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Dynamics of predator-dependent nonlinear disease transmission in infected prey with diverse functional response

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Abstract. The disease in prey causes the indirect effect on the disease transmission of prey-predator interactions; this phenomenon of predator-dependent disease transmission scenario can arise as a consequence of anti-predator defence behaviour, debilitating the immune system of the prey. This concept is implemented in the proposed nonlinear mathematical prey-predator model, where an infectious disease infects only prey populations. The interaction between the susceptible prey and predator is assumed to be governed by Crowley-Martin type functional response and Holling I type functional response for the predation of infected prey. The susceptible prey becomes infected when contact occurs with the infected prey. The existence, uniqueness, boundedness, and feasibility and stability conditions of the fixed points of the system are analyzed. Hopf bifurcation analysis for the system is perceived and presented through bifurcation diagrams for different parameter values. Lastly, numerical exercises and graphical demonstrations are given to help our investigative findings.

1. Introduction

Many researchers contribute a lot of things in the field of prey-predator interaction due to disease transmission. The epidemiology problem has been painting attention to many scholars and experts ([1]-[41], [42]). Although a rich literature on eco-epidemiological models, predator-dependent disease transmission has rarely been considered in eco-epidemiological models. Mathematically, we consider a classical prey-predator model in which the prey population is subjected to an infectious disease. The predator can consume both infected and healthy prey (without becoming infected); however, the attack rates on infected and healthy prey are different. The disease transmission coefficient is considered as a function of the total amount of predator(s) density. Various mechanisms can allow for predator-dependent disease transmission. For instance, this can be a consequence of anti-predator behaviour. For example, in the presence of predators, a freshwater snail spends a long time hiding inside its shell. This makes it more vulnerable to parasites since the organism cannot expel the blood necessary for proper immune system functioning. Another widespread mechanism causing an increase in the transmission rate is a result of the grouping of prey in

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the presence of predators through the formation of fish schools, avian flocks, herds of herbivores, etc.. A dense 'packing' of individuals in groups would signify a larger transmission rate due to increased contacts. As a result, the prey population can become more vulnerable to a disease in the presence of predators, so the transmission rate of disease becomes a function of the total amount of the predator. Let us assume, for the sake of simplicity, that the incidence rate (i.e., the number of infected individuals per unit of time) can be described via a classical mass action relation, λIS, in an $S-I$ model, where S is the number of susceptible and I is the number of infected individuals in the prey population. In this case, the transmission coefficient λ can be a function of the number of predators P in the ecosystem, i.e., $\lambda(P)$. We call this phenomenon predator-dependent disease transmission, and, as is further shown, this can play a crucial role in shaping predator-prey-disease interactions.

In the recent era, some renowned authors ([43] – [49], [55], [61] – [69], [71] – [73]) studied functional responses to understand the importance of interaction between the prey and predator in the eco-system. They used some functional responses such as Cowley-Martin type functional response to make the model system more realistic and controllable in the eco-system. Ghanbari et al. [50] used Atangana-Baleanu fractional response to understand the nature of the three-species predator-prey model. A comparative study of explosive predator and mutualistic prey is done by Batabyal et al. [52]. The main aim of eco-epidemiological system is to eradicate the disease transmission in the system and many research papers are studied how the eco-epidemiological system can be made disease-free by regulating the system parameters. In this context we like to mention that Packer et al. [53] suggested that predation actually helps to reduce the infection load in prey population. They placed several experimental observations also to support their claim. Sih et al. [54] reviewed predator-removal experiments where they removed the predator population from the system and observed its effect on the prey population that are infected by some transmissible disease.

Depend on the above literature's, we discuss the role of predation on both susceptible and infected prey species for controlling the disease transmission. Our model uses the disease transmission term in the healthy and infected prey population, focusing on the effects of predation rate and infection rate to get the impact of disease in our system. Our analytical and numerical studies suggest that along with the infection rate, predation rate also plays a vital role in controlling the system's dynamics. We recognised two exciting cases: the disease can be abolished from prey species by regulating the predation on healthy prey. On the other hand, the system will be disease-free with appropriate control on predation of infected prey. The paper is organized as follows: Section 2 outlines the mathematical model with some basic assumptions. In Section 3: we discuss the positivity and boundedness of the prey-predator system. Section 4: we study the stability of equilibrium points. Section 5: Hopf bifurcation and permanence and impermanence of the system. The permanence of the prey-predator system is carried out in Section 6. Section 7: Numerical results and discussion. At the end of the paper, we conclude with the findings of this study and future directions for further development in Section 8.

2. Modelling of Disease Transmission Framework

In this work on the prey-predator system, the infected prey's disease spread model has been projected and deliberate. Here, $S(t)$, $I(t)$ and $P(t)$ denote the numbers of Susceptible Prey (SP), Infected Prey (IP), and Predator populations, respectively. Moreover, all parameters of our given mathematical model are positive. We impose the following assumptions to formulate the mathematical model.

(A_1) It is assumed that a parasite is infectious, and it spreads among prey only.

(A_2) In the absence of disease, the prey population grows according to a logistic curve with growth rate r ($r > 0$) and carrying capacity k ($k > 0$).

(A_3) In the presence of disease, the prey follows the susceptible-infected cycle only.

(A_4) Infected prey populations do not recover from the disease while the infected prey is removed by death with a constant positive death rate δ_1.

(A_5) Here, the disease in the prey population is transmitted from susceptible to infected prey population governed by a modified incidence function of the form $(\lambda_0 + aP)\frac{SI}{S+I}$, i.e., we assume predator dependent disease transmission.

(A_6) Disease does not spread from the infected prey to predator by feeding or other way.

(A_7) Predators consume both susceptible and infected prey populations. We also consider that the predator is a specialist, i.e., the prey population constitutes its only food source.

(A_8) We have considered Cowley-Martin type functional response for the predation of susceptible prey population. The Crowley-Martin response function is of the form: $\frac{\alpha_1 SP}{(1+bS)(1+cP)}$, which was first proposed by [46]. Here α_1, b and c positive parameters describe the effects of capture rate, handling time, and the magnitude of interference among predators on the feeding rate, respectively. This is a function of the biomass of both prey and predator due to predator interference. If the prey biomass is high, then also the predator feeding rate can decrease by higher predator biomass. Therefore, the effects of predator interference on feeding rate remain essential all the time whether an individual predator is handling or searching for prey at a given instant.

(A_9) The interaction between infected prey and predator is assumed to be governed by Holling type I functional response. Here the predator functional response on infected prey population is given by βIP.

(A_{10}) Predator is reduced by death with a constant positive death rate δ_2.

Based on the above ideas, we consider the following prey-predator model with nonlinear disease transmission rate

$$\begin{aligned}
\frac{dS}{dt} &= rS(1-\tfrac{S+I}{k}) - (\lambda_0 + aP)\tfrac{SI}{S+I} - \tfrac{\alpha_1 SP}{(1+bS)(1+cP)} \\
\frac{dI}{dt} &= (\lambda_0 + aP)\tfrac{SI}{S+I} - \beta_1 IP - \delta_1 I \\
\frac{dP}{dt} &= \tfrac{\alpha_2 SP}{(1+bS)(1+cP)} + \beta_2 IP - \delta_2 P
\end{aligned} \tag{1}$$

with initial conditions

$$S(0) = S_0 > 0,\ I(0) = I_0 > 0 \text{ and } P(0) = P_0 > 0. \tag{2}$$

where λ_0 be the disease transmission rate in the absence of a predator; a describes the effect of the presence of predator; b is the positive constant describing the handling time of the predator to consume the susceptible prey; c be a non-negative constant describing the magnitude of interference among predators; α_1 the predation rate on susceptible prey; α_2 the growth rate of a predator due to susceptible prey; β_1 the predation rate on infected prey; β_2 the growth rate of a predator due to infected prey.

3. Positivity and Boundedness

Theorem 1. *Entire solution of system (1) with initial conditions (2) exists in the interval* $[0, \infty)$, *for all* $t \geq 0$.

Proof. Since the right-hand side of the system (1) is completely continuous and locally Lipschitzian on C, the solution $(S(t), I(t), P(t))$ of (1) with initial conditions (2) exists and is unique on $[0, \zeta)$, where $0 < \zeta < +\infty$.

