $p_1+p_4>0, p_3+p_6+p_8>0$ and $(p_1+p_4)(p_2+p_5+p_7)-(p_3+p_6+p_8)>0$.

Case 2: Taking $\tau>0$, and we investigate the effects of time delay τ on stability. We are currently trying to find a biologically meaningful periodic response. For the existence of a periodic solution of

system (1.5), we substitute $\lambda = i\sigma(\sigma > 0)$ in (3.18) and obtain the following pair of transcendental equations:

$$P_1\cos(\sigma\tau) + P_2\sin(\sigma\tau) + P_3\cos(2\sigma\tau) + P_4\sigma\sin(2\sigma\tau) = P_5$$
$$-P_1\sin(\sigma\tau) + P_2\cos(\sigma\tau) - P_3\sin(2\sigma\tau) + P_4\cos(2\sigma\tau) = P_6$$

where $P_1 = p_8 - p_6\sigma^2,\ P_2 = p_7\sigma - p_5\sigma^3,\ P_3 = p_{11} - \sigma^2 p_9,\ P_4 = \sigma p_{10}, P_5 = p_1\sigma^3 - p_3\sigma,$ $P_6 = p_2\sigma^2 - \sigma^4 - p_4$

which gives

$$\sin(\sigma\tau) = \frac{P_1P_3 + P_1P_5 + P_2P_4 + P_2P_6}{P_5^2 + P_6^2 - P_3^2 - P_4^2} \text{ and } \cos(\sigma\tau) = \frac{P_1P_4 - P_1P_6 + P_2P_5 - P_2P_3}{P_5^2 + P_6^2 - P_3^2 - P_4^2}. \tag{3.20}$$

Squaring and adding the above equations we get

$$\sigma^{16} + l_7\sigma^{14} + l_6\sigma^{12} + l_5\sigma^{10} + l_4\sigma^8 + l_3\sigma^6 + l_2\sigma^4 + l_1\sigma_2 + l_0 = 0, \tag{3.21}$$

$$l_7 = 2\left(p_1^2 - 2p_2\right) - p_5^2,$$

$$l_6 = \left(p_1^2 - 2p_2\right)^2 - (p_6 - p_5p_{10})^2 - 2p_5\left(p_3p_6 - p_8p_{10} - p_6p_{10} + p_7p_2 - p_7p_9 + p_4p_5 + p_5p_{11}\right) + 2\left(p_2^2 + 2p_4 - 2p_1p_3 - p_9^2\right)$$

$$l_5 = 2\left(p_3^2 - 2p_2p_4 + 2p_{11}p_9 - p_{10}^2\right) + 2\left(p_1^2 - 2p_2\right)\left(p_2^2 + 2p_4 - 2p_1p_3 - p_9^2\right) - 2\left(p_6 - p_5p_{10}\right)\left(p_6p_9 - p_2p_6 + p_5p_3 - p_5p_{10} - p_7p_1 - p_8\right) - 2p_5\left(p_1p_6 - p_7 - p_5p_2 - p_5p_9\right) - \left(p_3p_6 - p_8p_{10} - p_6p_{10} + p_7p_2 - p_7p_9 + p_4p_5 + p_5p_{11}\right)^2$$

$$l_4 = \left(p_2^2 + 2p_4 - 2p_1p_3 - p_9^2\right)^2 + 2\left(p_1^2 - 2p_2\right)\left(p_3^2 - 2p_2p_4 + 2p_{11}p_9 - p_{10}^2\right) + 2\left(p_4^2 - p_{11}^2\right) - \left(p_6p_9 - p_2p_6 + p_5p_3 - p_5p_{10} - p_7p_1 - p_8\right)^2 - 2p_5\left(p_8p_{10} - p_7p_4 - p_7p_{11}\right) - 2\left(p_6 - p_5p_{10}\right)\left(p_2p_8 - p_8p_9 + p_4p_6 - p_3p_7 - p_6p_{11} + p_7p_{10}\right) - 2\left(p_3p_6 - p_8p_{10} - p_6p_{10} + p_7p_2 - p_7p_9 + p_4p_5 + p_5p_{11}\right)\left(p_1p_6 - p_7 - p_5p_2 - p_5p_9\right)$$

$$l_3 = 2\left(p_1^2 - 2p_2\right)\left(p_4^2 - p_{11}^2\right) + 2\left(p_2^2 + 2p_4 - 2p_1p_3 - p_9^2\right)\left(p_3^2 - 2p_2p_4 + 2p_{11}p_9 - p_{10}^2\right) - 2\left(p_6p_9 - p_2p_6 + p_5p_3 - p_5p_{10} - p_7p_1 - p_8\right)\left(p_2p_8 - p_8p_9 + p_4p_6 - p_3p_7 - p_6p_{11} + p_7p_{10}\right) - 2\left(p_3p_6 - p_8p_{10} - p_6p_{10} + p_7p_2 - p_7p_9 + p_4p_5 + p_5p_{11}\right)\left(p_8p_{10} - p_7p_4 - p_7p_{11}\right) - 2\left(p_6 - p_5p_{10}\right)\left(p_{11}p_8 - p_8p_4\right) - \left(p_1p_6 - p_7 - p_5p_2 - p_5p_9\right)^2,$$

$$l_2 = \left(p_3^2 - 2p_2p_4 + 2p_{11}p_9 - p_{10}^2\right)^2 - 2\left(p_1p_6 - p_7 - p_5p_2 - p_5p_9\right)\left(p_8p_{10} - p_7p_4 - p_7p_{11}\right) - 2\left(p_2^2 + 2p_4 - 2p_1p_3 - p_9^2\right)\left(p_4^2 - p_{11}^2\right) - \left(p_2p_8 - p_8p_9 + p_4p_6 - p_3p_7 - p_6p_{11} + p_7p_{10}\right)^2 - 2\left(p_6p_9 - p_2p_6 + p_5p_3 - p_5p_{10} - p_7p_1 - p_8\right)\left(p_{11}p_8 - p_8p_4\right)$$

$$l_1 = 2\left(p_3^2 - 2p_2p_4 + 2p_{11}p_9 - p_{10}^2\right)\left(p_4^2 - p_{11}^2\right) - \left(p_8p_{10} - p_7p_4 - p_7p_{11}\right)^2 - 2\left(p_2p_8 - p_8p_9 + p_4p_6 - p_3p_7 - p_6p_{11} + p_7p_{10}\right)\left(p_{11}p_8 - p_8p_4\right)$$

$$l_0 = \left(p_4^2 - p_{11}^2\right)^2 - \left(p_{11}p_8 - p_8p_4\right)^2.$$

$(H(ii))$: It is assumed that σ_* is a positive root of Eq (3.21).
Then, from (3.20), we have

$$\tau_k = \frac{1}{\sigma_*}\arctan\left[\frac{P_1P_3 + P_1P_5 + P_2P_4 + P_2P_6}{P_1P_4 - P_1P_6 + P_2P_5 - P_2P_3}\right]_{\sigma=\sigma_*} + \frac{2k\pi}{\sigma_*}, \quad k = 0, 1, 2, 3...$$

We will now investigate the transversality condition of the Hopf bifurcation. When (3.18) is differentiated with regard to τ, it yields

$$\left(\frac{d\lambda}{d\tau}\right)^{-1} = \frac{4\lambda^3 + 3l_1\lambda^2 + 2l_2\lambda + l_3 + \left(3l_5\lambda^2 + 2l_6\lambda + l_7\right)e^{-\lambda\tau} + (2l_9\lambda + l_{10})\,e^{-2\lambda\tau}}{\lambda\left[(l_5\lambda^3 + l_6\lambda^2 + l_7\lambda + l_8)\,e^{-\lambda\tau} + (l_9\lambda^2 + l_{10}\lambda + l_{11})\,e^{-2\lambda\tau}\right]} - \frac{\tau}{\lambda}.$$

We get $\Re\left[\frac{d\lambda}{d\tau}\right]^{-1}_{\lambda=i\sigma_*,\tau=\tau_*} = \frac{E_1E_3 + E_2E_4}{E_1^2 + E_2^2}$,

where

$E_1 = (p_8\sigma_* - p_6\sigma_*^3)\cos\sigma_*\tau_* + (p_7\sigma_*^2 - p_5\sigma_*^4)\sin\sigma_*\tau_* - 2p_{10}\sigma_*^2\cos 2\sigma_*\tau_* + (p_{11}\sigma_* - 2p_9\sigma_*^3)\sin 2\sigma_*\tau_*,$
$E_2 = (p_6\sigma_*^3 - p_8\sigma_*)\cos\sigma_*\tau_* + (p_5\sigma_*^4 - p_7\sigma_*^2)\sin\sigma_*\tau_* + 2(p_9 + \sigma_*^3 + p_{11}\sigma_*)\cos 2\sigma_*\tau_* + 2p_{10}\sigma^2\sin 2\sigma\tau,$
$E_3 = p_3 - 3p_1\sigma_*^2 + (p_7 - 3p_5\sigma_*^2)\cos\sigma_*\tau_* + 2p_6\sigma_*\sin\sigma_*\tau_* + p_{10}\cos 2\sigma_*\tau_* + 2p_9\sigma_*\sin 2\sigma_*\tau_*,$
$E_4 = 2p_2\sigma_* - 4\sigma_*^3 + 2p_6\sigma_*\cos\sigma_*\tau_* + (3p_5\sigma_*^2 - p_7)\sin\sigma_*\tau_* + 2p_9\sigma_*\cos 2\sigma_*\tau_* - p_{10}\sin 2\sigma_*\tau_*.$

Therefore, the transversality condition $\Re\left[\frac{d\lambda}{d\tau}\right]_{\lambda=i\sigma_0,\tau=\tau_0} > 0$ holds as if $(H(iii)) : E_1E_3 + E_2E_4 > 0$. Here $\tau_0 = \tau_k|_{k=0}$.

Thus, we can state the following theorem.

Theorem 6. *Let us assume that for system* (1.5)*, the conditions* $(H(i)), (H(ii)), (H(iii))$ *satisfy. When* $\tau \in [0, \tau_*)$*, the interior equilibrium point* E^* *is locally asymptotically stable; when* $\tau = \tau_*$*, a Hopf-bifurcation takes place at the equilibrium point* E^**.*

4. Direction and stability of Hopf-bifurcation

The circumstances under which the system experiences Hopf-bifurcation for the time delay τ at various equilibrium points were determined in the preceding section.In this section, we use the center manifold theorem and normal form theory in accordance with Hassard's idea [38] to compute the direction, stability, and period of the bifurcated periodic solutions at $\tau = \tau_*$.

Assuming $\tau = \tau_* + \mu, \mu \in R$, the Hopf bifurcation value of system (1.5) is $\mu = 0$. If we rescale the time delay $t \to (\frac{t}{\tau})$, we may rewrite system (1.5) as

$$\dot{s}(t) = L_\mu s_t + F(\mu, w_t), \tag{4.1}$$

where $s(t) = (s_1(t), s_2(t), s_3(t), s_4(t))^T \in R^4$, $s_t(\psi) = s(t+\psi)$ and $L_\mu : C \to R^4, F : R\times C \to R^4$ are given by

$$L_\mu s_t = (\tau_* + \mu)\begin{pmatrix} a_{11} & a_{12} & a_{13} & 0 \\ 0 & a_{22} & -\alpha y^* & b \\ 0 & \alpha y^* & a_{33} & -\beta z \\ 0 & 0 & \beta z & a_{44} \end{pmatrix}\begin{pmatrix} s_{1t}(0) \\ s_{2t}(0) \\ s_{3t}(0) \\ s_{4t}(0) \end{pmatrix}$$

$$+(\tau_*+\mu)\begin{pmatrix}0&0&0&0\\b_{21}&b_{22}&0&0\\b_{31}&0&b_{33}&0\\0&0&0&0\end{pmatrix}\begin{pmatrix}s_{1t}(-1)\\s_{2t}(-1)\\s_{3t}(-1)\\s_{4t}(-1)\end{pmatrix}, \tag{4.2}$$

$$F(\mu,s_t)=(\tau_*+\mu)\begin{pmatrix}f_1s_{1t}^2(0)+f_2s_{1t}(0)s_{2t}(0)+f_3s_{1t}(0)w_{3t}(0)+\dots\\g_1s_{1t}^2(-1)+g_2s_{1t}(-1)s_{2t}(-1)+g_3s_{2t}(0)w_{3t}(0)+\dots\\h_1s_{1t}^2(-1)+h_2s_{1t}(-1)s_{3t}(-1)+h_3s_{2t}(0)w_{3t}(0)+\dots\\j_1s_{3t}(0)s_{4t}(0)+j_2s_{1t}^2(0)s_{2t}(0)+\dots\end{pmatrix},$$

