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Stability Analysis and Optimal Measure for Controlling Eco-Epidemiological Dynamics of Prey-Predator Model

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Abstract: An eco-epidemiological model representing the interactions between prey and predator populations affected by a disease in an ecosystem is presented. The model is governed by a five-dimensional nonlinear system of ordinary differential equations coupling both ecological and epidemiological features of interacting populations. The well-posedness of the model is established with respect to positivity and boundedness of solutions. Conditions for asymptotic stability of different equilibrium points are extensively investigated to determine the existence and coexistence of prey and predator species using local linearization and Lyapunov functions techniques. Additionally, the analysis of the model is extended to assess the effects of three time-dependent control functions, such as disease prevention, treatment and alternative resource for predator, on the population dynamics of the prey-predator coexistence in the system.

Keywords: ecology, epidemiology, prey-predator model, stability, optimal control problem, simulations

1. INTRODUCTION

Interaction between organisms of different species is a common phenomenon in an ecosystem. It is an intrinsic feature which cannot be downplayed in the study of ecological system. This interaction, especially between prey and predator species, often affects or changes the population sizes of organisms in the system [11]. Since the advent of the classical Lotka and Volterra prey-predator models [20, 31], many ecosystem models, which can be defined as mathematical representations of ecological system of prey and predator interactions, have been studied in literature to better understand the real system (see, e.g. [5, 11, 13, 17, 27]). In [11], analysis of modified Lotka-Volterra model was carried out by taking into consideration an environmental case containing two related populations of prey and predator species having influence on the size of each other. Kumar and Kumari [17] studied the impact of fear on a chaotic model describing the interactions of the species in the food chain of one prey and two predators. Prasad *et al.* [27] investigated the role of mutual interference between predators on the dynamics of additional food provided predator-prey system.

In recent decades, a number of studies have considered ecosystems of interacting populations among which diseases spread. The resulting ecological systems are described by eco-epidemiological models [7–9, 21, 29]. Das [9] studied the predator-prey model with parasitic infection transmitting among the predator population only. Nandi *et al.* [21] developed a predator-prey system where prey species only are infected with a viral disease resulting into susceptible and two-stage infected classes. Bera *et al.* [8] presented theoretical analysis supported by simulation of the dynamical behaviors of a prey-predator system where both populations are affected by diseases.

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Interest has been growing recently in the application of optimal control theory to the dynamics of eco-epidemiological models for prey-predator system. AL-Nassir [6] studied a two-dimensional continuous prey-predator system incorporating an optimal control variable with the aim of reducing the number of the predator density in the population. In [10], the authors extended a prey-predator mathematical model with infection and harvesting on prey to include one time-dependent control for preventing infection in the prey population only. In another development, Hugo *et al.* [16] applied optimal control theory in minimizing the spread of Newcastle disease among chickens (prey) and humans (predator) populations using three control variables, such as vaccination of prey, education campaign and treatment of infected humans.

In this work, a five-dimensional nonlinear prey-predator model is studied to gain further insights into the dynamics of eco-epidemiological interactions between prey and predator populations. The analysis of the model is extended to investigate the impact of three optimal control variables, namely disease prevention in both prey and predator populations, treatment control for prey, and provision of alternative resources control for predator population. The rest of the work is organized as follows: The model is formulated and its well-posedness is established in Section 2. In Section 3, existence and stability of possible equilibrium points are investigated. In Section 4, the model is extended to explore optimal control dynamics with numerical simulations. Section 5 is devoted to concluding remarks.

2. MODEL FORMULATION

The dynamic eco-epidemiological prey-predator system is formulated by sub-dividing the prey population at time, t, into susceptible $X_s(t)$, infected $X_i(t)$ and recovered $X_r(t)$. On the other hand, the predator population is sub-divided into two, namely susceptible $Y_s(t)$ and infected $Y_i(t)$. It is assumed that the susceptible prey population increases logistically with intrinsic growth rate r and carrying capacity k. Following effective contact with the infected prey, the susceptible prey becomes infected at rate β_1. To avoid extinction of the prey population due to disease, the infected prey is allowed to recover at per capita rate γ. The natural mortality rate for the prey population is given by μ_1. Further, it is assumed that the susceptible predator, being healthy, has the capacity to attack and consume both susceptible and infected prey, while the recovered prey is assumed to be under cover from predation. It is also assumed that predators depend on prey for sustenance. The susceptible predator contracts disease from infected predator at rate β_2, while the infected predator, being unhealthy, has the capacity to attack and consume infected prey only as assumed in [8]. It has been assumed that only infected prey population recovers due to their accessibility to treatment. Hence, recovered class for predators is not considered. This assumption is based on some eco-epidemiological cases, for examples: carnivorous animals (predator) and humans (prey), and hawks (predator) and chicks (prey). The predation and conversion rates for the predator population are given, respectively, by P_i and C_i, for $i = 1, 2, 3$. The natural mortality rate for predator population is given by μ_2. Concise description of the variables and the parameters of the prey-predator system is presented in Table 2.1 and the nonlinear ordinary differential equations describing the interacting prey and predator populations is given by

