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Research Article

Eco-Epidemic Dynamics With Infected Communicable Infection From Prey to Predator: Effect of Recovery Delay for Predator

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Abstract. In this paper, the system including communicable infection from prey to predator, represented by the growing rate of the prey as a healing of a predator's, has been utilized to develop and analyze a four-dimensional, non-linear eco-epidemic model. In the context of the analysis, the exploration of all potential equilibrium points, along with an examination of their local stability conditions, has been conducted both with and without considering time delays. Additionally, the model's positivity and boundedness have been confirmed. The positive and negative impact of the proposed model on the prey-predator population has been investigated aided by sensitivity analysis where the presence and validence of Bifurcation were elucidated by the numerical studies. The parameters were identified which explained the influence of disease and recovery delay over the model population. A right base for the perception of the behavioural effects of the prey-predator on eco-epidemiology has been prepared through the theoretical result. Based on the analytical result, numerical simulations were done so that the authenticity of the author's numerical analytical approach using parameter values could be verified.

Keywords. Eco-epidemic model, Fundamental reproduction number, Local stability, Time delay, Sensitivity analysis

Mathematics Subject Classification (2020). 34D20, 92B05, 92D25, 34C23, 37Gxx

1. Introduction

Ecology and epidemiology, as separate fields of study, have gained considerable prominence. In contemporary research, there is a growing focus on the interdisciplinary domain known as eco-epidemiology. This discipline delves into the study of how hosts interact with a range of pathogens, viruses, and illnesses, impacting both human and wildlife communities on a population and ecosystem scale. A practical application of this research lies in enhancing our comprehension of dynamic systems through the utilization of mathematical models. In the existing literature, two distinct areas stand out: theoretical ecology and epidemiology, as highlighted by Arino *et al*. [2] in their work on infection dynamics (also see, Sarangi and Raw [2, 15]). In many instances, ecological systems are intricately linked to their historical context, and models can be tailored to reflect real-world scenarios by incorporating a time lag or delay. The introduction of a time-delay parameter in a mathematical model can have a profound impact on stability, often introducing increased complexity to the system. A substantial body of literature explores ecological models that incorporate time delays in their governing equations. Researchers have extensively examined time-delayed eco-epidemiological models, as evidenced by studies such as those conducted by Qi and Zhao [13] and Ruan [14] on dynamical on system dynamics on stability considerations. In their research focused on modeling and analyzing an eco-epidemic system of the predator-prey type, incorporating time delays. While numerous models developed by Qi and Zhao [13] and Tripathi and Singh [17] have explored diseases in prey populations, there is a noticeable gap in understanding models involving diseases in predator populations. To address this gap, the researchers constructed a delayed eco-epidemiological model wherein the predator population is influenced by an infectious disease. Infected predators are assumed to be weakened, rendering them unable to capture prey, and only susceptible predators partake in hunting using the functional response of Holling type-I. Hethcote *et al*. [9], Kumar and Sinha [10], Sharma and Samanta [16], and Venturino [19] have dedicated their consideration towards the diseases spread studies within prey-predator populations. The potency of the Allee effect is inversely related to the level of chaotic behavior observed in ecological models (Sarangi and Raw [15]). Functional response describes how predators consume prey over time. Different functional responses, such as the Holling type-I, Holling type-II, and ratio-dependent functional responses, are utilized in mathematical analyses of eco-epidemiological systems. Among the ratio-dependent functional response is oft regarded as very emphatic in modeling prey-predator interactions (Arditi and Ginzburg [1]). Other studies have considered scenarios in which healthy prey exhibit greater activity than infected ones, making them less susceptible to predation by predators. Certain mathematicians like Belvisi and Venturino [3], and Haque and Venturino [7] have developed models incorporating diseases in predator populations, positing that infected predators are unable to effectively hunt healthy prey. These investigations seek to elucidate the efficacy of infection delay at the dynamics of prey-predator interactions within populations. In the realm of predator-prey interactions, diseases can manifest by spreading solely within the prey or predator population or by affecting both simultaneously. This scenario exemplifies disease spreading among prey populations. Conversely, diseases in predators, such as fox rabies, can be transmitted among foxes (Vulpis) and to their prey, rabbits, through biting in regions like Europe and North America. Further instances of such dynamics are documented by Tripathi *et al*. [18]. From a mathematical epidemiology standpoint, particular attention

is warranted in understanding the dynamics of infected predators to discern whether the presence of prey enables the survival of a portion of the predator population. On the contrary, in the natural world, species coexist within ecosystems, and an increasing body of research indicates that population growth is significantly influenced by the competition for resources and space, along with the impact of infectious diseases. Therefore, the integration of these factors is crucial for a comprehensive understanding of population dynamics. To provide a clearer overview, here is a revised version of the structure: Section 2: Model formulation of the eco-epidemiological model with detailed explanations. Section 3: Positivity and boundedness analysis of the model's solution. Section 4: Exploration of eventual equilibria for the deterministic system. Section 5: Investigation of stability behavior, incorporating the fundamental reproduction number and various bifurcations. Section 6: Formulation of the model and studies on the existence and uniqueness of global positive solutions, along with conditions for species extinction. Section 7: Formulation for the delay model studies. Section 8: Sensitivity analysis to determine the impact of system parameters on the reproduction number. Section 9: Numerical simulations supporting analytical findings and including biological interpretation results from both deterministic systems. Section 10: Discussion and summary of research outcomes.

2. Model formulation

This section suggests a non-linear four-dimensional eco-epidemic model, we consider a prey-predator model which is:

(i) There are four types of population, namely, the susceptible prey ($S(t)$), infected prey ($I(t)$), the susceptible predator ($X(t)$), and the infected predator ($Y(t)$).

