Disease Control Through Harvesting - Conclusion drawn from a mathematical study of a Predator-Prey model with disease in both the population

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Abstract. Disease control by managers is a crucial response to emerging epidemics and in the context of global change, emerging risks associated with parasites, invasive species and infectious diseases are an important issue especially for developing countries. Our objective is to provide a mathematical framework to study the response of a predator-prey model to a disease in both the populations and harvesting of each species. We have worked out the conditions for local stability of the equilibrium points as well as persistence of the system. We have derived the ecological and the disease basic reproduction numbers. These enable us to determine the community structure of the

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system. Harvesting may play a crucial role in a host-parasite system and reasonable harvesting can remove parasite burden from their host. Our numerical results reveal that the reasonable harvesting prevents the oscillations of the species. We conclude that harvest can be an effective strategy for controlling the spread of disease.

**Key words:** Disease in both populations, limit cycles, reproduction numbers, harvesting, permanence

1 Introduction

Disease in ecological species is an important area and many investigator are now paying more interest to study such dynamics both from experimental and theoretical perspective. Due to ethical issues, it is not possible to work with ecological species, theoretical observations play an important tool in such cases.

Theoretical studies on prey population only (for example, (1; 2; 3; 4; 5)) or predation population only are plenty, but the study where both the population affected by parasites are limited.

Exploitation of biological resources and harvesting of the species is a common practice in fishery, forestry, agriculture and wildlife management. To the best of our knowledge the mathematical model on this area was first introduced by (6). Modifying Clark’s model (7) studied combined harvesting and considered the perspectives of bio-economics as well as dynamics optimization of a two species fishery. Harvesting or constant quota of harvesting has been studied by many researchers (for examples, see (6; 8; 9; 10; 11)) in predator-prey models.

Control of disease is one of the major challenge. Harvesting is a common strategy for controlling the disease. The disease which spread in a very short span of time and the infection rate is very high, induction of culling as a harvesting policy is most appropriate. A lot of theoretical studies have already been done in this aspect.

Before presenting the aim and objective of the present investigation, we like to provide a short reviewed on such studies. The problem of nonselective harvesting of a predator-prey fishery with infection on prey population has been studied by (12). (13) proposed and analyzed a mathematical model of a harvested predator-prey system with infection on the prey population. They arrived at a conclusion that harvesting of the infected prey population prevents the limit cycle oscillations and may be used as a biological control for the persistence of the infected predator-prey dynamics. (14) considered an eco-epidemiological model with disease in the prey population where the prey population is subjected to harvesting. (15) proposed and analyzed eco-epidemiological model with harvesting in the predator population, they observed quasi-periodic and chaotic behavior of the system. (16) studied an eco-epidemiological model with harvesting in both the susceptible and infected prey species. They concluded that reasonable harvesting can remove a parasite from their host. Their study also indicated that impulsive harvesting can control the cyclic behaviour of the system population leading to the persistence of all species and obtain disease-free stable equilibrium. (17) studied a ratio-dependent eco-epidemiological model where the infected prey population is subjected to harvesting and they derived biological feasible conditions for non-existence of periodic solutions around the interior equilibrium. Recently (18) analyzed a predator-prey model with prey harvesting where the
disease is spreading in both the populations. They derived the conditions for which the disease free equilibrium state of the system is stable.

In general the disease spread from the infected prey to the susceptible prey and consequently to the predator population via predation related activities. Theoretical studies on predator-prey population where disease is spreading on both the species are of great importance (2; 19; 20; 21). Unfortunately such studies are very limited, as far our knowledge goes nobody has explicitly put harvesting term in a predator-prey model with disease in both the populations. We believe that such study can provide some useful observations which may be used in both fishery and conservation biology.

The paper is organized as follows. In the section (2), we outline the basic mathematical model. Dynamics of the submodels are presented in the section (3). We study the dynamics of the full model in section (4). We perform an extensive numerical simulation in section (5). The article ends with a conclusion in section (6).

2 Development of the model

We make the following assumptions to formulate the mathematical model:

A1: We consider a predator-prey system where the total prey and predator population densities are denoted by \(N\) and \(Z\), respectively. It is assumed that both the prey and the predator are susceptible to some transmissible disease (like viral disease) and in the presence of disease, the prey and the predator populations are divided into two classes-(i) susceptible prey \((x)\) and infected prey \((y)\); (ii) susceptible predator \((z_1)\) and infected predator \((z_2)\).

A2: We assume that the susceptible prey \((x)\) is capable of reproducing with logistic law having carrying capacity \(K\) and intrinsic birth rate \(r\).

\[
\frac{dx}{dt} = rx \left(1 - \frac{x}{K}\right).
\]

A3: We consider that disease spreads among both the prey and the predator species. The susceptible prey and the susceptible predator become infected due to attack of many viruses or parasite respectively. The contact process is admittedly debatable. Some researchers have argued that a proportional mixing rate is more appropriate than that of simple mass action. But data of Greenwood experiment suggests that there is no change of qualitative properties upon the contact process whether it follows the law of mass action or proportional mixing rate (22). Let \(\lambda\) be the rate of the force of infection among the prey population and \(\beta\) be the rate of the force of infection among the predator population.

A4: It is assumed that the predator population predates both the prey population. Holling type-II functional form is considered for the predation of the susceptible prey and Holling type-I (mass action form) is taken for the predation of the infected prey, since infected prey population is more vulnerable than susceptible prey and they are easier to catch.

A5: It is also assumed that each species is harvested with a constant rate.

From the above assumptions we can write down the following set of ordinary differential equations:
\[
\begin{align*}
\frac{dx}{dt} &= rx \left(1 - \frac{x}{K}\right) - \lambda xy - \frac{\alpha x (z_1 + z_2)}{1 + ax} - h_1x, \\
\frac{dy}{dt} &= \lambda xy - \delta y(z_1 + z_2) - \mu y - h_2y, \\
\frac{dz_1}{dt} &= e_1 \alpha z_1 y + e_2 \delta z_1 y - \gamma z_1 - \beta z_1 z_2 - h_3z_1, \\
\frac{dz_2}{dt} &= e_3 \alpha z_2 y + e_4 \delta z_2 y - \psi z_2 + \beta z_1 z_2 - h_4z_2.
\end{align*}
\]

System (2.1) has to be analyzed with the following initial conditions:

\[ x(0) > 0, \quad y(0) > 0, \quad z_1(0) > 0, \quad z_2(0) > 0. \]

<table>
<thead>
<tr>
<th>Variables/Parameters</th>
<th>Biological meaning</th>
</tr>
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<tbody>
<tr>
<td>(x)</td>
<td>Density of susceptible prey</td>
</tr>
<tr>
<td>(y)</td>
<td>Density of infected prey</td>
</tr>
<tr>
<td>(z_1)</td>
<td>Density of susceptible predator</td>
</tr>
<tr>
<td>(z_2)</td>
<td>Density of infected predator</td>
</tr>
<tr>
<td>(r)</td>
<td>Birth rate of susceptible prey</td>
</tr>
<tr>
<td>(K)</td>
<td>Environmental carrying capacity</td>
</tr>
<tr>
<td>(\lambda)</td>
<td>Rate of infection in prey population</td>
</tr>
<tr>
<td>(\beta)</td>
<td>Rate of infection in predator population</td>
</tr>
<tr>
<td>(\alpha)</td>
<td>Attack rate of predator on susceptible prey</td>
</tr>
<tr>
<td>(\delta)</td>
<td>Attack rate of predator on infected prey</td>
</tr>
<tr>
<td>(a)</td>
<td>Half saturation constant</td>
</tr>
<tr>
<td>(\mu)</td>
<td>Death rate of infected prey</td>
</tr>
<tr>
<td>(\gamma)</td>
<td>Death rate of susceptible predator</td>
</tr>
<tr>
<td>(\psi)</td>
<td>Death rate of infected predator</td>
</tr>
<tr>
<td>(e_1)</td>
<td>Conversion efficiency on susceptible prey of susceptible predator</td>
</tr>
<tr>
<td>(e_2)</td>
<td>Conversion efficiency on infected prey of susceptible predator</td>
</tr>
<tr>
<td>(e_3)</td>
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</tr>
<tr>
<td>(e_4)</td>
<td>Conversion efficiency on infected prey of infected predator</td>
</tr>
<tr>
<td>(h_1)</td>
<td>Harvesting effort for susceptible prey</td>
</tr>
<tr>
<td>(h_2)</td>
<td>Harvesting effort for infected prey</td>
</tr>
<tr>
<td>(h_3)</td>
<td>Harvesting effort for susceptible predator</td>
</tr>
<tr>
<td>(h_4)</td>
<td>Harvesting effort for infected predator</td>
</tr>
</tbody>
</table>

**Table 1** Variables and parameters used in Model (2.1)

The model description schematic diagram is presented in Figure (1) and the variables and parameters used in model (2.1) is presented in the Table (1).
Fig. 1  Schematic diagram of a prey-predator model with disease and harvesting in both the populations, see presentation of the model in (2.1).

3 Dynamics of submodels

In order to understand the full dynamics of (2.1), we should have a complete picture of the dynamics of the following four submodels.

1. The SI-model in the absence of predation in (2.1) is presented as (Susceptible Prey-Infected Prey model)

\[
\begin{align*}
\frac{dx}{dt} &= rx\left(1 - \frac{x}{h_1}\right) - \lambda xy - h_1 x, \\
\frac{dy}{dt} &= \lambda xy - \mu y - h_2 y. 
\end{align*}
\] (3.1)

For convenience, we introduce a basic reproduction number \( R_{00} \) defined by

\[ R_{00} = \frac{r}{h_1}. \]

Here \( r \) is the intrinsic growth rate of the susceptible prey and \( h_1 \) is the harvesting rate of the susceptible prey. So, \( \frac{1}{h_1} \) is considered as average lifespan of the susceptible prey and \( R_{00} \) is treated as mean numbers of the newborn susceptible preys which can be interpreted as the ecological basic reproduction number. \( R_{00} \) implies that all species go to extinction and consequently all species extinction state will be stable. We note also that this term, first formulated and explained by (23), is the average number of the prey converted to the predator biomass in a course of the predator’s life span (3).