$$\begin{aligned}
\frac{dX_s}{dt} =& rX_s\left(1-\frac{X_s}{k}\right)-\beta_1 X_s X_i - P_1 X_s Y_s - \mu_1 X_s \\
\frac{dX_i}{dt} =& \beta_1 X_s X_i - P_2 X_i Y_s - P_3 X_i Y_i - (\mu_1+\gamma)X_i \\
\frac{dX_r}{dt} =& \gamma X_i - \mu_1 X_r \\
\frac{dY_s}{dt} =& C_1 P_1 X_s Y_s + C_2 P_2 X_i Y_s - \beta_2 Y_s Y_i - \mu_2 Y_s \\
\frac{dY_i}{dt} =& \beta_2 Y_s Y_i + C_3 P_3 X_i Y_i - \mu_2 Y_i.
\end{aligned} \tag{2.1}$$

Table 2.1. The variables and parameters of the malaria model (2.1)

Variable	**Description**
$X_s(t)$	Susceptible (healthy) prey
X_i(t)	Infected (unhealthy) prey
X_r(t)	Recovered prey
$Y_s(t)$	Susceptible (healthy) predator
$Y_i(t)$	Infected (unhealthy) predator
Parameter	**Description**
r	Intrinsic growth rate
k	Carrying capacity
β_1	Disease transmission rate in prey
β_2	Disease transmission rate in predator
$P_i, i=1,2,3$	Predation coefficients
$C_i, i=1,2,3$	Conversion rates
γ	Recovery rate of infected prey
μ_1	Natural mortality rate of prey
μ_2	Natural mortality rate of predator

2.1. Well-posedness of the model

The mathematical and eco-epidemiological relevance of the prey-predator system (2.1) depends on the well-posedness of the model. Keeping in mind that all the parameters of the model are non-negative. Here, the boundedness and positivity of solutions of the model are investigated to establish the well-posedness of the model.

2.1.1. Boundedness of solutions

The following result is required to establish the boundedness of solutions of the model (2.1).

Lemma 2.1:
The susceptible prey population $X_s(t)$ is bounded.

Proof
It is clear from the first equation of the model (2.1) that

$$\frac{dX_s}{dt} \leq rX_s - \frac{X_s^2}{k}. \tag{2.2}$$

Solving the nonlinear first order differential inequality of Bernoulli type (2.2) yields

$$X_s(t) \leq \frac{kX_s(0)}{ke^{-rt} + X_s(0)(1 - e^{-rt})}.$$

It follows that the $\limsup X_s(t) < k$ as $t \to \infty$. □

Theorem 2.1:
The solutions of the system (2.1) *are uniformly bounded.*

Proof
Let the total prey and predator populations be represented, respectively, by X and Y, so that the total population of both species be $T = X + Y$. Then it follows that

$$\frac{dT}{dt} \leq rX_s - \mu_1 X - \mu_2 Y. \tag{2.3}$$

Using Lemma 2.1 and let $\mu = \min\{\mu_1, \mu_2\}$, then (2.3) becomes

$$\frac{dT}{dt} + \mu T \leq rX_s \leq rk,$$

which on integration gives

$$T(t) \leq \frac{rk}{\mu}(1 - e^{-\mu t}) + T(0)e^{-\mu t}.$$

Consequently, $\limsup T(t) < (rk/\mu) + \epsilon$, $\forall\ \epsilon > 0$ as $t \to \infty$. This ends the proof. □

2.1.2. Positivity of solutions

Theorem 2.2:
The solution set $\{X_s, X_i, X_r, Y_s, Y_i\}$ *of the eco-epidemiological model* (2.1) *with non-negative initial conditions,* $X_s(0)$*,* $X_i(0)$*,* $X_r(0)$*,* $Y_s(0)$*,* $Y_i(0)$ *in* Ω*, remain non-negative for all time* $t > 0$*.*

Proof
Since $X_s(t) < k$ as shown in Lemma 2.1, then $(1 - X_s/k) \geq 0$ with equality if the size of the susceptible prey is exactly its carrying capacity. Hence, the first equation of the model (2.1) implies that

$$\frac{dX_s(t)}{dt} + (\beta_1 X_i + P_1 Y_s + \mu_1)X_s(t) \geq 0, \tag{2.4}$$

which on using integrating factor gives

$$\frac{d}{dt}\left[X_s(t)\exp\left(\int_0^t (\beta_1 X_i(\phi) + P_1 Y_s(\phi))d\phi + \mu_1 t\right)\right] \geq 0. \tag{2.5}$$