where,

$$f_1=-\frac{2r}{K}+\frac{2mxy\left(3a_1-x^2\right)}{\left(a_1+x^2\right)^3}+\frac{2nxz\left(3a_2-x^2\right)}{\left(a_2+x^2\right)^3},\ f_2=-\frac{m\left(a_1-x^2\right)}{\left(a_1+x^2\right)^2},$$

$$f_3=-\frac{n\left(a_1-x^2\right)}{\left(a_2+x^2\right)^2},\ f_4=\frac{2mxy\left(3a_1-x^2\right)}{\left(a_1+x^2\right)^3},\ f_5=\frac{2nxz\left(3a_2-x^2\right)}{\left(a_2+x^2\right)^3}$$

$$f_6=\frac{my\left[\left(a_1+x^2\right)(a_1-x)-2x^2\left(3a_1-x\right)\right]}{\left(a_1+x^2\right)^4}+\frac{nz\left[\left(a_2+x^2\right)(a_2-x)-2x^2\left(3a_2-x\right)\right]}{\left(a_2+x^2\right)^4}$$

$$g_2=\frac{e_1m\left(a_1-x^2\right)}{\left(a_1+x^2\right)^2},\ g_1=-\frac{2e_1mxy\left(3a_1-x^2\right)}{\left(a_1+x^2\right)^3},\ g_4=-\frac{2e_1mx\left(3a_1-x^2\right)}{\left(a_1+x^2\right)^3}$$

$$g_5=-\frac{e_1my\left(a_1-x\right)}{\left(a_1+x^2\right)^3}+\frac{2e_1myx^2\left(3a_1-x^2\right)}{\left(a_1+x^2\right)^3},\ g_3=-\alpha,\ h_2=\frac{e_2n\left(a_2-x^2\right)}{\left(a_2+x^2\right)^2}$$

$$h_1=-\frac{2e_2mxz\left(3a_2-x^2\right)}{\left(a_2+x^2\right)^3},\ h_5=-\frac{2e_2nx\left(3a_2-x^2\right)}{\left(a_2+x^2\right)^3},\ h_3=\alpha,\ h_4=-\beta$$

$$h_6=-\frac{e_2nz\left(a_2-x\right)}{\left(a_2+x^2\right)^3}+\frac{2e_2nzx^2\left(3a_2-x^2\right)}{\left(a_2+x^2\right)^3},\ j_1=\beta.$$

The elements of a 4×4 matrix $\eta(\psi,\mu),\psi\in[-1,0]$ are of limited variation function, and as per the Riesz representation theorem [37], they exist such as

$$L_\mu\phi=\int_{-1}^{0}d\eta(\psi,\mu)\phi(\theta),\ \text{for}\ \phi\in C=C([-1,0],R^4). \tag{4.3}$$

Essentially, we have a choice

$$\eta(\psi,\mu)=(\tau_*+\mu)\begin{pmatrix}a_{11}&a_{12}&a_{13}&0\\0&a_{22}&-\alpha y^*&b\\0&\alpha z^*&a_{22}&-\beta w^*\\0&0&\beta z^*&a_{44}\end{pmatrix}\delta(\psi)+(\tau_*+\mu)\begin{pmatrix}0&0&0&0\\b_{21}&b_{22}&0&0\\b_{31}&0&b_{33}&0\\0&0&0&0\end{pmatrix}\delta(\psi+1). \tag{4.4}$$

In this case the Dirac delta function is denoted by δ.
For $\phi\in C([-1,0],R^4)$, define

$$B(\mu)\phi=\begin{cases}\frac{d\phi(\psi)}{d\psi}, & -1\le\psi<0\\ \int_{-1}^{0}d\eta(\psi,\mu)\phi(\psi), & \psi=0\end{cases} \tag{4.5}$$

$$Q(\mu)\phi = \begin{cases} 0 & -1 \le \psi < 0, \\ f(\mu, \phi) & \psi = 0. \end{cases} \tag{4.6}$$

After that, the differential equation will be the same as (1.5).

$$\dot{s}(t) = B\mu s_t + Q(\mu)s_t, \text{ where } v_t(\psi) = v(t+\psi),\ \psi \in [-1, 0]. \tag{4.7}$$

For $\theta \in C^1([0,1], (\mathbb{R}^4)^*)$, we define

$$B^*\theta(u) = \begin{cases} -\frac{d\theta(u)}{du} & 0 < u \le 1, \\ \int_{-1}^{0} d\eta^T(u,0)\theta(-u) & u = 0. \end{cases} \tag{4.8}$$

For $\phi \in C([0,1], \mathbb{R}^4)$ and $\theta \in C^1([0,1], (\mathbb{R}^4)^*)$, define a bilinear inner product

$$< \theta, \phi >= \bar{\theta}(0)\phi(0) - \int_{-1}^{0}\int_{\xi=0}^{\psi} \theta^T(\xi - \psi)d\eta(\psi)\phi(\xi)d\xi, \tag{4.9}$$

where $\eta(\psi) = \eta(\psi, 0), B = B(0)$, and B^* are adjoint operators. The eigenvalues of $B(0)$ are $\pm i\sigma_*\tau_*$, which implies that they are also eigenvalues of B^*. It is simple to confirm that the vectors $q(\psi) = (1, \alpha_1, \beta_1, \gamma_1)^T e^{i\sigma_*\tau_*\psi}$ $(\psi \in [-1, 0])$ and $q^*(s) = \frac{1}{D}(1, \alpha_1^*, \beta_1^*, \gamma_1^*)^T e^{i\sigma_*\tau_* s}$ $(s \in [-1, 0])$ are the eigenvectors of $B(0)$ and B^* corresponding to the eigenvalues $i\sigma_*\tau_*$ and $-i\sigma_*\tau_*$, respectively. Then, $B(0)q(\psi) = i\tau_*\sigma_* q(\psi)$. Additionally it follows from the definition of $B(0)$ in (4.4), (4.5), (4.7) that we have

$$\tau_* \begin{bmatrix} i\sigma^* - a_{11} & -a_{12} & -a_{13} & 0 \\ -b_{21}e^{-i\sigma_*\tau_*} & i\sigma_* - a_{22} - b_{22}e^{-i\sigma_*\tau_*} & \alpha y^* & b \\ -b_{31}e^{-i\sigma_*\tau_*} & -\alpha z^* & i\sigma_* - a_{33} - b_{33}e^{-i\sigma_*\tau_*} & -\beta z^* \\ 0 & 0 & \beta w^* & a_{44} \end{bmatrix} q(0) = \begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}.$$

Then we obtain

$$\alpha_1 = \frac{-a_{13}b_{21}(i\sigma_* - a_{44})e^{-i\sigma_*\tau_*} + (i\sigma_* - a_{11})(i\sigma_*\alpha y^* - a_{44}\alpha y^* - b\beta w^*)}{a_{12}(i\sigma_*\alpha y^* - a_{44}\alpha y^* - b\beta w^*) + a_{13}(i\sigma_* - a_{44})(a_{22} + b_{22}e^{-i\sigma_*\tau_*} - i\sigma_*)},$$

$$\beta_1 = \frac{a_{12}b_{21}e^{-i\sigma_*\tau_*} + (a_{11} - i\sigma_*)(i\sigma_*\alpha - a_{22} - b_{22}e^{-\sigma_*\tau_*})}{a_{12}(i\sigma_*\alpha y^* - a_{44}\alpha y^* - b\beta w^*) + a_{13}(i\sigma_* - a_{44})(a_{22} + b_{22}e^{-i\sigma_*\tau_*} - i\sigma_*)}.$$

$$\gamma_1 = \frac{\beta w^*\left[a_{12}b_{21}e^{-i\sigma_*\tau_*} + (a_{11} - i\sigma_*)(i\sigma_*\alpha - a_{22} - b_{22}e^{-\sigma_*\tau_*})\right]}{a_{12}(i\sigma_*\alpha y^* - a_{44}\alpha y^* - b\beta w^*) + a_{13}(i\sigma_* - a_{44})(a_{22} + b_{22}e^{-i\sigma_*\tau_*} - i\sigma_*)}.$$

According to the B^* definition, we may also compute $\alpha_1^*, \beta_1^*, \gamma_1^*$. We must ascertain the value of D in order to guarantee that $< q^*(s), q(\psi) >= 1$ and $< q^*(s), \bar{q}(\psi) >= 0$. Thus, we get from $< q^*(s), q(\psi) >= 1$,

$$\bar{D} = 1 + \alpha_1\bar{\alpha_1^*} + \beta_1\bar{\beta_1^*} + \gamma_1\bar{\gamma_1^*} + \tau_* e^{-i\sigma_*\tau_*}\left(b_{21}\bar{\alpha_1^*} + b_{31}\bar{\beta_1^*} + b_{22}\alpha_1\bar{\alpha_1^*} + b_{33}\beta\bar{\beta_1^*}\right).$$

Using a calculation procedure resembling to that of Song and Wei [39] and the algorithm outlined in Hassard [38] to derive the features of Hopf-bifurcation, we obtain

$$g_{20} = \frac{2\tau_*}{\bar{D}}\left[f_1 + \alpha_1 f_2 + \beta_1 f_3 + \bar{\alpha_1^*}\left(g_1 e^{-2i\sigma_*\tau_*} + g_2\alpha_1 e^{-2i\sigma_*\tau_*} + g_3\alpha_1\beta_1\right)\right.$$

$$+\bar{\beta}_1^*\left(h_1e^{-2i\sigma_*\tau_*}+h_2\beta_1e^{-2i\sigma_*\tau_*}+h_3\alpha_1\beta_1+c_4\gamma\beta\right)+\bar{\gamma}_1^*d_{0011}\gamma_1\beta_1\Big],$$

$$g_{11}=\frac{\tau_*}{\bar{D}}\Big[f_3(\beta_1+\bar{\beta}_1)+\bar{\alpha}_1^*\left(2g_1+g_2(\alpha_1+\bar{\alpha}_1)+g_3(\bar{\alpha}_1\beta_1+\alpha_1\bar{\beta}_1)\right)$$
$$+\bar{\beta}_1^*\left(2h_1+h_2(\beta_1+\bar{\beta}_1)+h_3(\bar{\alpha}_1\beta_1+\alpha\bar{\beta}_1)+h_4(\bar{\gamma}_1\beta_1+\gamma_1\bar{\beta}_1)\right)$$
$$+2f_1+f_3(\alpha_1+\bar{\alpha}_1)+d_{0011}(\bar{\gamma}_1\beta_1+\gamma_1\bar{\beta}_1)\bar{\gamma}_1^*\Big],$$

$$g_{02}=\frac{2\tau_*}{\bar{D}}\Big[f_1+\bar{\alpha}_1f_2+\bar{\beta}_1f_3+\bar{\alpha}_1^*\left(g_1e^{2i\sigma_*\tau_*}+g_2\bar{\alpha}_1e^{2i\sigma_*\tau_*}+g_3\bar{\alpha}_1\bar{\beta}_1\right)$$
$$+\bar{\beta}_1^*\left(h_1e^{2i\sigma_*\tau_*}+h_2\bar{\beta}_1e^{2i\sigma_*\tau_*}+h_3\bar{\alpha}_1\bar{\beta}_1+h_4\bar{\gamma}_1\bar{\beta}_1\right)+d_{0011}\bar{\gamma}_1\bar{\beta}_1\Big],$$