We also define another basic reproduction numbers \( R_{01} \) given by

\[ R_{01} = \frac{\lambda x_1}{\mu + h_2}. \]

Here \( \lambda x_1 \) is the infection rate of a new infective prey appearing in a totally susceptible prey population and \( \frac{1}{\mu + h_2} \) is the duration of infectivity of an infective prey, product of which is the disease basic reproduction number at \( x^e = x_1 = \frac{r(1 - \frac{1}{h_1})}{\mu} \). \( R_{01} \leq 1 \) implies that disease will be eradicated from the prey population.
2. The PP-model in the absence of the disease in (2.1) is presented as (Susceptible Prey-Susceptible Predator model)

\[
\frac{dx}{dt} = rx \left(1 - \frac{x}{K}\right) - \frac{axz}{1+ax} - h_1x, \\
\frac{dz}{dt} = e\frac{axz}{1+ax} - \gamma_1 - h_3z.
\]  

(3.2)

For convenience, we introduce a demographic reproduction number for susceptible predator \(R_{02}\) defined by

\[
R_{02} = \frac{e_1ax_1}{(\gamma + h_3)(1 + ax_1)}
\]

where \(e_1ax_1\) is the birth rate of the susceptible predator due to consumption of the susceptible prey and \(\gamma + h_3\) is the death rate of the susceptible predator due to harvesting and natural death death. \(R_{02} < 1\) implies that the susceptible predator population will be washed out.

3. The SIP-model in the absence of the disease in predator in (2.1) is presented as (Susceptible Prey-Infected Prey-Susceptible Predator model)

\[
\frac{dx}{dt} = rx \left(1 - \frac{x}{K}\right) - \lambda xy - \frac{axz}{1+ax} - h_1x, \\
\frac{dy}{dt} = \lambda xy - \delta yz_1 - \mu y - h_2y, \\
\frac{dz_1}{dt} = e\frac{axz}{1+ax} + e_2\delta y_1 - \gamma_1 - h_3z_1.
\]  

(3.3)

For convenience, we introduce a demographic reproduction number for susceptible predator \(R_{03}\) defined by

\[
R_{03} = \frac{1}{\gamma + h_3} \left( \frac{e_1ax_1}{1 + ax_1} + e_2\delta y_1\right)
\]

where \(e_1ax_1\) is the birth rate of the susceptible predator due to consumption of the both preys at the equilibrium \((x^{SI}, y^{SI}, 0)\) and \(\gamma + h_3\) is the removal rate of the susceptible predator at that point. \(R_{03} < 1\) implies that the susceptible predator will be washed out.

Again, we introduce a disease basic reproduction number of an infected prey when entering a disease-free environment where the predator and prey coexist.

\[
R_{04} = \frac{\lambda x_{iP}^{PP}}{\mu + h_2 + \delta y_{iP}^{PP}}
\]

where \(\lambda x_{iP}^{PP}\) is the infection rate of an infective prey and \(\mu + h_2 + \delta y_{iP}^{PP}\) is the removal rate of the infection prey at the equilibrium \((x^{PP}, y_{iP}^{PP})\). \(R_{04}\) implies that the disease will be wiped out from the prey species.

4. The PSI-model in the absence of the disease in prey in (2.1) is presented as (Susceptible Prey-Susceptible Predator-Infected Predator model)

\[
\frac{dx}{dt} = rx \left(1 - \frac{x}{K}\right) - \frac{axz}{1+ax} - h_1x, \\
\frac{dx_1}{dt} = e\frac{axz}{1+ax} - \gamma_1 - \beta_1z_2 - h_3z_1, \\
\frac{dz}{dt} = e\frac{axz}{1+ax} - \psi z_2 + \beta_1z_2 - h_4z_2.
\]  

(3.4)

Here we introduce a demographic reproduction number \(R_{05}\) for the infected predator at the equilibrium where only susceptible prey exists as a locally stable equilibrium in this case.

\[
R_{05} = \frac{e_1ax_1}{(1 + ax_1)(\psi + h_4)}
\]
where $\frac{e_1\alpha}{1+\alpha^2}\gamma$ is the birth rate of the infected predator by consuming susceptible prey and $\psi + h_4$ is the removal rate due to natural death and harvesting. Again we define the basic reproduction number $R_{00}$ for the infected predator at the equilibrium where susceptible prey and susceptible predator coexists as a locally stable equilibrium.

$$R_{00} = \frac{1}{\psi + h_4} \left( \frac{e_1\alpha}{1+\alpha^2}\gamma + \beta_0 \right)$$

where $\frac{e_1\alpha}{1+\alpha^2}\gamma + \beta_0$ is the net growth of infected predator due to consumption of susceptible prey and transmission of disease in susceptible predator. $R_{00} < 1$ implies that the disease will be eradicated from the prey population. Now the local stability of the equilibrium where susceptible prey and infected predator coexists can be determined by the basic reproduction number $R_{01}$, which is given by

$$R_{01} = \frac{e_1\alpha}{1+\alpha^2}\gamma \frac{\psi + h_3 + \beta_0}{(\gamma + h_3 + \beta_0)^2}$$

where $\frac{e_1\alpha}{1+\alpha^2}\gamma$ is the growth rate of the newborn susceptible predator and $\gamma + h_3 + \beta_0$ is the removal rate of the predators at that equilibrium. Hence $R_{01}$ gives the ecological basic reproduction number of the predator population when disease is endemic in the predator population. $R_{01} < 1$ implies that susceptible predator goes to extinction.

### 3.1 Equilibria and local stability

It is easy to check that both submodels (3.1) and (3.2) have $(0,0)$ and $(x_I,0) = \left( \frac{K(\gamma-h_1)}{r}, 0 \right)$ as their boundary equilibria.

For convenience, for Model (3.1), we denote

$$E_0^S = (0,0), E_1^S = (x_I,0) \text{ and } E_1^S = (x^S_i, y^S_i) = \left( \frac{\mu + h_2}{\lambda}, \frac{r(K\lambda - \mu - h_2) - h_1K\lambda}{K\lambda} \right)$$

while for Model (3.2), we denote

$$E_0^{PP} = (0,0), E_1^{PP} = (x_I,0) \text{ and } E_1^{PP} = (x^{PP}_I, y^{PP}_I) = \left( \frac{\gamma + h_4}{e_1\alpha - a(\gamma + h_3)}, \frac{r(K - \theta_I) - Kh_1}{(1 + \alpha^2)K} \right)$$

where $E_1^{SI}$ and $E_1^{PP}$ are interior equilibria for the submodel (3.1) and (3.2), respectively, provided their existence.

**Proposition 1.** [Local stability of equilibria for submodels (3.1) and (3.2)] The local stability and existence of equilibria of submodels (3.1) and (3.2) is summarized in Table (2).

**Table 2**  Sufficient conditions for the existence and stability of equilibria for system (3.1) and (3.2)

<table>
<thead>
<tr>
<th>Equilibria</th>
<th>Existence condition</th>
<th>Stability condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>$E_0^S$</td>
<td>Always exists</td>
<td>$R_{00} &lt; 1$</td>
</tr>
<tr>
<td>$E_1^S$</td>
<td>$r &gt; h_1$</td>
<td>$R_{00} &gt; 1$ and $R_{01} &lt; 1$</td>
</tr>
<tr>
<td>$E_1^{SI}$</td>
<td>$r(K\lambda - \mu - h_2) - h_1K\lambda &gt; 0$</td>
<td>Always LAS if exists</td>
</tr>
<tr>
<td>$E_0^{PP}$</td>
<td>Always exists</td>
<td>$R_{00} &lt; 1$</td>
</tr>
<tr>
<td>$E_1^{PP}$</td>
<td>$r &gt; h_1$</td>
<td>$R_{00} &gt; 1$ and $R_{02} &lt; 1$</td>
</tr>
<tr>
<td>$E_1^{PP}$</td>
<td>$e_1\alpha - a(\gamma + h_3) &gt; 0$, $r(K - \theta_I^{PP}) - h_1K &gt; 0$, $\frac{r_{PP}^{PP}}{K} &gt; \frac{a\alpha^2}{1+\alpha^2}$</td>
<td></td>
</tr>
</tbody>
</table>

International Journal of Biomathematics and Systems Biology 7

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Proof. The Jacobian matrix of the submodel (3.1) at its equilibrium \((x^*, y^*)\) is presented as follows

\[
J_{SI}(x^*, y^*) = \begin{bmatrix}
  r - \frac{2a_i}{1 + a_i} - \lambda y^* - h_1 & -\lambda x^* \\
  \lambda y^* & \lambda x^* - \mu - h_2
\end{bmatrix}
\]  

while the Jacobian matrix of the submodel (3.2) at its equilibrium \((x^*, z^*_1)\) is presented as follows

\[
J_{PP}(x^*, z^*_1) = \begin{bmatrix}
  r - \frac{2a_i}{1 + a_i} - \frac{a_i c_i}{(1 + a_i)^2} - h_1 & -\frac{a_i c_i}{(1 + a_i)^2} \\
  \frac{a_i c_i}{(1 + a_i)^2} & \gamma - h_3
\end{bmatrix}
\]

After substituting \((x^*, y^*) = E_{SI}^0, u = 0, 1, i\) into \((3.5)\), we obtain the eigenvalues for each equilibrium:

1. The equilibrium \(E_{SI}^0 = (0, 0)\) is locally asymptotically stable if \(R_{00} < 1\) and it is saddle if \(R_{00} > 1\) since both eigenvalues associated with \((3.5)\) at \(E_{SI}^0\) can be represented as follows:

\[
\lambda_1 = r - h_1 \begin{cases}
< 0 & \text{if } R_{00} < 1 \\
> 0 & \text{if } R_{00} > 1.
\end{cases}
\]

2. The equilibrium \(E_{SI}^1 = (x_1, 0)\) is locally asymptotically stable if \(R_{01} > 1\) and \(R_{01} < 1\) since both eigenvalues associated with \((3.5)\) at \(E_{SI}^1\) can be represented as follows:

\[
\lambda_1 = h_1 - r \begin{cases}
< 0 & \text{if } R_{01} > 1 \\
> 0 & \text{if } R_{01} < 1.
\end{cases}
\]

3. The equilibrium \(E_{SI}^i = (x_i, y_i)\) is locally asymptotically stable whenever it exists since eigenvalues are the roots of the quadratic equation

\[
p^2 + \frac{\theta}{K}p + \lambda^2 = 0.
\]

Both the roots of this quadratic equation are either negative or complex conjugate with negative real parts.