Further integration of (2.5) gives

$$X_s(t) \geq X_s(0)\exp\left[-\left(\int_0^t (\beta_1 X_i(\phi) + P_1 Y_s(\phi))d\phi + \mu_1 t\right)\right] > 0, \quad \forall t > 0. \tag{2.6}$$

All other variables can be proved to be positive in a similar approach. □

Hence, it is sufficient to consider the dynamics of the flow generated by the system (2.1) in a feasible region defined by

$$\Omega = \left\{ (X_s, X_i, X_r, Y_s, Y_i) \in \mathbb{R}_+^5 : T < \frac{rk}{\mu} + \epsilon \right\}.$$

In the region Ω, the prey-predator model (2.1) can be said to be eco-epidemiologically well-posed.

3. ANALYSIS OF THE MODEL

In this section, the prey-predator model is analysed around the possible equilibrium points.

3.1. Existence of equilibrium points

The prey-predator model (2.1) has the following possible equilibrium points:

3.1.1. Trivial equilibrium point (E_0)
This is a steady state in the absence of prey and predator populations. Hence, no interactions exist. The equilibrium point is given by

$$E_0 = (0, 0, 0, 0, 0). \tag{3.7}$$

3.1.2. Axial equilibrium point (E_1)
This is a steady state where only the susceptible prey is present. In other words, it is the disease-free *cum* predator-free equilibrium point. It exists provided that the intrinsic growth rate r exceeds the natural mortality rate μ_1. The equilibrium point is given by

$$E_1 = \left(k \left[1 - \frac{\mu_1}{r} \right], 0, 0, 0, 0 \right). \tag{3.8}$$

3.1.3. Disease-free equilibrium point (E_2)
This is a steady state where there is no disease in both prey and predator populations. It exists provided the inequality $r \left(1 - \dfrac{\mu_2}{kC_1P_1} \right) > \mu_1$ holds. Hence, it is obtained as

$$E_2 = \left(\frac{\mu_2}{C_1P_1}, 0, 0, \frac{1}{P_1} \left[r \left(1 - \frac{\mu_2}{kC_1P_1} \right) - \mu_1 \right], 0 \right). \tag{3.9}$$

3.1.4. Predator-free equilibrium point (E_3)
This is a steady state where there is no predator in the system. It exists provided the inequality $r \left(1 - \left(\frac{\mu_1 + \gamma}{k\beta_1} \right) \right) > \mu_1$ holds. The equilibrium is given as

$$E_3 = \left(\frac{\mu_1 + \gamma}{\beta_1}, \frac{1}{\beta_1} \left[r \left(1 - \left(\frac{\mu_1 + \gamma}{k\beta_1} \right) \right) - \mu_1 \right], \frac{\gamma}{\beta_1\mu_1} \left[r \left(1 - \left(\frac{\mu_1 + \gamma}{k\beta_1} \right) \right) - \mu_1 \right], 0, 0 \right). \tag{3.10}$$

3.1.5. Infected prey-free equilibrium (E_4)
This is a steady state where the infected prey does not exist. The equilibrium will exist if $k > \frac{k}{r} \left(\frac{P_1\mu_2}{\beta_2} + \mu_1 \right)$ and $\frac{C_1P_1}{\beta_2} \left[k - \frac{k}{r} \left(\frac{P_1\mu_2}{\beta_2} + \mu_1 \right) \right] > \mu_2$. Thus, it is given by

$$E_4 = \left(k - \frac{k}{r} \left(\frac{P_1\mu_2}{\beta_2} + \mu_1 \right), 0, 0, \frac{\mu_2}{\beta_2}, \frac{C_1P_1}{\beta_2} \left[k - \frac{k}{r} \left(\frac{P_1\mu_2}{\beta_2} + \mu_1 \right) \right] - \mu_2 \right). \tag{3.11}$$

3.1.6. Infected predator-free equilibrium (E_5)
This is a steady state where the infected predator is absent in the ecosystem. The equilibrium is given by

$$E_5 = (X_s^*, X_i^*, X_r^*, Y_s^*, 0), \tag{3.12}$$

such that $X_s^* > 0, X_i^* > 0, X_r^* > 0, Y_s^* > 0$, and where

$$X_s^* = \frac{C_2[P_2(r-\mu_1) - P_1(\mu_1+\gamma)] - \beta_1\mu_2}{C_2P_2\left(\frac{1}{k} + \frac{P_1\beta_1}{P_2}\right) - C_1P_1\beta_1},$$

$$X_i^* = \frac{\mu_2}{C_2P_2} - \frac{C_1P_1}{C_2P_2}\left[\frac{C_2[P_2(r-\mu_1) - P_1(\mu_1+\gamma)] - \beta_1\mu_2}{C_2P_2\left(\frac{1}{k} + \frac{P_1\beta_1}{P_2}\right) - C_1P_1\beta_1}\right],$$

