# Article

# Dynamical behavior of an eco-epidemiological model incorporating Holling type-II functional response with prey refuge and constant prey harvesting

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# Abstract

In the present investigation, we examine the consequences of a predator-prey model that includes a constant harvesting technique in a population of susceptible prey. A particular kind of flipping functional approach is present in our proposed prey-predator system; in this response, the predator consumes on susceptible and sick prey, however it shifts its attention to a new sort of prey when its supply of that kind of prey decreases. By employing boundedness, positivity, equilibrium analysis and stability analysis, the essential mathematical characteristics of the model are explored. Attention is directed on the prey refuge in further explorations of the Hopf bifurcation close to the coexistence equilibrium point. This paper's unique contribution is that it examines the dynamics of predator-prey systems from an eco-epidemiological perspective while simultaneously considering the impacts of prey refuge and constant-rate harvesting. To ascertain the critical values of the bifurcation parameters, if present, and to validate the primary findings, numerical simulations are executed. Our numerical simulations reveal that the presence of prey refuge increases the severity of sickness, which is how the three-species eco-epidemiological system creates chaos. However, we find that both the prey refuge and harvesting can manage the resulting chaotic dynamics.

**Keywords** eco-epidemic model; susceptible prey; infected prey; predator; Hopf bifurcation; harvesting; prey refuge.

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# **1** Introduction

There is consensus that infections affect both natural systems and population density. In order to better understand the eco-system, it is crucial to study how epidemiological factors affect the dynamics of prey-predator interactions. The area of study on mathematical modelling of epidemics has been greatly influenced by the pioneering work of Kermack and McKendrick (1927) on SIRS systems. Anderson and May (1981) were the first to investigate the invasion, persistence, and spread of infectious diseases by using an

eco-epidemiological model that simulates the relationship between prey and predators. Models for epidemics and eco-epidemiology have been developed and studied by several researchers in the last few years (Wang et al., 2016; Gao et al., 2018; Meskaf et al., 2020; Shaikh et al., 2020; Maji et al., 2018; Maji et al., 2019; Mondal et al., 2019; Yang et al., 2018; Saha et al., 2018; Saha et al., 2019; Mondal et al., 2019; Mondal et al., 2020). Haque and Chattopadhyay (2007) studied a prey-predator system in which the prey was infected with a contagious sickness. In the discussion of a Leslie-Gower predator-prey model (Ashok Mondal et al., 2019) included a disease-developed predator population.

A novel component of prey-predator models is research on prey concealing behaviour. This study has important implications for the dynamics of prey-predator interactions, which is of great significance in theoretical ecology and applied mathematics. For the most part, in nature, prey groups may find refuge from their predators in certain places. Both mitigating the risk of extinction as a result of predation and reducing the oscillations of prey predators are two important functions that these refugia often provide. Consequently, they might be a significant tool for enhancing biodiversity in ecosystems while simultaneously regulating population growth, biomass, and productivity. Much research has focused on how predator-prey interactions are affected by prey refuges. Predators and prey often engage in interactions that include geographical refuge that helps to protect prey from predators and reduces the chances of extinction due to predation (Kar et al., 2005; Huang et al., 2006). A model that displayed divergent oscillations without a refuge was shown by Hassel (1974) to exhibit stable equilibrium behavior after adding a huge refuge. Refuge seems to have a stabilising influence on predator-prey interactions, according to mathematical models and observations.

Harvesting is another emerging field of study in ecology. More and more evidence suggests that harvesting significantly affects the dynamic development of a species' population. Constant rate harvesting has been the subject of several studies (Mondal et al., 2019a; Chaudhuri, 1986; Chaudhuri et al., 1996; Goh et al., 1974; Ianelli et al., 2003; Martin et al., 2001; Mesterton-Gibbons, 1988; Mesterton-Gibbons, 1996; Xiao et al., 2005; Venkataiah et al., 2024). It is difficult to manage renewable resources, and it is much more difficult to build reliable mathematical models that account for the impact of harvesting on populations of vegetables or animals. This is the case because establishing an ideal model would need accounting for a wide range of variables that affect the survival of the harvested population as well as the cost-benefit criteria. It would be necessary to take into account the size, growth rate, carrying capacity, predators, rivals, harvesting cost, and selling price of each population along with their associated factors. The impact of harvesting on various prey-predator models has been extensively studied. To explore the dynamical behaviour of predators and prev Brauer et al. (1979, 1979a) specifically set up a predator-prev system that took into account the ongoing harvesting of prev populations. They also reported that harvesting reduces the region of asymptotic stability in the predator-prey plane. Leard et al. (2008) explored a predator-prey system that includes non-constant prey harvesting as a Michaelis-Menten type. Martin et al. (2001) states that in predator-prey systems including latency and constant-rate harvesting for the prey, the maximum sustainable yield (MSY) is determined by the environmental carrying capacity. With this background, we modeled a single predator-prey interaction mathematically using a Susceptible-Infected (SI) epidemic method to depict the prey's illness.