Similarly, after substituting \((x^*, z^*_1) = E_{PP}^0, u = 0, 1, i\) into \((3.6)\), we obtain the eigenvalues for each equilibrium:

1. The equilibrium \(E_{PP}^0 = (0, 0)\) is locally asymptotically stable if \(R_{00} < 1\) and it is saddle if \(R_{00} > 1\) since both eigenvalues associated with \((3.6)\) at \(E_{PP}^0\) can be represented as follows:

\[
\lambda_1 = r - h_1 \begin{cases}
< 0 & \text{if } R_{00} < 1 \\
> 0 & \text{if } R_{00} > 1.
\end{cases}
\]

2. The equilibrium \(E_{PP}^1 = (x_1, 0)\) is locally asymptotically stable if \(R_{01} > 1\) and \(R_{01} < 1\) since both eigenvalues associated with \((3.6)\) at \(E_{PP}^1\) can be represented as follows:

\[
\lambda_1 = h_1 - r \begin{cases}
< 0 & \text{if } R_{01} > 1 \\
> 0 & \text{if } R_{01} < 1.
\end{cases}
\]

3. The equilibrium \(E_{PP}^i = (x_i, y_i)\) is locally asymptotically stable if \(\frac{r x_i}{K} > \frac{a_i c_i}{(1 + a_i)^2}\) since eigenvalues are the roots of the quadratic equation

\[
p^2 + \left(\frac{r x_i}{K} - \frac{a_i c_i}{(1 + a_i)^2}\right)p + \frac{a_i c_i}{(1 + a_i)^2} = 0.
\]

Both the roots of this quadratic equation are either negative or complex conjugate with negative real parts if the condition \(\frac{r x_i}{K} > \frac{a_i c_i}{(1 + a_i)^2}\) holds.
Again, both submodels (3.3) and (3.4) have \((0,0,0)\) and \((x_1,0,0) = \left( \frac{K_{e2} \delta_0}{\bar{r}}, 0, 0 \right)\) as their boundary equilibria.

For convenience, for Model (3.3), we denote

\[
E_{0,0}^{\text{SIP}} = (0,0,0), \quad E_{1,0}^{\text{SIP}} = (x_1,0,0), \quad E_{2,0}^{\text{SIP}} = (x_2^{\text{SIP}},0,0), \quad E_{3,0}^{\text{SIP}} = (x_4^{\text{SIP}},0,0)
\]

and

\[
E_{i,0}^{\text{SIP}} = \left( x_{i,0}^{\text{SIP}}, x_{i,0}^{\text{SIP}}, x_{i,0}^{\text{SIP}} \right)
\]

where \(x_{i,0}^{\text{SIP}}\) is the unique root of the quadratic equation

\[
are_2 \delta_2 x^2 + [r_2 \delta + \alpha \lambda (\gamma + h_3) + K e_2 (\alpha \lambda + a \delta h_1) - K (\gamma + h_1) + K \delta (h_1 - r) - K e_2 (\mu + h_2) = 0
\]

if \(r_2 \delta + \alpha \lambda (\gamma + h_3) + K e_2 (\alpha \lambda + a \delta h_1) > K (\gamma + h_1) + K \delta (h_1 - r) - K e_2 (\mu + h_2)\)

while for Model (3.4), we denote

\[
E_{0,0}^{\text{PSI}} = (0,0,0), \quad E_{1,0}^{\text{PSI}} = (x_1,0,0), \quad E_{2,0}^{\text{PSI}} = (x_2^{\text{PSI}},0,0), \quad E_{3,0}^{\text{PSI}} = (x_3^{\text{PSI}},0,0)
\]

\[
E_{i,0}^{\text{PSI}} = \left( x_{i,0}^{\text{PSI}}, x_{i,0}^{\text{PSI}}, x_{i,0}^{\text{PSI}} \right)
\]

here \(x_{i,0}^{\text{PSI}}\) is the positive root of the cubic equation

\[
a^2 \beta \delta(x_{i,0}^{\text{PSI}})^3 + [2a^2 \beta^2 K h_1 + 2ar \beta^2 - a^2 \beta^2 K (x_{i,0}^{\text{PSI}})^2 + [\alpha \bar{K} (x_{i,0}^{\text{PSI}})^3] + (\alpha a \bar{K} (x_{i,0}^{\text{PSI}})^3 + (x_{i,0}^{\text{PSI}})^3 + \alpha \bar{K} (x_{i,0}^{\text{PSI}})^3 + \beta K h_1 - r \beta K = 0
\]

and the equilibria \(E_{i,0}^{\text{PSI}}\) exist for \(a \bar{K} - a - \alpha \bar{K} (x_{i,0}^{\text{PSI}})^3 > 0\) and \(r \bar{K} - x_{i,0}^{\text{PSI}} - r h_1 > 0\). Here, \(E_{i,0}^{\text{SIP}}\) and \(E_{i,0}^{\text{PSI}}\) are interior equilibria for the submodel (3.3) and (3.4), respectively, provided their existence.

Proposition 2. [Local stability of equilibria for submodels (3.3) and (3.4)] The local stability and existence of equilibria of submodels (3.3) and (3.4) is summarized in Table (3).

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<tr>
<td>(E_{2,0}^{\text{SIP}})</td>
<td>(r \bar{K} - \mu - h_2 &gt; 0, K \delta &gt; 0)</td>
<td>(R_{03} &lt; 1)</td>
</tr>
<tr>
<td>(E_{3,0}^{\text{SIP}})</td>
<td>(e_1 \alpha - a (\gamma + h_3) &gt; 0, r \bar{K} - x_{i,0}^{\text{SIP}} - h_1 K &gt; 0) (R_{04} &lt; 1, \frac{r \bar{K} - x_{i,0}^{\text{SIP}}}{K} &gt; \frac{\alpha \bar{K} (x_{i,0}^{\text{SIP}})^3}{(1 + \alpha \bar{K})^2})</td>
<td>(e_2 = \frac{r \bar{K} - x_{i,0}^{\text{SIP}}}{K} &gt; \frac{\alpha \bar{K} (x_{i,0}^{\text{SIP}})^3}{(1 + \alpha \bar{K})^2})</td>
</tr>
<tr>
<td>(E_{0,0}^{\text{PSI}})</td>
<td>Always exists</td>
<td>(R_0 &lt; 1)</td>
</tr>
<tr>
<td>(E_{1,0}^{\text{PSI}})</td>
<td>(r &gt; h_1)</td>
<td>(R_{01} &gt; 1, R_{02} &lt; 1)</td>
</tr>
<tr>
<td>(E_{2,0}^{\text{PSI}})</td>
<td>(e_1 \alpha - a (\gamma + h_3) &gt; 0, r \bar{K} - x_{i,0}^{\text{PSI}} - h_1 K &gt; 0)</td>
<td>(R_{03} &lt; 1, \frac{r \bar{K} - x_{i,0}^{\text{PSI}}}{K} &gt; \frac{\alpha \bar{K} (x_{i,0}^{\text{PSI}})^3}{(1 + \alpha \bar{K})^2})</td>
</tr>
<tr>
<td>(E_{3,0}^{\text{PSI}})</td>
<td>(e_1 \alpha - a (\gamma + h_3) &gt; 0, r \bar{K} - x_{i,0}^{\text{PSI}} - h_1 K &gt; 0)</td>
<td>(R_{03} &lt; 1, \frac{r \bar{K} - x_{i,0}^{\text{PSI}}}{K} &gt; \frac{\alpha \bar{K} (x_{i,0}^{\text{PSI}})^3}{(1 + \alpha \bar{K})^2})</td>
</tr>
</tbody>
</table>
Proof. The local stability of equilibrium can be determined by the eigenvalues $\lambda_i, i = 1, 2, 3$ of the Jacobian matrix of system (3.3) evaluated at the equilibrium. The Jacobian matrix of the submodel (3.3) at its equilibrium point $(x^*, y^*, z_1^*)$ is presented as follows:

$$J_{3.3} = \begin{bmatrix} r - 2x^* - \lambda y^* - \frac{\alpha y^*}{1 + ax^*} - h_1 & -\lambda x^* & -\frac{\alpha y^*}{1 + ax^*} \\ \lambda y^* & \lambda x^* - \delta z_1^* - \mu - h_2 & -\delta y^* \\ \frac{e_2 \alpha y^*}{1 + ax^*} & \frac{e_2 \delta z_1^*}{1 + ax^*} + e_2 \delta y^* - \gamma - h_3 \end{bmatrix}$$ (3.7)

while the Jacobian matrix of the submodel (3.4) at its equilibrium point $(x^*, z_1^*, z_2^*)$ is presented as follows:

$$J_{3.4} = \begin{bmatrix} r - 2x^* - \frac{\alpha (z_1^* + z_2^*)}{1 + ax^*} - h_1 & -\alpha x^* - \gamma - 2z_1^* - h_2 & -\alpha z_1^* \\ \frac{e_1 \alpha z_1^*}{1 + ax^*} & \frac{e_1 \alpha z_1^*}{1 + ax^*} - \gamma - 2z_1^* - h_3 & -\beta z_1^* \\ \beta z_2^* & \frac{e_1 \alpha z_1^*}{1 + ax^*} - \psi + \beta z_1^* - h_4 \end{bmatrix}$$ (3.8)

After substituting $E_{3.3}^{SP} = (x^*, y^*, z_1^*), u = 0$ into (3.7), we obtain the eigenvalues for each boundary equilibrium:

1. The equilibrium $E_{0}^{SP} = (0, 0, 0)$ is locally asymptotically stable if $R_{00} < 1$ and saddle if $R_{00} > 1$ since all eigenvalues associated with (3.7) at $E_{0}^{SP}$ can be represented as follows:

$$\lambda_1 = r - h_1 \begin{cases} < 0 & \text{if } R_{00} < 1 \\ > 0 & \text{if } R_{00} > 1 \end{cases}, \quad \lambda_2 = -(\mu + h_2) < 0 \quad \text{and} \quad \lambda_3 = - (\gamma + h_3) < 0.$$

2. The equilibrium $E_{1}^{SP} = (x_1^*, 0, 0)$ is locally asymptotically stable if $R_{01} > 1, R_{01} < 1$ and $R_{02} < 1$ since all eigenvalues associated with (3.7) at $E_{1}^{SP}$ can be represented as follows:

$$\lambda_1 = h_1 - r \begin{cases} < 0 & \text{if } R_{01} > 1 \\ > 0 & \text{if } R_{01} < 1 \end{cases}, \quad \lambda_2 = \lambda x_1 - (\mu + h_2) \begin{cases} < 0 & \text{if } R_{01} < 1 \\ > 0 & \text{if } R_{01} > 1 \end{cases}, \quad \lambda_3 = \frac{e_1 \alpha x_1}{1 + ax_1} - (\gamma + h_3) \begin{cases} < 0 & \text{if } R_{02} < 1 \\ > 0 & \text{if } R_{02} > 1 \end{cases},$$

where the sign of $\lambda_3$ indicates its eigenvector pointing toward ($< 0$) or away from ($> 0$) the equilibrium in $x$-axis ($i=1$), $y$-axis ($i=2$), and $z_1$-axis ($i=3$), respectively.