$$g_{21}=\frac{2\tau_*}{\bar{D}}\Big[3f_6+f_4\left(2\alpha_1+\bar{\alpha}_1\right)+f_5\left(2\beta_1+\bar{\beta}_1\right)+f_1\left(2W_{11}^{(1)}(0)+W_{20}^{(1)}(0)\right)$$
$$+f_2\left(W_{11}^{(2)}(0)+\frac{1}{2}W_{20}^{(2)}(0)+\frac{1}{2}\bar{\alpha}_1W_{20}^{(1)}(0)+\alpha_1W_{11}^{(1)}(0)\right)$$
$$+f_3\left(W_{11}^{(3)}(0)+\frac{1}{2}W_{20}^{(3)}(0)+\frac{1}{2}\bar{\beta}_1W_{20}^{(1)}(0)+\beta_1W_{11}^{(1)}(0)\right)$$
$$+\bar{\alpha}_1^*\left(\left(g_4\left(\bar{\alpha}_1+2\alpha_1\right)+g_5\right)e^{-i\sigma_*\tau_*}+g_1\left(e^{i\sigma_*\tau_*}W_{20}^{(1)}(-1)+2e^{-i\sigma_*\tau_*}W_{11}^{(1)}(-1)\right)\right)$$
$$+\bar{\alpha}_1^*g_2\left(e^{-i\sigma_*\tau_*}\left(W_{11}^{(2)}(-1)+\alpha_1W_{11}^{(1)}(-1)\right)+\frac{1}{2}e^{i\sigma_*\tau_*}\left(W_{20}^{(2)}(-1)+\bar{\alpha}_1W_{20}^{(2)}(-1)\right)\right)$$
$$+\bar{\alpha}_1^*g_3\left(\alpha_1W_{11}^{(3)}(0)+\frac{1}{2}\bar{\alpha}_1W_{20}^{(3)}(0)+\beta_1W_{11}^{(2)}(0)+\frac{1}{2}\bar{\beta}_1W_{20}^{(2)(0)}\right)$$
$$+h_1\left(e^{i\sigma_*\tau_*}W_{20}^{(1)}(-1)+2e^{-i\sigma_*\tau_*}W_{11}^{(1)}(-1)\right)+\left(h_5\left(\bar{\beta}_1+2\beta_1\right)+3\bar{\beta}_1^*h_6\right)e^{-i\sigma_*\tau_*}$$
$$+\bar{\beta}_1^*h_2\left(e^{-i\sigma_*\tau_*}\left(W_{11}^{(3)}(-1)+\beta_1W_{11}^{(1)}(-1)\right)+\frac{1}{2}e^{i\sigma_*\tau_*}\left(W_{20}^{(3)}(-1)+\bar{\beta}_1W_{20}^{(2)}(-1)\right)\right)$$
$$+\bar{\beta}_1^*h_3\left(\alpha_1W_{11}^{(3)}(0)+\frac{1}{2}\bar{\alpha}_1W_{20}^{(3)}(0)+\beta_1W_{11}^{(2)}(0)+\frac{1}{2}\bar{\beta}_1W_{20}^{(2)}\right)$$

$$+\bar{\beta}_1^*h_4\left(\gamma_1W_{11}^{(3)}(0)+\frac{1}{2}\bar{\gamma}_1W_{20}^{(3)}(0)+\beta_1W_{11}^{(4)}(0)+\frac{1}{2}\bar{\beta}_1W_{20}^{(4)}\right)$$
$$+\bar{\gamma}_1^*d_{0110}\left(\gamma_1W_{11}^{(3)}(0)+\frac{1}{2}\bar{\gamma}_1W_{20}^{(3)}(0)+\beta_1W_{11}^{(4)}(0)+\frac{1}{2}\bar{\beta}_1W_{20}^{(4)}\right)\Big].$$

where $W_{20}(\theta)=\dfrac{ig_{20}}{\sigma_*\tau_*}q(0)e^{i\theta\sigma_*\tau_*}+\dfrac{i\bar{g}_{02}}{3\sigma_0\tau_*}\bar{q}(0)e^{-i\theta\sigma_*\tau_*}+J_1e^{2i\theta\sigma_*\tau_*},$

$$W_{11}(\theta)=-\frac{ig_{11}}{\sigma_*\tau_*}q(0)e^{i\theta\sigma_*\tau_*}+\frac{i\bar{g}_{11}}{\sigma_*\tau_*}\bar{q}(0)e^{-i\theta\sigma_*\tau_*}+J_2,$$

$J_1=(J_1^1,J_1^2,J_1^3,J_1^4)^T$, and $J_2=(J_2^1,J_2^2,J_2^3,J_1^4)^T$, where of them is a constant vector in R^4. Following computation, we obtain $J_1=2K_1^{-1}K_2$ and $J_2=2K_3^{-1}K_4$ with

$$K_1=\begin{bmatrix}2i\sigma_*-a_{11} & -a_{12} & -a_{13} & 0\\ -b_{21}e^{-2i\sigma_*\tau_*} & 2i\sigma_0-a_{22}-b_{22}e^{-\sigma_*\tau_*} & -\alpha y^* & -b\\ -b_{31}e^{-2i\sigma_*\tau_*} & \alpha z^* & 2i\sigma_*-a_{33}-b_{33}e^{-\sigma_*\tau_*} & \beta z^*\\ 0 & 0 & -\beta z^* & 2i\sigma_*-a_{44}\end{bmatrix},$$

$$K_2 = \begin{bmatrix} f_1 + \alpha_1 f_2 + \beta_1 f_3 \\ g_1 e^{-2i\sigma_*\tau_*} + g_2\alpha_1 e^{-2i\sigma_*\tau_*} + g_3\alpha_1\beta_1 \\ h_1 e^{-2i\sigma_*\tau_*} + h_3\beta_1 e^{-2i\sigma_*\tau_*} + h_4\alpha_1\beta_1 + h_2\gamma\beta \\ j_1\gamma_1\beta_1 \end{bmatrix},$$

$$K_3 = \begin{bmatrix} -a_{11} & -a_{12} & -a_{13} & 0 \\ -b_{21} & -a_{22} - b_{22} & -\beta y^* & -b \\ -b_{31} & \alpha z^* & -a_{33} - b_{33} & \beta z^* \\ 0 & 0 & -\beta z^* & -a_{44} \end{bmatrix},$$

$$K_4 = \begin{bmatrix} 2f_1 + f_2(\alpha_1 + \bar{\alpha}_1) + f_3(\beta_1 + \bar{\beta}_1) \\ 2g_1 + g_2(\alpha_1 + \bar{\alpha}_1) + g_3(\bar{\alpha_1}\beta_1 + \alpha\bar{\beta}_1) \\ 2h_1 + h_3(\beta_1 + \bar{\beta}_1) + h_4(\bar{\alpha_1}\beta_1 + \alpha\bar{\beta}_1) + h_2(\bar{\gamma_1}\beta_1 + \gamma_1\bar{\beta}_1) \\ j_1(\bar{\gamma_1}\beta_1 + \gamma_1\bar{\beta}_1) \end{bmatrix}.$$

The parameters and delay may therefore be used to represent each g_{ij}. As a result, we determine the following values:

$$\begin{aligned} R_1(0) &= \frac{i}{2\tau_*\sigma_*}\left(g_{11}g_{20} - 2|g_{11}|^2 - \frac{|g_{02}|^2}{3}\right) + \frac{g_{21}}{2}, \\ \nu_1 &= -\frac{\Re\{R_1(0)\}}{\Re\{\lambda'(\tau_*)\}}, \\ \nu_2 &= 2\Re(R_1(0)), \\ T_2 &= -\frac{\Im\{R_1(0)\} + \eta_1\Im(\lambda'(\tau_*))}{\tau_*\sigma_*}. \end{aligned}$$

Theorem 7. *If $\nu_1 > 0 (\nu_1 < 0)$, then the Hopf bifurcation in system (1) is supercritical (subcritical). If $\nu_2 < 0 (\nu_2 > 0)$, then the bifurcating periodic solutions are stable (unstable). If $T_2 > 0\,(T_2 < 0)$, then the bifurcating periodic solutions increase (decrease).*

5. Numerical simulation

In this part, we use the four sets of parameter values R1, R2, and R3 to conduct some numerical simulations that validate and expand upon our analytical findings.

Take starting point $x(0) = 1$, $y(0) = 1$, $z(0) = 1$, $w(0) = 1$ with the set of parameters values R1=$(r, k, m, n, e_1, e_2, a_1, a_2, b, c, H_1, H_2, \alpha, \beta) = (5, 1, 2.5, 1.2, 2.1, 1, 0.8, 1.1, 0.2, 0.1, 1.5, 1.4, 0.5, 0.1)$. The disease-free and treatment-free equilibrium point $E_2(0.25, 1.30, 0, 0)$ is obtained for that combination of factors. Additionally, meet the requirements in Theorem 3.3, and we'll be able to determine the crucial time delay of $\tau_* = 0.22$, at which a Hopf-bifurcation emerges itself. Accordingly, for $\tau = 0.1 (< 0.22 = \tau_*)$ (see Figure 1), the system (1.5) is asymptotically stable at E_1, while for $\tau = 0.3 > (> 0.22 = \tau_*)$ (see Figure 2), it gets unstable. For the set of parameters values R1, the parameteric bifurcation diagram for vulnerable predator and prey populations is shown in Figure 3, and it illustrates how the system evolves when τ's numerical values fluctuate within $[0.2, 0.25]$. It suggests that the dynamics of the system will fluctuate via a Hopf bifurcation point at $\tau = 0.22$ if we raise the value of the τ parameter. Now with $\tau = 0.3$, the system (1.5) becomes stable when we increase

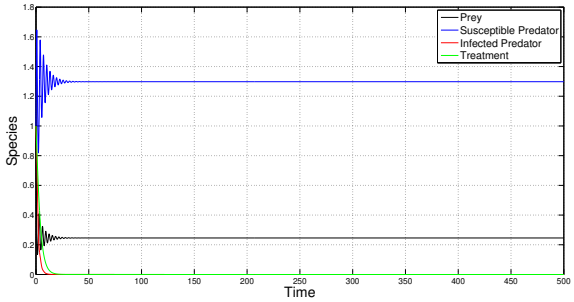
the parameter H_1 while maintaining the values of the remaining factors as in R1, as demonstrated in Figure 4. However, when we raise H_1 even more, the healthy predator vanishes and only the existence of prey species is shown in Figure 5. Additionally, we have a critical value of factor τ for every single value of $H_1(< 2.1)$.

Now, if we replace the values of the parameters $n = 2.2, H_2 = 0.5$ in R1 with starting point $x(0) = 1,\ y(0) = 1,\ z(0) = 1,\ w(0) = 1$, we get the helathy predator-free and treatment-free equilibrium point $E_3(0.266, 0, 1.952, 0)$. Additionally, we meet the requirements of Theorem 3.4, and we'll be able to determine the crucial time delay of $\tau_* = 1.09$, at which a Hopf-bifurcation manifests itself. Accordingly, for $\tau = 1.05(< 1.09 = \tau_*)$ (see Figure 6), the system (1.5) is asymptotically stable at E_3, and for $\tau = 1.5(> 1.09 = \tau_*)$ (see Figure 7), it gets unstable. For the set of parameters values R1 with $n = 2.2$, the parameteric bifurcation diagram for the populations of infected predators and prey is shown in Figure 8. It illustrates how the system evolves when the numerical values of τ fluctuate within $[1.05, 1.14]$. It suggests that the behaviour of the model will fluctuate via a Hopf bifurcation point at $\tau_* = 1.09$ if we raise the value of the τ parameter. The system (1.5) becomes stable if we take $\tau = 0.2$ and increase the value of the parameter H_2 while maintaining the values of the remaining factors at the same level as in R1 with $n = 2.2$. This will cause the sick predator to gradually disappear from the system and leave only healthy predators and prey. Here, the $0.05 \leq H_2 < 0.7$ system becomes stable and only sick predator and prey species are present, which is similar to the Figure 6. After $0.7 < h_2 < 1.11$, then prey, healthy, and sick predator are only present but if we increase then sick predator vanishes and only healthy predator and prey species are survived which is similar to the Figure 1.

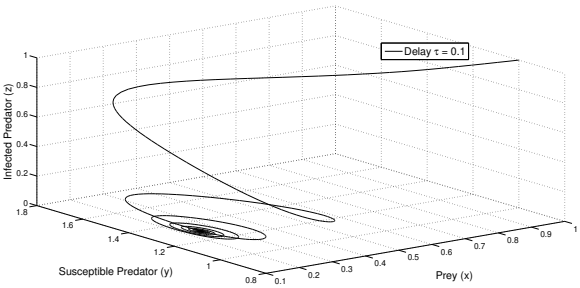
Take starting point $x(0) = 1,\ y(0) = 1,\ z(0) = 1,\ w(0) = 1$ with the set of parameters values R2=$(r, k, m, n, e_1, e_2, a_1, a_2, b, c, H_1, H_2, \alpha, \beta) = (5, 1, 2.5, 2.2, 2.1, 1.8, 1.2, 1.1, 1, 0.5, 1, 0.51, 0.5, 1)$. We obtain the treatment-free equilibrium point $E_5(0.138, 0.945, 0.53, 0)$ for this combination of parameters R2. Additionally, meet the requirements of Theorem 3.5, and we'll be able to determine the crucial time delay of $\tau = 0.53$, at which a Hopf-bifurcation exhibits itself. Accordingly, for $\tau = 0.1 < 0.135$ (see Figure 9) the system (1.5) is asymptotically stable at E_5, and for $\tau = 0.2 > 0.135$ (see Figure 10), it becomes unstable. For the set of parameters values R2, the parameteric bifurcation diagram for susceptible, infected, and prey predator populations is shown in Figure 11. It illustrates how the system evolves when τ's numerical values fluctuate within $[0.09, 0.15]$. It suggests that the behavior of the model will fluctuate via a Hopf bifurcation point at $\tau = 0.115$ if we raise the value of the τ parameter.