(ii) $N(t) = I(t) + S(t)$ denoted the number of prey at time t. The prey population experiences logistic growth, influenced by the intrinsic growth rate denoted as r. Consequently, the differential equation with logistic growth $\frac{dN}{dt} = rN\left(1 - \frac{N}{K}\right) = rS\left(1 - \frac{S}{K}\right)$ accurately models the dynamics of the total prey population (where K = the carrying capacity).

(iii) The disease system does not immediately manifest the population of infected predators. Instead, there exists a time delay, referred to as the incubation period (τ), between the occurrence of events within the system and the onset of infection. This delay is recognized as an integral component of the dynamics.

(iv) Predators afflicted with the disease do not undergo recovery; instead, they succumb to the infection at a rate denoted as d_1.

We employ the Holling type-II functional response to describe both predation and disease transmission dynamics. The model, incorporating the assumptions outlined above, is represented in the following manner:

$$\left.\begin{aligned}
\frac{dS}{dt} &= rS\left(1 - \frac{S}{k}\right) - \frac{\beta IS}{1 + bS} - \frac{\alpha SX}{1 + b_1 S}, \\
\frac{dI}{dt} &= \frac{\beta IS}{1 + bS} - \gamma XI - dI, \\
\frac{dX}{dt} &= \frac{\alpha_1 SX}{1 + b_1 S} - d_1 X + \gamma_2 Y(t - \tau), \\
\frac{dY}{dt} &= \gamma_1 IX - \gamma_2 Y(t - \tau) - d_2 Y
\end{aligned}\right\} \tag{2.1}$$

with initial condition $S(0) > 0,\ I(0) > 0,\ X(0) > 0,\ Y(0) > 0$. To know the expression of the suggested model (2.1). The biological importance of each parameter and its values are distributed in Table 1. Here, it may be applicable that the parameters used in Table 1 are for the infectious diseases that are spread as a result of natural disasters. The interpretation of the four key players of the model is given like this.

Table 1. Model parameter values, biological interpretation

Parameter	Description
K	Carrying capacity
r	Per capita intrinsic growth rate per day
β	Transmission rate between susceptible and infected individuals per day
γ, γ_1	Daily predation rates on infected prey by susceptible and infected predators
d_1, d_2	Total mortality rate of both susceptible and infected individual
α, α_1	Conversion rate
d	Infected mortality rate per day
b, b_1	Constant half-saturation per unit of area
τ	Incubation period
γ_2	Overall mortality rate of susceptible and infected individuals

3. Theoretical Studies of Model

In this section, we discussed the positivity and bounded analysis of the proposed model, discussed in Subsection 3.1, and 3.2:

3.1 Positive Invariance

Theorem 3.1. *For all $t \geq 0$, the solutions of the considered system* (2.1) *with the initial condition are positive.*

Proof. Assume that the solutions of the suggested model (i)-(iv) with non-negative initial populations are $(S(t), I(t), X(t), Y(t))$.

Using properties (i) and (ii), prey population is non-negative,

$$\left.\begin{aligned} &\frac{dS}{dt} = rS\left(1 - \frac{S}{K} - \frac{\beta IS}{1+bS} - \frac{\alpha SX}{1+b_1 S}\right), \\ &S(t) \geq S(0)\exp\int_0^t \left(r\left(1 - \frac{S}{K}\right) - \frac{\beta I}{1+bS} - \frac{\alpha X}{1+b_1 S}\right) dt. \end{aligned}\right\} \tag{3.1}$$

Using property (ii),

$$\left.\begin{aligned} &\frac{dI}{dt} = \frac{\beta SI}{1+bS} - \gamma XI - dI, \\ &I(t) \geq I(0)\exp\int_0^t \left(\frac{\beta S}{1+bS} - \gamma X - d\right) dt. \end{aligned}\right\} \tag{3.2}$$

Using property (iii), can be written as

$$\left.\begin{aligned} &\frac{dX}{dt} = \frac{\alpha_1 SX}{1+b_1 S} - d_1 X + \gamma_2 Y(t-\tau), \\ &X(t) \geq X(0)\exp\{-d_1 t\}. \end{aligned}\right\} \tag{3.3}$$

Using property (iv), can be written as

$$\left.\begin{aligned} &\frac{dY}{dt} = \gamma_1 IX - \gamma_2 Y(t-\tau) - d_2 Y, \\ &Y(t) \geq Y(0)\{e^{-(\gamma_2+d_2)t}\}. \end{aligned}\right\} \tag{3.4}$$

□

3.2 Boundedness of the System

Theorem 3.2. *All solutions of system* (2.1) *starting in* R_+^4 *confined to the region*

$$R_+^4 = \left\{(S,I,X,Y) \mid 0 \leq S(t)+I(t)+X(t)+Y(t) \leq \frac{rk}{\delta}\right\}.$$

Remain bounded regardless of the initial conditions, where $\delta = \min\{r,d,d_1,d_2\}$.

Proof. Let's define $N(t) = S(t)+I(t)+X(t)+Y(t)$. Now, if we $N(t)$ for t, we obtain the rate of change of the total population over time,

$$\frac{dN(t)}{dt} \leq rS\left(1-\frac{S}{K}\right) - \delta(S+I+X+Y). \tag{3.5}$$

For $\delta = \min\{r,d,d_1,d_2\}$, we obtained that

$$\frac{dN}{dt} + \delta N \leq rK,$$

as $t \to \infty$, we have

$$0 \leq N(t) \leq N(0)e^{-\delta t} + \frac{rK}{\delta}. \tag{3.6}$$

After solving the aforementioned expression we observe that, $0 < N(t) < N(0)e^{-\delta t} + \frac{rK}{\delta}$. If we let $t \to \infty$, then we obtain, $0 < N(t) < \frac{rK}{\delta}$. Consequently, $N(t)$ remains bounded. Thus, the solution of the system with the initial condition is uniformly bounded in R_+^4. □

Section 4 provides a discussion on the classification and circumstances for the possibility of steady states for the system represented by equation (2.1).