This paper's unique contribution is that it examines the dynamics of predator-prey systems from an eco-epidemiological perspective while simultaneously considering the impacts of prey refuge and constant-rate harvesting. Few scholars have thought about combining prey refuge and harvesting in an epidemic model, even though both have been explored independently in two-prey, one-predator systems. According to our findings, the system's stability may be modulated by adjusting the prey refuge, harvesting rate, and various competition coefficients.

The structure of this paper is as follows: Section 2 provides an overview of mathematical model

development. Section 3 elaborates on the bifurcation investigation of the temporal system (3), the presence and boundedness of solutions, and the local and global stability analysis of the biologically possible equilibrium points. The study of the spatiotemporal system is covered in Section 4; local stability of the system's equilibrium points (2) and persistence qualities of solutions are covered in Section 5. Section 6 presents the findings of the numerical simulation. Section 7 concludes with some findings.

# **2** Mathematical Model Formulation

The suggested system includes two categories of species: prey and predators. To formulate the model, we make the assumption that there are two compartments within the overall prey population: susceptible prey and infected prey, with population density denoted by X(t) and Y(t), respectively, and predator population density denoted by Z(t). The absence of infection and predator causes the prey's growth rate to follow a logistic distribution with a constant r. Assuming the sickness is incurable, we may say that infected prey will

either perish at a disease-induced mortality rate of  $m_1$  or be eliminated from the population before they can

reproduce. This is why we have chosen not to include word related to birth in the infectious class. Holling type II (prey dependent) functional response  $\theta XY/L_1 + X$  allows the infection to propagate among healthy prey when a predator is not present. The density of the predator population, indicated as Z(t) is considered a specialist variable in this context as the predator's development pace is solely reliant on the amount of susceptible and infected prey. This study also gives the refuge effect in prey populations the consideration it deserves. The impact on the prey's harvest has been carefully considered.

$$\frac{dX}{dT} = rX\left(1 - \frac{X}{k}\right) - \frac{\theta XY}{L_1 + X} - \frac{\delta_1(1 - m)XZ}{L_2 + (1 - m)X} - PX , 
\frac{dY}{dT} = \frac{\theta XY}{L_1 + X} - \frac{\delta_2 YZ}{L_2 + Y} - m_1 Y , 
\frac{dZ}{dT} = \frac{\gamma_1 \delta_1(1 - m)XZ}{L_2 + (1 - m)X} + \frac{\gamma_2 \delta_2 YZ}{L_2 + Y} - m_2 Z ,$$
(1)

with initial conditions X(0) > 0, Y(0) > 0, Z(0) > 0. All the parameters of the system are positive. The variables' biological descriptions have been provided in Table 1. To achieve dimensionless in the model, we now employ  $x = \frac{X}{k}, y = \frac{Y}{k}, z = \frac{Z}{k}, t = rT$ .

Using these substitutions in our epidemiological model we get

$$\frac{dx}{dt} = x(1-x-P_1) - \frac{\theta_1 xy}{A_1 + x} - \frac{B_1(1-m)xz}{A_2 + (1-m)x},$$

$$\frac{dy}{dt} = \frac{\theta_1 xy}{A_1 + x} - \frac{B_2 yz}{A_2 + y} - M_1 y,$$

$$\frac{dz}{dt} = \frac{C_1(1-m)xz}{A_2 + (1-m)x} + \frac{C_2 yz}{A_2 + y} - M_2 z,$$
where  $P_1 = \frac{P}{r}, \theta_1 = \frac{\theta}{r}, A_1 = \frac{L_1}{k}, A_2 = \frac{L_2}{k}, B_1 = \frac{\delta_1}{r}, B_2 = \frac{\delta_2}{r}, M_1 = \frac{m_1}{r}, M_2 = \frac{m_2}{r}, C_1 = \gamma_1 B_1, C_2 = \gamma_2 B_2$ 

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having as x(0) > 0, y(0) > 0, z(0) > 0 and 0 < m < 1 starting conditions.