Lastly, taking initial point $x(0) = 1,\ y(0) = 1,\ z(0) = 1,\ w(0) = 1$ with the set of parameters values R3=$(r, k, m, n, e_1, e_2, a_1, a_2, b, c, H_1, H_2, \alpha, \beta) = (5, 1, 2.5, 1.2, 2.1, 1, 0.8, 1.1, 0.2, 0.1, 1.5, 1.4, 0.5, 0.1)$. There is an interior equilibrium point $E^*(0.227, 0.5, 1.5, 0.4)$ for this combination of values. Additionally, meet the requirements in Theorem 3.3, and we'll be able to determine the critical delay of $\tau = 0.61$, at which a Hopf-bifurcation displays itself. Accordingly, the system (1.5) is unstable if $\tau = 1 > 0.61$ (see Figure 13) and asymptotically stable at E^* for $\tau = 0.5 < 0.61$ (see Figure 12). For the set of parameters values R3, the parameteric bifurcation diagram for all populations is shown in Figure 14, and it displays the way the system evolves when τ's numerical values fluctuate within $[0.57, 0.66]$. It suggests that the behavior of the system will fluctuate via a Hopf bifurcation point at $\tau = 0.58$ if we raise the value of the τ parameter. Given $\tau = 1$, increasing the value of the component α while maintaining the values of the remaining factors as in R3 results in a Hopf-bifurcation, with

$\alpha^* = 1.05$ serving as the crucial value of α. Figure 15 illustrates the stable state of the system (1.5) when the quantity of α is further increased. Figure 16 illustrates the unstable state of the system when the value of $\alpha = 0.5$ is decreased. Furthermore, we demonstrate that by substituting this value for the parameter $\alpha = 0.05, \beta = 2$ in R3, we can increase system chaos by increasing the gestation time delay value, as shown in Figure 17, or decrease it to see less chaotic behavior, as in Figure 18. Figure 19 shows the parameteric bifurcation diagram for all populations for the set of parameters values R3 with $\alpha = 0.05, \beta = 2$. It illustrates how the system gets more chaotic as τ's numerical values increase from $\tau = 0.43$. Again decreament in the value of the parameter of gestation time delay system becomes stable. To verify the chaotic nature of the system (1.5), we calculate the maximum Lyapunov exponent at $\tau = 2.1$ using the approach developed by Wolf et al. [40] (see Figures 20 and 21).

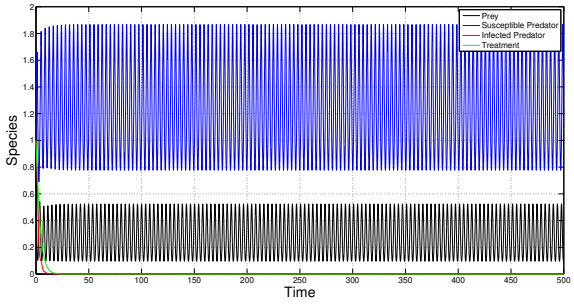


(a)

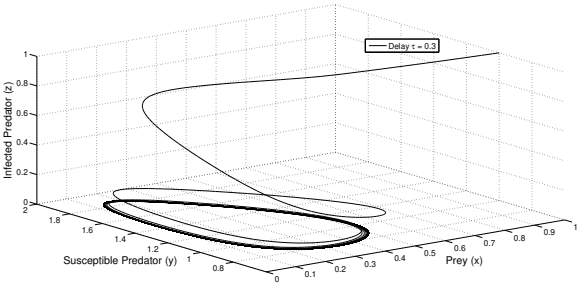


(b)

Figure 2. (a) Time series solution and, (b) parametric graph of the system (5) about the disease and treatment-free equilibrium point E_2 that is locally asymptotically stable when $\tau = 0.1(< 0.22 = \tau^*)$ with set of parameter values in R1.

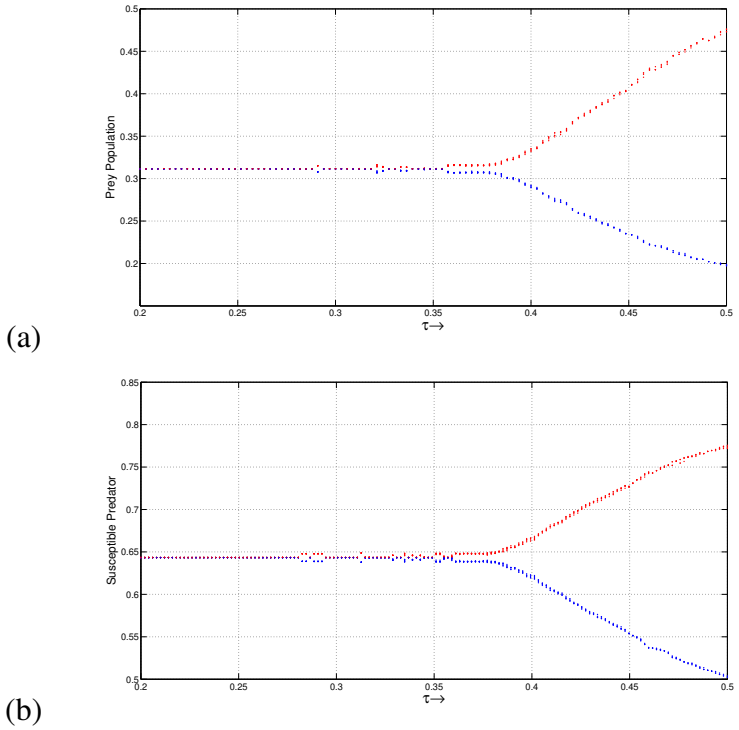


(a)



(b)

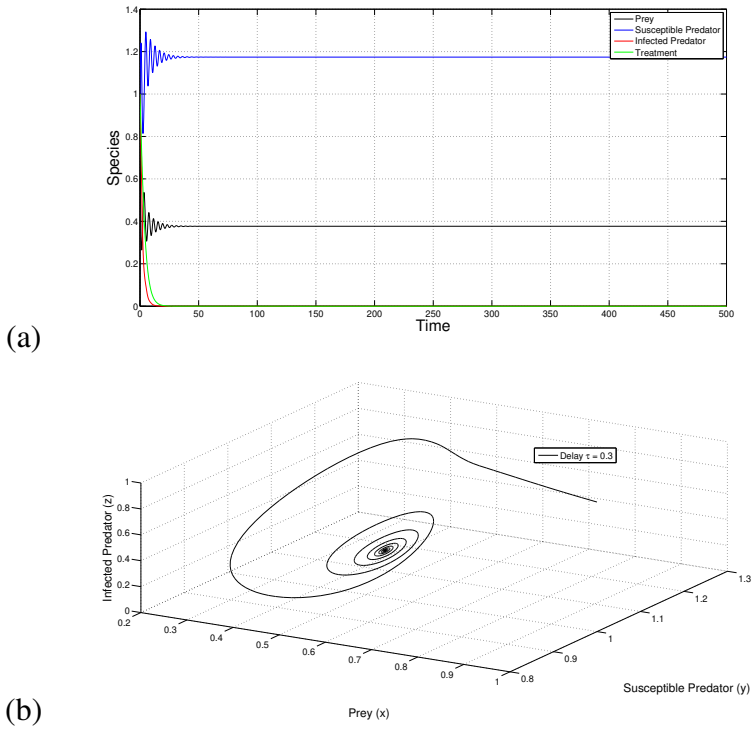
Figure 3. The settings are the same as in Figure 1, with the exception that $\tau = 0.3(> 0.22 = \tau^*)$, then E_2 loses its stability. Here, (b) and (a) denote the phase and oscillation patterns of the population, respectively.



(a)

(b)

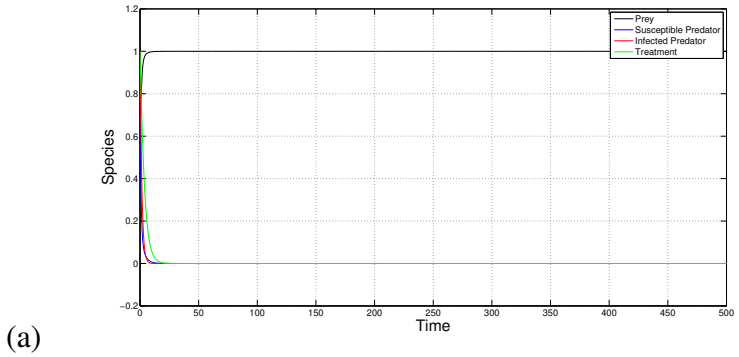
Figure 4. Diagram illustrating the prey and susceptible predator populations' bifurcation in terms of delay with respect to the bifurcating parameter, τ.



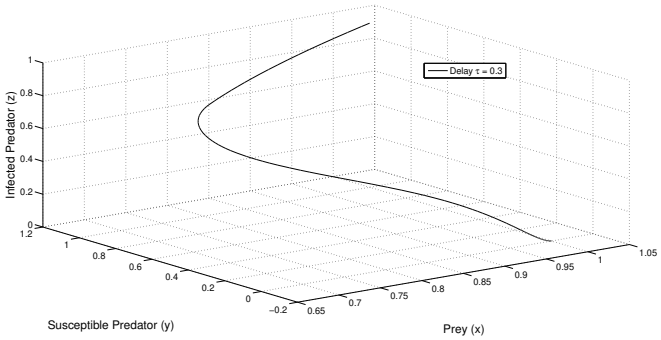
(a)

(b)

Figure 5. The settings are the same as in Figure 2, with the exception that $H_1 = 2.1(> 1.78 = H_{1_2})$, then E_2 becomes locally asymptotically stable. Here, (a) time series solution and, (b) parametric graph of the system (5).

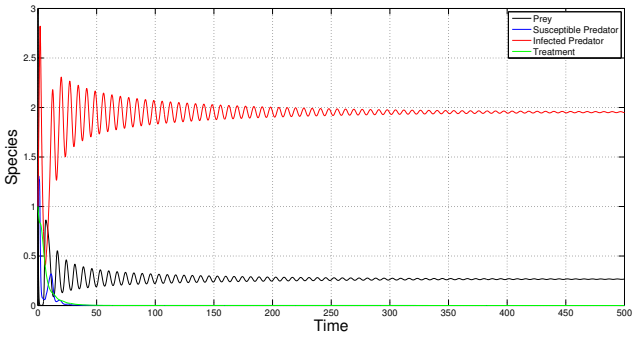


(a)

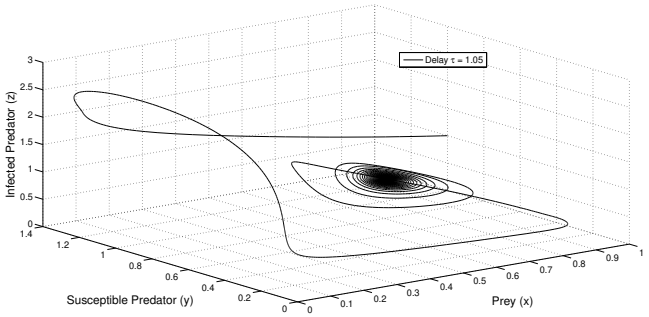


(b)

Figure 6. The settings are the same as in Figure 2, with the exception that $H_1 = 4.5(> 1.78 = H_{1_2})$ resulting in healthy predator vanishes only when prey species survived. Here, (a) time series solution and; (b) parametric graph of the system (5).

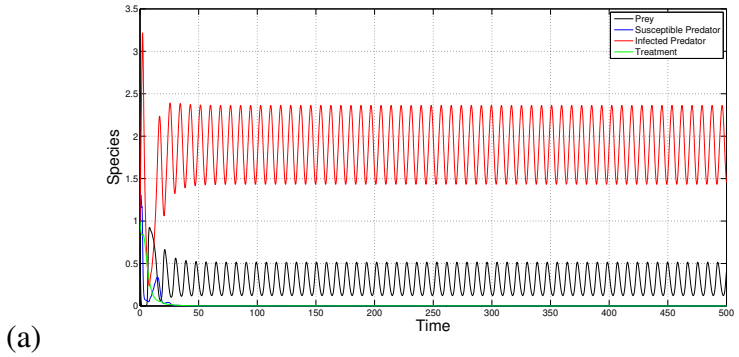


(a)

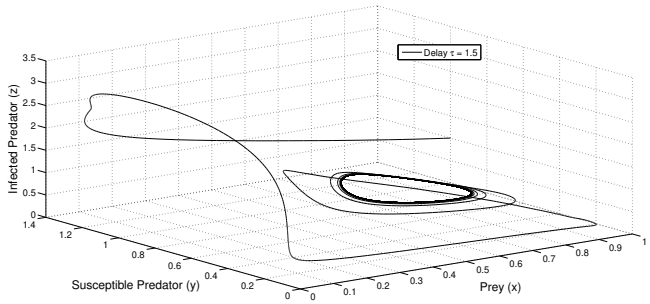


(b)

Figure 7. The settings are the same as in Figure 1, with the exception that $n = 2.2, h_2 = 0.5$, then about the healthy predator-free and treatment-free equilibrium point E_3 is locally asymptotically stable when $\tau = 1.05(< 1.09 = \tau^*)$. Here, (a) time series solution and, (b) parametric graph of the system (5).