$$X_r^* = \frac{\gamma X_i^*}{\mu_1},$$

$$Y_s^* = \frac{\beta_1 X_s^* - (\mu_1+\gamma)}{P_2}.$$

3.1.7. Interior equilibrium point (E_6)
This is a steady state where all the populations for prey and predator species are present in the system. The equilibrium is given by

$$E_6 = (X_s^{**}, X_i^{**}, X_r^{**}, Y_s^{**}, Y_i^{**}), \tag{3.13}$$

such that $X_s^{**} > 0, X_i^{**} > 0, X_r^{**} > 0, Y_s^{**} > 0, Y_i^{**} > 0$, and where

$$X_s^{**} = \frac{1}{k}\left[(r-\mu_1) - X_i^{**}\left(\beta_1 - \frac{C_3P_1C_3}{\beta_2}\right)\right],$$

$$X_i^{**} = \frac{\beta_2[(P_2-P_3)\mu_2 + \beta_2(\mu_1+\gamma) - (r-\mu_1)(\beta_1\beta_2 - C_1P_1P_3)]}{\beta_2(P_2P_3C_3 + C_2P_2P_3) + (\beta_1\beta_2 - C_1P_1P_3)(\beta_1\beta_2 - C_3P_1P_3)},$$

$$X_r^{**} = \frac{\gamma X_i^{**}}{\mu_1},$$

$$Y_s^{**} = \frac{\mu_2 - C_3P_3X_i^{**}}{\beta_2},$$

$$Y_i^{**} = \frac{\beta_1 X_s^{**} - P_2Y_s^{**} - (\mu_1+\gamma)}{P_3}.$$

3.2. Stability analysis

The Jacobian matrix of the prey-predator system (2.1) is given by

$$\mathbb{J} = \begin{bmatrix} J_1 & -\beta_1X_s & 0 & -P_1X_s & 0 \\ \beta_1X_i & J_2 & 0 & -P_2X_i & -P_3X_i \\ 0 & \gamma & -\mu_1 & 0 & 0 \\ C_1P_1Y_s & C_2P_2Y_s & 0 & J_3 & -\beta_2Y_s \\ 0 & 0 & 0 & \beta_2Y_i & \beta_2Y_s + C_3P_3X_i - \mu_2 \end{bmatrix}, \tag{3.14}$$

where

$$J_1 = r - \frac{2rX_s}{k} - \beta_1 X_i - P_1 Y_s - \mu_1,$$
$$J_2 = \beta_1 X_s - P_2 Y_s - P_3 Y_i - (\mu_1 + \gamma),$$
$$J_3 = C_1 P_1 X_s + C_2 P_2 X_i - \beta_2 Y_i - \mu_2.$$

The stability of each of the equilibrium points is analysed by finding the eigenvalues of the Jacobian matrix (3.14) evaluated at each point.

3.2.1. Stability of E_0

The Jacobian matrix (3.14) is evaluated at the trivial equilibrium point (3.7). Hence, solving the corresponding characteristic equation $|\mathbb{J}(E_0) - \lambda \mathbb{I}_5)| = 0$, where λ is the eigenvalue and $\mathbb{I}_5$ is the identity matrix of order five, the following eigenvalues are obtained: $\lambda_1 = r - \mu_1, \lambda_2 = -\mu_1, \lambda_3 = -\mu_2, \lambda_4 = -(\mu_1 + \gamma), \lambda_5 = -\mu_2$.
It can be seen that all the eigenvalues are unconditionally negative except λ_1. Thus, E_0 will be stable if $r < \mu_1$ and unstable if $r > \mu_1$. This result is theorized as follows.

Theorem 3.1:
The trivial equilibrium point E_0 of the prey-predator model (2.1)*, given by* (3.7)*, is stable if $r < \mu_1$ and unstable if $r > \mu_1$.*

The implication of Theorem 3.1 is that the coexistence of prey-predator population will not occur if the intrinsic growth rate r does not exceed the natural mortality rate μ_1. In other words, stable equilibrium point E_0 will result into extinction of prey and predator species in the ecosystem.

3.2.2. Stability of E_1

The characteristic equation of the matrix (3.14) evaluated at the axial equilibrium point E_1 is given by $|\mathbb{J}(E_1) - \lambda \mathbb{I}_5)| = 0$, which gives the following eigenvalues: $\lambda_1 = \mu_1 - r$, $\lambda_2 = -\mu_1$, $\lambda_3 = \beta_1 k \left(1 - \frac{\mu_1}{r}\right) - (\mu_1 + \gamma)$, $\lambda_4 = C_1 P_1 k \left(1 - \frac{\mu_1}{r}\right) - \mu_2$, $\lambda_5 = -\mu_2$.
The stability result is theorized as follows.