# **3** Mathematical Analyses

In this part the eco-epidemiological paradigm (2) is mathematically explored. The following theory guarantees the basic system's validity and constraint.

**Theorem 1.** Every solution in system (2) that begins within  $\Re^3_+$  stays positive indefinitely.

**Proof:** Using the first equation in (2), we get

$$\frac{dx}{dt} = x \left[ (1 - x - P_1) - \frac{\theta_1 y}{A_1 + x} - \frac{B_1 (1 - m) z}{A_2 + (1 - m) x} \right]$$

Thus, 
$$x(t) = x(0) \exp\left[\int_{0}^{t} \left\{ \left(1 - x(\gamma) - P_{1}\right) - \frac{\theta_{1}y(\gamma)}{A_{1} + x(\gamma)} - \frac{B_{1}(1 - m)z(\gamma)}{A_{2} + (1 - m)x(\gamma)} \right\} d\gamma \right] > 0 \text{ for } x(0) > 0$$

Similarly,

$$y(t) = y(0) \exp\left[\int_{0}^{t} \left\{\frac{\theta_{1}x(\gamma)}{A_{1} + x(\gamma)} - \frac{B_{2}z(\gamma)}{A_{2} + y(\gamma)} - M_{1}\right\} d\gamma\right] > 0 \text{ for } y(0) > 0$$
$$z(t) = z(0) \exp\left[\int_{0}^{t} \left\{\frac{C_{1}(1 - m)x(\gamma)}{A_{2} + (1 - m)x(\gamma)} + \frac{C_{2}y(\gamma)}{A_{2} + y(\gamma)} - M_{2}\right\} d\gamma\right] > 0 \text{ for } z(0) > 0$$

#### Table 1 Definitions of model parameters.

Parameters	Description in a biological sense
r	Increase pace of vulnerable prey
k	Susceptible prey carrying capacity
$\theta$	Amount of infection
$\delta_1,\delta_2$	Maximum predation rates of $X, Y$
$L_{1}, L_{2}$	Half stabilization and saturation constant respectively
m	Impact of the susceptible population's shelter
$m_1, m_2$	The mortality rate of diseased prey and predator
Р	Harvesting amount of vulnerable prey
$\gamma_1, \gamma_2$	Rates of predator attacks per prey with and without shelter correspondingly

**Theorem 2.** The solution to the system (2) that begins within  $\mathfrak{R}^3_+$  is bounded.

**Proof.** Consider  $\psi'(t) = \rho_1 x'(t) + \rho_2 y'(t) + \rho_3 z'(t)$ 

$$= \rho_{1} \left[ x \left( 1 - x - P_{1} \right) - \frac{\theta_{1} x y}{A_{1} + x} - \frac{B_{1} \left( 1 - m \right) x z}{A_{2} + \left( 1 - m \right) x} \right] + \rho_{2} \left[ \frac{\theta_{1} x y}{A_{1} + x} - \frac{B_{2} y z}{A_{2} + y} - M_{1} y \right] \\ + \rho_{3} \left[ \frac{C_{1} \left( 1 - m \right) x z}{A_{2} + \left( 1 - m \right) x} + \frac{C_{2} y z}{A_{2} + y} - M_{2} z \right] \right] \\ \leq \rho_{1} x + \rho_{2} \frac{\theta_{1} x y}{A_{1} + x} - \rho_{2} M_{1} y - \rho_{3} M_{2} z \\ \leq 2\rho_{1} x + \rho_{2} \frac{\theta_{1} x y}{A_{1} + x} - \rho_{1} x - \rho_{2} M_{1} y - \rho_{3} M_{2} z \\ \leq 2\rho_{1} + \rho_{2} \frac{\theta_{1}}{A_{1} + 1} - \wp \psi \quad \text{where} \quad \wp = \min \left\{ \rho_{1}, \rho_{2} M_{1}, \rho_{3} M_{2} \right\} \\ \psi'(t) + \wp \psi \leq 2\rho_{1} + \rho_{2} \frac{\theta_{1}}{A_{1} + 1} = \Delta \\ \Rightarrow \psi(t) \leq \frac{\Delta}{\wp} \left( 1 - e^{-\wp t} \right) + \psi \left( x(0), y(0), z(0) \right) e^{-\wp t} ast \rightarrow \infty, 0 < \psi(t) \leq \frac{\Delta}{\wp}$$