4. Equilibrium Analysis

The system (2.1) has a trivial equilibrium point equilibrium point $E_0(0,0,0,0)$ and equilibrium point $E_1(k,0,0,0)$ always exist. The predator-free equilibrium point $E_2(\widehat{S},\widehat{I},0,0)$ exists.

- The predator-free equilibrium point $E_2(\widehat{S},\widehat{I},0,0)$ exists,
  where $\widehat{S} = \frac{d}{\beta - db}$, $\widehat{I} = \frac{r(\beta K - bdK - d)}{(\beta - bd)^2}K$, only when

$$\beta > \left\{db + \frac{d}{K}\right\}. \tag{4.1}$$

- The equilibrium without any disease $E_3(S_0,0,X_0,0)$ exists,
  where $S_0=\frac{d_1}{\alpha_1-d_1b_1}$, $X_0=\frac{\alpha_1 r(K\alpha_1-Kb_1d_1-d_1)}{\alpha K(\alpha_1-b_1d_1)^2}$, only when
  $$\alpha_1>\left\{d_1b_1+\frac{b_1}{K}\right\}. \tag{4.2}$$
- The interior equilibrium point $E_4(S^\star,I^\star,X^\star,Y^\star)$,
  where $Y^\star=\left(\frac{\gamma_1 I^\star X^\star}{\gamma_2+d_2}\right)$, $X^\star=\left(\frac{\beta S^\star-d-dbS^\star}{\gamma(1+bS^\star)}\right)$ and $I^\star=\frac{(\gamma_2+d_2)(d_1+d_1b_1S^\star-\alpha_1S^\star)}{\gamma_1\gamma_2(1+b_1S^\star)}$,
  $$-AS^{\star 3}+BS^{\star 2}+CS^\star+D=0,$$
  where $A=-bb_1\gamma\gamma_1\gamma_2 r$,
  $B=\gamma\gamma_1\gamma_2(bb_1Kr-br-b_1r)$,
  $C=K\beta\gamma(d_2\alpha_1-b_1d_1d_2-b_1d_1\gamma_2+\alpha_1\gamma_2)+K\gamma_1\gamma_2(bd\alpha-\beta\alpha+br\gamma+b_1r\gamma)-r\gamma\gamma_1\gamma_2$,
  $D=K\gamma_2\gamma_1(r\gamma+\alpha_1 d)-d_1K\gamma(\gamma_2-d_2)$.

Using Descartes's rule of signs one positive root and two negative roots.

We will calculate the fundamental reproduction number in Section 5.

5. Fundamental Reproduction Number

The fundamental reproduction number R_0, alternatively known as the basic reproduction ratio or rate, serves as an epidemiological measure employed to characterize the degree of contagion or transmissibility exhibited by infectious agents. R_0 - The fundamental reproduction number can be calculated using the upcoming generation matrix formula. Regarding this, the following theorem (2.1),

$$R_0=\frac{\beta d_1\alpha K(\alpha_1-b_1d_1)^2}{(\alpha_1+bd_1-b_1d_1)[(\alpha_1-b_1d_1)^2\alpha Kd+K\alpha_1 r(K\alpha_1-Kb_1d-d_1)]}.$$

In Section 6, the discussion primarily revolves around analyzing the stability and bifurcation patterns of the equilibria in the system described by eq. (2.1). This investigation particularly emphasizes situations where there is no time delay involved.

6. Examination of the Investigated Model Without the Time Delay Reveals

If there is no delay, the system described by eq. (2.1) transforms to:

$$\left.\begin{aligned}
\frac{dI}{dt}&=\frac{\beta IS}{1+bS}-\gamma XI-dI,\\
\frac{dS}{dt}&=rS\left(1-\frac{S}{k}\right)-\frac{\beta IS}{1+bS}-\frac{\alpha SX}{1+b_1S},\\
\frac{dX}{dt}&=\frac{\alpha_1 SX}{1+b_1S}-d_1X+\gamma_2Y,\\
\frac{dY}{dt}&=\gamma_1IX-\gamma_2Y-d_2Y.
\end{aligned}\right\} \tag{6.1}$$

The system is considered under the initial conditions: $S(0)\geq 0$, $I(0)\geq 0$, $X(0)\geq 0$, $Y(0)\geq 0$.

6.1 Examining Local Stability

The local stability of the proposed model has been estimated under Theorems 6.1–6.4 as follows:

Theorem 6.1. *The system* (2.1) *is trivial equilibrium* $E_0(0,0,0)$ *is inherently unstable.*

Proof. At E_0, the Jacobian matrix of system (2.1). The eigenvalues of the variational matrix are $\lambda_1 = -d$, $\lambda_2 = -d_1$, $\lambda_3 = -(\gamma_2 + d_2)$, $\lambda_4 = r$. As there is a minimum of one positive eigenvalue, that $E_0(0,0,0,0)$ is an unstable trivial equilibrium. □

Theorem 6.2. *If* $K < \min\{\frac{d}{\beta - d}, \frac{d_1}{\alpha - d_1 b_1}\}$, *then the axial equilibrium* $E_1(K,0,0,0)$ *of system* (2.1) *is locally asymptotically stable; otherwise, it is unstable.*

Proof. The Jacobian matrix corresponding to the axial equilibrium $E_1(K,0,0,0)$ is as follows: The eigenvalue are $\lambda_1 = -r$, $\lambda_2 = \frac{\beta K}{1+bK} - d$, $\lambda_3 = \frac{\alpha K}{1+b_1 K} - d_1$, $\lambda_4 = -(\gamma_2 + d_2)$. The eigenvalue λ_2 and λ_3 are negative if $K(\beta - b) < d$ and $\alpha K < d_1(1 + b_1 K)$ holds. Hence, axial equilibrium E_1 is locally asymptotically stable if $K < \min\{\frac{d}{\beta - d}, \frac{d_1}{\alpha - d_1 b_1}\}$, else unstable. □