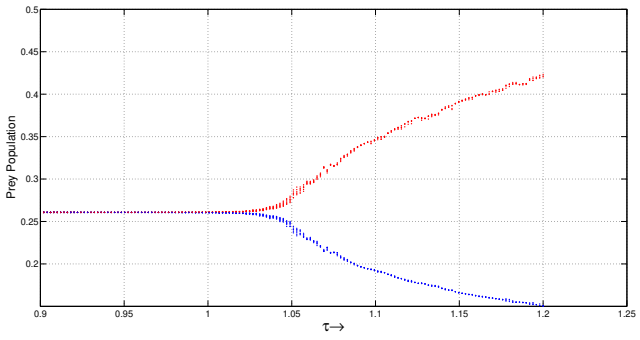


(a)



(b)

Figure 8. The settings are the same as in Figure 5, then about the healthy predator-free and treatment-free equilibrium point E_3 loses its stability when $\tau = 1.5(< 1.09 = \tau_3^*)$. Here, (b) and (a) denote the phase and oscillation patterns of the population, respectively.



(a) (b)

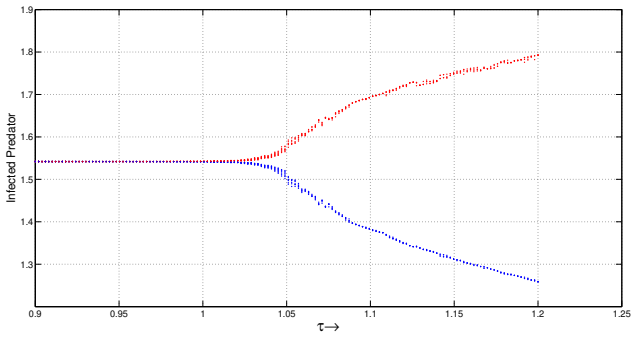
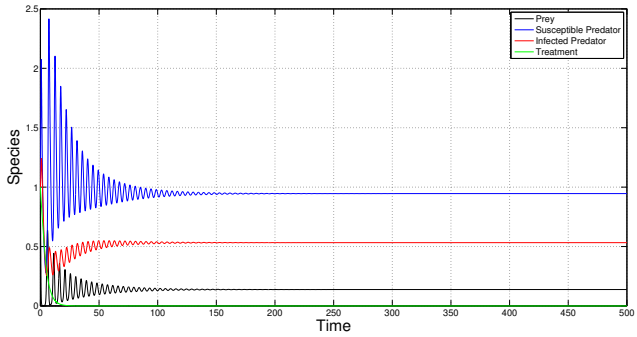
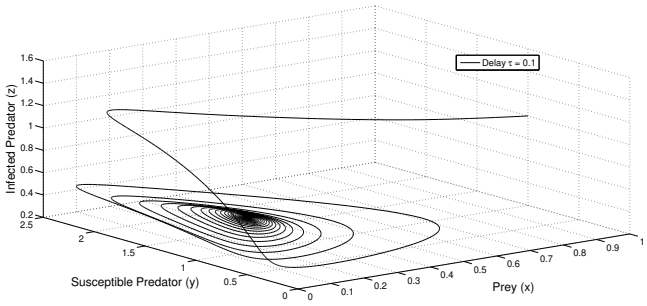


Figure 9. Diagram illustrating how the populations of infected predators and prey split according to the bifurcating parameter, τ, in terms of delay.

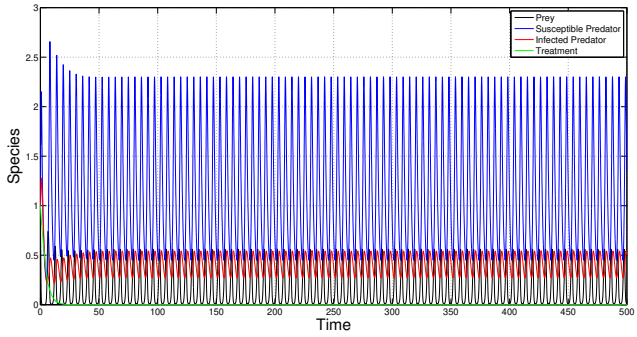


(a)

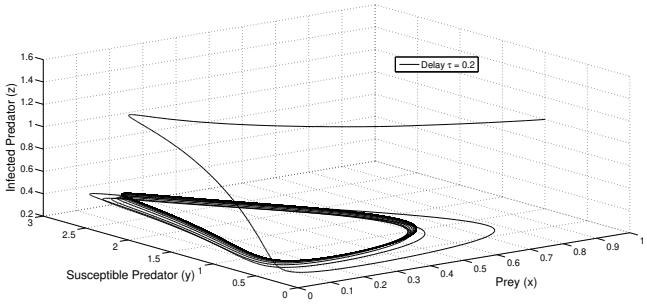


(b)

Figure 10. (a) Time series solution and, (b) parametric graph of the system (5) about how the treatment-free equilibrium point E_5 is locally asymptotically stable when $\tau = 0.1 (< 0.135 = \tau_0)$ with set of parameter values in R2.



(a)



(b)

Figure 11. The settings are the same as in Figure 7, with the exception that $\tau = 0.2 (> 0.135 = \tau_0)$, then E_5 loses its stability. Here, (b) and (a) denote the phase and oscillation patterns of the population, respectively.

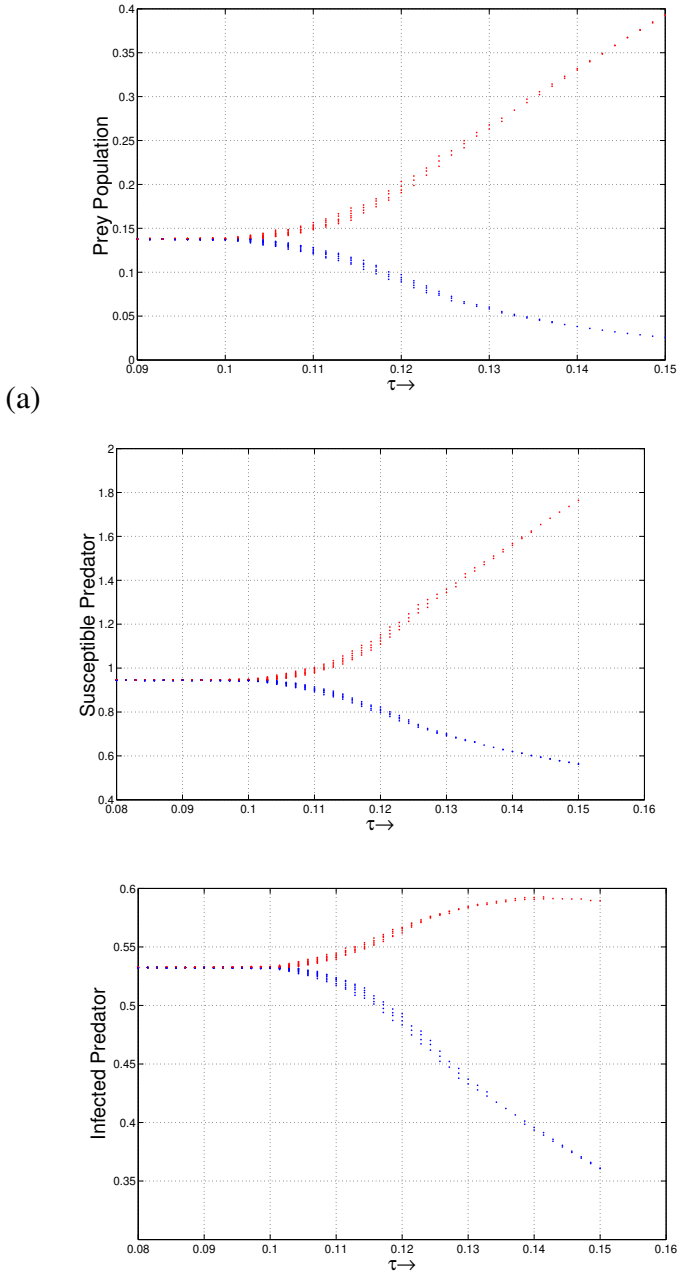
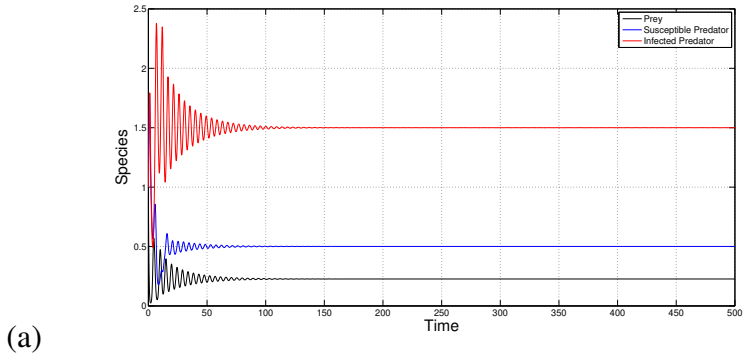
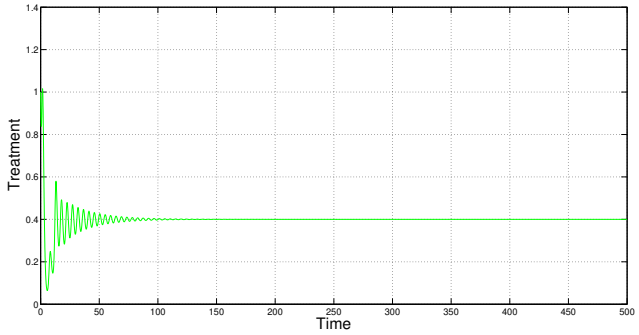


Figure 12. The bifurcation diagram represents the relationship between the populations of susceptible predators, infected predators, and prey with respect to τ, the bifurcating parameter for the delay factor.



(a) (b)



(c)

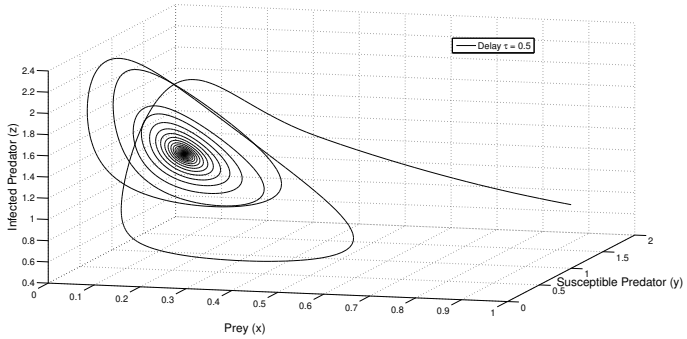


Figure 13. (a) and (b) time series solution and, (c) parametric graph of the system (1.5) about the interior equilibrium point E^* is locally asymptotically stable when $\tau = 0.5(< 0.61 = \tau_*)$ with set of parameter values in R3.