From system (1) with initial conditions (2) , we have

$$\begin{aligned}
S(t) &= S_0 e^{\int_0^t \left\{ r(1-(S(\xi)+I(\xi))/k) - (\lambda_0 + aP(\xi))\frac{I(\xi)}{S(\xi)+I(\xi)} - \frac{a_1 P(\xi)}{(1+bS(\xi))(1+cP(\xi))} \right\} d\xi} > 0 \\
I(t) &= I_0 e^{\int_0^t \left\{ (\lambda_0 + aP(\xi))\frac{S(\xi)}{S(\xi)+I(\xi)} - \beta_1 P(\xi) - \delta_1 \right\} d\xi} > 0 \\
P(t) &= P_0 e^{\int_0^t \left\{ \frac{a_2 S(\xi)}{(1+bS(\xi))(1+cP(\xi))} + \beta_2 I(\xi) - \delta_2 \right\} d\xi} > 0
\end{aligned}$$

which completes the proof. □

Theorem 2. *The solutions of the given mathematical model (1) are entirely bounded.*

Proof. From the first equation of model (1), we have

$$\begin{aligned}
\frac{dS}{dt} &= rS(t)\left(1 - \frac{S(t)+I(t)}{k}\right) - (\lambda_0 + aP(t))\frac{S(t)I(t)}{S(t)+I(t)} - \frac{\alpha_1 S(t)P(t)}{(1+bS(t))(1+cP(t))} \\
\frac{dS}{dt} &\leq rS(t)\left(1 - \frac{S(t)}{k}\right).
\end{aligned}$$

This gives, $S(t) \leq \frac{kc_1e^{rt}}{c_1e^{rt}+1}$ or, $S(t) \leq \frac{kc_1}{c_1+e^{-rt}}$, where c_1 is an arbitrary constant. Again since r is a positive quantity then $S(t) \leq k$ as $t \to \infty$.

Now, we perform a function like as $W(t) = S(t) + I(t) + P(t)$

Differentiating both sides with respect to t, we have

$$\frac{dW(t)}{dt} = \frac{dS(t)}{dt} + \frac{dI(t)}{dt} + \frac{dP(t)}{dt}$$

$$\frac{dW(t)}{dt} = rS(t)\left(1 - \frac{S(t)+I(t)}{k}\right) - (\lambda_0 + aP(t))\frac{S(t)I(t)}{S(t)+I(t)} - \frac{\alpha_1 S(t)P(t)}{(1+bS(t))(1+cP(t))}$$

$$+(\lambda_0 + aP(t))\frac{S(t)I(t)}{S(t)+I(t)} - \beta_1 I(t)P(t) - \delta_1 I(t) + \frac{\alpha_2 S(t)P(t)}{(1+bS(t))(1+cP(t))} + \beta_2 I(t)P(t) - \delta_2 P(t)$$

$$= rS(t) - (\delta_1 I(t) + \delta_2 P(t)) - I(t)P(t)(\beta_1 - \beta_2) - \frac{rS(t)(S(t)+I(t))}{k} - \frac{S(t)P(t)(\alpha_1 - \alpha_2)}{(1+bS(t))(1+cP(t))}$$

Since, $I(t)P(t)(\beta_1 - \beta_2) > 0$ if $\beta_1 > \beta_2$, $rS(t)\left(\frac{S(t)+I(t)}{k}\right) > 0$, and lastly $\frac{S(t)P(t)}{(1+bS(t))(1+cP(t))}(\alpha_1 - \alpha_2) > 0$ if $\alpha_1 > \alpha_2$, we have

$$\frac{dW(t)}{dt} \leq 2rS(t) - (rS(t) + \delta_1 I(t) + \delta_2 P(t)) \tag{3}$$

Assuming $\eta = \min\{r, \delta_1, \delta_2\}$, then from the relation (3), we have

$$\frac{dW(t)}{dt} + \eta W \leq 2rk \tag{4}$$

Using the theory of differential inequality, we have

$$0 < W(t) \leq W(0)e^{-\eta t} + \frac{2rk}{\eta}(1 - e^{-\eta t})$$

and for $t \to \infty$, we have $0 < W \leq \frac{2rk}{\eta}$. Therefore, all the solutions of the system $(S(t), I(t), P(t))$ enters in $\Omega = \{(S(t), I(t), P(t)) \in \mathbb{R}^3_+ : 0 < W \leq \frac{2rk}{\eta}\}$ or approaches it asymptotically. Hence, it is a positively invariant set of system.

Thus, in Ω the system (1) is well-posed eco-epidemiologically and mathematically.

Hence, it is sufficient to study the dynamics of the system (1) in Ω. □

4. Equilibrium points: their existence and stability

In this section, we will study the existence and stability behaviour of the system (1). The equilibrium points of the model system (1) are: (I) The trivial equilibrium point $E_0 = (0, 0, 0)$, (II) The infection and predator-free Axial equilibrium point $E_1 = (k, 0, 0)$, (III) The predator-free equilibrium point (i) $E_2 = \left(\overline{S}, \overline{I}, 0\right)$, where $\overline{S} = \frac{\delta_1 k(r-\lambda_0+\delta_1)}{r\lambda_0}$, $\overline{I} = \frac{k(\lambda_0-\delta_1)(r-\lambda_0+\delta_1)}{r\lambda_0}$, (ii) The infection-free equilibrium point $E_2 = \left(\overline{S}, 0, \overline{P}\right)$, was $\overline{P} = \frac{(\alpha_2-b\delta_2)\overline{S}-\delta_2}{c\delta_2(1+b\overline{S})}$ and $\overline{S}$ is the positive root of the following cubic equation.

$$K_0S^3 + 3K_1S^2 + 3K_2S + K_3 = 0$$

where $K_0 = rbc\alpha_2$, $K_1 = \frac{1}{3}rc\alpha_2(1 - bk)$, $K_2 = \frac{1}{3}[\alpha_1 k(\alpha_2 - b\delta_2) - rck\alpha_2]$, $K_3 = -\alpha_1 k\delta_2$.

The above equation has exactly one positive root if $G + 4H^3 > 0$, where $G = K_0^2K_3 - 3K_0K_1K_2 + 2K_3^3$ and $H = K_0K_2 - K_1^2$.

Using Cardan's method, we obtain the root as $\frac{1}{A}(J - K_1)$ where J denotes the real value of $\left[\frac{1}{2}\left(-G + \sqrt{G^2 + 4H^3}\right)\right]^{\frac{1}{3}}$, and (IV) Interior equilibrium point $E^* = (S^*, I^*, P^*)$.

4.1. Existence of interior equilibrium $E^*(S^*, I^*, P^*)$

This section will analyze the existence of the non-trivial interior equilibrium of the model system (1). At an interior equilibrium, the followings hold:

$$S > 0, I > 0, P > 0 \text{ and } \frac{dS}{dt} = \frac{dI}{dt} = \frac{dP}{dt} = 0. \tag{5}$$

Now, solving the equations of (5) at the equilibrium point we get,

$$\begin{aligned}
&r\left(1 - \frac{S+I}{k}\right) - (\lambda_0 + aP)\frac{I}{S+I} - \frac{\alpha_1 P}{(1+bS)(1+cP)} = 0\\
&(\lambda_0 + aP)\frac{S}{S+I} - \beta_1 P - \delta_1 = 0\\
&\frac{\alpha_2 S}{(1+bS)(1+cP)} + \beta_2 I - \delta_2 = 0,
\end{aligned}$$

and (S^*, I^*, P^*) be the positive root of the above three equations.

4.2. Local stability analysis

In this section, we shall study the local stability analysis of the system (1) at various equilibrium points.

4.2.1. Trivial equilibrium

Now, the variational matrix of system (1) at $E_0(0,0,0)$ is given by

$$V(E_0) = \begin{pmatrix} r & 0 & 0 \\ 0 & -\delta_1 & 0 \\ 0 & 0 & -\delta_2 \end{pmatrix}$$

Therefore, eigenvalues of the characteristic equation of $V(E_0)$ are $\lambda_1 = r > 0$, $\lambda_2 = -\delta_1 < 0$, $\lambda_3 = -\delta_2 < 0$. This implies E_0 is always unstable since one eigenvalue is positive and the other two are negative.