Theorem 6.3. *For system* (6.1)*, it is found that, given certain assumptions, the free for-disease equilibrium* $E_3(S_0,0,X_0,0)$ *is locally asymptotically stable if:*

$$(\alpha_1 - b_1 d_1)^2(\beta d_1 \alpha K - \alpha K d(\alpha_1 - d_1 b_1 + b d_1) - K\alpha r(K\alpha_1 - K b_1 d_1 + b d_1)(\alpha_1 - d_1 b_1 + b d_1) < 0, \tag{6.2}$$

else, it becomes unstable.

Proof. The $V(E_3)$ of model (2.1) at $E_3(S_0,0,X_0,0)$, the Jacobian matrix is as follows:

$$V(E_3) = \begin{bmatrix} b_{11} & b_{12} & b_{13} & 0 \\ 0 & 0 & 0 & 0 \\ b_{31} & 0 & 0 & b_{34} \\ 0 & b_{42} & 0 & b_{44} \end{bmatrix},$$

where $b_{11} = r(1 - \frac{2S_0}{K}) - \frac{\alpha X_0}{(1+b_1 S_0)^2}$, $b_{12} = \frac{-\beta S_0}{(1+bS_0)}$, $b_{13} = \frac{-\alpha S_0}{1+b_1 S_0}$, $b_{31} = \frac{\alpha X_0}{(1+b_1 S_0)^2}$, $b_{34} = \gamma_2$, $b_{42} = \gamma_1 X_0$, $b_{44} = -(\gamma_2 + d_2)$.

The population is free of the disease if the fundamental reproduction number $R_0 = \frac{\beta d_1}{d(\alpha + bd_1 - b_1 d_1)} < 1$, which is feasible when, in addition to conditions (4.1) and (4.2) is satisfied as well. Thus, the free of disease equilibrium $E_4(S_0,0,X_0,0)$, if, (4.1) and (6.2) holds then, it is locally asymptotically stable. □

Theorem 6.4. *The coexistence equilibrium* $E_0(S^*,I^*,X^*,Y^*)$ *of system* (6.1) *obtains locally asymptotically stability.*

Proof. The coexistence equilibrium $E_4(S^\star,I^\star,X^\star,Y^\star)$, the jacobian matrix of (6.1):

$$V(E_4) = \begin{bmatrix} c_{11} & c_{12} & c_{13} & 0 \\ c_{21} & 0 & c_{23} & 0 \\ c_{31} & 0 & c_{33} & c_{34} \\ 0 & c_{42} & c_{43} & c_{44} \end{bmatrix},$$

where $c_{11} = -\frac{r}{K} + \frac{\beta I d}{(1+bS^\star)^2} + \frac{\alpha b_1 X^\star}{(1+b_1 S^\star)^2}$, $c_{12} = \frac{-\beta}{(1+bS^\star)}$, $c_{13} = \frac{-\alpha}{1+bS^\star}$, $c_{21} = \frac{\beta}{(1+bS^\star)^2}$, $c_{23} = -\gamma$,

$c_{31} = \frac{\alpha_1}{(1+b_1S^\star)^2}$, $c_{33} = \frac{\gamma_2 Y}{(X^\star)^2}$, $c_{34} = \frac{-\gamma}{X^\star}$, $c_{42} = \frac{\alpha_1}{(1+b_1S^\star)^2}$, $c_{43} = \gamma_1 I^\star$, $c_{44} = -(\gamma_2 + d_2)$.

The characteristic equation of the Jacobian matrix $J(E_4)$ is as follows,

$$\lambda^4 + C_1\lambda^3 + C_2\lambda^2 + C_3\lambda + C_4 = 0, \tag{6.3}$$

where $C_1 = -(c_{11} + c_{33} + c_{44})$, $C_2 = c_{11}(c_{33} + c_{44}) + c_{33}c_{44} + c_{13}c_{31} - c_{12}c_{21} - c_{34}c_{43}$,
$C_3 = c_{11}(c_{33}c_{44} - c_{34}c_{43}) + c_{12}c_{21}(c_{33} + c_{44}) + c_{23}c_{34}c_{42} + c_{31}(c_{13}c_{44} + c_{12}c_{23})$,
$C_4 = c_{23}(c_{11}c_{34}c_{42} - C_{12}c_{31}c_{44}) + c_{12}c_{21}(c_{34}c_{43} - C_{33}c_{44})$.

Then evaluation of the determinant of the characteristic equation $|A - \lambda I| = 0$ gives a fourth-order algebraic equation of the form $\lambda^4 + C_1\lambda^3 + C_2\lambda^2 + C_3\lambda + C_4 = 0$. Using the Hurwitz criterion the coexistence will be stable if the following conditions are satisfied. According to the Routh-Hurwitz criterion, the characteristic equation (6.3) will possess eigenvalues with negative real parts under the conditions: $C_1 > 0$, $C_3 > 0$, $C_4 > 0$, $C_1C_2 - C_3 > 0$, and $C_1C_2C_3 > C_3^2 + C_1^2C_4$. If these conditions are not met, the system is considered unstable. □

Section 7 delves into the examination of local stability and Transcritical bifurcation phenomena within the context of delay model (2.1), focusing particularly on the coexistence state. Furthermore, the discourse will encompass discussions regarding the disease-free state.