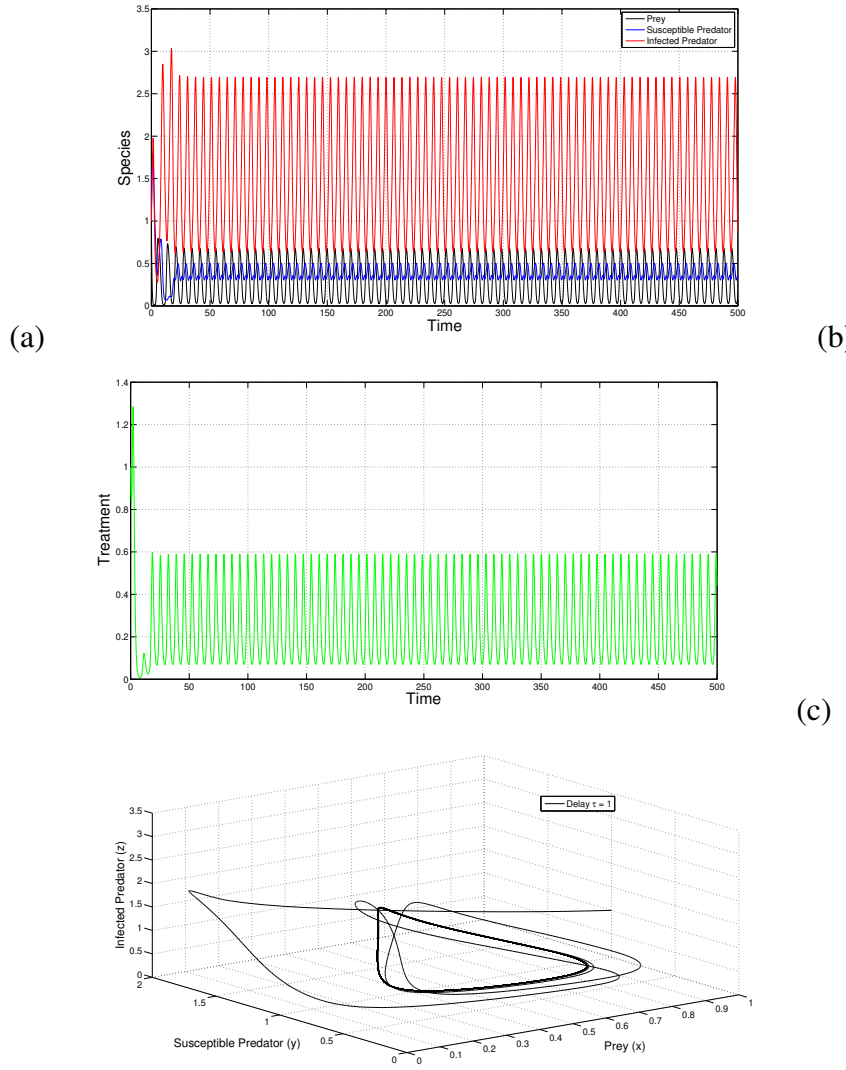
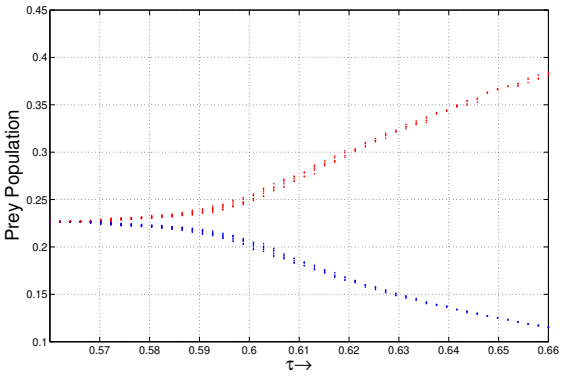
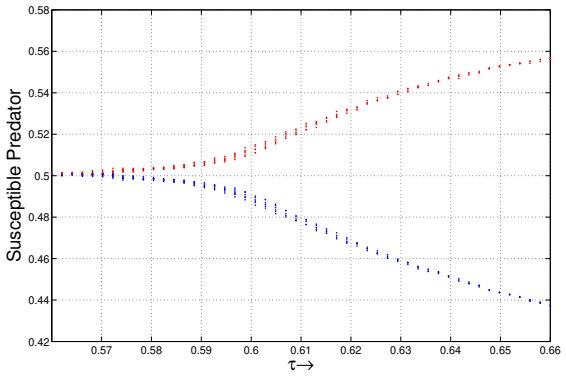


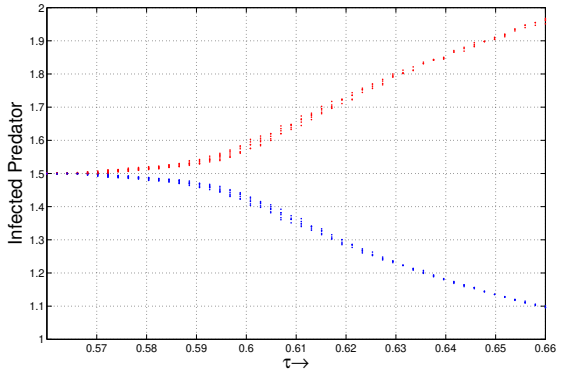
Figure 14. The settings are the same as in Figure 9, with the exception that $\tau = 1 (> 0.61 = \tau^*)$, then interior equilibrium E^* loses its stability. Here, (b) and (a) denote the phase and oscillation patterns of the population, respectively.



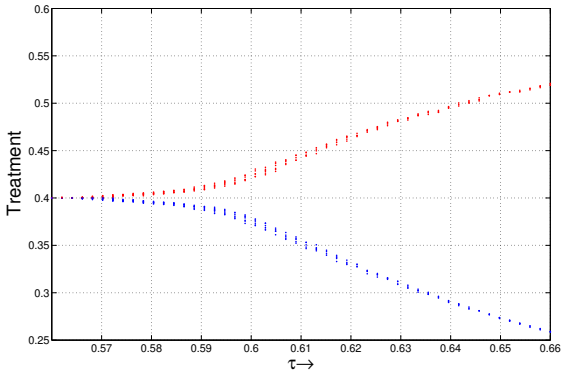
(a)



(b)

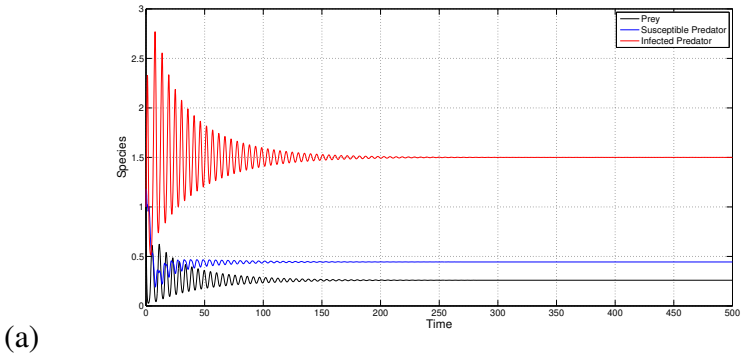


(c)

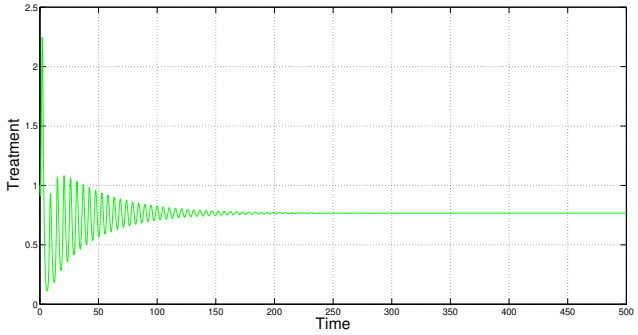


(d)

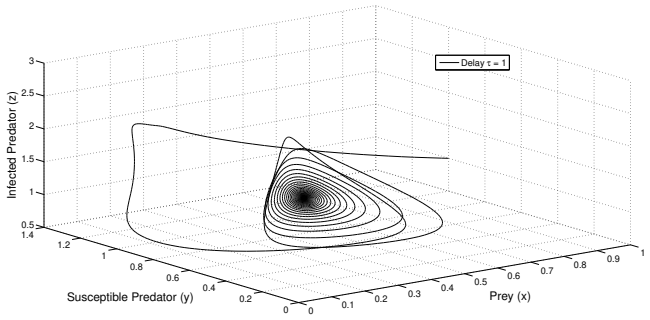
Figure 15. Bifurcation diagram for system (1.5) in relation to the bifurcating parameter, τ, for the delay parameter.



(a)

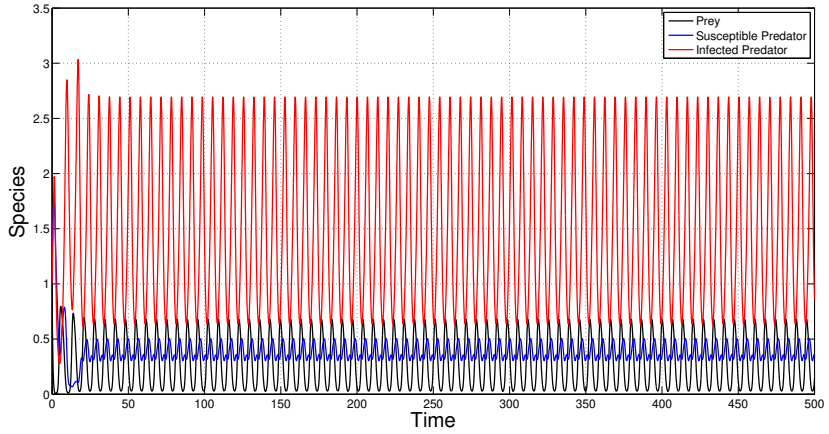


(b)

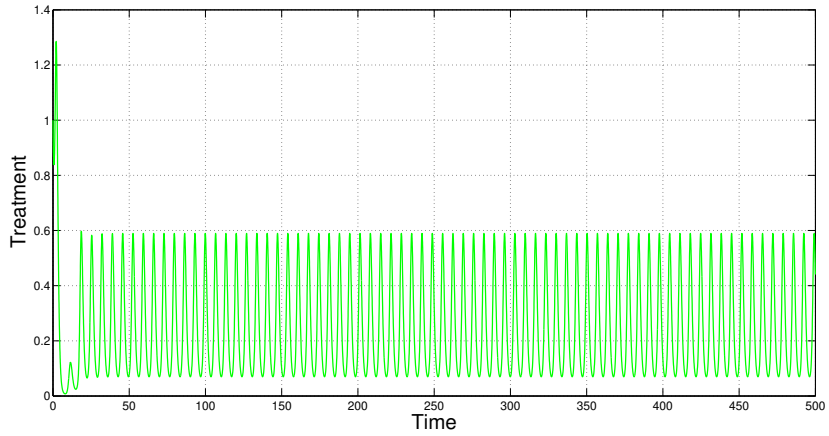


(c)

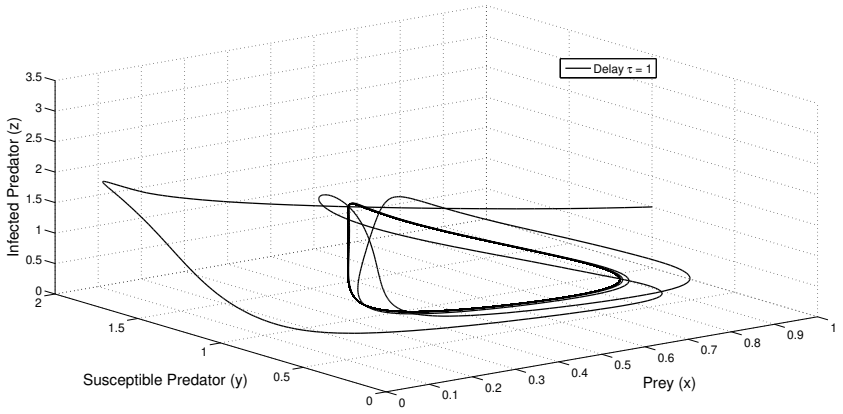
Figure 16. The settings are the same as in Figure 10, with the exception that $\alpha = 1.2(> 1.05 = \alpha_*)$, then E^* becomes locally asymptotically stable. Here, (a) time series solution and, (b) parametric graph of the system (5).