As a result, solutions for model (2) exist in the region

$$\Theta = \left\{ \left(x, y, z\right) : 0 < x\left(t\right) \le 1, 0 < y\left(t\right) \le 1, 0 < z\left(t\right) \le 1; 0 < \psi\left(t\right) \le \frac{\Delta}{\wp} \right\}.$$

#### **4 Dynamical Behaviour**

The stability of system (2) was explored in relation to its equilibrium points in this section. For system (2) we obtained three equilibrium points.

- 1. Trivial equilibrium point  $E_0(0,0,0)$
- 2. Auxiliary equilibrium point  $E_1(x^*, 0, 0)$ , where  $x^* = 1 P_1$ , which is exists if  $P_1 < 1$

3. Infection free equilibrium point  $E_2(x^*, 0, z^*)$ , where  $x^* = \frac{M_2 A_2}{(1-m)(C_1 - M_2)}$  and  $y^* = \frac{C_1 A_2 [(1-P_1)(1-m)(C_1 - M_2) - M_2 A_2]}{(1-P_1)(1-m)(C_1 - M_2)}$  which is exists if  $(1-P_1)(1-m)(C_1 - M_2)$ 

$$y^* = \frac{C_1 A_2 \lfloor (1 - P_1)(1 - m)(C_1 - M_2) - M_2 A_2 \rfloor}{B_1 (1 - m)^2 (C_1 - M_2)^2}, \text{ which is exists if } (1 - P_1)(1 - m)(C_1 - M_2) > M_2 A_2.$$

4. Positive equilibrium point  $E_3(x^*, y^*, z^*)$ , finding  $x^*, y^*, z^*$  explicitly in terms of parameters is more tedious and this we verified numerically for suitable parameter values.

By using the eigen values of the corresponding community matrix, one can ascertain the local stability conditions of the equilibrium points. The community matrix for system (1) can be expressed as follows:

$$J = \begin{bmatrix} l_{11} & l_{12} & l_{13} \\ l_{21} & l_{22} & l_{23} \\ l_{31} & l_{32} & l_{33} \end{bmatrix}$$
  
where  $l_{11} = 1 - 2x - P_1 - \frac{\theta_1 A_1 y}{(A_1 + x)^2} - \frac{B_1 (1 - m) A_2 z}{(A_2 + (1 - m) x)^2}, \quad l_{12} = -\frac{\theta_1 x}{A_1 + x}, \quad l_{13} = -\frac{B_1 (1 - m) x}{A_2 + (1 - m) x}$ 

$$l_{21} = \frac{\theta_1 A_1 y}{(A_1 + x)^2}, \quad l_{22} = \frac{\theta_1 x}{A_1 + x} - \frac{B_2 A_2 z}{(A_2 + y)^2} - M_1, \quad l_{23} = -\frac{B_2 y}{A_2 + y}$$
$$l_{31} = \frac{C_1 (1 - m) z A_2}{(A_2 + (1 - m) x)^2}, \quad l_{32} = \frac{C_2 A_2 z}{(A_2 + y)^2}, \quad l_{33} = \frac{C_1 (1 - m) x}{A_2 + (1 - m) x} + \frac{C_2 y}{A_2 + y} - M_2$$

**Theorem 3.** The trivial equilibrium point  $E_0(0,0,0)$  is stable if  $1 < P_1$ , otherwise it is a saddle point.

**Proof.** At trivial equilibrium point  $E_0(0,0,0)$ , the community matrix is defined as

$$J_{E_0} = \begin{bmatrix} 1 - P_1 & 0 & 0 \\ 0 & -M_1 & 0 \\ 0 & 0 & -M_2 \end{bmatrix}$$

Therefore, the eigen values are  $1 - P_1, -M_1, -M_2$ . Here two eigen values are negative and  $1 - P_1 < 0$  if  $1 < P_1$ .

**Theorem 4.** The equilibrium point  $E_1(x^*, 0, 0)$  is stable for  $P_1 < 1$ ,  $(\theta_1 - M_1)(1 - P_1) < M_1A_1$  and

 $(C - mC_1 + mM_2 - M_2)(1 - P_1) < M_2A_2$ , otherwise it is unstable.