7. Investigation of the Delay Model

The model (2.1) now includes a non-linear incidence rate, governing how susceptible prey transform into infected individuals. In this context, the predator exclusively consumes the infected prey-predator based on the population's functional response. Introducing a time lag is pivotal for the conversion of susceptible predators into infected ones, thereby transforming the present model into a delayed eco-epidemic model. The local stability and the occurrence of bifurcation phenomena have been discussed in Section 7.1.

7.1 Analyzing the Coexistence Equilibrium's Local Stability and Bifurcation Phenomena

At the coexistence equilibrium $E_4(S^*, I^*, X^*, Y^*)$, for system (6.1), the Jacobian matrix is:

$$V(E_4) = \begin{bmatrix} c_{11} & c_{12} & c_{13} & 0 \\ c_{21} & 0 & c_{23} & 0 \\ c_{31} & 0 & c_{33} & c_{34} + \gamma e^{-\lambda\tau} \\ 0 & c_{42} & c_{43} & c_{44} - \gamma e^{-\lambda\tau} \end{bmatrix},$$

where $c_{11}, c_{12}, c_{13}, c_{21}, c_{23}, c_{31}, c_{33}, c_{34}c_{42}, c_{43}, c_{44}$ are provided in Theorem 6.4. Utilizing matrix row operations, specifically, $R_3 -> R_3 + R_4$, $R_1 -> c_{23}R_2 - c_{13}R_2$ and $R_4 -> c_{21}R_4 - R_3$, $R_3 -> c_{31}R_2 - c_{21}R_3$ we modify the final matrix into the following form,

$$V(E_4) = \begin{bmatrix} d_{11} & d_{12} & 0 & 0 \\ d_{21} & 0 & d_{23} & 0 \\ 0 & d_{32} & d_{33} & d_{34} \\ 0 & 0 & d_{43} & d_{44} \end{bmatrix},$$

where $d_{11} = c_{23}(c_{11} - c_{23})$, $d_{12} = c_{12}c_{23}$, $d_{21} = c_{21}$, $d_{23} = c_{23}$, $d_{32} = c_{42}c_{21}$, $d_{33} = c_{21}(c_{33} + c_{42}) - c_{23}c_{31}$, $d_{34} = c_{21}(c_{34} + c_{44})$, $d_{43} = c_{21}(c_{43} - c_{42} - c_{33}) + c_{23}c_{31}$, $d_{44} = -c_{21}(c_{34} + \gamma e^{-\delta\tau})$.

We express characteristic equation of model (2.1) at E_4 as following transcendental equation,

$$\lambda^4 + F_1\lambda^3 + F_2\lambda^2 + F_3\lambda + F_4 + (F_5\lambda^3 + F_6\lambda^2 + F_7\lambda + F_8) = 0, \tag{7.1}$$

where $F_1 = -(d_{11} + d_{33})$, $F_2 = d_{11}d_{33} - d_{23}d_{32} - d_{12}d_{21} - d_{34}d_{43}$, $F_3 = d_{11}d_{23}d_{32} + d_{11}c_{34}d_{43} + d_{12}d_{21}d_{33}$, $F_4 = -d_{12}d_{21}d_{34}d_{43}$, $F_5 = -d_{44}$, $F_6 = d_{11}d_{44} - d_{33}d_{44}$, $F_7 = d_{12}d_{21}d_{44} + d_{23}d_{32}d_{44} - d_{11}d_{33}d_{44}$, $F_8 = -d_{11}d_{23}d_{32}d_{44} - d_{12}d_{21}d_{33}d_{44}$.

The local stability of the equilibrium E_4 has also been studied, utilizing lemmas of Kumar *et al*. [11], and Ruan [14] for the transcendental polynomial equation. The transcendental polynomial equation of the first degree is as follows:

$$F(\lambda) = (v + \lambda + qe^{-\lambda\tau}) = 0\,. \tag{7.2}$$

According to [11, 14], if:

(*A*1) $q + v > 0$,

(*A*2) $v^2 - q^2 < 0$,

(*A*3) $v^2 - q^2 > 0$.

Consider the lemma that follows [11, 14].

Lemma 7.1. *Concerning equation* (7.2) *states the following:*

(1) *Assuming that* (*A*1)-(*A*3) *are met, every root of* (7.2) *will have negative real portions for any* $\tau \geq 0$.

(2) *Here, equation* (7.2) *has two totally imaginary roots,* $\pm\omega$, *if conditions* (*A*1)-(*A*3) *are satisfied and* $\tau = \tau_j^+$. *Every root of* (7.2), *with the exception of* $\pm\omega$, *exhibits negative real portions at* $\tau = \tau_j^+$.

Proof. Case I: Let $\tau = 0$, we have

$$\lambda^4 + \lambda^3(F_1 + F_5) + \lambda^2(F_2 + F_6) + \lambda(F_3 + F_7) + F_4 + F_8) = 0 \tag{7.3}$$

if $\quad F_1 + F_5 > 0, \quad F_2 + F_6 > 0, \quad F_3 + F_4 > 0,$

$$(F_1 + F_5)(F_2 + F_6) - (F_3 + F_7) > 0.$$

All values are negative real roots the steady state is asymptotically locally stable.

Case II: Let $\tau > 0$, So the value is negative or real root hence, it becomes locally asymptotically stable.