4.2.2. Axial equilibrium

The variational matrix of system (1) at $E_1(k,0,0)$ is given by,

$$V(E_1) = \begin{pmatrix} -r & -r-\lambda_0 & -\frac{\alpha_1 k}{1+bk} \\ 0 & \lambda_0 - \delta_1 & 0 \\ 0 & 0 & \frac{\alpha_2 k}{1+bk} - \delta_2 \end{pmatrix}$$

Therefore, eigenvalues of the characteristic equation of $V(E_1)$ are $\lambda_1 = -r < 0$, $\lambda_2 = \lambda_0 - \delta_1$, $\lambda_3 = \frac{\alpha_2 k}{1+bk} - \delta_2$. Hence, all the eigenvalues will be negative if $\lambda_2 < 0$, i.e., $\lambda_0 < \delta_1$ and $\lambda_3 < 0$, i.e., $\alpha_2 k < \delta_2(1+bk)$. Thus E_1 is locally asymptotically stable if $\lambda_0 < \delta_1$ and $\alpha_2 k < \delta_2(1+bk)$.

4.2.3. Planar equilibrium

The variational matrix of system (1) at $E_2(\bar{S}, \bar{I}, 0)$ is given by,

$$V(E_2) = \begin{pmatrix} m_1 & m_2 & m_3 \\ m_4 & m_5 & m_6 \\ 0 & 0 & m_7 \end{pmatrix}$$

where $m_1 = r - \frac{2r\bar{S}}{k} - \frac{r\bar{I}}{k} - \lambda_0 \frac{\bar{I}^2}{(\bar{S}+\bar{I})^2}$, $m_2 = -\frac{r\bar{S}}{k} - \lambda_0 \frac{\bar{S}^2}{(\bar{S}+\bar{I})^2}$, $m_3 = -\frac{a\bar{S}\bar{I}}{\bar{S}+\bar{I}} - \frac{\alpha_1 \bar{S}}{1+b\bar{S}}$, $m_4 = \lambda_0 \frac{\bar{I}^2}{(\bar{S}+\bar{I})^2}$, $m_5 = \lambda_0 \frac{\bar{S}^2}{(\bar{S}+\bar{I})^2} - \delta_1$, $m_6 = a\frac{\bar{S}\bar{I}}{\bar{S}+\bar{I}} - \beta_1 \bar{I}$, and $m_7 = \frac{\alpha_2 \bar{S}}{1+b\bar{S}} + \beta_2 \bar{I} - \delta_2$.

Therefore, the characteristic equation of $V(E_2)$ is $(m_7-\lambda)\left\{\lambda^2+A_{11}\lambda+A_{12}\right\}=0$, where $A_{11}=-(m_1+m_5)$ and $A_{12}=m_1m_5-m_2m_4$.

Hence, the one eigenvalue of the above characteristic equation is m_7, which is negative as $\delta_2>\frac{\alpha_2\overline{S}}{1+b\overline{S}}+\beta_2\overline{I}$ and the other two eigenvalues should be negative if $A_{11}>0$ and $A_{12}>0$.

Hence, E_2 is locally asymptotically stable if $A_{11}>0$, $A_{12}>0$ and $\delta_2>\frac{\alpha_2\overline{S}}{1+b\overline{S}}+\beta_2\overline{I}$.

Here, the variational matrix of system (1) at $E_3\left(\overline{S},0,\overline{P}\right)$ is given by,

$$V(E_3)=\begin{pmatrix} p_1 & p_2 & p_3\\ 0 & p_4 & 0\\ p_5 & p_6 & p_7\end{pmatrix}$$

where $p_1=r-\frac{2r\overline{S}}{k}-\frac{\alpha_1\overline{P}}{(1+c\overline{P})(1+b\overline{S})^2}$, $p_2=-\frac{r\overline{S}}{k}-\left(\lambda_0+a\overline{P}\right)$, $p_3=-\frac{\alpha_1\overline{S}}{(1+b\overline{S})(1+c\overline{P})^2}$, $p_4=\left(\lambda_0+a\overline{P}\right)-\beta_1\overline{P}-\delta_1$, $p_5=\frac{\alpha_2\overline{P}}{(1+c\overline{P})(1+b\overline{S})^2}$, $p_6=\beta_2\overline{P}$ and $p_7=\frac{\alpha_2\overline{S}}{(1+b\overline{S})(1+c\overline{P})^2}-\delta_2$.

Now, the characteristic equation for $V(E_3)$ is $(p_4-\lambda)\left\{\lambda^2+B_{11}\lambda+B_{12}\right\}=0$, where $B_{11}=-(p_1+p_7)$ and $B_{12}=p_1p_7-p_3p_5$.

Hence, the one eigenvalue of the above characteristic equation is p_4, which is negative as $\delta_1>\lambda_0+(a-\beta_1)\overline{P}$ and the other two eigenvalues should be negative if $B_{11}>0$ and $B_{12}>0$.

So, the predator-free equilibrium point $E_3(\overline{S},0,\overline{P})$ is locally asymptotically stable if $\delta_1>\lambda_0+(a-\beta_1)\overline{P}$, $B_{11}>0$ and $B_{12}>0$, otherwise the system (1) will be unstable.

4.2.4. Interior equilibrium

Now, the variational matrix of system (1) at $E^*(S^*,I^*,P^*)$ is given by,

$$V(E^*)=\begin{pmatrix} a_{11} & a_{12} & a_{13}\\ a_{21} & a_{22} & a_{23}\\ a_{31} & a_{32} & a_{33}\end{pmatrix}$$

where $a_{11}=r-\frac{2rS^*}{k}-\frac{rI^*}{k}-(\lambda_0+aP^*)\frac{I^{*2}}{(S^*+I^*)^2}-\frac{\alpha_1P^*}{(1+cP^*)(1+bS^*)^2}$, $a_{12}=-\frac{rS^*}{k}+(\lambda_0+aP^*)\frac{S^{*2}}{(S^*+I^*)^2}$, $a_{13}=-\left(\frac{aI^*}{S^*+I^*}+\frac{\alpha_1}{(1+bS^*)(1+cP^*)^2}\right)S^*$, $a_{21}=(\lambda_0+aP^*)\frac{I^{*2}}{(S^*+I^*)^2}$, $a_{22}=(\lambda_0+aP^*)\frac{S^{*2}}{(S^*+I^*)^2}-\beta_1P^*-\delta_1$, $a_{23}=\frac{aS^*I^*}{S^*+I^*}-\beta_1I^*$, $a_{31}=\frac{\alpha_2P^*}{(1+cP^*)(1+bS^*)^2}$, $a_{32}=\beta_2P^*$ and $a_{33}=\frac{\alpha_2S^*}{(1+bS^*)(1+cP^*)^2}+\beta_2I^*-\delta_2$.

Therefore, the characteristic equation of $V(E^*)$ is given by,

$$\eta^3+B_1\eta^2+B_2\eta+B_3=0 \tag{6}$$

where, $B_1=-(a_{11}+a_{22}+a_{33})$, $B_2=-(a_{12}a_{21}+a_{13}a_{31}+a_{23}a_{32}-a_{11}a_{22}-a_{11}a_{33}-a_{22}a_{33})$ and $B_3=-(a_{11}a_{22}a_{33}+a_{12}a_{23}a_{31}+a_{13}a_{21}a_{32}-a_{13}a_{31}a_{22}-a_{12}a_{21}a_{33}-a_{11}a_{23}a_{32})$.