**Proof:** At 
$$E_1(x^*, 0, 0) J_{E_1} = \begin{bmatrix} 1 - 2x - P_1 & -\frac{\theta_1 x}{A_1 + x} & -\frac{B_1(1 - m)x}{A_2 + (1 - m)x} \\ 0 & \frac{\theta_1 x}{A_1 + x} - M_1 & 0 \\ 0 & 0 & \frac{C_1(1 - m)x}{A_2 + (1 - m)x} - M_2 \end{bmatrix}$$
 (3)

Therefore, the eigen values are  $\lambda_1 = P_1 - 1$ ,  $\lambda_2 = \frac{\theta_1 (1 - P_1)}{A_1 + (1 - P_1)} - M_1$ ,  $\lambda_3 = \frac{C_1 (1 - m)(1 - P_1)}{A_2 + (1 - m)(1 - P_1)} - M_2$ . The above

eigen values are negative if conditions of Theorem4 are hold. Hence equilibrium point  $E_1(x^*, 0, 0)$  is stable otherwise it is unstable.

**Theorem 5.** The equilibrium point  $E_2(x^*, 0, z^*)$  is stable if  $P_1 < 1$ ,  $x_2^* < \frac{(B_2 z_2 + M_2 A_2)A_1}{A_2 \theta_1 - (B_2 z_2 + M_2 A_2)}$ , otherwise it is

unstable.

**Proof:** At  $E_2(x^*, 0, z^*)$ 

$$J_{E_{2}} = \begin{bmatrix} 1 - 2x - P_{1} - \frac{B_{1}(1-m)A_{2}z}{(A_{2} + (1-m)x)^{2}} & -\frac{\theta_{1}x}{A_{1} + x} & -\frac{B_{1}(1-m)x}{A_{2} + (1-m)x} \\ 0 & \frac{\theta_{1}x}{A_{1} + x} - \frac{B_{2}A_{2}z}{(A_{2} + y)^{2}} - M_{1} & 0 \\ \frac{C_{1}(1-m)zA_{2}}{(A_{2} + (1-m)x)^{2}} & \frac{C_{2}z}{A_{2}} & 0 \end{bmatrix}$$
(4)

So,  $\lambda_1 = \frac{\theta_1 x}{A_1 + x} - \frac{B_2 A_2 z}{(A_2 + y)^2} - M_1$  is one of the eigen values, while the remaining two are the roots of the

equation  $\lambda^2 - l_{11}\lambda - l_{13}l_{31} = 0$ . Thus, in case when  $l_{11} < 0$  and  $l_{13}l_{31} < 0$ , the two eigen values necessarily possess a negative real portion. But one eigen value is  $\lambda_1 < 0$  if condition mentioned in Theorem 3 hold. **Theorem 6.** The equilibrium point  $E_2(x^*, y^*, z^*)$  is locally asymptotically stable if  $D_1 > 0, D_3 > 0$  and  $\Delta > 0$ .

**Proof:** 

$$\operatorname{At} E_{3}(x^{*}, y^{*}, z^{*}) J_{E_{2}} = \begin{bmatrix} -x + \frac{\theta_{1}xy}{\left(A_{1} + x\right)^{2}} + \frac{B_{1}\left(1 - m\right)^{2}xz}{\left(A_{2} + \left(1 - m\right)x\right)^{2}} & -\frac{\theta_{1}x}{A_{1} + x} & -\frac{B_{1}\left(1 - m\right)x}{A_{2} + \left(1 - m\right)x} \\ & \frac{\theta_{1}A_{1}y}{\left(A_{1} + x\right)^{2}} & \frac{B_{2}yz}{\left(A_{2} + y\right)^{2}} & -\frac{B_{2}y}{\left(A_{2} + y\right)} \\ & \frac{C_{1}\left(1 - m\right)zA_{2}}{\left(A_{2} + \left(1 - m\right)x\right)^{2}} & \frac{C_{2}A_{2}z}{\left(A_{2} + y\right)^{2}} & 0 \end{bmatrix}$$
(5)

The laten equation of above community matrix is  $\lambda^3 + D_1\lambda^2 + D_2\lambda + D_3 = 0$ 

where  $D_1 = -(l_{11} + l_{22})$ ,  $D_2 = l_{11}l_{22} - l_{12}l_{21} - l_{13}l_{31} - l_{23}l_{32}$  and  $D_3 = l_{32}(l_{11}l_{23} - l_{13}l_{21}) + l_{31}(l_{13}l_{22} - l_{12}l_{23})$ . Consider  $\Delta = D_1D_2 - D_3$  and so by Routh-Hurwitz criterion all the roots of above latent equation have negative real parts if  $D_1 > 0$ ,  $D_3 > 0$  and  $\Delta > 0$ . Thus, the positive equilibrium point  $E_3$  is locally asymptotically stable.