Theorem 3.2:
The axial equilibrium point E_1 of the prey-predator model (2.1)*, given by* (3.8)*, is stable if the inequalities: $\mu_1 < r$, $\beta_1 k \left(1 - \frac{\mu_1}{r}\right) < (\mu_1 + \gamma)$ and $C_1 P_1 k \left(1 - \frac{\mu_1}{r}\right) < \mu_2$ hold.*

The implication of Theorem 3.2 is that the stable E_1 will support the existence of prey population only in the ecosystem provided these threshold conditions are satisfied:
$\mathcal{R}_{01} < 1$, $\mathcal{R}_{02} < 1$ and $\mathcal{R}_{03} < 1$, where

$$\mathcal{R}_{01} = \frac{\mu_1}{r},\ \mathcal{R}_{02} = \frac{\beta_1 k \left(1 - \frac{\mu_1}{r}\right)}{\mu_1 + \gamma},\ \text{and } \mathcal{R}_{03} = \frac{C_1 P_1 k \left(1 - \frac{\mu_1}{r}\right)}{\mu_2}.$$

Now, suppose $\mathcal{R}_0 = \max\{\mathcal{R}_{01}, \mathcal{R}_{02}, \mathcal{R}_{03}\}$, and considering the prey sub-model only such that the axial equilibrium point E_1 becomes $\tilde{E}_1 = (X_s^0, 0, 0)$, where $X_s^0 = k\left[1 - \frac{\mu_1}{r}\right]$. The following global stability result is proved.

Theorem 3.3:
The equilibrium point $\tilde{E}_1$ is globally asymptotically stable if $\mathcal{R}_0 < 1$.

Proof
Since $P_1 = 0$ in the absence of predation, then $\mathcal{R}_0 = \max\{\mathcal{R}_{01}, \mathcal{R}_{02}\}$. Consider the Lyapunov

function (combination of Goh-Volterra [2, 3, 13, 15, 22] and linear types) given by

$$\mathcal{L} = X_s - X_s^0 - X_s^0 \ln \frac{X_s}{X_s^0} + X_i. \tag{3.15}$$

The time derivative of Lyapunov function (3.15) along the trajectory of the prey only sub-model is given by

$$\dot{\mathcal{L}} = \left(1 - \frac{X_s^0}{X_s}\right) \frac{dX_s}{dt} + \frac{dX_i}{dt}. \tag{3.16}$$

Using $\frac{dX_s}{dt} = rX_s\left(1 - \frac{X_s}{k}\right) - \beta_1 X_s X_i - \mu_1 X_s$ and $\frac{dX_i}{dt} = \beta_1 X_s X_i - (\mu_1 + \gamma)X_i$ in (3.16) with $X_s \leq X_s^0$, it follows that

$$\begin{aligned} \dot{\mathcal{L}} &= (X_s - X_s^0)\left(r\left[1 - \frac{X_s}{k}\right] - \beta_1 X_i - \mu_1\right) + \beta_1 X_s X_i - (\mu_1 + \gamma)X_i \\ &\leq r\left[1 - \frac{X_s^0}{k}\right] - \beta_1 X_i - \mu_1 + \beta_1 X_s^0 X_i - (\mu_1 + \gamma)X_i. \end{aligned} \tag{3.17}$$

Since $r\left[1 - \frac{X_s^0}{k}\right] = \mu_1$ at the disease-free steady state of the prey only sub-model, then it follows from (3.17), by further simplifications, that

$$\dot{\mathcal{L}} \leq -\beta_1 X_i - (\mu_1 + \gamma)\left(1 - \frac{\beta_1 X_s^0}{\mu_1 + \gamma}\right) X_i. \tag{3.18}$$

Since $\mathcal{R}_{02} = \dfrac{\beta_1 X_s^0}{\mu_1 + \gamma}$, consequently (3.18) becomes

$$\dot{\mathcal{L}} \leq -\beta_1 X_i - (\mu_1 + \gamma)\left(1 - \mathcal{R}_{02}\right) X_i.$$

Therefore, $\dot{\mathcal{L}} \leq 0$ when $\mathcal{R}_{02} < 1$ with equality, $\dot{\mathcal{L}} = 0$, if and only if $X_i = 0$. It follows from LaSalle's Invariance Principle [19] that the largest invariant set contained in $\{(X_s, X_i, X_r) \in \mathbb{R}_+^3 : \dot{\mathcal{L}} = 0\}$ is the singleton set $\{\tilde{E}_1\}$. Hence, the equilibrium point $\tilde{E}_1 = (k[1 - (\mu_1/r)], 0, 0)$ is globally asymptotically stable. □

The implication of Theorem 3.3 is that the presence of disease in the prey population can be eliminated if $\mathcal{R}_{02} < 1$, irrespective of the initial sizes of the infected prey in the ecosystem. Thus, every solution of the prey only sub-model will always converge to the point $\tilde{E}_1$ whenever $\mathcal{R}_{02}$ is less than unity. This theoretical result is demonstrated in Figure 3.1.