Hence, by the Routh-Hurwitz criterion, it follows that all eigenvalues of the characteristic equation (6) have negative real parts if and only if

$$B_1>0,\ B_3>0 \ \text{ and } \ B_1B_2-B_3>0. \tag{7}$$

Therefore, the system (1) shows local asymptotic stability at E^* when conditions (7) are satisfied. Thus, from the previous discussions, we come to the following result:

Theorem 3. *The equilibrium point E_0 is always unstable.*

Theorem 4. *The equilibrium point E_1 is locally asymptotically stable if $\lambda_0<\delta_1$ and $\alpha_2k<\delta_2(1+bk)$.*

Theorem 5. *The equilibrium point E_2 is locally asymptotically stable if $A_{11}>0$, $A_{12}>0$ and $\delta_2>\frac{\alpha_2\overline{S}}{1+b\overline{S}}+\beta_2\overline{I}$.*

Theorem 6. *The equilibrium point E_3 is locally asymptotically stable if $B_{11}>0$, $B_{12}>0$ and $\delta_1>\lambda_0+(a-\beta_1)\overline{P}$.*

Theorem 7. *The equilibrium point E^* is locally asymptotically stable if $B_1>0$, $B_3>0$ and $B_1B_2-B_3>0$.*

4.3. Global stability analysis

In this section, we shall study the global stability behaviour of the system (1) at the interior equilibrium $E^*(S^*, I^*, P^*)$. Let us define,

$$L = \left(S - S^* - S^* \ln \frac{S}{S^*}\right) + Q\left(I - I^* - I^* \ln \frac{I}{I^*}\right) + R\left(P - P^* - P^* \ln \frac{P}{P^*}\right) \tag{8}$$

where Q and R are positive constants.

Here $L(S, I, P) \geq 0$ since $\theta - 1 \geq \ln \theta$ for $\theta > 0$ and $L(S^*, I^*, P^*) = 0$. Differentiating L along with the solutions of system (1) with respect to time parameter t, we get

$$\begin{aligned}
\frac{dL}{dt} &= \left(\frac{S - S^*}{S}\right)\frac{dS}{dt} + Q\left(\frac{I - I^*}{I}\right)\frac{dI}{dt} + R\left(\frac{P - P^*}{P}\right)\frac{dP}{dt} \\
&= \left[r(1 - \frac{S + I}{k}) - (\lambda_0 + aP)\frac{I}{S + I} - \frac{\alpha_1 P}{(1 + bS)(1 + cP)}\right](S - S^*) \\
&+ Q\left[(\lambda_0 + aP)\frac{S}{S + I} - \beta_1 P - \delta_1\right](I - I^*) + R\left[\frac{\alpha_2 S}{(1 + bS)(1 + cP)} + \beta_2 I - \delta_2\right](P - P^*)
\end{aligned}$$

After some simplifications we get,

$$\begin{aligned}
\frac{dL}{dt} &= -(S - S^*)\left[\frac{r}{k}\{(S + I) - (S^* + I^*)\} + \{(\lambda_0 + aP)\frac{I}{S + I} - (\lambda_0 + aP^*)\frac{I^*}{S^* + I^*}\}\right. \\
&\left. + a_1\left(\frac{P}{(1 + bS)(1 + cP)} - \frac{P^*}{(1 + bS^*)(1 + cP^*)}\right)\right] \\
&- Q\,(I - I^*)\left[\beta_1\,(P - P^*) + \{(\lambda_0 + aP)\frac{S}{S + I} - (\lambda_0 + aP^*)\frac{S^*}{S^* + I^*}\}\right] \\
&- R\,(P - P^*)\left[\frac{cbS\,(S^*P - S^*P^*) - c\,(SP^* - S^*P) - (S - S^*)}{(1 + bS)(1 + cP)(1 + bS^*)(1 + cP^*)} - \beta_2\,(I - I^*)\right]
\end{aligned}$$

Now, we see that $\frac{dL}{dt}$ is negative definite in the region:
$G = \{(S, I, P) : S > S^*, I > I^* \text{ and } P > P^* \text{ or } S < S^*, I < I^* \text{ and } P < P^*\}$ and consequently L is a Lyapunov function with respect to all solutions in G. Summarizing the above discussions we come to the following result:

Theorem 8. *If E^* is locally asymptotically stable then E^* is globally asymptotically stable in $G = \{(S, I, P) : S > S^*, I > I^* \text{ and } P > P^* \text{ or}$*

5. Hopf bifurcation at $E^*(S^*, I^*, P^*)$

The characteristic equation of system (1) at $E^*(S^*, I^*, P^*)$ is given by

$$\eta^3 + B_1(r)\eta^2 + B_2(r)\eta + B_3(r) = 0 \tag{9}$$

where, $B_1(r) = (\delta_1+\delta_2) - r - \left(\beta_2 - (\lambda_0+aP^*) - \frac{r}{k}\right)I^* - \left(\frac{\alpha_2}{1+bS^*} - \frac{2r}{k} + \lambda_0 + aP^*\right)S^* + \left(\frac{\alpha_1}{(1+bS^*)^2} + \beta_1\right)P^*$,

$$
\begin{aligned}
B_2(r) &= \left(r - \frac{2rS^*}{k} - \left(\frac{r}{k} - \lambda_0 - aP^*\right)I^* - \frac{\alpha_1 P^*}{(1+bS^*)^2}\right)((\lambda_0+aP^*)S^* - \beta_1 P^* - \delta_1) \\
&+ \left(aI^* + \frac{\alpha_1}{1+bS^*}\right)\frac{\alpha_2 P^* S^*}{(1+bS^*)^2} + \left(r - \frac{2rS^*}{k} - \left(\frac{r}{k} - (\lambda_0+aP^*)\right)I^* - \frac{\alpha_1 P^*}{(1+bS^*)^2}\right)\left(\frac{\alpha_2 S^*}{1+bS^*} + \beta_2 I^* - \delta_2\right) \\
&+ \left(\frac{r}{k} + \lambda_0 + aP^*\right)(\lambda_0+aP^*)S^*I^* + ((\lambda_0+aP^*)S^* - \beta_1 P^* - \delta_1)\left(\frac{\alpha_2 S^*}{1+bS^*} + \beta_2 I^* - \delta_2\right) \\
&- ((aS^* - \beta_1)\beta_2 P^* I^*), \\
B_3(r) &= \left(r - r\frac{2S^*+I^*}{k} - (\lambda_0+aP^*)I^* - \frac{\alpha_1 P^*}{(1+bS^*)^2}\right)(((\lambda_0+aP^*)S^* - \beta_1 P^* - \delta_1)\left(\frac{\alpha_2 S^*}{1+bS^*} + \beta_2 I^* - \delta_2\right) - (aS^* - \beta_1)\beta_2 I^* P^*) \\
&+ \left(\frac{r}{k} + \lambda_0 + aP^*\right)(\lambda_0+aP^*)\left(\frac{\alpha_2 S^*}{1+bS^*} + \beta_2 I^* - \delta_2\right)S^*I^* - \left(\frac{r}{k} + \lambda_0 + aP^*\right)\frac{(aS^* - \beta_1)\alpha_2}{(1+bS^*)^2}S^*I^*P^* \\
&+ \left(aI^* + \frac{\alpha_1}{1+bS^*}\right)\frac{\alpha_2 P^*}{(1+bS^*)^2}((\lambda_0+aP^*)S^* - \beta_1 P^* - \delta_1)S^* - \left(aI^* + \frac{\alpha_1}{1+bS^*}\right)(\lambda_0+aP^*)\beta_2 S^* I^* P^*.
\end{aligned}
$$

To check out the instability of the system (1), let us consider r as the bifurcation parameter. For this purpose, let us first state the following theorem:

Theorem 9. *(Hopf Bifurcation Theorem) If $B_1(r)$, $B_2(r)$, $B_3(r)$ are smooth functions of r in an open interval about $r_0 \in R$ such that the characteristic equation (9) has*

(i) a pair of complex eigenvalues $\eta = \varkappa(r) \pm i\varrho(r)$ (with $\varkappa(r), \varrho(r) \in R$) so that they become purely imaginary at $r = r_0$ and $\left(\frac{d\varkappa}{dr}\right)_{r=r_0} \neq 0$,

(ii) the other eigenvalue is negative at $r = r_0$, then a Hopf bifurcation occurs around E^* at $r = r_0$ (i.e., a stability change of E^* accompanied by the creation of a limit cycle at $r = r_0$).