# **5** Hopf Bifurcation At Positive Equilibrium Point $E_3$

In this subsection, we explore whether the system dynamics vary with respect to m through Hopf bifurcation analysis.

**Theorem 7.** When the prey refuge m exceeds a threshold value, the dynamical system experiences a Hopf bifurcation over the interior equilibrium  $E_3$ . Hopf bifurcation develops at  $m = m^*$  if the following two criteria

are met concurrently  $H_1(m^*) > 0, H_3(m^*) > 0$  and  $H_1(m^*) H_2(m^*) = H_3(m^*)$  with

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$$H_{1}(m^{*})H_{2}'(m^{*}) \neq H_{3}'(m^{*}) + H_{1}'(m^{*})H_{2}(m^{*}).$$

**Proof.** For  $m = m^*$  then the latent equation of the system (2) for  $E_3$  is expressed as

$$\left(\lambda^2 + \mathrm{H}_2\right)\left(\lambda + \mathrm{H}_1\right) = 0.$$

We can obtain latent values from above equation are  $\lambda = i\sqrt{H_2}$ ,  $\lambda = -i\sqrt{H_2}$ ,  $\lambda = -H_1$ . Of these three latent values, two that are entirely imaginary and one that is entirely negative. Taking *m* into account as bifurcation parameter allows us to alter the roots in the following way:

$$\lambda_1(m) = \Upsilon_1(m) + i\Upsilon_2(m), \quad \lambda_2(m) = \Upsilon_1(m) - i\Upsilon_2(m), \quad \lambda_3(m) = -\mathrm{H}_1.$$
(6)

We can solve above equation by substituting  $\lambda_1(m) = \Upsilon_1(m) + i\Upsilon_2(m)$  first, differentiate it with respect to *m*,

and then separating the real and imaginary parts.

Then, we have,

$$G_{1}(m)\Upsilon_{1}'(m) - G_{2}(m)\Upsilon_{2}'(m) + G_{3} = 0$$

$$G_{2}(m)\Upsilon_{1}'(m) - G_{1}(m)\Upsilon_{2}'(m) + G_{4} = 0$$
(7)

Where 
$$G_1(m) = 3(\Upsilon_1^2 - \Upsilon_2^2) + 2H_1\Upsilon_1 + H_2$$
,  $G_2(m) = 6\Upsilon_1\Upsilon_2 + 2H_1\Upsilon_2$ ,  $G_3(m) = H_1'(\Upsilon_1^2 - \Upsilon_2^2) + H_2'\Upsilon_1 + H_3'$ ,

$$G_{4}(m) = 2H_{1}'\Upsilon_{1}\Upsilon_{2} + H_{2}'\Upsilon_{2}$$
Observing that  $\Upsilon_{1}(m^{*}) = 0, \Upsilon_{2}(m^{*}) = \sqrt{H_{2}(m^{*})}$ 
we get
$$G_{1}(m^{*}) = -2H_{2}(m^{*}), G_{2}(m^{*}) = 2H_{1}(m^{*})\sqrt{H_{2}(m^{*})}, G_{3}(m^{*}) = H_{3}'(m^{*}) - H_{1}'(m^{*})H_{2}(m^{*})$$
and
$$G_{4}(m^{*}) = H_{2}'(m^{*})\sqrt{H_{2}(m^{*})}.$$
Now
$$d_{4}(D_{2} + 2(m^{*}))G_{4}(m^{*}) + G_{1}(m^{*})G_{3}(m^{*})$$
(8)

$$\frac{d}{dm} (\operatorname{Re} \lambda(m)) G_{1}(m^{*}) \Big|_{m=m^{*}} = \frac{2(-)^{-4} (-)^{-1} (-)^{-3} ($$

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Hence, when  $m = m^*$ , the system shows Hopf bifurcation.