Equilibrium E_5 become unstable, put $\lambda = i\omega$ (eq. (7.3)). Comparing real and imaginary parts of

$$\left.\begin{aligned}
&\omega^4 - F_1\omega^3 i - F_2\omega^2 + F_3 i\omega + F_4 + (-F_5 i\omega^3 - F_6\omega^2 + F_7 i\omega + F_8)e^{i\omega\tau} = 0,\\
&(-F_6\omega^2 + F_8)\cos\omega\tau + (-F_5\omega^3 - F_7\omega)\sin\omega\tau = -(\omega^4 + F_2\omega^2 - F_4),\\
&(-F_5\omega^3 + F_7\omega)\cos\omega\tau + (F_6\omega^2 - F_8)\sin\omega\tau = -(F_1\omega^3 - F_2\omega),\\
&(-F_6\omega^2 + F_8)\cos\omega\tau + (-F_5\omega^3 - F_7\omega)\sin\omega\tau = -(\omega^4 + F_2\omega^2 - F_4),\\
&(-F_5\omega^3 + F_7\omega)\cos\omega\tau + (F_6\omega^2 - F_8)\sin\omega\tau = -(F_1\omega^3 - F_2\omega).
\end{aligned}\right\} \tag{7.4}$$

Upon solving the system of eq. (7.4), we obtain the following results,

$$\cos[\tau\omega] = \frac{-(\omega^4 + F_2\omega^2 - F_4)(-F_6\omega^2 + F_8) + (F_1\omega^3 - F_2\omega)(-F_5\omega^3 + F_7\omega)}{(F_6\omega^2 - F_8)(-F_6\omega^2 + F_8) - (-F_5\omega^3 + F_7\omega)(F_7\omega - F_5\omega^3)}.$$

The above implies that,

$$\tau_k{}^{+} = \frac{1}{\omega}\arccos\left[\frac{V_1\omega^6 + V_2\omega^4 + V_3\omega^2 - V_4}{-(F_6\omega^2 - F_8)^2 + (F_5\omega^3 - F_7\omega)^2}\right] + \frac{2\pi K}{\omega},$$

where $V_1 = -(F_6 + F_1F_5)$, $V_2 = (F_1F_7 + F_3F_5 + F_8)$, $V_3 = (F_2F_6 - F_2F_8 - F_6F_4 - F_3F_7)$, $V_4 = F_4F_8$.

It is evident that τ^* is a function of ω. By squaring and adding the two equations, we obtain the algebraic eq. (7.4), in ω,

$$\begin{aligned}&\omega^8 + (F_1^2 - 2F_2 - F_5^2)\omega^6 + (F_2^2 - 2F_4 - 2F_5F_7 - 2F_1F_3 - F_6^2)\omega^4\\ &\quad + (F_3^2 - F_7^2 - 2F_2F_4 + 2F_5F_8)\omega^2 - F_8^2 + F_4^2 = 0.\end{aligned} \tag{7.5}$$

Now, (7.5) possesses at least one positive root if $F_4^2 - F_8^2 < 0$. Suppose ω is a root of equation (7.5); thus, $-{}^{+}i\omega$ is a root of the characteristic (7.2). Upon differentiating (7.2), we obtain

$$\left(\frac{d\lambda}{d\tau}\right)^{-1} = \frac{(4\lambda^3 + 3F_1\lambda^2 + 2F_2\lambda + F_3)e^{\lambda\tau} + (3F_5\lambda^2 + 2F_6\lambda + F_7)}{\lambda(F_5\lambda^3 + F_6\lambda^2 + F_7\lambda + F_8)} - \frac{\tau}{\lambda}. \tag{7.6}$$

Substitute $\lambda = i\omega$ in (7.6), we have

$$\left(\frac{d\lambda}{d\tau}\right)^{-1} = \frac{LU - VM}{(L^2 + M^2)},$$

where $L = F_5\omega^2 - F_5\omega$, $M = (F_8 - F_6\omega^2)$, $U = (F_4\omega^3 - 2F_2\omega)\sin[\omega\tau] + (-3F_1\omega^2 + F_3)\cos[\omega\tau] + (-3F_5\omega^2 + F_7)$, $V = -(4\omega^3 - 2F_2\omega)\cos[\omega\tau] + (-3F_1\omega^2 + F_3)\sin[\omega\tau] + 2F_6\omega$.

Moreover, the critical requirement for the occurrence of bifurcation at $\tau = \tau^+$, is $Re\left(\frac{d\lambda}{d\tau}\right)^{-1}_{\tau=\tau^+} \neq 0$. This condition is met only if $LU \neq VM$. Thus, bifurcation transpires at $\tau = \tau^+$, if $L(\omega)U(\omega) - M(\omega)V(\omega) \neq 0$. This concludes the proof. □

8. Analysis of Sensitivity

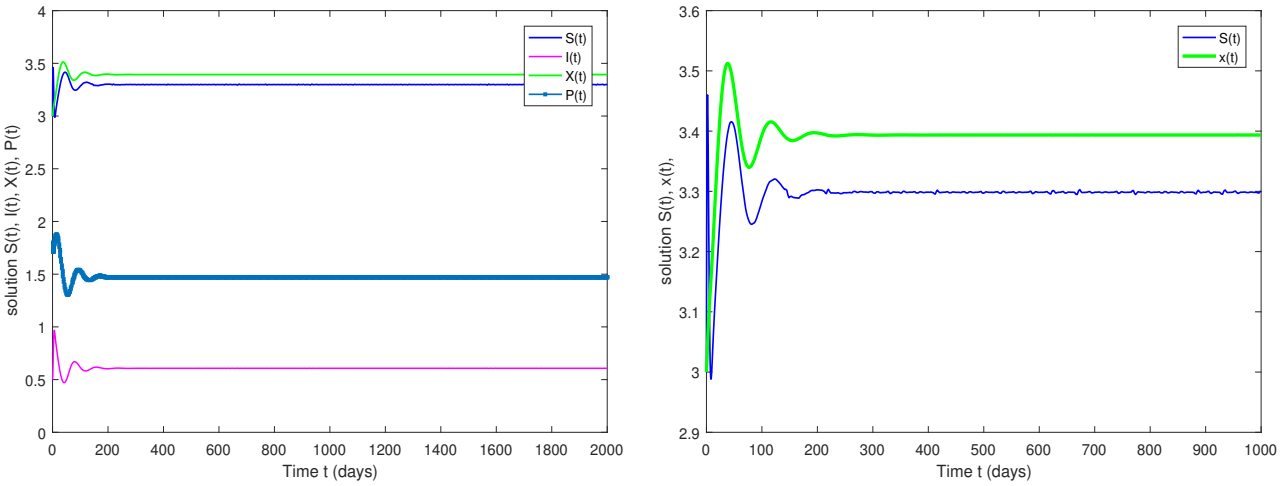
Considering the parameter values specified in [4,5], we set the following parameters value are: $\alpha = 2.5$, $\beta = 0.5$, $r = 4$, $d_1 = 0.07$, $d = 0.02$, $K = 100$, $\alpha_1 = 0.6$, $b = 50$, $b_1 = 20$. These values fall within the observed range, except where model solutions were not obtained. Sensitivity analysis reveals that parameters α, β, b_1, d, and d_1 positively influence R_0, while r, b, and α_1 have a negative impact on R_0. Additionally, the remaining parameters do not significantly affect R_0.