Theorem 10. *The system (1) possesses a Hopf bifurcation around E^* when a passes through r_0 provided $B_1(r_0), B_2(r_0) > 0$ and $B_1(r_0)B_2(r_0) = B_3(r_0)$.*

Proof. For $r = r_0$, the characteristic equation of the prey-predator system (1) at E^* becomes $(\eta+B_1)(\eta^2+B_2) = 0$, providing roots $\eta_1 = i\sqrt{B_2}, \eta_2 = -i\sqrt{B_2}$, and $\eta_3 = -B_1$. Thus, there exists a pair of purely imaginary eigenvalues and a strictly negative eigenvalue. Additionally, $B_1(r)$, $B_2(r)$, $B_3(r)$ are smooth functions of r. Therefore, for r in a neighbourhood of r_0, the roots have the form $\eta_1(r) = \xi_1(r) + i\xi_2(r)$, $\eta_2(r) = \xi_1(r) - i\xi_2(r)$, $\eta_3 = -\xi_3(r)$, where $\xi_i(r), i = 1, 2, 3$ are real.
Next, we shall verify the transversality condition

$$\frac{d}{dr}(Re(\eta_i(r)))|_{r=r_0} \neq 0, i = 1, 2.$$

Putting the value $\eta(r) = \xi_1(r) + i\xi_2(r)$ into the characteristic equation (9), we have

$$(\xi_1 + i\xi_2)^3 + B_1(\xi_1 + i\xi_2)^2 + B_2(\xi_1 + i\xi_2) + B_3 = 0 \tag{10}$$

Now, differentiating both sides of equation (10) with respect to r, we get

$$\left(3(\xi_1 + i\xi_2)^2 + 2B_1(\xi_1 + i\xi_2) + B_2\right)\left(\dot{\xi_1} + i\dot{\xi_2}\right) + \dot{B_1}(\xi_1 + i\xi_2)^2 + \dot{B_2}(\xi_1 + i\xi_2) + \dot{B_3} = 0 \tag{11}$$

Comparing the real and imaginary parts of both sides of (11) we get,

$$D_1\dot{\xi_1} - D_2\dot{\xi_2} + D_3 = 0 \tag{12}$$

and

$$D_2\dot{\xi_1} + D_1\dot{\xi_2} + D_4 = 0, \tag{13}$$

where $D_1 = 3(\xi_1^2 - \xi_2^2) + 2B_1\xi_1 + B_2$, $D_2 = 6\xi_1\xi_2 + 2B_1\xi_2$, $D_3 = \dot{B_1}(\xi_1^2 - \xi_2^2) + \dot{B_2}\xi_1 + \dot{B_3}$, $D_4 = 2\dot{B_1}\xi_1\xi_2 + \dot{B_2}\xi_2$.

Solving (12) and (13) we get,

$$\dot{\xi_1} = -\frac{D_2D_4 + D_1D_3}{D_1^2 + D_2^2} \tag{14}$$

Now, $D_3 = \dot{B_1}(\xi_1^2 - \xi_2^2) + \dot{B_2}\xi_1 + \dot{B_3} \neq \dot{B_1}(\xi_1^2 - \xi_2^2) + \dot{B_2}\xi_1 + \dot{B_1}B_2 + \dot{B_2}B_1$
At $r = r_0$,

(i) For $\xi_1 = 0$, $\xi_2 = \sqrt{B_2}$, we have $D_1 = -2B_2$, $D_2 = 2B_1\sqrt{B_2}$, $D_3 \neq B_1\dot{B_2}$, $D_4 = \dot{B_2}\sqrt{B_2}$, hence $D_2D_4 + D_1D_3 \neq 2B_1\dot{B_2}B_2 - 2B_1\dot{B_2}B_2 = 0$. So, $D_2D_4 + D_1D_3 \neq 0$ at $r = r_0$, when $\xi_1 = 0, \xi_2 = \sqrt{B_2}$.

(ii) For $\xi_1 = 0$, $\xi_2 = -\sqrt{B_2}$, we have $D_1 = -2B_2$, $D_2 = -2B_1\sqrt{B_2}$, $D_3 \neq B_1\dot{B_2}$, $D_4 = -\dot{B_2}\sqrt{B_2}$, therefore $D_2D_4 + D_1D_3 \neq 2B_1\dot{B_2}B_2 - 2B_1\dot{B_2}B_2 = 0$. So, $D_2D_4 + D_1D_3 \neq 0$ at $r = r_0$, when $\xi_1 = 0$, $\xi_2 = -\sqrt{B_2}$.
Therefore, $\frac{d}{dr}(Re(\eta_i(r)))|_{r=r_0} = -\frac{D_2D_4+D_1D_3}{D_1^2+D_2^2}|_{r=r_0} \neq 0$
and $\xi_3(r_0) = -B_1(r_0) < 0$.
Hence by Theorem (9), the result follows. □

6. Permanence of the system

To prove the permanence of the system (1), we shall use the average Liapunov function.

Theorem 11. *Suppose that the system (1) satisfies the following conditions: (i) $\lambda_0 > \delta_1$ and $\frac{\alpha_2 k}{1+bk} > \delta_2$, (ii) $\frac{\alpha_2\overline{S}}{1+b\overline{S}} + \beta_2\overline{I} > \delta_2$, (iii) $\left(\lambda_0 + a\overline{P}\right) > \beta_1\overline{P} + \delta_1$. Then the system (1) is permanent.*

Proof. Let us consider the average Lyapunov function in the form $F(S, I, P) = S^{\gamma_1}I^{\gamma_2}P^{\gamma_3}$ where each γ_i ($i = 1, 2, 3$) is assumed to be positive. In the interior of $\mathbb{R}^3_+$, we have

$$\frac{\dot{F}}{F} = \phi(S, I, P) = \gamma_1\left[r(1 - \frac{S+I}{k}) - (\lambda_0 + aP)\frac{I}{S+I} - \frac{\alpha_1 P}{(1+bS)(1+cP)}\right]$$
$$+\gamma_2\left[(\lambda_0 + aP)\frac{S}{S+I} - \beta_1 P - \delta_1\right] + \gamma_3\left[\frac{\alpha_2 S}{(1+bS)(1+cP)} + \beta_2 I - \delta_2\right].$$

To prove the permanence of the system, we shall have to show that $\phi(S, I, P) > 0$ for all boundary equilibria of the system. The values of $\phi(S, I, P)$ at the boundary equilibria E_0, E_1, E_2 and E_3 are the following:

$E_0 : r\gamma_1 - \delta_1\gamma_2 - \delta_2\gamma_3$,
$E_1 : (\lambda_0 - \delta_1)\gamma_2 + \left(\frac{\alpha_2 k}{1+bk} - \delta_2\right)\gamma_3$,
$E_2 : \left(\frac{\alpha_2\overline{S}}{1+b\overline{S}} + \beta_2\overline{I} - \delta_2\right)\gamma_3$,
and $E_3 : \left(\left(\lambda_0 + a\overline{P}\right) - \beta_1\overline{P} - \delta_1\right)\gamma_3$.

Now, $\phi(0, 0, 0) > 0$ is automatically satisfied for some γ_i ($i = 1, 2, 3$). Also, if the inequalities (i)-(iii) hold, ϕ is positive at E_1, E_2 and E_3. Therefore, the system (1) is permanent. Hence the theorem. □

Remark 1. *The conditions (i) $E_1 : \lambda_0 - \delta_1 > 0$ and $\frac{\alpha_2 k}{1+bk} - \delta_2 > 0$, (ii) $E_2 : \frac{\alpha_2\overline{S}}{1+b\overline{S}} + \beta_2\overline{I} - \delta_2 > 0$, (iii) $E_3 : \left(\lambda_0 + a\overline{P}\right) - \beta_1\overline{P} - \delta_1 > 0$, guarantee that the boundary equilibrium points E_1, E_2 and E_3 are unstable.*

7. Numerical Example

It is quite challenging to have the numerical value of the system's parameters based on real-world observations. On the other hand, it is necessary to have some idea regarding the sensitivity of the parameters in connection with the observed natural system. Therefore, the major results described by the simulations presented should be considered from a qualitative rather than a quantitative point of view with some hypothetical data. However, numerous scenarios covering the breath of the biological feasible parameter space were conducted. The results are shown above to display the breadth of dynamical results collected from all tested scenarios. MATLAB and Mathematica software are used for simulation experiments for the sole purpose of solving the system numerically to obtain the results after numerical simulations.