3. According to Proposition (1), the equilibrium $E_{2}^{SP} = (x_{1}^{SP}, y_{1}^{SP}, 0)$, is locally asymptotically stable only if it is locally asymptotically stable in the submodel (3.1) and

$$\frac{dz_1}{dt} |_{E_{2}^{SP}} = \frac{e_1 \alpha z_{1}^{SP}}{1 + az_{1}^{SP}} + e_2 \phi_{1}^{SP} - \delta z_{1}^{SP} - \mu - h_2 < 0 \Rightarrow R_{03} < 1.$$

4. Similarly, the equilibrium $E_{3}^{SP} = (x_{1}^{SP}, 0, y_{1}^{SP})$ is locally asymptotically stable if it is locally asymptotically stable in the submodel (3.2) and

$$\frac{dy}{dt} |_{E_{3}^{SP}} = \lambda y_{1}^{SP} - \delta y_{1}^{SP} - \mu - h_2 < 0 \Rightarrow R_{04} < 1.$$

5. For the equilibrium point $E_{4}^{SP} = (x_{1}^{SP}, y_{1}^{SP}, z_{1}^{SP}, z_{2}^{SP})$, eigenvalues are the roots of the cubic equation

$$\rho^3 + \Phi_1 \rho^2 + \Phi_2 \rho + \Phi_3 = 0$$

where

$$\Phi_1 = \frac{e_1 \alpha z_1^{SP}}{1 + ax_1^{SP}} - \frac{e_1 \alpha z_1^{SP}}{1 + ax_1^{SP}}, \quad \Phi_2 = \delta z_1^{SP} + \delta y_1^{SP} - \mu - h_2,$$

and

$$\Phi_3 = \left( \frac{e_2 x_1^{SP}}{1 + ax_1^{SP}} \right) \left( \frac{e_2 x_1^{SP}}{1 + ax_1^{SP}} \right) \left( \frac{e_2 x_1^{SP}}{1 + ax_1^{SP}} \right) \left( \frac{e_2 x_1^{SP}}{1 + ax_1^{SP}} \right).$$

Now

$$\Phi_1 \Phi_2 - \Phi_3 = \left( \frac{e_2 x_1^{SP}}{1 + ax_1^{SP}} \right)^3 \left( \frac{e_2 x_1^{SP}}{1 + ax_1^{SP}} \right) - \frac{e_2 x_1^{SP}}{1 + ax_1^{SP}} - \frac{e_2 x_1^{SP}}{1 + ax_1^{SP}} - \frac{e_2 x_1^{SP}}{1 + ax_1^{SP}}.$$
\[- (e_2 - \frac{e_1}{1 + a_1x_1^{lPSI}}) \frac{\lambda \delta x_1^{lPSI} r_x^{PSI} S_P S_I lPSI}{1 + a_1x_1^{lPSI}}. \]

Therefore, the equilibrium $E_{lPSI}$ is locally asymptotically stable if

$$\frac{r_x^{PSI}}{K} > \frac{a_3x_1^{lPSI} r_x^{PSI} S_P S_I lPSI}{(1 + a_1x_1^{lPSI})^2} \text{ and } e_2 = \frac{e_1}{1 + a_1x_1^{lPSI}}.$$ 

Similarly, after substituting $E_{x_1^{PP}} = (x^*, z_1^{PP}, z_2^{PP})$, $u = 0, 1$ into (3.8), we obtain the eigenvalues for each equilibrium:

1. The equilibrium $E_{lPSI}^0 = (0, 0, 0)$ is locally asymptotically stable if $R_{00} < 1$ and saddle if $R_{00} > 1$ since all eigenvalues associated with (3.8) at $E_{lPSI}^0$ can be represented as follows:

$$\lambda_1 = r - h_1 \begin{cases} < 0 \text{ if } R_{00} < 1, \\ > 0 \text{ if } R_{00} > 1, \end{cases} \quad \lambda_2 = -(\gamma + h_3) < 0 \quad \text{and} \quad \lambda_3 = -(\psi + h_4) < 0.$$

2. The equilibrium $E_{lPSI} = (x_1^{PP}, 0, 0)$ is locally asymptotically stable if $R_{01}, R_{02} < 1$ and $R_{05} < 1$ since all eigenvalues associated with (3.8) at $E_{lPSI}^1$ can be represented as follows:

$$\lambda_1 = h_1 - r \begin{cases} < 0 \text{ if } R_{01} < 1, \\ > 0 \text{ if } R_{01} > 1, \end{cases} \quad \lambda_2 = \frac{e_1 \alpha x_1}{1 + a_1} - \gamma - h_3 \begin{cases} < 0 \text{ if } R_{02} < 1, \\ > 0 \text{ if } R_{02} > 1, \end{cases} \quad \lambda_3 = \frac{e_1 \alpha x_1}{1 + a_1} - \psi - h_4 \begin{cases} < 0 \text{ if } R_{05} < 1, \\ > 0 \text{ if } R_{05} > 1. \end{cases}$$

3. According to Proposition (1), the equilibrium $E_{lPSI}^2 = (x_1^{PP}, z_1^{PP}, 0)$, is locally asymptotically stable only if it is locally asymptotically stable in the submodel (3.2) and

$$\frac{d z_1^{PP}}{dt} |_{E_{lPSI}^2} = e_3 \frac{\alpha z_1^{PP}}{1 + a_3} - \gamma - \alpha z_1^{PP} - h_3 < 0 \Leftrightarrow R_{16} < 1.$$ 

4. For the equilibrium point $E_{lPSI}^3 = (\psi, 0, 3)$ all eigenvalues associated with (3.8) at $E_{lPSI}^3$ can be represented as follows:

$$\lambda_2 = \frac{e_1 \alpha x_1}{1 + a_3} - \gamma - \alpha z_1^{PSI} - h_3 < 0 \Leftrightarrow R_{07} < 1.$$

Other two eigenvalues $\lambda_1$ and $\lambda_3$ are roots of the quadratic equation

$$\rho^2 + \left(\frac{r_x^{PSI}}{K} \frac{a_3x_1^{lPSI} r_x^{PSI} S_P S_I lPSI}{(1 + a_1x_1^{lPSI})^2}\right) \rho + \frac{e_1 \alpha x_1 x_1^{lPSI} r_x^{PSI} S_P S_I lPSI}{(1 + a_1x_1^{lPSI})^2} = 0.$$ 

Thus, $E_{lPSI}^3$ is locally asymptotically stable if $R_{07} < 1$ and $e_3^{lPSI} = \frac{\alpha x_1^{lPSI} r_x^{PSI} S_P S_I lPSI}{(1 + a_1x_1^{lPSI})^2}$.

5. For the equilibrium point $E_{lPSI}^4 = (x_1^{PSI}, z_1^{PSI}, z_2^{PSI})$, eigenvalues are the roots of the cubic equation

$$\rho^3 + \Omega_1 \rho^2 + \Omega_2 \rho + \Omega_3 = 0,$$

where

$$\Omega_1 = \frac{r_x^{PSI}}{K} - \frac{a_x^{PSI}}{(1 + a_x^{lPSI})^2}, \quad \Omega_2 = \frac{e_3 \alpha x_1}{1 + a_3} x_1^{lPSI} + \frac{\alpha^2 x_1 x_1^{lPSI} r_x^{PSI} S_P S_I lPSI}{(1 + a_1x_1^{lPSI})^3},$$

and

$$\Omega_3 = \frac{e_3 \alpha x_1}{1 + a_3} x_1^{lPSI} \left(\frac{r_x^{PSI}}{K} - \frac{a_x^{PSI}}{(1 + a_x^{lPSI})^2}\right).$$

Now $\Omega_1 \Omega_2 - \Omega_3 > 0$

hence the equilibrium point $E_{lPSI}^4$ is locally asymptotically stable if the following conditions holds

$$\frac{r_x^{PSI}}{K} > \frac{a_3x_1^{lPSI} r_x^{PSI} S_P S_I lPSI}{(1 + a_1x_1^{lPSI})^2}.$$
4 Dynamics of the full model

After obtaining the complete dynamics of predator-free (SI), disease-free (PP), only predator disease free (SIP) and only prey disease free (PSI) models of the full model (2.1) in the previous section (3), we continue to study the dynamics of the full model.

It is easy to check that the system (2.1) has the following boundary equilibria:

\[ E_0 = (0,0,0,0), \quad E_1 = (x_1,0,0,0), \quad E_2 = (x^S, x^S, 0, 0), \quad E_3 = (x^{PP}, 0, z^{PP}, 0), \]

\[ E_4 = (x^{SI}, x^{SI}, 0, 0), \quad E_5 = (x^{PSI}, 0, z^{PSI}, 0), \]

\[ E_6 = (\xi, 0, 0, z), \quad E_7 = (\hat{x}, \hat{y}, 0, z), \]

where \( \hat{x} \) is the unique positive root of the quadratic equation:

\[
\frac{\mu + h_4}{\alpha + \alpha e} \frac{(r(K - \delta) - Kh_1)(1 + \alpha e)}{K} + \lambda K(\psi + h_1 + h_1 K e_4 - rde_4(aK - 1))\hat{x} + \lambda K(\psi + h_1 + h_1 K e_3 - rde_3 - \alpha e_3 K(\mu + h_1) = 0.
\]

Here, \( E_1, E_2, E_3, E_4, E_5, E_6 \) and \( E_7 \) are nontrivial boundary equilibria for the full model (2.1), provided their existence.

Before going to discuss about the stability of equilibrium points of the full model (2.1), we define some threshold quantities to understand the dynamics of the full model.

First, we define \( R_{08} \) as

\[
R_{08} = \frac{1}{\psi + h_4} \left( \frac{e_3 \alpha e^S}{1 + \alpha e} + e_4 \delta^S \right)
\]

where \( \frac{e_3 \alpha e^S}{1 + \alpha e} + e_4 \delta^S \) is the birth rate of the infected predator due to consumption of the both preys at the equilibrium where susceptible and infected preys coexist. \( \psi + h_4 \) is the total removal of the infected predator due to harvesting and natural mortality. \( R_{08} \) gives the disease basic reproduction number at \( E_1 \). \( R_{08} < 1 \) implies that the disease will be eradicated from the predator population.

Second, we define the basic reproduction number \( R_{09} \) is given by

\[
R_{09} = \frac{1}{\psi + h_4} \left( \frac{e_3 \alpha e^{SI}}{1 + \alpha e} + e_4 \delta^{SI} \right)
\]

which determines the local stability of \( E_4 \). Here \( e_4 \delta^{SI} \) is the net growth rate of the infected predator due to disease transmission in susceptible predator and all the other quantities define above. Hence \( R_{09} \) gives the disease basic reproduction number and \( R_{09} < 1 \) implies disease will be eradicated from the predator population.