Table 2. Sensitivity analysis

Parameters	RV
α	1.00017
α_1	−1.97317
β	1
b	−1.2963
b_1	2.270347
d	0.000166549
d_1	1.97317
r	−1.00017
K	0

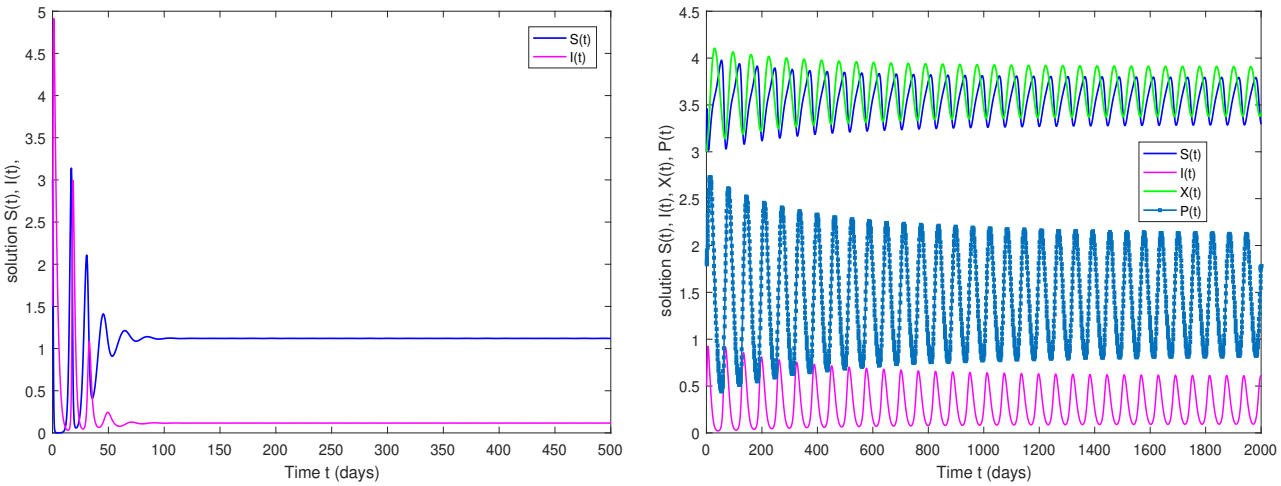
9. Numerical Experimentation

In this section, we present numerical examples to illustrate our analytical results. Due to the challenges associated with obtaining precise parameter values from real-world observations, we employ biologically plausible data for numerical computations. It is important to note that our focus is on qualitative rather than quantitative results. For numerical simulations, we primarily utilize MATLAB and MATHEMATICA software. The parameter values used in our simulations are as follows:



(a) (b)

Figure 1. When $\tau = 0$ for set of value of parameters: (a) is a interior equilibrium for behaviour is stable, and (b) is a disease-free population $\alpha = 0.1$, $d1 = 0.05$, $\gamma_2 = 0.1$, $b = 0.02$, $d = 0.03$, $b_1 = 0.03$, $\alpha_1 = 0.02$, $d_2 = 0.05$



(a) (b)

Figure 2. When $\tau = 0$, i.e., without delay: (a) predator-free equilibrium, and (b) is a time series For set of value of parameters $\alpha = 0.2$, $d_1 = 0.05$, $\gamma_2 = 0.05$, $b = 0.02$, $d = 0.05$, $b_1 = 0.05$, $\alpha_1 = 0.01$, $d_2 = 0.02$, $\beta = 0.4$, $r = 1.8$, $k = 6$, $\gamma_1 = 0.095$, $\gamma = 0.35$