Table 1: Parameter values and initial densities

Parameter	Value	Parameter/Population	Value
r	2	β_1	0.5
k	5.5	β_2	0.4
λ_0	0.5	δ_1	0.1
a	0.6	δ_2	0.5
α_1	0.5	$S(0)$	1.0
α_2	0.4	$I(0)$	1.0
b	0.1	$P(0)$	2.0
c	0.1		

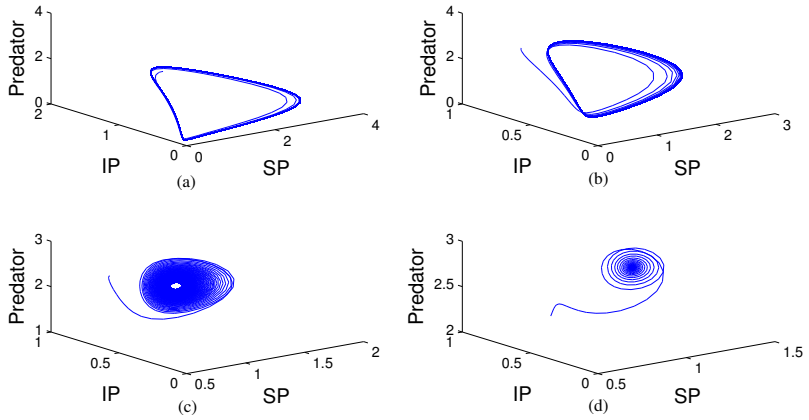

Fig 1: Phase portrait for different value of (a) $r = 1.0$, (b) $r = 1.5$, (c) $r = 2.0$, (d) $r = 2.3$ using other parameters and initial value from Table 1.

The Phase portrait in (S, I, P)-view of the model system (1) at different values of r is presented in the Fig 1. The stable limit cycle in SIP-view of the model system (1) at $r = 1.0$ and $r = 1.5$ are presented in the Fig 1(a) and Fig 1(b) respectively. Similarly Fig 1(c) and Fig 1(d) represents the stable focus in SIP-view for $r = 2.0$ and $r = 2.3$, respectively.

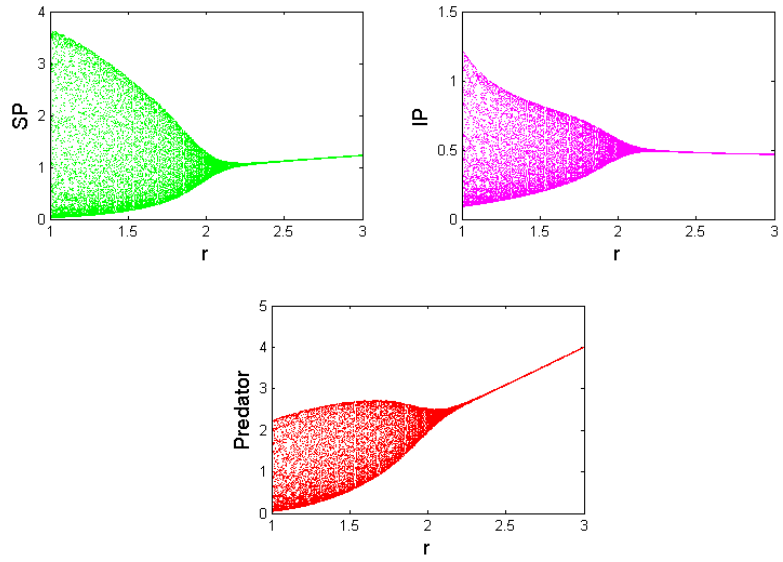

Fig 2: Bifurcation diagram of the system (1) for r in $1 \leq r \leq 3$ for the data given in Table 1 to SP, IP and Predator.

Fig 2 represents the bifurcation diagrams generated for the successive of the population densities S, I, and P in the ranges $[0.0, 4.0]$, $[0.0, 1.5]$, and $[0.0, 5.0]$ respectively as a function of intrinsic growth rate r in the range $1 \leq r \leq 3$ and parameter values are given in the Table 1. It is observed that, when r increases the then all population densities are tends to stable system. In our model system (1) we consider r as a bifurcation parameter.

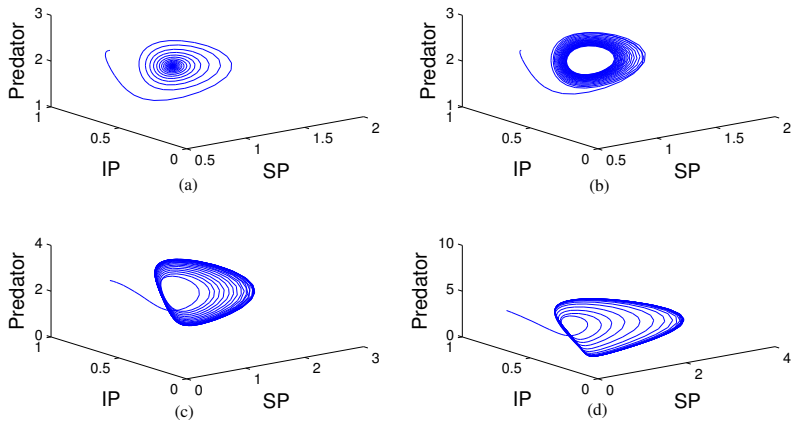

Fig 3: Phase portrait for different value of (a) $k = 4.5$, (b) $k = 5.5$, (c) $k = 6.5$, (d) $k = 7.5$ and other fixed inputs given in Table 1.

The Phase portrait in (S, I, P)-view of the system (1) at different values of k is presented in the Fig 3. The stable focus in SIP-view of the model (1) at $k = 4.5$ be presented in the Fig 3(a). Similarly Fig 3(b), Fig 3(c) and Fig 3(d) represents the stable limit cycle in SIP-view for $k = 5.5$, $k = 6.5$ and $k = 7.5$, respectively.

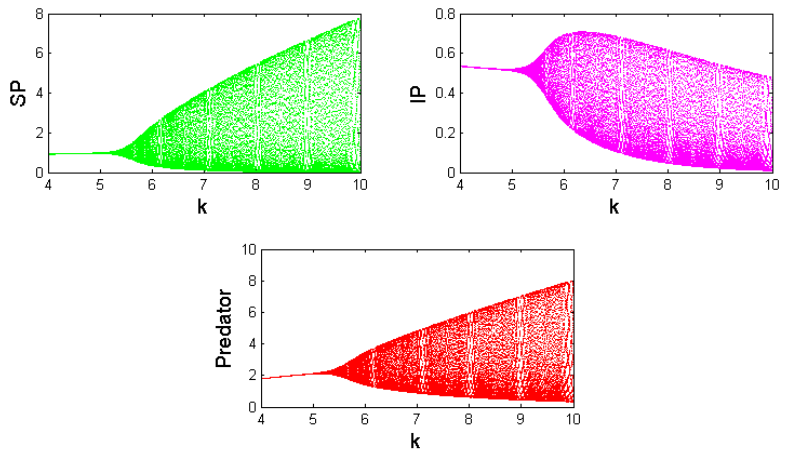

Fig 4: Bifurcation diagram of the model (1) for k in $4 \leq k \leq 10$ for the parameters given in Table 1 to SP, IP and Predator.

The bifurcation diagrams generated for the successive of the population densities S, I, and P in the ranges $[0.0, 8.0]$, $[0.0, 6.0]$, and $[0.0, 10.0]$ respectively as a function of environmental carrying capacity k in the range $4 \leq k \leq 10$ (see Fig 4.) and parameter values are given in the Table 1. It is observed that, when k increases the then all of the population densities are tends to form a limit cycle system.

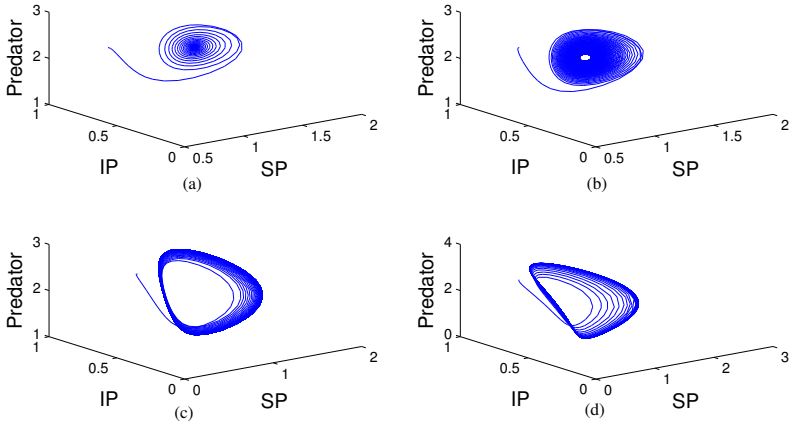

Fig 5: Phase portrait for different value of (a) $\lambda_0 = 0.4$, (b) $\lambda_0 = 0.5$, (c) $\lambda_0 = 0.6$ and (d) $\lambda_0 = 0.7$, and taking other as fixed input from Table 1.