Third, we define the basic reproduction number \( R_{10} \) given by

\[
R_{10} = \frac{\lambda \alpha^S}{\mu + h_2 + \delta (z^S_i + z^{PSI}_i)}
\]

which determines the local stability of the equilibrium \( E_6 \). Here \( \lambda \alpha^S \) is the infection rate of a new infective prey appearing in a totally the susceptible prey population and \( \mu + h_2 + \delta (z^S_i + z^{PSI}_i) \) is the removal rate of the infected prey at \( E_5 \). So, \( R_{10} \) is considered as the disease basic reproduction number in the prey population. \( R_{10} < 1 \) implies disease in the prey population will be wiped out.

The local stability of \( E_6 \) determined by the basic reproduction numbers \( R_{11} \) and \( R_{12} \) given by
\[
R_{11} = \frac{\lambda x}{\mu + h + \delta z} \quad \text{and} \quad R_{12} = \frac{e_1 \alpha x}{(\gamma + h_3 + \beta z_2)(1 + \alpha z)}
\]

Here \(\lambda x\) is the infection rate of an infectious prey and \(\mu + h + \delta z\) is the removal rate of the infectious prey at \(E_6\). Consequently \(R_{11}\) is the disease basic reproduction number of an infectious prey when entering a prey disease-free environment where the infected predator and the susceptible prey coexist. \(\frac{e_1 \alpha x}{(\gamma + h_3 + \beta z_2)(1 + \alpha z)}\) is the growth rate of the newborn susceptible predator and \(\gamma + h_3 + \beta z_2\) is the removal rate of the predators at \(E_6\). Hence \(R_{12}\) gives the ecological basic reproduction number of the predator population when disease is endemic in the predator population. \(R_{11} < 1\) implies that disease in the prey population will be eradicated while \(R_{12} < 1\) implies that the susceptible predator goes to extinction. Hence the combination of these conditions results in \(E_6\) being locally asymptotically stable.

The local stability of \(E_7\) is determined by the basic reproduction number \(R_{13}\) given by

\[
R_{13} = \frac{1}{(\gamma + h_3 + \beta z_2)} \left( \frac{e_1 \alpha x}{1 + \alpha z} + e_2 \delta y \right)
\]

Here \(\frac{e_1 \alpha x}{1 + \alpha z} + e_2 \delta y\) is the birth rate of the newborn susceptible predator due to consumption of the both preys and \(\gamma + h_3 + \beta z_2\) is the removal rate of the susceptible predator at \(E_7\). So, \(R_{13}\) can be interpreted as the ecological basic reproduction number at \(E_7\). \(R_{13} < 1\) implies that the susceptible predator will be eradicated from the system.

**Proposition 3.** [Boundary equilibria and stability] Sufficient conditions for the existence and the local stability of boundary equilibria for system (2.1) are summarized in Table (4).

<table>
<thead>
<tr>
<th>Equilibria</th>
<th>Existence condition</th>
<th>Stability condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>(E_0)</td>
<td>Always exists</td>
<td>(R_{00} &lt; 1)</td>
</tr>
<tr>
<td>(E_1)</td>
<td>(r &gt; h_1)</td>
<td>(R_{00} &gt; 1, R_{01} &lt; 1, R_{02} &lt; 1) and (R_{05} &lt; 1)</td>
</tr>
<tr>
<td>(E_2)</td>
<td>(r(K \lambda - \mu - h_2) - h_1 K \lambda &gt; 0)</td>
<td>(R_{03} &lt; 1) and (R_{08} &lt; 1)</td>
</tr>
<tr>
<td>(E_3)</td>
<td>(e_1 \alpha - a(\gamma + h_3) &gt; 0, r(K - \gamma_P P) - h_1 K &gt; 0)</td>
<td>(R_{04} &lt; 1, R_{06} &lt; 1, \frac{r \gamma P}{K} &gt; \frac{\alpha \gamma_P P}{(1 + a \gamma_P)^2} )</td>
</tr>
<tr>
<td>(E_4)</td>
<td>(\frac{\mu + h_2}{K} &lt; x_{SP} &lt; \frac{\gamma + h_3}{e_1 \alpha - a(\gamma + h_3)})</td>
<td>(R_{09}, \frac{r \gamma P}{K} &gt; \frac{\alpha \gamma_P P}{(1 + a \gamma_P)^2} )</td>
</tr>
<tr>
<td>(E_5)</td>
<td>(\frac{\psi}{e_1 \alpha - a(\gamma + h_3)} &lt; x_{SP} &lt; \frac{\psi + h_4}{e_1 \alpha - a(\psi + h_3)})</td>
<td>(R_{10} &lt; 1, \frac{r \gamma P}{K} &gt; \frac{\alpha \gamma_P P}{(1 + a \gamma_P)^2} )</td>
</tr>
<tr>
<td>(E_6)</td>
<td>(e_3 \alpha - a(\psi + h_4) &gt; 0, r(K - \gamma - h_1) &gt; 0)</td>
<td>(R_{11} &lt; 1, R_{12} &lt; 1, \frac{\gamma P}{K} &gt; \frac{\alpha \gamma_P P}{(1 + a \gamma_P)^2} )</td>
</tr>
<tr>
<td>(E_7)</td>
<td>(\frac{\mu + h_2}{K} &lt; \hat{x} &lt; \frac{\psi + h_4}{a(\psi + h_3) - e_1 \alpha} )</td>
<td>(R_{13} &lt; 1, \frac{\gamma P}{K} &gt; \frac{\alpha \gamma_P P}{(1 + a \gamma_P)^2} )</td>
</tr>
</tbody>
</table>

**Proof.** The local stability of equilibrium can be determined by the eigenvalues \(\lambda_i, i = 1, 2, 3, 4\) of the Jacobian matrix of system (2.1) evaluated at the equilibrium. The Jacobian matrix of the system (2.1) at its equilibrium \((x', y', z_1', z_2')\) is presented as follows:

\[
\begin{bmatrix}
  r - \frac{2x^*}{K} - \lambda x^* - \alpha \frac{z_1^* + z_2^*}{(1 + ax)^2} - h_1 & -\lambda x^* & -\lambda x^* & -\lambda x^* \\
  -\lambda x^* & \lambda x^* - \delta (z_1^* + z_2^*) - \mu - h_2 & -\delta y^* & -\delta y^* \\
  \frac{e_1 \alpha x^*}{(1 + ax)^2} & e_2 \delta y^* & \frac{e_1 \alpha x^*}{(1 + ax)^2} + e_2 \delta y^* & -\beta z_2^* \\
  e_4 \delta z_2^* & \beta z_2^* & \frac{e_1 \alpha x^*}{(1 + ax)^2} + e_4 \delta y^* & -\psi - \beta z_2^* - h_4
\end{bmatrix}
\]  

(4.1)
After substituting $E_u = (x^*, y^*, z^*_1, z^*_2)$, $u = 0, 1$ into (4.1), we obtain the eigenvalues for each boundary equilibrium:

1. The equilibrium $E_0 = (0, 0, 0, 0)$ is locally asymptotically stable if $R_{00} < 1$ and saddle if $R_{00} > 1$ since all eigenvalues associated with (4.1) at $E_0$ can be represented as follows:

$$
\lambda_1 = r - h_1 \begin{cases} < 0 & \text{if } R_{00} < 1 \\
< 0 & \text{if } R_{00} > 1 
\end{cases},
\lambda_2 = -(\mu + h_2), \lambda_3 = -(\gamma + h_3) < 0 \text{ and } \lambda_4 = -(\psi + h_4) < 0.
$$

2. The equilibrium $E_1 = (x_1, 0, 0, 0)$ is locally asymptotically stable if $R_{00} > 1$, $R_{01} < 1$, $R_{02} < 1$ and $R_{05} < 1$ since all eigenvalues associated with (4.1) at $E_1$ can be represented as follows:

$$
\lambda_1 = h_1 - r \begin{cases} < 0 & \text{if } R_{00} > 1 \\
< 0 & \text{if } R_{00} < 1 
\end{cases},
\lambda_2 = x_1 - (\mu + h_2) \begin{cases} < 0 & \text{if } R_{01} < 1 \\
< 0 & \text{if } R_{01} > 1 
\end{cases},
\lambda_3 = \frac{e_1 \alpha x_1}{1 + \alpha x_1} - (\gamma + h_3) \begin{cases} < 0 & \text{if } R_{02} < 1 \\
< 0 & \text{if } R_{02} > 1 
\end{cases} \text{ and } \lambda_4 = \frac{e_3 \alpha x_3}{1 + \alpha x_3} - (\psi + h_4) \begin{cases} < 0 & \text{if } R_{05} < 1 \\
< 0 & \text{if } R_{05} > 1 
\end{cases},
$$

where the sign of $\lambda_i$ indicates its eigenvector pointing toward ($< 0$) or away from ($> 0$) the equilibrium in $x$-axis ($i = 1$), $y$-axis ($i = 2$), $z_1$-axis ($i = 3$) and $z_2$-axis ($i = 4$), respectively.

3. According to Proposition (2), the equilibrium $E_2 = (x_2^*, y_2^*, 0, 0)$, is locally asymptotically stable if it is locally asymptotically stable in the submodel (3.3) and

$$
d\frac{d\bar{z}_2}{dt} = \frac{e_2 \alpha x_2^*}{1 + \alpha x_2^*} + e_2 \gamma \bar{y}_2^* - \psi - h_4 < 0 \Leftrightarrow R_{00} < 1.
$$

4. Similarly, the equilibrium $E_3 = (x_3^*, 0, y_3^*, 0)$, is locally asymptotically stable if it is locally asymptotically stable in the submodel (3.4) and

$$
d\frac{dy}{dt} = \lambda x_3^* - \delta y_3^* - \mu - h_2 < 0 \Leftrightarrow R_{04} < 1.
$$

5. Again, the equilibrium $E_4 = (x_4^*, y_4^*, z_4^*, 0)$, is locally asymptotically stable if it is locally asymptotically stable in the submodel (3.3) and

$$
d\frac{d\bar{z}_4}{dt} = \frac{e_3 \alpha x_4^*}{1 + \alpha x_4^*} + e_3 \gamma \bar{y}_4^* - \psi - h_4 < 0 \Leftrightarrow R_{00} < 1.
$$

6. Again, the equilibrium $E_5 = (x_5^*, 0, y_5^*, z_5^*)$, is locally asymptotically stable if it is locally asymptotically stable in the submodel (3.4) and

$$
d\frac{dy}{dt} = \lambda x_5^* - \delta (x_5^* + z_5^*) - \mu - h_2 < 0 \Leftrightarrow R_{10} < 1.
$$