3.2.3. Stability of E_2

Considering that there are only two disease classes in the prey-predator system (2.1), then using the notations in the next generation operator method [30], it follows that

$$F = \begin{bmatrix} \frac{\beta_1 \mu_2}{C_1 P_1} & 0 \\ 0 & \frac{\beta_2}{P_1} m_1 \end{bmatrix} \text{ and } V = \begin{bmatrix} \frac{P_2}{P_1} m_1 + \mu_1 + \gamma & 0 \\ 0 & \mu_2 \end{bmatrix},$$

where $m_1 = r\left(1 - \frac{\mu_2}{kC_1P_1}\right) - \mu_1$. Hence, the basic reproduction number of the full system (2.1), denoted by $\mathbf{R}_0^*$, is given by

$$\mathbf{R}_0^* = \rho(FV^{-1}) = \max\left\{\frac{\beta_1 \mu_2}{C_1 P_1\left(\frac{P_2}{P_1} m_1 + \mu_1 + \gamma\right)}, \frac{\beta_2 m_1}{P_1 \mu_2}\right\}. \tag{3.19}$$

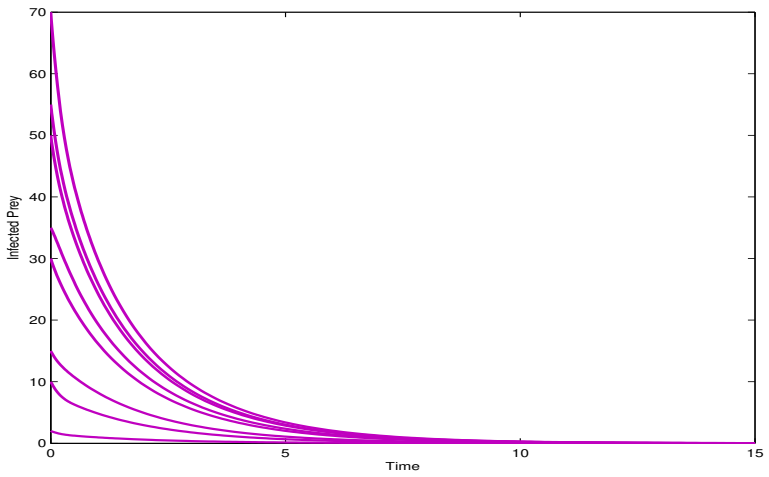


Fig. 3.1. Convergence of solution trajectories of prey sub-model to the equilibrium point $\tilde{E}_1$ at different initial sizes. The parameter values used are: $r = 11.2$, $\beta_1 = 0.04$, $\mu_1 = 0.85$, $\gamma = 0.75$, $k = 30$, so that $\mathcal{R}_{01} = 0.0759$ and $\mathcal{R}_{02} = 0.6931$, implying that $\mathcal{R}_0 = \mathcal{R}_{02} < 1$.

Therefore, by Theorem 2 of [30], the following stability result is claimed.

Theorem 3.4:
The disease-free equilibrium point E_2 *of the prey-predator model* (2.1)*, given by* (3.9)*, is stable provided that* $\mathbf{R}_0^* < 1$.

The implication of Theorem 3.4 is that the presence of few species around the equilibrium point E_2 will support the coexistence of both prey and predator species in a disease-free ecosystem whenever the basic reproduction number, $\mathbf{R}_0^*$, is less than unity. The global dynamics of the prey-predator around the disease-free equilibrium point E_2 is explored next.

Theorem 3.5:
The disease-free equilibrium point of the prey-predator model (2.1)*, given by* (3.9)*, is globally asymptotically stable if* $\mathbf{R}_0^* > 1$.

Proof
The proof is based on using the Lyapunov function defined by

$$\mathcal{F} = \frac{P_1}{P_2 m_1 + P_1(\mu_1 + \gamma)} X_i + \frac{1}{\mu_2} Y_i, \tag{3.20}$$