The Phase portrait in (S, I, P)-view of the system (1) at different values of λ_0 is presented in the Fig 5. The stable focus in SIP-view of the model system (1) at $\lambda_0 = 0.4$ be presented in the Fig 5(a). Similarly Fig 5(b), Fig 5(c) and Fig 5(d) represents the stable limit cycle in SIP-view for $\lambda_0 = 0.5$, $\lambda_0 = 0.6$ and $\lambda_0 = 0.7$, respectively.

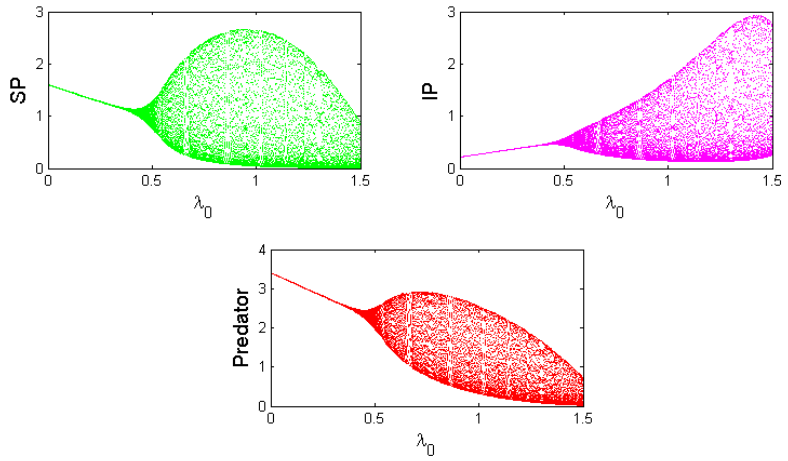

Fig 6: Bifurcation diagram of the system (1) for λ_0 in the range $0 \leq \lambda_0 \leq 1.5$ with respect to SP, IP and Predator for the input data given in Table 1.

Fig 6 represents the bifurcation diagrams generated for the successive of the population densities SP, IP, and Predator in the ranges [0.0, 3.0], [0.0, 3.0], and [0.0, 4.0], respectively as a function of disease transmission rate in the absence of predator λ_0 in the range $0 \leq \lambda_0 \leq 1.5$ and parameter values are given in the Table 1. It is observed that, when λ_0 increases the then all of the population densities are tends to limit cycle system.

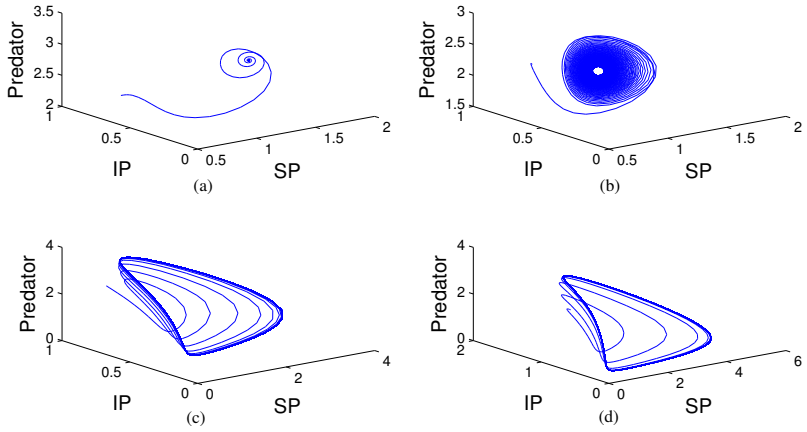

Fig 7: Phase portrait for different value of a for (a) $a = 0.5$, (b) $a = 0.6$, (c) $a = 0.7$, (d) $a = 0.8$ and keeping other input fixed as given in Table 1.

The Phase portrait in (S, I, P)-view of the model (1) at different values of a is presented in the Fig 7. The stable focus in SIP-view of the system (1) at $a = 0.5$ be presented in the Fig 7(a). Similarly Fig 7(b), Fig 7(c) and Fig 7(d) represents the stable limit cycle in SIP-view for $a = 0.6$, $a = 0.7$ and $a = 0.8$, respectively.

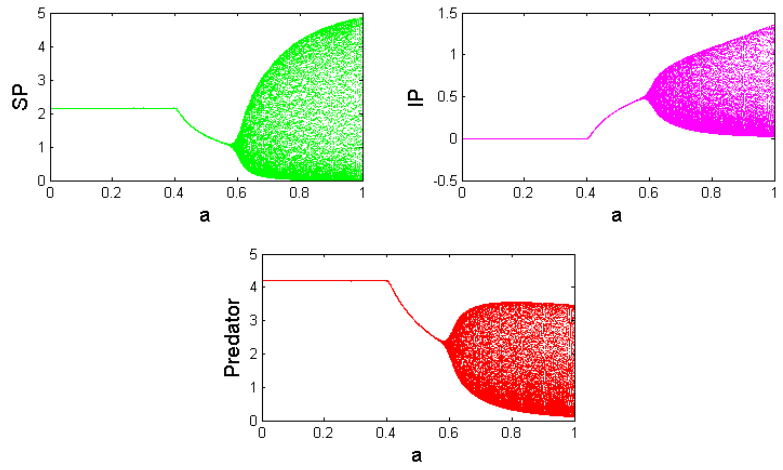

Fig 8: Bifurcation diagram of the model (1) for a in the range $0 \leq a \leq 1$ to SP, IP and Predator for the parameters given in Table 1.

The bifurcation diagrams generated for the successive of the population densities S, I, and P in the ranges $[0.0, 5.0], [0.0, 1.5]$, and $[0.0, 5.0]$, respectively as a function of effect of the presence of predator a in the range $0 \leq a \leq 1$ (see Fig 8.) and parameter values are given in the Table 1. It is observed that, when a increases the then all population densities are tends to form a limit cycle system.

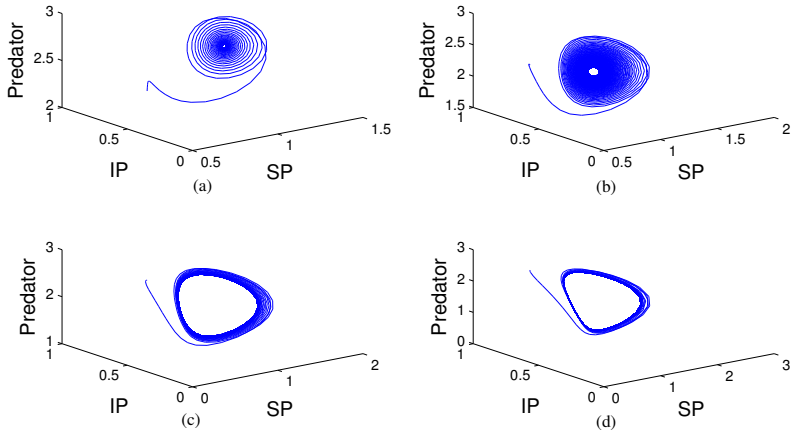

Fig 9: Phase portrait in 3D view for different value of α_1 and keeping other parameters and initial value from Table 1: For (a) α_1 =0.4, (b) α_1 = 0.5, (c) α_1 = 0.6 and (d) α_1 = 0.7.

The Phase portrait in (S, I, P)-view of the model system (1) at different values of α_1 is presented in the Fig 9. The stable focus in SIP-view of the model (1) at $\alpha_1 = 0.4$ be presented in the Fig 9(a). Similarly Fig 9(b), Fig 9(c) and Fig 9(d) represents the stable limit cycle in SIP-view for $\alpha_1 = 0.5$, $\alpha_1 = 0.6$ and $\alpha_1 = 0.7$, respectively.