7. The equilibrium $E_6 = (x, 0, 0, z_2^*)$ is locally asymptotically stable if $R_{11} < 1$, $R_{12} < 1$ and $\frac{r \bar{z}_2}{K} > \frac{e_4 \alpha x_2^*}{(1 + \alpha x_2^*)}$ since all eigenvalues associated with (4.1) at $E_6$ can be represented as follows:

$$
\lambda_2 = \lambda \bar{z}_2 - \delta \bar{z}_2 - \mu - h_2 < 0 \Leftrightarrow R_{11} < 1 \lambda_3 = \frac{e_1 \alpha \bar{x}_2^*}{1 + \alpha \bar{x}_2^*} - \gamma - h_3 - \beta \bar{z}_2 < 0 \Leftrightarrow R_{12} < 1,
$$

and the other two eigenvalues $\lambda_1$ and $\lambda_4$ are the roots of the quadratic equation

$$
\rho^2 + \left( \frac{r \bar{z}_2}{K} \frac{e_4 \alpha x_2^*}{(1 + \alpha x_2^*)^2} \right) \rho + \frac{e_1 \alpha x_2^*}{(1 + \alpha x_2^*)} = 0.
$$

Hence the equilibrium $E_6$ is locally asymptotically stable if the following conditions hold

$$
R_{11} < 1, \quad R_{12} < 1 \quad \text{and} \quad \frac{r \bar{z}_2}{K} > \frac{e_4 \alpha x_2^*}{(1 + \alpha x_2^*)}.
$$
8. The equilibrium \( E_7 = (\xi, \eta, 0, \zeta_2) \) is locally asymptotically stable if \( R_{13} < 1, \quad \frac{r_7}{K} > \frac{\alpha \lambda^2 \zeta_2}{(1 + ax)^2} \) and \( e_2 = \frac{e_1}{1 + ax} \) since all eigenvalues associated with (4.1) at \( E_7 \) can be represented as follows:

\[
\lambda_3 = \frac{e_1 \alpha^2 \lambda^2}{1 + ax} + e_2 \delta^2 - \gamma - h_3 - \beta \zeta_2 < 0 \Longleftrightarrow R_{13} < 1,
\]

and the other three eigenvalues \( \lambda_1, \lambda_2 \) and \( \lambda_3 \) are the roots of the cubic equation

\[
\rho^3 + \Theta_1 \rho^2 + \Theta_2 \rho + \Theta_3 = 0,
\]

where \( \Theta_1 = \frac{r_7}{K} - \frac{\alpha \lambda^2 \zeta_2}{(1 + ax)^2} \), \( \Theta_2 = \delta^2 e_2 \zeta_2 + \lambda^2 \delta^2 + e_1 \alpha^2 \delta \zeta_2 \), and \( \Theta_3 = e_2 \delta^2 \zeta_2 \left( \frac{r_7}{K} - \frac{\alpha \lambda^2 \zeta_2}{(1 + ax)^2} \right) + \left( \frac{e_3}{1 + ax} + \frac{e_4}{1 + ax} \right) \frac{\lambda \delta \lambda \zeta_2}{1 + ax} \). Hence the equilibrium \( E_7 \) is locally asymptotically stable if the following conditions hold

\[
R_{13} < 1, \quad \frac{r_7}{K} > \frac{\alpha \lambda^2 \zeta_2}{(1 + ax)^2} \quad \text{and} \quad e_2 = \frac{e_1}{1 + ax}.
\]

4.1 Stability of interior equilibria

The interior equilibrium point is given by \( E^* = (x^*, y^*, z_1^*, z_2^*) \), where \( (x^*, y^*, z_1^*, z_2^*) \) is a solution of the system of equations

\[
\begin{align*}
& r \left( 1 - \frac{1}{K} \right) - \lambda y - \frac{\alpha (z_1 + z_2)}{1 + ax} - h_1 = 0, \\
& \lambda x - \delta (z_1 + z_2) - h_2 - \mu = 0, \\
& e_1 \alpha + e_2 \delta y - \gamma - \beta z_2 - h_3 = 0, \\
& e_3 \alpha + e_4 \delta y - \gamma + \beta z_1 - h_4 = 0.
\end{align*}
\]

The Jacobian \( J = (J_{ij})_{4 \times 4} \) of the system (2.1) at any arbitrary point \((x, y, z_1, z_2)\) whose entries are

\[
\begin{align*}
J_{11} &= r \left( 1 - \frac{2x}{K} \right) - \lambda y - \frac{\alpha (z_1 + z_2)}{(1 + ax)^2} - h_1, \\
J_{12} &= -\lambda x, \\
J_{13} &= -\lambda, \\
J_{14} &= -\frac{\alpha \lambda}{1 + ax}, \\
J_{22} &= \lambda x, \\
J_{23} &= \lambda, \\
J_{24} &= -\delta y, \\
J_{32} &= e_2 \delta, \\
J_{33} &= e_1 \alpha, \\
J_{34} &= e_2 \delta, \\
J_{41} &= \frac{e_1 \alpha \delta}{(1 + ax)^2}, \\
J_{42} &= e_2 \delta, \\
J_{43} &= e_1 \alpha, \\
J_{44} &= e_2 \delta.
\end{align*}
\]

**Theorem 1.** The interior point \( E^*(x^*, y^*, z_1^*, z_2^*) \) for the system (2.1) is locally asymptotically stable if the following conditions are hold as follows:

\[
\sigma_1 > 0, \quad \sigma_2 - \sigma_3 > 0, \quad \sigma_4 (\sigma_1 \sigma_2 - \sigma_3) - \sigma_5 > 0,
\]

where \( \sigma_i \)'s are given in the proof of the theorem.
Proof. The entities of the Jacobian matrix \( A = (A_{ij})_{4 \times 4} \) at the interior point \( E^*(x^*, y^*, z^*_1, z^*_2) \) are

\[
A_{11} = \frac{-rx^*}{K} + \frac{\alpha xx^*(z^*_1 + z^*_2)}{(1 + \alpha x^*)^2}, \quad A_{12} = -\lambda x^*, \quad A_{13} = \frac{-\alpha x^*}{1 + \alpha x^*}, \quad A_{14} = \frac{-\alpha x^*}{1 + \alpha x^*}
\]

\[
A_{21} = \lambda x^*, \quad A_{22} = 0, \quad A_{23} = -\delta y^*, \quad A_{24} = -\delta y^*
\]

\[
A_{31} = \frac{e_1 \alpha z^*_1}{(1 + \alpha x^*)^2}, \quad A_{32} = e_2 \delta z^*_1, \quad A_{33} = 0, \quad A_{44} = -\beta z^*_1
\]

\[
A_{41} = \frac{e_0 \alpha z^*_2}{(1 + \alpha x^*)^2}, \quad A_{42} = e_2 \delta z^*_2, \quad A_{43} = \beta z^*_2, \quad A_{44} = 0
\]

The characteristic equation of Jacobian matrix is given by

\[
\rho^4 + \sigma_1 \rho^3 + \sigma_2 \rho^2 + \sigma_3 \rho + \sigma_4 = 0
\]

where

\[
\sigma_1 = -A_{11},
\sigma_2 = -A_{34}A_{43} - A_{23}A_{32} - A_{24}A_{42} - A_{12}A_{21} - A_{13}A_{31} - A_{14}A_{41},
\sigma_3 = A_{12}A_{23}A_{31} + A_{13}A_{24}A_{41} - A_{12}A_{21}A_{13}A_{31} - A_{12}A_{21}A_{13}A_{31},
\sigma_4 = A_{12}A_{23}A_{31} + A_{13}A_{24}A_{41} - A_{12}A_{21}A_{13}A_{31} - A_{12}A_{21}A_{13}A_{31},
\sigma_5 = A_{12}A_{23}A_{31} + A_{13}A_{24}A_{41} - A_{12}A_{21}A_{13}A_{31} - A_{12}A_{21}A_{13}A_{31}.
\]

Using the Routh-Hurwitz criteria we observe that the system (2.1) is stable around the positive equilibrium point \( E^* \) if the conditions stated in the theorem hold.

Now we shall find out the conditions for which \( E^* \) enters into Hopf bifurcation as \( \beta \) varies over \( \mathbb{R} \). Routh-Hurewitz Criterion and Hopf-bifurcation: Let \( \Psi : (0, \infty) \to \mathbb{R} \) be the following continuously differentiable function of \( \beta \):

\[
\Psi(\beta) := \sigma_1(\beta)\sigma_2(\beta)\sigma_3(\beta) - \sigma_2^2(\beta) - \sigma_4(\beta)\sigma_5(\beta)
\]

The assumptions for Hopf bifurcation to occur are the usual ones and these require that the spectrum \( \sigma(\beta) = \{ \rho : D(p) = 0 \} \) of the characteristic equation such is that

(A) There exists \( \beta^* \in (0, \infty) \), at which a pair of complex eigenvalues \( \rho(\beta^*), \rho(\beta^*) \in \sigma(\beta) \) are such that

\[
\text{Re} \rho(\beta^*) = 0, \quad \text{Im} \rho(\beta^*) = \omega_0 > 0,
\]

and the transversality condition

\[
\frac{d \text{Re} \rho(\beta)}{d \beta} \bigg|_{\beta^*} \neq 0;
\]

(B) All other elements of \( \sigma(\beta) \) have negative real parts.

**Theorem 2.** The system (2.1) around the interior equilibrium \( E^* \) enters into Hopf-bifurcation at \( \beta = \beta^* \in (0, \infty) \) if and only if

1. \( \Psi(\beta^*) = 0 \)
2. \( \sigma_1^2(\beta^*) > 2(\sigma_2^2 - 2\sigma_4^2)(\sigma_1^2 - 2\sigma_4^2) \)

and all other eigenvalues are of negative real parts, where \( \rho(\beta) \) is purely imaginary at \( \beta = \beta^* \).
Proof. By the condition $\Psi(\beta^*) = 0$, the characteristic equation can be written as
\[
\left( p^2 - \frac{\sigma_1}{\sigma_1} \right) \left( p^2 + \sigma_1 p + \frac{\sigma_1 \sigma_1}{\sigma_1} \right) = 0
\]
If it has four roots, say $\rho_j$, $(i=1,2,3,4)$ with the pair of purely imaginary roots at $\beta = \beta^*$ as $\rho_1 = \bar{\rho}_2$, then we have
\[
\begin{align*}
\rho_1 + \rho_4 &= -\sigma_1, \\
\rho_2 \rho_3 &= \sigma_2, \\
\rho_2 \rho_4 &= -\sigma_3, \\
\rho_2^2 \rho_4 p_4 &= \sigma_4.
\end{align*}
\]
(4.2)
where $\omega_0 = \text{Im} \rho_1 (\beta^*)$. By above $\omega_0 = \sqrt{\frac{\sigma_1}{\sigma_1}}$. Now, if $\rho_3$ and $\rho_4$ are complex conjugate, then from (4.2), it follows that $2\text{Re} \rho_3 = -\sigma_1$; if they are real roots, then by (4.2) and (4.2) $\rho_3 < 0$ and $\rho_4 < 0$. To complete the discussion, it remains to verify the transversality condition.