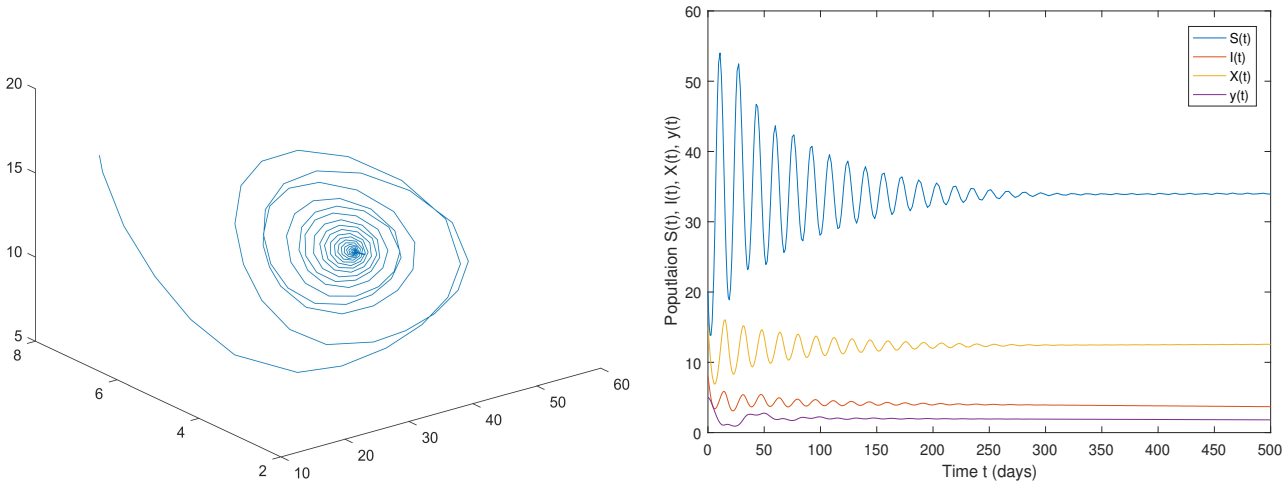


Figure 3. When $\tau = 12$, i.e., with delay $\alpha = 0.086$, $\alpha_1 = 0.028$, $\beta = 0.065$, $k = 100$, $d = 0.6$, $d_1 = 0.5$, $d_2 = 0.04$, $b = 0.066$, $b_1 = 0.028$, $r = 0.95$, $\gamma = 0.0065$, $\gamma_1 = 0.0049$, $\gamma_2 = 0.082$, $\tau = 12$

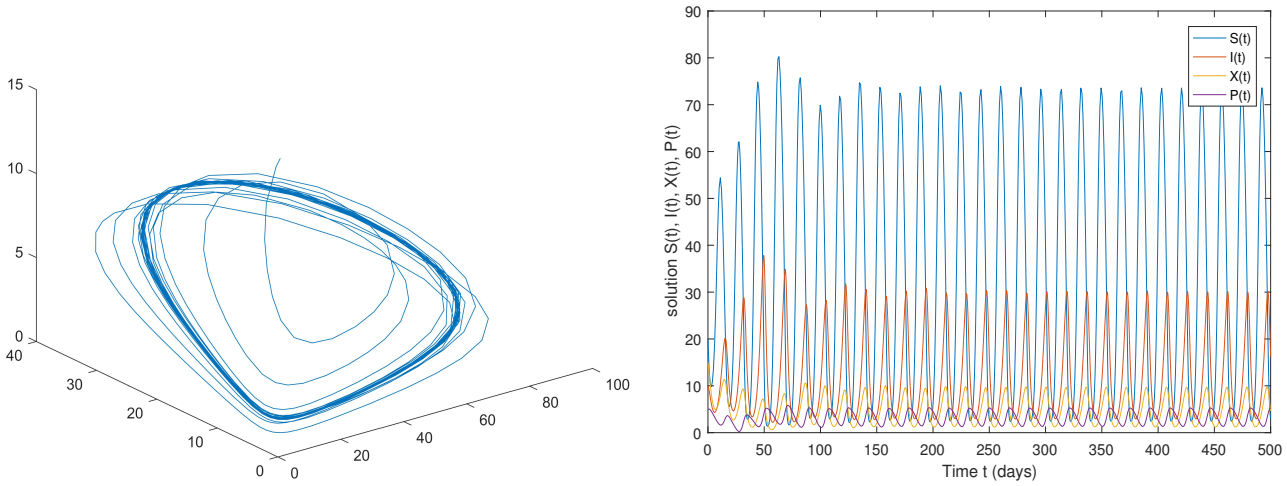


Figure 4. When $\tau > 25$, the system shows threshold value, i.e., with delay $\alpha = 0.086$, $\alpha_1 = 0.028$, $\beta = 0.075$, $k = 100$, $d = 0.6$, $d_1 = 0.5$, $d_2 = 0.04$, $b = 0.066$, $b_1 = 0.028$, $r = 0.95$, $\gamma = 0.0065$, $\gamma_1 = 0.0049$, $\gamma_2 = 0.082$, $\tau = 25$

10. Discussion and Conclusion

In this research paper, we investigate a predator-prey model that integrates infectious diseases within the predator population, considering the impact of delay-induced predator behaviors and a fear effect represented by the parameter k, which leads to a reduction in the prey's birth rate. Our study involves a thorough stability analysis of equilibria for the proposed system (denoted as (2.1)), revealing the occurrence of bifurcation. The paper introduces a comprehensive four-compartment eco-epidemiological model, providing valuable insights into both ecological and epidemiological aspects. The analysis covers every biologically feasible sustainable state of the model and evaluates the serenity of each equilibrium. The coexistence of species is shown to depend on specific parametric constraints, with the recovery predator playing a pivotal role in overall system dynamics. Furthermore, the paper explores limit cycle and periodic

oscillation behaviors observed at the bifurcation point around the interior equilibrium, as illustrated in phase portraits. The primary objective is to make significant contributions to the advancement of research in ecology, eco-epidemiology, and related fields, with a particular emphasis on biodiversity protection and effective management strategies. Additionally, the paper suggests potential directions for future research, proposing the incorporation of gestation delay to enhance the existing model. This envisioned addition could provide further insights and broaden the applicability of the model in understanding ecological and epidemiological dynamics. The paper also discusses the eco-epidemic model involving the predator population about disease, incorporating recovery delay. While most eco-epidemic models have previously focused on gestation delay, this study uniquely considers recovery delay in predators as well as disease transmission delay. Initially, the paper establishes the positivity and boundness of the suggested system's solutions. Subsequently, it identifies all possible equilibrium points and deduces their local stability criteria in the absence and presence of recovery delay, thereby enriching the understanding of the system's behavior.

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Competing Interests

The authors declare that they have no competing interests.

Authors' Contributions

All the authors contributed significantly in writing this article. The authors read and approved the final manuscript.

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