(a)

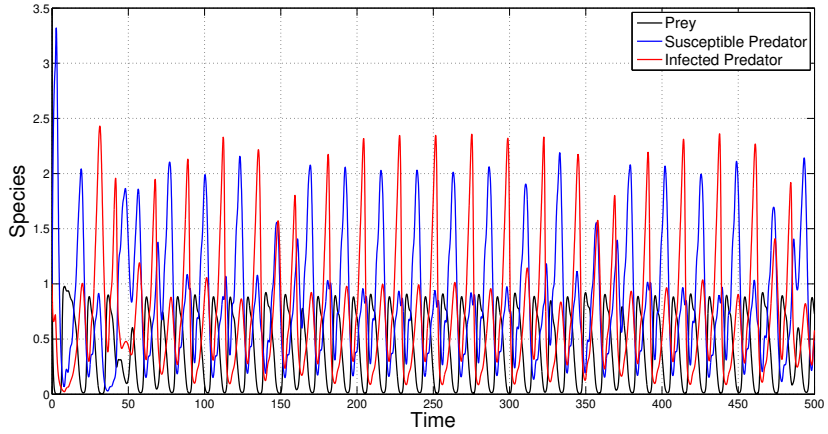


(b)

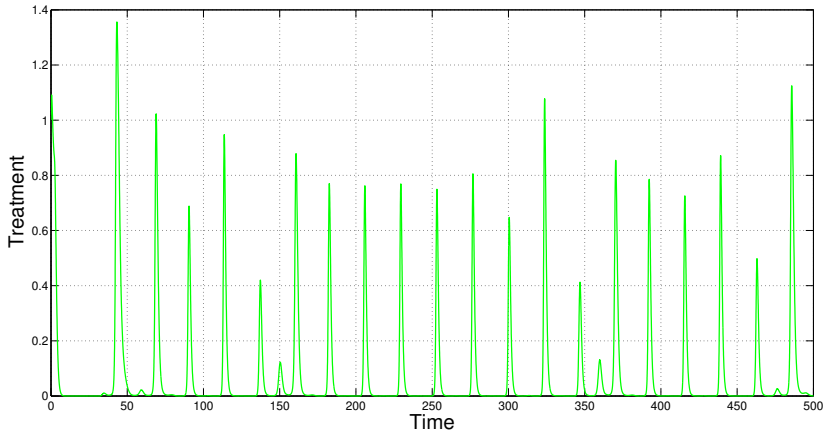


(c)

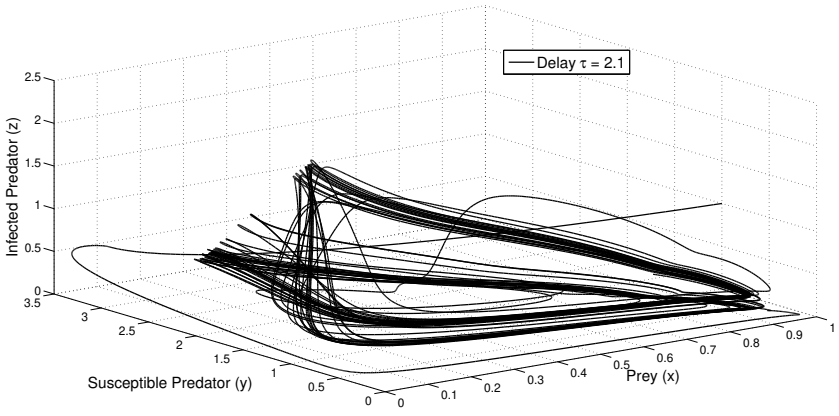
Figure 17. The settings are the same as in Figure 10, with the exception that $\alpha = 0.5(< 1.05 = \alpha_*)$, then E^* loses its stability. Here, (b) and (a) denote the phase and oscillation patterns of the population, respectively.



(a)

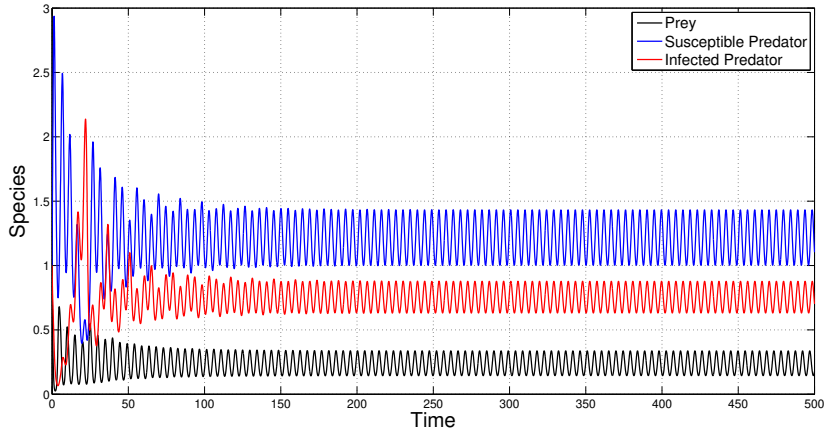


(b)

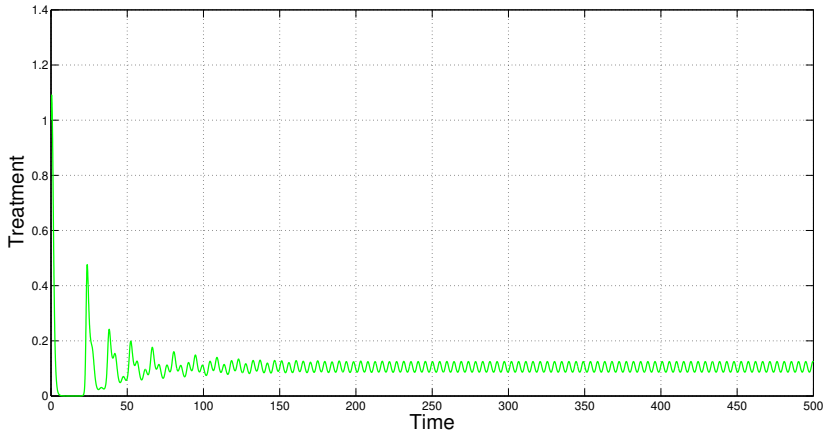


(c)

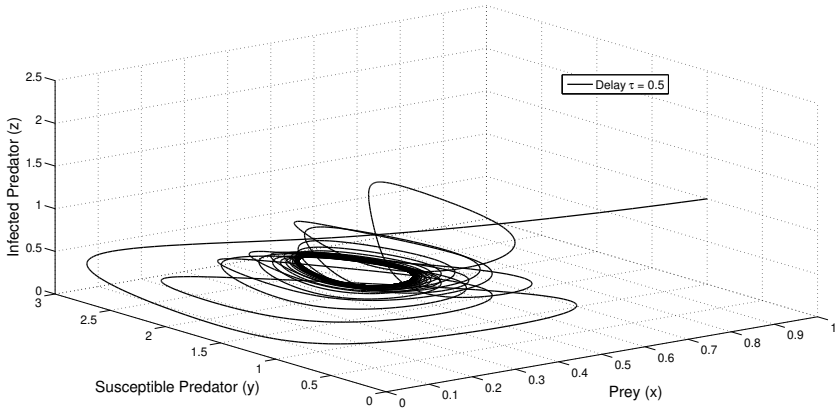
Figure 18. Considering the parameters value in R3 with the exception that $\alpha = 0.05, \beta = 2$ for $\tau = 2.1$ system (1.5) becomes chaotic.



(a)



(b)



(c)

Figure 19. The settings are the same as in Figure 13. If we decrese the value of the gestation time delay, then chaotic behave becomes diminises. Again decreament in the value of the parameter of gestation time delay system becomes stable.

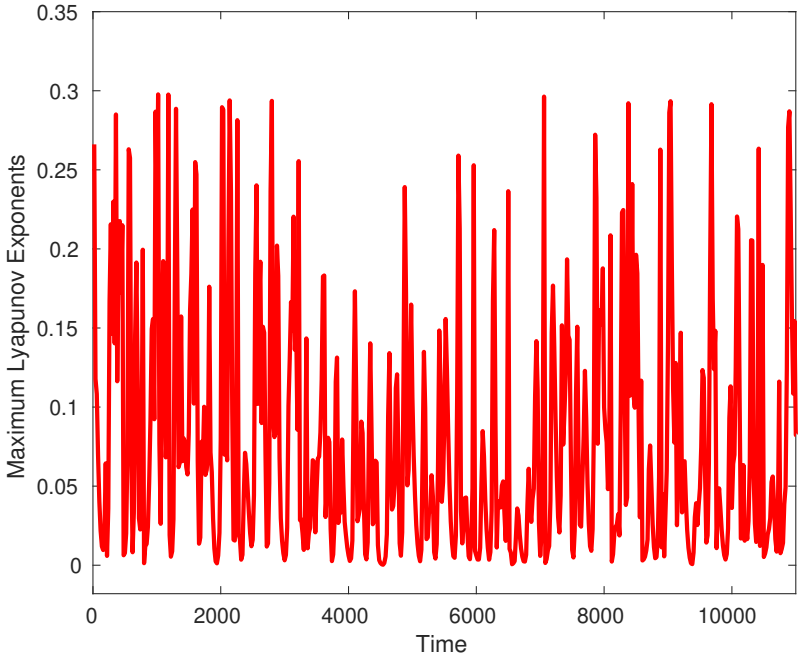
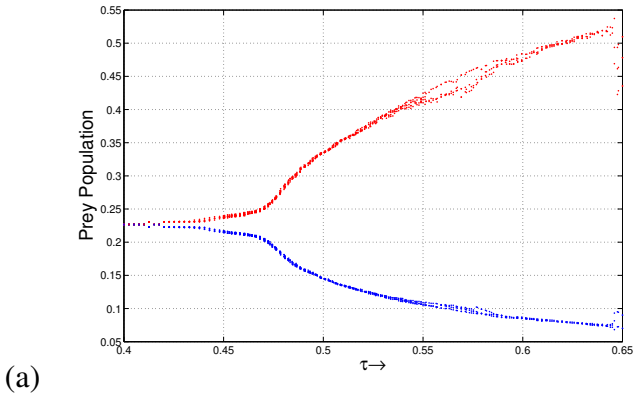
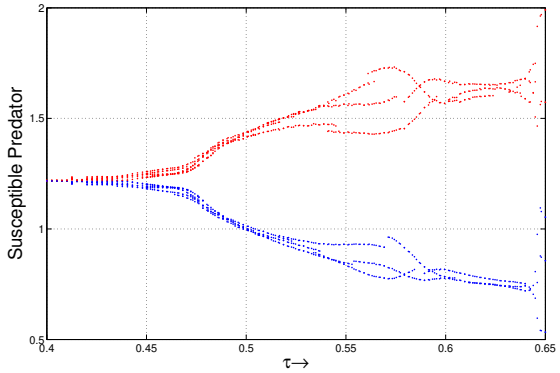


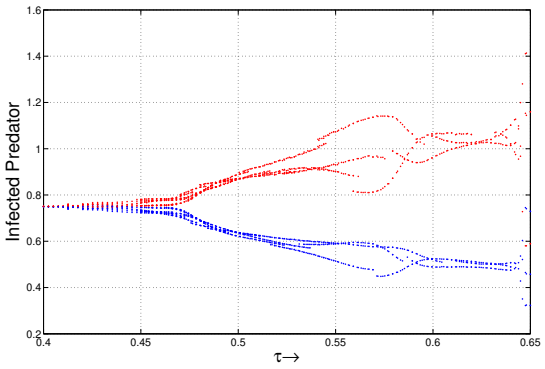
Figure 20. Maximum Lyapunov exponent of the system (1.5) at $\tau = 2.1$. Parameters are taken from R_3. The positive values of the maximum Lyapunov exponent confirm the existence of chaotic oscillation.



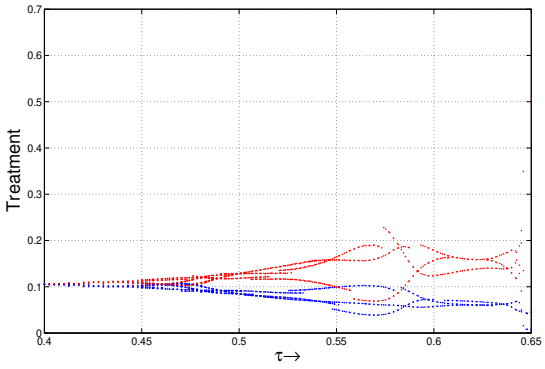
(a)



(b)



(c)



(d)

Figure 21. Bifurcation diagram for system (1.5) in relation to the bifurcating parameter, τ, for the delay parameter. It illustrates that the system gets more chaotic as τ's numerical values increase from $\tau = 0.43$.

6. Conclusions

In the current study, we have examined the structure and functioning of a prey-predator model in which predator species are harvested while infection spread occurs solely among the predators. A time delay is used to indicate gestation delay of the predators with Holling type III functional responses and also the infected predator population is treated. We came up with the presence of equilibria and gave their positive conditions for the suggested delayed model (1.5). Additionally, we provided boundedness for the delayed model's solution (1.5). There are seven potential nonnegative equilibria in this prey-predator model, and we also present a thorough examination of the stability of each potential nonnegative equilibria. Trivial equilibrium is inherently unstable among them since it requires the existence of either prey species or both predator and prey species at first. By using Theorem 3.2, we can observe that if the rates at which susceptible and infected predators are harvested exceed $\frac{e_1 mK}{a_1+K^2}$ and $\frac{e_2 nK}{a_2+K^2}$, respectively, then the predator-free and treatment-free equilibrium point is feasible as well as asymptotically stable.