As $\Psi(\beta^*)$ is a continuous function of all its roots, so there exists an open interval $\beta \in (\beta^* - \varepsilon, \beta^* + \varepsilon)$ where $\rho_1$ and $\rho_2$ are complex conjugate for $\beta$. Suppose, their general forms in this neighborhood are
\[
\begin{align*}
\rho_1 (\beta) &= \chi (\beta) + i \nu (\beta), \\
\rho_2 (\beta) &= \chi (\beta) - i \nu (\beta).
\end{align*}
\]
Now, we shall verify the transversality condition
\[
\left. \frac{d\text{Re} (\rho_j (\beta))}{d\beta} \right|_{\beta = \beta^*} \neq 0, \quad j = 1, 2.
\]
Substituting $\rho_j (\beta) = \chi (\beta) \pm i \nu (\beta)$, into (4.2) and calculating the derivative, we have
\[
\begin{align*}
K(\beta) \chi' (\beta) - L(\beta) \nu' (\beta) + M(\beta) &= 0, \\
L(\beta) \chi' (\beta) + K(\beta) \nu' (\beta) + N(\beta) &= 0
\end{align*}
\]
where
\[
\begin{align*}
K(\beta) &= 4 \chi^3 - 12 \chi \nu^2 + 3 \sigma_1 (\chi^2 - \nu^2) + 2 \sigma_2 \chi + \sigma_3, \\
L(\beta) &= 12 \chi \nu + 6 \sigma_1 \chi \nu - 4 \chi^3 + 2 \sigma_2 \chi, \\
M(\beta) &= \sigma_1 \chi^3 - 3 \sigma_1 \chi \nu^2 + \sigma_2 (\chi^2 - \nu^2) + \sigma_3 \chi, \\
N(\beta) &= 3 \sigma_1 \chi^2 \nu - \sigma_1 \nu^3 + 2 \sigma_2 \chi \nu + \sigma_3 \chi.
\end{align*}
\]
Solving for $\chi' (\beta^*)$ we have
\[
\left[ \frac{d\text{Re} (\rho_j (\beta))}{d\beta} \right]_{\beta = \beta^*} = \chi' (\beta^*) \neq 0
\]
if $\sigma_1^2 \sigma_2 (\sigma_1 - 3 \sigma_3) > 2(\sigma_2 \sigma_2^2 - 2 \sigma_2^2) (\sigma_1 \sigma_2^2 - \sigma_1^2 \sigma_2^2)^2$.

Thus the transversality conditions hold and hence Hopf bifurcation occurs at $\beta = \beta^*$.

Hence the theorem.

Theorem 3. If the conditions $R_{0i} > 1$ and $\hat{R}_{0j} > 1$ for $i = 1$ to 7 and $j = 2$ to 4 are satisfied, then system 2.1 is uniformly persistent.
Proof. We consider the average Lyapunov function of the form

\[ V(x, y, z_1, z_2) = x^\alpha_1 y^\alpha_2 z_1^\alpha_3 z_2^\alpha_4 \]

where each \( \alpha_i \) (i=1,2,3,4) is assumed positive. In the interior of \( \mathbb{R} \), we have

\[
\dot{V} = \Psi(x, y, z_1, z_2) \\
= \alpha_1 \left[ r (1 - \frac{y}{\tilde{y}}) - \lambda y - \frac{\alpha_1 \alpha_2 \psi}{1 + \alpha_1} - h_1 \right] + \alpha_2 \left[ \lambda y - \tilde{z}_1 + z_2 - \mu - h_2 \right] \\
+ \alpha_3 \left[ \frac{\alpha_3 \theta_3}{1 + \alpha_3} + \epsilon_2 \delta y - \gamma - h_3 - \beta_3 \right] + \alpha_4 \left[ \frac{\alpha_4 \theta_4}{1 + \alpha_4} + \epsilon_4 \delta y - \psi - h_4 + \beta_4 \right]
\]

We can show that \( \Psi(x, y, z_1, z_2) > 0 \) for all equilibria and suitable choice of \( \alpha_i > 0 \) (i=1, 2, 3, 4) to prove permanent coexistence. The following conditions should be satisfied for all equilibrium points:

\[
\Psi(E_1) = \alpha_2 \left[ \lambda \beta - \mu - h_1 \right] + \alpha_3 \left[ \frac{\alpha_3 \theta_3}{1 + \alpha_3} - \gamma - h_3 \right] + \alpha_4 \left[ \frac{\alpha_4 \theta_4}{1 + \alpha_4} - \psi - h_4 \right] > 0
\]

\[
\Psi(E_2) = \alpha_2 \left[ \lambda \beta_1 - \tilde{z}_1 + z_2 - \mu - h_2 \right] + \alpha_3 \left[ \frac{\alpha_3 \theta_3}{1 + \alpha_3} - \gamma - h_3 - \beta_3 \right] > 0
\]

\[
\Psi(E_3) = \alpha_2 \left[ \lambda \beta_2 - \tilde{z}_1 + z_2 - \mu - h_2 \right] + \alpha_3 \left[ \frac{\alpha_3 \theta_3}{1 + \alpha_3} - \gamma - h_3 - \beta_3 \right] + \alpha_4 \left[ \frac{\alpha_4 \theta_4}{1 + \alpha_4} - \psi - h_4 + \beta_4 \right] > 0
\]

\[
\Psi(E_4) = \alpha_2 \left[ \lambda \beta_3 - \tilde{z}_1 + z_2 - \mu - h_2 \right] + \alpha_3 \left[ \frac{\alpha_3 \theta_3}{1 + \alpha_3} - \gamma - h_3 - \beta_3 \right] + \alpha_4 \left[ \frac{\alpha_4 \theta_4}{1 + \alpha_4} - \psi - h_4 + \beta_4 \right] > 0
\]

\[
\Psi(E_5) = \alpha_2 \left[ \lambda \beta_4 - \tilde{z}_1 + z_2 - \mu - h_2 \right] + \alpha_3 \left[ \frac{\alpha_3 \theta_3}{1 + \alpha_3} - \gamma - h_3 - \beta_3 \right] + \alpha_4 \left[ \frac{\alpha_4 \theta_4}{1 + \alpha_4} - \psi - h_4 + \beta_4 \right] > 0
\]

After some algebraic calculations, it can be easily shown that above conditions are satisfied if the hypothesis of the above theorem holds.

5 Numerical results and discussion

In this section, we have performed extensive numerical experiments to study the role of harvesting on the model dynamics. It is established that mathematical models involve simplifications, sacrificing certain biological complexities only to promote mathematical tractability. It is quite obvious there are some issues, which is beyond to establish experimentally due to some ethical problems and/or huge experimental costs. But mathematical models through hypothetical simulations provide knowledge on the inaccessible parameters in terms of accessible parameters. Those findings certainly help the experimentalists/system manager to design an experiment or to manage the system accordingly. We will first observe the dynamics of the system for variation of the force of infection in the prey population and after that we will observe the impact of disease propagation in the predator population. Finally, we will observe the role of harvesting in our proposed system. In whole numerical experiment, we use a set of hypothetical parameter values: \( r = 2.0, K = 300, \alpha = 0.015, \mu = 0.3, \delta = 0.04, \gamma = 0.18, \psi = 0.25, h_1 = 0.05, h_2 = 0.04, h_3 = 0.02, h_4 = 0.01, e_1 = 0.12, e_2 = 0.8, a = 0.002. \)

Figure (2) illustrates that the infected predator does not persist, but other three species coexist in a stable position for \( \lambda = 0.035 \) and \( \beta = 0.0005. \) Hence disease in the predator population will be wiped out for the above set of parameter values. Now we will fix the value of \( \beta \) at 0.0005 and observe the dynamical behavior of the system for variation of \( \lambda. \) It is clear from Figure (3) that the infected predator does not survive, but both the preys (susceptible and infected) and the susceptible predator survives in an oscillatory position for \( \lambda = 0.05. \) This result is already well-established in the eco-epidemiological model with disease in the prey species. To observe a clear dynamics of the system we give a bifurcation diagram for variation of \( \lambda \) keeping fixed \( \beta = 0.0005. \) Figure (4) illustrates that both the preys and the susceptible predator settles down to oscillatory position from steady stable distribution for increasing \( \lambda. \)
Fig. 2 Figure depicts extinction of infected predator and the stable distribution of both prey and susceptible predator of system(1) for \( \lambda = 0.035 \), \( \beta = 0.0005 \) and \( r = 2.0 \), \( k = 300 \), \( \alpha = 0.015 \), \( \mu = 0.3 \), \( \delta = 0.04 \), \( \gamma = 0.18 \), \( \psi = 0.25 \), \( h_1 = 0.05 \), \( h_2 = 0.04 \), \( h_3 = 0.02 \), \( h_4 = 0.01 \), \( e_1 = 0.12 \), \( e_2 = 0.8 \), \( a = 0.002 \).

It is observed from Figure (5) that all four species coexist in an oscillatory position for \( \lambda = 0.08 \) and \( \beta = 0.0007 \). It is interesting to note that for increasing the values of \( \beta \) from 0.0005 to 0.0007, we observe that the infected predator survives with other three species. To have a clear picture we provide a bifurcation diagram for variation of \( \lambda \) keeping \( \beta = 0.0007 \). Figure (6) illustrates all four species settle down to oscillations for variation of \( \lambda \) and \( \beta = 0.0007 \). It is obvious from Figure (5) that introduction of disease in the predator population helps all four species to coexist. From Figure (7), we observe that the susceptible predator goes to extinction for \( \beta = 0.0008 \) and \( \lambda = 0.08 \). So, coexistence of all four species is possible in the interval \( \beta \) but for fix \( \beta = 0.0007 \), the coexistence of all species is possible for variation \( \lambda \) (see Figure (6)).
Fig. 4 Figure depicts that both prey and susceptible predator coexist for $\lambda > 0$ and only infected predator survives with other three species for $\lambda \in [0.04, 0.06]$ and other parameters fixed as given in Figure 2.

Fig. 5 Figure shows the oscillatory coexisting of all four species for $\beta = 0.0007$, $\lambda = 0.08$ and other parameters fixed as given in Figure 2.
Fig. 6 Figure depicts the dynamics of all four species for varying $\lambda$ and $\beta = 0.0007$ and other parameters fixed as given in Figure 2.