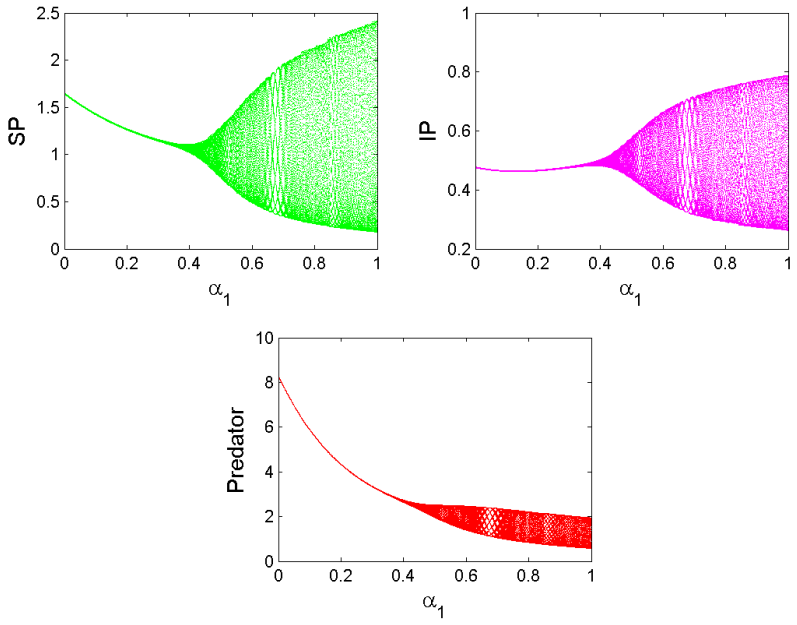

Fig 10: Bifurcation diagram of the model system (1), as a function of α_1 is plotted in the range $0 \leq \alpha_1 \leq 1$ for the parameters given in Table 1 with respect to Susceptible prey (SP), Infected prey (IP) and Predator.

Fig 10 represents the bifurcation diagrams generated for the successive of the population densities S, I, and P in the ranges $[0.0, 2.5], [0.0, 1.0]$, and $[0.0, 10.0]$ respectively as a function of predation rate on susceptible prey α_1 in the range $0 \leq \alpha_1 \leq 1$ and parameter values are given in the Table 1. It is observed that, when α_1 increases the then all of the population densities are tends to form a limit cycle system.

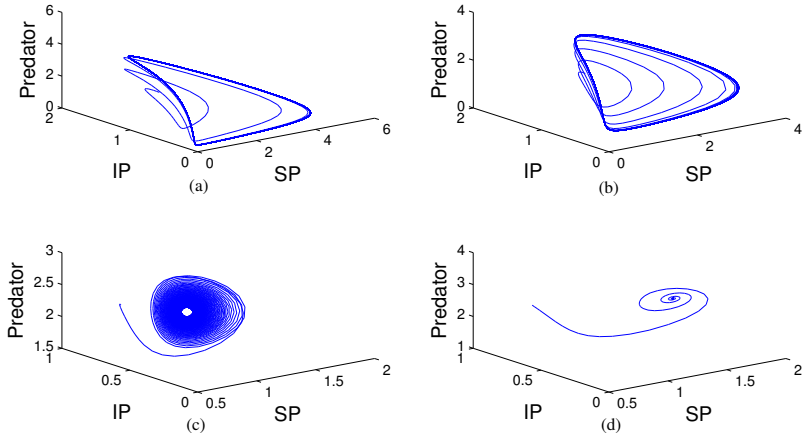

Fig 11: Phase portrait in 3D view for different value of β_1 and keeping other parameters and initial value from Table 1: For (a) $\beta_1 = 0.3$, (b) $\beta_1 = 0.4$, (c) $\beta_1 = 0.5$ and (d) $\beta_1 = 0.6$.

The Phase portrait in (S, I, P)-view of the model (1) at different values of β_1 is presented in the Fig 11.

The stable limit cycle in SIP-view of the model (1) at $\beta_1 = 0.3$, $\beta_1 = 0.4$ and $\beta_1 = 0.5$ are presented in the Fig 11(a), Fig 11(b) and Fig 11(c), respectively. Similarly Fig 11(d) represents the stable focus in SIP-view for $\beta_1 = 0.6$.

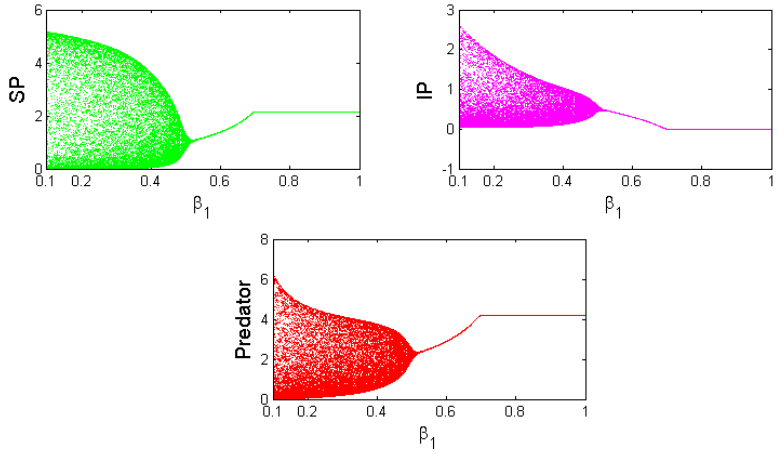

Fig 12: Bifurcation diagram of the model system (1), as a function of β_1 is plotted in the range $0.1 \leq \beta_1 \leq 1$ for the parameters given in Table 1 with respect to Susceptible prey (SP), Infected prey (IP) and Predator.

Fig 12 represents the bifurcation diagrams generated for the successive of the population densities S, I, and P in the ranges $[0.0, 6.0]$, $[0.0, 3.0]$, and $[0.0, 8.0]$, respectively as a function of transmission coefficient from infected prey to predator β_1 in the range $0.1 \leq \beta_1 \leq 1$ and other parameter values are given in the Table 1. It is observed that, when β_1 increases the then all of the population densities are tends to stable system.

8. Conclusion

In this manuscript, a prey-predator model is presented incorporating an infectious disease on prey, and it is transmitted only in the prey population by nonlinear disease transmission rate as a function of a predator. We also consider Cowley-Martin and Holling type I functional responses for susceptible and infected prey predation, respectively. The mode of disease spread follows a simple mass-action law.

Our theoretical investigation using a relatively simple eco-epidemiological model (1) demonstrates that a predator-dependent transmission rate may have an essential role in shaping prey-predator disease interactions. Firstly, a predator-dependent disease transmission rate can enhance the transmission of the disease (e.g. Fig 7 and Fig 8). Second, a predator-dependent disease transmission rate can promote the survival of a (specialist) predator (e.g. Fig 5,Fig 6,Fig 7 and Fig 8), which is otherwise impossible. Presence of predators creates a special environment with high contamination properties which can guarantee their own survival. Finally, the effects of a predator on the transmission rate will destabilise in eco-systems with high carrying capacity levels k .

Other very vital parameters are the predation parameters α_1 and β_1, which cannot be ignored in our presented system. We observed that the predation rate on healthy prey α_1 has saved the predator from extinction. It is observed that the system becomes disease-free by increasing the predation rate β_1 and the predation of infected. We have observed from the system that susceptible prey can be saved from extinction, and the disease does not propagate in healthy prey populations by predating infected prey.

This mathematical model method and the present paper's results may enhance the research methodology of pattern formation in prey-predator systems. To conclude on this point, that predator-dependent disease transmission is more appropriate to describe the contact process.

Further studies are necessary to analyze the behaviour of eco-epidemic models with more realistic features. Finally, the model of this present paper initiates the possibility of future work: (i) Some work should be done on recovering prey population's case, considering the healthy prey population, (ii) Alternative food sources of a predator can be an essential phenomenon, (iii) Some can consider the case when the contact of the prey population can infect the predator population, (iv) One can be using the concept of a stochastic model to express the eco-system, (v) Model with time delay is an important characteristic to be considered.

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