A disease-free and treatment-free equilibrium point can only be feasible if the carrying capacity of the prey species exceeds the density of the prey population and the harvesting rate of susceptible predators is below a certain threshold. It has been shown in Theorem 3.3 that the disease-free and treatment-free equilibrium must meet a number of requirements in order to be locally asymptotically stable. Furthermore, it has been demonstrated that the time delay τ may, in some circumstances, destabilize the system's (1.5) equilibrium point that is free of sickness and treatment, leading to population fluctuations. Hopf-bifurcation of the system (1.5) is seen, and when the time delay is sufficiently small, the system is shown to be locally asymptotically stable. In this instance, the at-risk predator and prey may cohabit in an ideal, stable condition free from the threat of illness, which is crucial to the predator's harvesting model. The system's stability (1.5) depends on time delay when we raise the pace at which susceptible predators are harvested. However, if susceptible predators are harvested too much, healthy predators may disappear from the habitat, leaving only prey species, which is undesirable for predator harvesting which is shown in Figure 4.

Only when the prey species' carrying capacity surpasses the prey population density and the harvesting rate of infected predators falls below a specific threshold can a healthy predator-free and treatment-free equilibrium point become feasible. Theorem 3.4 has demonstrated the conditions that must be satisfied for the healthy predator-free and treatment-free equilibrium to be locally asymptotically stable. Moreover, it is shown that the time delay τ can, under some conditions, cause population fluctuations by destabilising the system's (1.5) equilibrium point, which is free of healthy predator and treatment. The system (1.5) exhibits hopf-bifurcation and, with a small enough time delay, it is demonstrated to be locally asymptotically stable. With a fixed time delay and a set of parameters, the system becomes stable when the infected predator harvesting rate is between 0.5 and 0.7. Only sick predators and prey species remain present. However, if the infected predator harvesting rate is increased, the sick predator disappears and only healthy predators and prey species remain, which is the ideal situation for harvesting.

It has been demonstrated that utilizing Table 1 to determine the treatment-free equilibrium point is only possible under specific circumstances. Theorem 3.5 demonstrates the conditions that must be satisfied for the treatment-free equilibrium to be locally asymptotically stable. Furthermore, it is demonstrated that, in some circumstances, the time delay τ can lead to population fluctuations by

upsetting the system's (1.5) equilibrium point, which is unaffected by treatments. It is shown that, with small enough time delays, the system (1.5) is locally asymptotically stable and shows hopf-bifurcation. The necessary and sufficient conditions for the stability and instability of the interior equilibrium in both the presence and absence of gestation time delays are already established. The time delay τ plays a crucial role in controlling the behavior of the suggested system for interior equilibrium. For the model system (1.5), Hopf bifurcation takes place when the time delay passes the critical value τ_*. It is found that, when the time delay value is properly low, system (1.5) is locally asymptotically stable. In this scenario, the densities of the four species—the diseased predator population, the healthy predator population, the prey population, and the recovered population—will trend toward stabilization, meaning that the illness can be contained and the populations of the three species will be in an optimal, stable condition. Also, the infection rate α leads to fluctuations in the population and destabilizes the system's interior equilibrium point (1.5). In addition, we demonstrate that by substituting this value for the parameter $\alpha = 0.05, \beta = 2$ in R3, the system becomes less chaotic as Figure 13 illustrates. Conversely, if the gestation time delay is decreased, the chaotic behavior increases. Once more, a decline in the gestation time delay system's parameter value leads to stability. Research has been done on the dynamics of predator-prey relationships, the consequences of infectious illnesses propagating across predator populations, the management of diseased predators, and gestational time delays (cf. [18–20]). These studies investigated the existence of Hopf bifurcation and the local asymptotic stability of the coexistence equilibrium, with the delay serving as the bifurcation parameter. The local asymptotic stability of border equilibrium, disease-free equilibrium, and axial equilibrium has received little attention. In this paper, we investigated the existence and local asymptotic stability of all feasible equilibriums from a mathematical perspective, to the optimum of our understanding. We looked at the possibility of a Hopf bifurcation in relation to the temporal delay caused by gestation and other characteristics. Finally, we would like to point out that our work instantly offers a precise hypothesis to be tested by empirical methods. We hope that these findings will spur experimental eco-epidemiologists to gather actual data in order to validate the theories generated during this investigation. Constructive critique and theoretical and experimental efforts are anticipated to elucidate the true mechanisms behind eco-epidemiology.

Author contributions

H. Mollah and S. Sarwardi: Conceptualization, methodology, formal analysis, writing-original draft preparation, writing-review and editing; S. Sarwardi and N. A. Almuallem: Software, validation; H. Mollah, S. Sarwardi and N. A. Almuallem: Investigation; S. Sarwardi: Supervision; N. A. Almuallem: Project administration. All authors have read and agreed to the published version of the manuscript.

Use of Generative-AI tools declaration

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

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Conflict of interest

The authors declare no conflict of interest.

References

1. A. J. Lotka, *Elements of physical biology*, Williams and Wilkins, Baltimore, 1925.
2. V. Volterra, Variations and fluctuations of a number of individuals in animl species living together, *Animal Ecology*, Mcgraw Hill, 1926.
3. N. Bairagi, P. K. Roy, J. Chattopadhyay, Role of infection on the stability of a predator-prey system with several response functions–a comparative study, *J. Theor. Biol.*, 2007. https://doi.org/10.1016/j.jtbi.2007.05.005
4. S. S. Maity, P. K. Tiwari, Z. Shuai, S. Pal, Role of space in an eco-epidemic predator-prey system with the effect of fear and selective predation, *J. Biol. Syst.*, World Scientific Publishing Co, 2023.
5. D. Barman, S. Roy, P. K. Tiwari, S. Alam, Two-fold impacts of fear in a seasonally forced predator-prey system with Cosner functional response, *J. Biol. Syst.*, World Scientific Publishing Co, 2023. https://doi.org/10.1142/S0218339023500183
6. R. Kumbhakar, S. Pal, N. Pal, P. K. Tiwari, Bistability and tristability in a predator-prey model with strong Allee effect in prey, *J. Biol. Syst.*, PB - World Scientific Publishing Co, 2023. https://doi.org/10.1142/S0218339023500110
7. S. Sarwardi, M. Haque, S. Hossain, Analysis of Bogdanov–Takens bifurcations in a spatiotemporal harvested-predator and prey system with Beddington–DeAngelis-type response function, *Nonlinear Dynam.*, 2020. https://doi.org/10.1007/s11071-020-05549-y
8. E. Venturino, Epidemics in predator-prey models: Disease in the predators, *IMA J. Math. Appl. Med. Biol.*, 2002. https://doi.org/10.1093/imammb/19.3.185
9. Z. Guo, W. Li, L. Cheng, Z. Li, An eco-epidemiological model with epidemic and response function in the predator, *J. Lanzhou Univ. Nat. Sci.*, 2009.
10. S. Sarwardi, M. Haque, E. Venturino, Global stability and persistence in LG-Holling type II diseased predator ecosystems, *J. Biol. Phys.*, 2011. https://doi.org/10.1007/s10867-010-9201-9
11. A. A. Shaikh, H. Das, N. Ali, Study of LG-Holling type III predator-prey model with disease in predator, *J. Appl. Math. Comput.*, 2017. https://doi.org/10.1007/s12190-017-1142-z
12. E. Francomano, M. F. Hilker, M. Paliaga, E. Venturino, Diseased social predators, *Bull. Math. Biol.*, 2017. https://doi.org/10.1007/s11538-017-0325-y
13. M. Haque, A predator-prey model with disease in the predator species only, *Nonlinear Anal. Real.*, 2010. https://doi.org/10.1016/j.nonrwa.2009.06.012

14. W. Lihong, F. Zhang, C. Jin, Analysis of an eco-epidemiological model with disease in the prey and predator, *Int. J. Math. Res.*, 2017.

15. S. Kant, V. Kumar, Stability analysis of predator-prey system with migrating prey and disease infection in both species, *Appl. Math. Model.*, 2016. https://doi.org/10.1016/j.apm.2016.10.003

16. L. Han, Z. Ma, H. W. Hethcote, Four predator prey models with infectious diseases, *Math. Comput. Model.*, **34** (2001), 849–858. https://doi.org/10.1016/S0895-7177(01)00104-2

17. Y. Hsieh, C. Hsiao, Predator–prey model with disease infection in both populations, *Math. Med. Biol.*, **25** (2008), 247–266. https://doi.org/10.1093/imammb/dqn017

18. A. Hugo, S. Massawe, O. Makinde, An eco-epidemiological mathematical model with treatment and disease infection in both prey and predator population, *J. Ecol. Nat. Environ.*, **4** (2012), 266–279. https://doi.org/10.5897/JENE12.013

19. B. Mondal, A. Sarkar, N. Sk, Treatment of infected predators under the influence of fear-induced refuge, *Sci. Rep.*, **13** (2023), 16623. https://doi.org/10.1038/s41598-023-43021-0

20. M. G. Cojocaru, T. Migot, A. Jaber. Controlling infection in predator-prey systems with transmission dynamics, *Infect. Dis. Model.*, **5** (2020), 1–11. https://doi.org/10.1016/j.idm.2019.12.002

21. J. Tripathi, S. Tyagi, S. Abbas, Global analysis of a delayed density dependent predator-prey model with Crowley-Martin functional response, *Commun. Nonlinear Sci.*, 2015.

22. L. Wang, R. Xu, G. Feng, Modelling and analysis of an eco-epidemiological model with time delay and stage structure, *J. Appl. Math. Comput.*, 2015. https://doi.org/10.1007/s12190-014-0865-3

23. A. M. Elaiw, N. A. Almuallem, Global properties of delayed-HIV dynamics models with differential drug efficacy in cocirculating target cells, *Appl. Math. Comput.*, 2015.

24. A. M. Elaiw, N. A. Almuallem, Global dynamics of delay-distributed HIV infection models with differential drug efficacy in cocirculating target cells, *Mat. Meth. Appl. Sci.*, 2015.

25. Q. Liu, M. Sun, T. Li, Analysis of an SIRS epidemic model with time delay on heterogeneous network, *Adv. Differ. Eqn.*, 2017. https://doi.org/10.1186/s13662-017-1367-z

26. P. Sen, S. Samanta, M. Y. Khan, S. Mandal, P. K. Tiwari, A seasonally forced eco-epidemic model with disease in predator and incubation delay, *J. Biol. Syst.*, World Scientific Publishing Co, 2023.

27. R. Xu, S. Zhang, Modelling and analysis of a delayed predator–prey model with disease in the predator, *Appl. Math. Comput.*, 2013.

28. X. Zhou, J. Cui, X. Shi, X. Song, A modified Leslie- Gower predator-prey model with prey infection, *Appl. Math. Comput.*, 2010.

29. G. P. Hu, X. L. Li, Stability and hopf bifurcation for a delayed predator-prey model with disease in the prey, *Chaos Soliton. Fract.*, 2012.

30. Z. Zhang, H. Yang, Hopf bifurcation control in a delayed predator-prey system with prey infection and modified Leslie-Gower scheme, *Abst. Appl. Anal.*, 2013. https://doi.org/10.1155/2013/704320

31. J. Zhang, W. Li, X. P. Yan, Hopf bifurcation and stability of periodic solutions in a delayed eco-epidemiological system, *Appl. Math. Comput.*, 2008. https://doi.org/10.1016/j.amc.2007.09.045

32. N. Bairagi, D. Jana, On the stability and Hopf bifurcation of a delay-induced predator-prey system with habitat complexity, *Appl. Math. Model.*, 2011. https://doi.org/10.1016/j.apm.2011.01.025

33. L. Deng, X. Wang, M. Peng, Hopf bifurcation analysis for a ratio-dependent predator-prey system with two delays and stage structure for the predator, *Appl. Math. Comp.*, 2014. https://doi.org/10.1016/j.amc.2014.01.025

34. X. Zhou, J. Cui, Stability and Hopf bifurcation analysis of an eco-epidemiological model with delay, *J. Franklin Inst.*, 2010.

35. Y. Kuang, Delay differential equation with application in population dynamics, 1993.

36. G. Birkhoff, G. C. Rota, Ordinary differential equations, *Ginn Boston.*, 1982.

37. J. K. Hale, S. M. V. Lunel, *Introduction to functional differential equations*, Springer-Verlag, New York, 1993. https://doi.org/10.1007/978-1-4612-4342-7

38. B. D. Hassard, N. D. Kazarinoff, Y. H. Wan, *Theory and applications of Hopf bifurcation*, Cambridge University Press Cambridge, New York, 1981.

39. Y. Song, J. Wei, Bifurcation analysis for Chen's system with delayed feedback and its application to control of chaos, *Chaos Soliton. Fract.*, 2004.

40. A. Wolf, J. B. Swift, H. L. Swinney, J. A. Vastano, Determining Lyapunov exponents from a time series, *Physica D*, 1985. https://doi.org/10.1016/0167-2789(85)90011-9

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