Fig. 7 Figure shows the extinction of susceptible predator and oscillatory coexistence of other three species for $\beta = 0.0008$, $\lambda = 0.08$ and other parameters fixed as given in Figure 2.
Fig. 8  Figure depicts the extinction of infected predator and other stable distribution of other three species for $h_1 = 1.4, \lambda = 0.08, \beta = 0.0007$ and other parameters fixed as given in Figure 2.

Fig. 9  Figure depicts the dynamics of all four species for varying $h_1, \lambda = 0.08, \beta = 0.0007$ and other parameters fixed as given in Figure 2.
Fig. 10 Figure depicts the stable distribution of other three species for $h_2 = 2.75$, $\lambda = 0.08$, $\beta = 0.0007$ and other parameters fixed as given in Figure 2.

In this connection, we like to mention that many investigators (2; 24; 25) observed that disease introduction into the predator population tends to destabilize established predator-prey communities. The same feature is also observed by Dobson (26); Fenton and Rands, (20) for microparasites with indirect life cycles. Macroparasitic models generally have a tendency to unstable dynamics, because they consider the parasite burden in the host in an additional equation (26; 27; 28). The paper by Hadeler and Freedman (2) is very interesting as it explicitly studies the case of an oscillating resident predator-prey community. Upon disease invasion, the amplitude of the oscillation increases, indicating further destabilization. Oscillation resident communities are particularly worthwhile to study, because prey-predator (or host-parasitoid, plant-herbivore, consumer-resource) systems are well known examples of inherently fluctuating populations (29). They represent interesting and relevant scenarios in biological control and resource management, because recurrent outbreaks in both terrestrial and aquatic ecosystems pose central problems in ecology (30; 31). Oscillations are also a concern of biological conservation because populations could reach such small abundances that they are likely to go extinct (32).

Now we will observe the role of harvesting in controlling oscillations and disease propagation. We have already seen oscillating co-existence of all four species for $\lambda = 0.08$ and $\beta = 0.007$. So, we fix the value of $\lambda$ and $\beta$ at $\lambda = 0.08$ and $\beta = 0.007$. Now we will observe the dynamics of the system for variation of the harvesting rate of species. From Figure (8), we observe that both the preys and the susceptible predator survives in stable position and disease in the predator population is wiped out for $h_1 = 1.4$. Figure (9) illustrates that oscillation of species is disappeared, and both the preys and the susceptible predator settle down to stable position but disease in the predator population is washed out for increasing the values of $h_1$. From Figure (10), it is found that all four species exist in a steady stable state for $h_2 = 2.75$. To observe clear dynamics we give a bifurcation diagram for $h_2$. Figure (11) illustrates that oscillating coexistence of all four species is disappeared for higher values of $h_2$ and settle down in a steady stable state. So we observe that harvesting has an important role in controlling oscillations. Many investigators already have used harvesting efforts as controlling parameter for disappearance of oscillations. (33) studied a predator-prey system with independent harvesting on both species and showed that the system enters into a stable position from limit cycle oscillation. They arrived at the conclusion that harvesting efforts may be used as a biological control for the persistence of the species. The another important role of harvesting is controlling disease propagation has been observed in our proposed system. From Figure (12), it is found that the infected prey and the infected predator do not exist for $h_2 = 2.8$ and $h_1 = 0.02$ and hence disease does not propagate in the prey and the predator populations.
Fig. 11  Figure depicts dynamics of all four species for varying $h_2, \lambda = 0.08, \beta = 0.0007$ and other parameters fixed as given in Figure 2.

Fig. 12  Figure shows that infected prey and infected predator goes to extinction for $h_1 = 0.02$ and $h_2 = 2.8, \lambda = 0.08$, $\beta = 0.0007$ and other parameters fixed as given in Figure 2.
(13) and (14), it is found that disease cannot be controlled through harvesting of only one species. For the same parameter values as in figure (12) if we consider only one species harvesting then infected prey and predator exists in the system thus disease cannot be controlled. Thus for controlling disease it is better to harvest both the prey and predator. So, harvesting has an important role in controlling oscillations as well as disease propagation in the species. Recently (16) used harvesting efforts in controlling of limit cycle oscillations as well as disease propagation in a predator-prey system with disease in prey only.

Our observations are also of relevance for biological control, as harvesting can be used as control agents of undesirable species such as biological invaders. This study interestingly suggests that harvesting can have been regulating effects on more than one trophic level and may be utilized for the management purposes in the multi-species systems. The harvesting cannot only control or eradicate the infected prey and the predator population but also allow the species to survive. For example, (16) observed that harvesting was not only possible to control the cyclic behavior of the system populations leading to the persistence of all species but other desired stable equilibrium, including disease-free can also be obtained. In our proposed system, we observe that harvesting of both the preys prevent limit cycle oscillation and combine effect of both harvesting also prevent disease propagation in the system. Here we explain the stabilizing mechanism in our system. The effect of harvesting is solely to increase the predator mortality which decreases the predator population size and the predation pressure on the prey. This in turn, increases the prey population size and the density dependence felt by the prey population which is a stabilizing factor. Thus harvesting indirectly couples the predator mortality with the prey population size. A similar inhibition of the predator population by high densities of the prey occurs in the presence of the toxic prey species (34).

In this paper, we have analyzed the ecological and the disease basic reproduction number. It can be defined as the expected number of off-spring a typical individual produces in its life or in epizootiology, as the expected number of secondary infectious produced by a single infective individual in a completely susceptible population during its entire infectious period. We will use reproduction numbers as helpful tools in determining the persistence or extinction of a species. This will allow us to categorize the community composition of prey, predators and disease. The threshold concept inherent in reproduction numbers has been used in previous studies of eco-epidemiological models (2; 3).

Fig. 13 Figure shows that infected prey and infected predator survives when only predator harvesting i.e. for \( h_1 = 0 \) and \( h_2 = 0, \lambda = 0.08, \beta = 0.0007 \) and other parameters fixed as given in Figure 2.
Fig. 14  Figure shows that infected prey and infected predator survives when only prey harvesting i.e. for $h_3 = 0$ and $h_4 = 0, \lambda = 0.08, \beta = 0.0007$ and other parameters fixed as given in Figure 2.

Now we will give a comparative study of our proposed system to the most of the earlier studies. (2) considered a predator-prey model with parasitic infection where the disease is allowed to cross the species barrier. Moreover, assuming that the predators could get infected by eating the prey and the prey could obtain the disease from parasites spread into the environment by the predators. They observed that in case where the predator cannot survive only on the prey in a disease-free environment, the parasitization could lead to persistence of the predator since the predators could only survive on the prey if some of the prey were more easily captured due to being diseased, provided a certain threshold for disease transmission is surpassed. Recently Hsieh and Hsiao (21) proposed a predator-prey model with disease in the both populations. They assumed that the predator is infected by consumption of the infected prey and the infected predator does not spread disease in the predator population. They analyzed the local stability of the equilibrium point by the help of the ecological as well as the disease basic reproduction numbers and concluded that the ecological reproduction number determines the coexistence of the predator and the prey while disease reproduction number dictates whether the disease will become endemic in the ecosystem. (19) considered a predator-prey model with disease circulating in both the populations and they also considered logistic growth of the infected prey population and assumed mass-action functional responses. They observed that the system will be disease free and all population will persist through proper predational strategies. In our proposed system we have considered a predator-prey model with disease in the both populations and we have also considered harvesting in each species. We have assumed both the prey and the predator are infected by some transmissible diseases (parasitic, viral and bacterial) but the predator can not get infection from the infected prey and only the infected predator spreads disease in the predator population and we have also assumed Holling type-II functional response for the susceptible prey and mass-action functional response for the infected prey. We have observed role of harvesting in controlling oscillations and disease propagation in the system. Hadeler and Freedman (2) and Hsieh and Hsiao (21) did not pay attention to the harvesting factor but harvesting is an important factor in our proposed system. Harvesting was studied by Bairagi et al. (16) and Chattopadhyay et al. (13) in the prey-predator system with disease in the prey population only and they observed that the reasonable harvesting can prevent oscillations and disease propagation. But in this study harvesting is considered in the predator-prey system where both populations are infected and we observe that reasonable harvesting helps system to enter into steady stable state from oscillation and it is also observed that combined effect of reasonable harvesting prevents disease propagation. We have also analyzed the community structure of the model system with the help of the basic reproduction numbers.
6 Conclusion

In the present article we have considered a four-dimensional predator-prey system with disease in the both populations and harvesting in each species. The main objective of this paper is to observe the role harvesting on the overall dynamics of the proposed system. We have derived the ecological and the disease basic reproduction numbers to analyze the local stability of the equilibrium points and obtained the conditions of permanence of the system. We have used reproduction numbers as helpful tools in determining the persistence or extinction of a species. We have studied the entire community classification completely on the basis of reproduction numbers. To observe global dynamics of our proposed model we have performed extensive numerical experiments. Limit cycle oscillation in a harvested predator-prey system are common in nature and control of such oscillation is utmost important from ecological as well as economical viewpoint. Our numerical results show that there exists an interval of the force of infection in the prey species, four species of our system coexist in oscillatory manner and the infected predator does not survive for higher values of the force of infection in the prey species. But propagation of disease transmission in the predator population helps to persist all four species in oscillating position for a threshold value of the force infection in the predator population and this observation is well agreement with other investigators (2; 24). The oscillatory behavior can be prevented by increasing the harvesting effort on both prey population. Thus, we may conclude that harvesting of the both prey may be used as a biological control for the persistence of all species in a stable steady state. In this connection we like to mention that (35) studied the effects of harvesting on Florida commercial sponge population. They concluded that unregulated harvesting might lead to a decline in the sponge population and affects the benthic community composition. Also there are certain reports about disease in the sponge population which effects the sponge to bleach from the base up until the whole sponge is completely white and then it just crumbles apart and ultimately kill it. The infection on sponge population also affects their predators. Thus the effect of disease in such a harvested prey-predator system is very much important in both ecological and economical point of view. Thus, a proper harvesting strategy is needed for the persistence of the species. We hope that our mathematical model and its result will be applicable in such system. The biological relevance of harvesting the infected populations and its applicability in real life situation has been in great demand over centuries. The apical factor for harvesting the infected populations is targeted to minimize the risk of infection transmissibility on endemic population. Endemic population under favorable environmental perturbation may replicate itself into an epidemic state. Henceforth, in the context of the problem culling/non-selective harvesting of the affected populations is a plausible phenomena to exclude disease from a population. Recent evidence of pre-emptive culling in the case of avian flu has been taken globally as mitigation strategies to resolve the pandemic problem globally. Such efforts are also being carried out in mitigating epidemics in fisheries, game reserves and other reasonable areas where conventional methods produces failure in containment of disease propagation.

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References