**Remarks:** Likewise,  $\theta_1$  may also be considered a bifurcating parameter.

# **6** Numerical Simulations

No analytical study is ever considered complete without some kind of numerical confirmation of the findings. This section presents a computer simulation of some of the system's solutions. In addition to verifying our analytical conclusions, these numerical solutions are quite useful in practice. The study focuses on critical characteristics such as prey refuge rate m and disease transmission rate  $\theta_1$ . We analyses System (2) using various values of the aforementioned parameters.

**Example 1.** Consider the set of parameter values as  $P_1 = 0.1095; \theta_1 = 0.5405; A_1 = 0.7789;$ 

 $A_2 = 1.3684; B_1 = 0.5716; B_2 = 1.2784; M_1 = 0.0676; M_2 = 0.2365; C_1 = 0.0646; C_2 = 0.1445$ . The requirement of

Theorem 4 is satisfied by this parameter selection; therefore,  $x^* = 0.672$ ,  $y^* = 0.3221$ ,  $z^* = 0.2408$ . We begin by analyzing how the system (2) dynamically responds to changes in the prey refuge amount m. If we take m = 0.2 system exhibits stale solution (Fig.1a) and increasing m value to 0.8 then the system exhibits unstable solution (Fig. 1b). The bifurcation illustrations for the prey-predator population in relation to m for the interval [0.1, 0.9] is shown in Fig. 2. The complicated dynamical characteristic in our suggested model (2), which extends from the stable focus to chaos, can be observed in the bifurcation diagram in relation to Fig. 2. The system then experiences a Hopf bifurcation with m acting as the bifurcation parameter, as deduced from Theorem 7. System (2) experiences a Hopf bifurcation of periodic solution around  $m = m^* = 0.25$ , as can be easily seen if we take m = 0.25.



Fig. 1a Time trajectories and phase portrait of the system (2) for m = 0.2



Fig. 1b Time trajectories and phase portrait of the system (2) for m = 0.8

**Example 2.** Now we introduce the rate of infection  $\theta_1$  and taking the same parameter values as Example1. If we take  $\theta_1 = 0.18270$  system exhibits stale solution (Fig. 3a) and increasing  $\theta_1$  value to 0.32270 then the system exhibits unstable solution (Fig. 3b). For the interval [0.09, 0.5], Fig. 4 shows the bifurcation diagram illustrating the prey-predator population with respect to  $\theta_1$ . The complicated dynamical characteristic in our suggested model (2) is shown in the bifurcation diagram with respect to m in Fig. 4, which goes from the stable focus to chaos. Then, under the influence of  $\theta_1$ , the system experiences a Hopf bifurcation. The Hopf bifurcation of the periodic solution in system (2) happens at  $\theta_1 = \theta_1^* = 0.19$  if we set  $\theta_1 = 0.19$ .





Fig. 2 The bifurcation illustration of System (2) in relation to m, while the remaining values remain the same as shown in Fig. 1.



Fig. 3a Time trajectories and phase portrait of the system (2) for  $\theta_1 = 0.18270$ 

#### 7 Discussion

Our study differs from previous ones in that it includes prey infection, which has a nonlinear prevalence level, an integrated functional response for vulnerable and contaminated prey populations, and improved Leslie-Gower predator dynamics. The system's dynamics are enhanced and the system becomes more realistic compared to previous models with the inclusion of these extra ecological components.

Using an eco-epidemiological model including three species, this study examines how the refuge effect affects the prey population. When the prey shelter influence is added to the prey population, the eco-epidemiological model's dynamical behaviour changes significantly. Our numerical simulation validates the stabilising impact on the prey hideout. By generating bifurcation diagrams, we were able to confirm it.

More importantly, the infection rate ( $\theta_1$ ) is also critical. A rising amount of  $\theta_1$  may transform a stable system

into an oscillating one via Hopf bifurcation; the numerical simulation shows that  $\theta_1$  has both stabilising and

The system's complicated and rich dynamics are shown by both analytical and numerical simulation

findings. In future studies, environmental factors like extra food, delay, Allee effect (Venkataiah et al., 2024a) and a development of territorial and spatiotemporal structure may be combined with vertical and horizontal spread of diseases to the predator population.



Fig. 3b Time trajectories and phase portrait of the system (2) for  $\theta_1 = 0.32270$ 



Fig. 4 The bifurcation illustration of System (2) in relation to  $\theta_1$ , while the remaining values remain the same as shown in Fig.1

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