which when differentiated with respect to time, t, gives

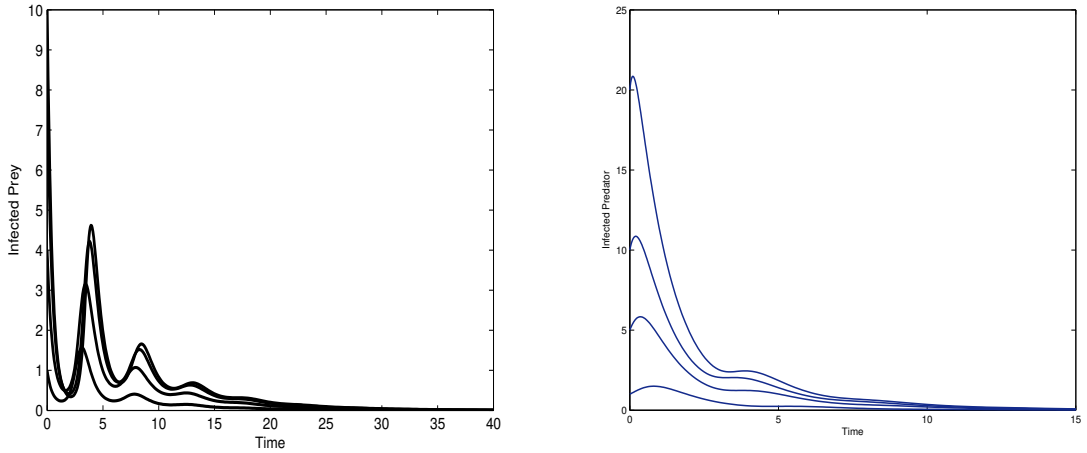
$$\begin{aligned}\frac{d\mathcal{F}}{dt} =& \frac{P_1}{P_2 m_1 + P_1(\mu_1 + \gamma)} [\beta_1 X_s X_i - P_2 X_i Y_s - P_3 X_i Y_i - (\mu_1 + \gamma) X_i] \\ &+ \frac{1}{\mu_2} (\beta_2 Y_s Y_i + C_3 P_3 X_i Y_i - \mu_2 Y_i).\end{aligned}$$

Since $X_s \leq \frac{\beta_1\mu_2}{C_1P_1}$ and $Y_s \leq \frac{1}{P_1}\left[r\left(1-\frac{\mu_2}{kC_1P_1}\right)-\mu_1\right]$, noting that $m_1 = r\left(1-\frac{\mu_2}{kC_1P_1}\right)-\mu_1$. Therefore, it follows, after simplification, that

$$\begin{aligned}
\frac{d\mathcal{F}}{dt} &\leq \left[\frac{\beta_1\mu_2}{C_1P_1\left(\frac{P_2}{P_1}m_1+\mu_1+\gamma\right)}-1\right]X_i - \frac{P_1P_3}{P_2m_1+P_1(\mu_1+\gamma)}X_iY_i \\
&\quad + \left[\frac{\beta_2m_1}{P_1\mu_2}-1\right]Y_i + \frac{1}{\mu_2}(C_3P_3X_iY_i) \\
&\leq \left[\max\left\{\frac{\beta_1\mu_2}{C_1P_1\left(\frac{P_2}{P_1}m_1+\mu_1+\gamma\right)}, \frac{\beta_2m_1}{P_1\mu_2}\right\}-1\right](X_i+Y_i) \\
&\quad - \left[\frac{P_1}{P_2m_1+P_1(\mu_1+\gamma)}-\frac{C_3}{\mu_2}\right]P_3X_iY_i \\
&= [\mathbf{R}_0^*-1](X_i+Y_i) - \left[\frac{P_1}{P_2m_1+P_1(\mu_1+\gamma)}-\frac{C_3}{\mu_2}\right]P_3X_iY_i.
\end{aligned}$$

Consequently, $d\mathcal{F}/dt \leq 0$ if $\mathbf{R}_0^* < 1$ and $X_i = 0$ or $Y_i = 0$. Thus, $X_s \to \mu_2/(C_1P_1)$, $X_r \to 0$ and $Y_s \to m_1/P_1$ as both $X_i \to 0$ and $Y_i \to 0$. Therefore, by LaSalle's invariance principle [19], the disease-free equilibrium point E_2 is globally asymptotically stable. □

The implication of Theorem 3.5 is that elimination of disease in the ecosystem is independent of initial sizes of infected prey and infected predator whenever $\mathbf{R}_0^* < 1$. This result is demonstrated in Figure 3.2(a) for infected prey and Figure 3.2(b) for infected predator population, where every solution trajectory converges to the disease-free equilibrium.



(a) (b)

Fig. 3.2. Convergence of solution trajectories of prey-predator model to the disease-free equilibrium point E_2 at different initial sizes. The parameter values used are: $mu_1 = 0.8$, $r = 3$, $\beta_1 = 0.4$, $P_1 = 1.0$, $\gamma = 0.35$, $P_2 = 0.2$, $P_3 = 1.0$, $\mu_2 = 0.85$, $k = 30$, $\beta_2 = 0.3$, $C_1 = 0.25$, $C_2 = 0.25$, $C_3 = 0.15$, so that $\mathbf{R}_0^* = 0.6565 < 1$.

3.2.4. Stability of E_3

Evaluating the Jacobian matrix (2.1) at predator-free equilibrium E_3 leads to the characteristic equation $|\mathbb{J}(E_3) - \lambda\mathbb{I}_5| = 0$. Thus, the following eigenvalues are obtained:

$$\lambda_1 = -\mu_1 \quad \lambda_2 = -\mu_2,$$

$$\lambda_3 = \frac{C_1P_1}{\beta_1}(\mu_1 + \gamma) - \frac{C_2P_2}{\beta_1}(m_2 + \mu_2),$$

$$\lambda_4 = -\frac{r}{2}\left(\frac{\mu_1+\gamma}{k\beta_1}\right) + \frac{1}{2}\sqrt{r^2\left(\frac{\mu_1+\gamma}{k\beta_1}\right)^2 + 4m_2(\mu_1+\gamma)},$$

$$\lambda_5 = -\frac{r}{2}\left(\frac{\mu_1+\gamma}{k\beta_1}\right) - \frac{1}{2}\sqrt{r^2\left(\frac{\mu_1+\gamma}{k\beta_1}\right)^2 + 4m_2(\mu_1+\gamma)},$$

where $m_2 = r\left(1 - \frac{\mu_1+\gamma}{k\beta_1}\right) - \mu_1$. The stability of E_3 is theorized as follows

Theorem 3.6:
The predator-free equilibrium point E_3 of the prey-predator model (2.1)*, given by* (3.10)*, is stable if the following conditions hold*

$$C_1P_1(\mu_1+\gamma) < C_2P_2(m_2+\mu_2) \text{ and } \sqrt{r^2\left(\frac{\mu_1+\gamma}{k\beta_1}\right)^2 + 4m_2(\mu_1+\gamma)} < r\left(\frac{\mu_1+\gamma}{k\beta_1}\right).$$

This result implies that stable E_3 will not be able to support the coexistence of both prey and predator populations. The presence of few species around the predator-free equilibrium point E_3 will make predator population to go extinct in the ecosystem.

3.2.5. Stability of E_4

The Jacobian matrix (3.14) is evaluated at the infected prey-free equilibrium point (3.11). Hence, solving the corresponding characteristic equation $|\mathbb{J}(E_4) - \lambda\mathbb{I}_5)| = 0$ gives one eigenvalue, $\lambda_1 = -\mu_1$, and the remaining four eigenvalues are obtainable from

$$\lambda^4 + W_1\lambda^3 + W_2\lambda^2 + W_3\lambda + W_4 = 0, \tag{3.21}$$

where

$$W_1 = r - \mu_1 - P_1\frac{\mu_2}{\beta_2},$$

$$W_2 = d_1d_2 - 2\mu_2(d_1+d_2) + \mu^2 + C_1P_1d_3 - C_1P_1\frac{\mu_2}{\beta_2}$$

with $d_1 = r - 2r\frac{d_3}{k} - P_1\frac{\mu_2}{\beta_2} - \mu_1$, $d_2 = \beta_1d_3 - P_2\frac{\mu_2}{\beta_2} - C_1P_1P_3\frac{d_3}{\beta_2} - (\mu_1+\mu_2+\gamma)$ and $d_3 = k - \frac{k}{r}\left(\frac{P_1\mu_2}{\beta_2} + \mu_2\right)$.

$$W_3 = C_1P_1\left[(d_2-\mu_2)\frac{\mu_2}{\beta_2} - d_3(d_2\mu_2 + d_1)\right] - d_2\mu_2(2d_1+\mu_2)$$

and

$$W_4 = d_2\mu_2^2\left(d_1 + \frac{P_1}{\beta_2}\right).$$

Applying Routh-Hurwitz's criterion, the roots of (3.21) will have negative real parts if $W_1 > 0$, $W_1W_2 > W_3$, $W_1W_2W_3 > W_1^2W_4 + W_3^2$. The stability result is hereby theorized as follows.

Theorem 3.7:
The infected prey-free equilibrium of the prey-predator model (2.1)*, given by* (3.11)*, is stable if* $W_1 > 0$*,* $W_1W_2 > W_3$ *and* $W_1W_2W_3 > W_1^2W_4 + W_3^2$.

The implication of Theorem 3.7 is that the presence of few species around the stable infected prey-free equilibrium E_4 will support the coexistence of both prey and predator populations in the ecosystem.

3.2.6. Stability of E_5

Two of the eigenvalues of the Jacobian matrix (3.14) evaluated at the infected predator-free equilibrium point E_5 are given by $\lambda_1 = -\mu_1$ and $\lambda_2 = -\mu_2$, while the remaining can be obtained from the characteristic equation

$$\lambda^3 + e_1\lambda^2 + e_2\lambda + e_3 = 0, \tag{3.22}$$

where $e_1 = -(f_1 + f_2 + f_3)$, with $f_1 = 2C_1P_1\tilde{f}_1 - \mu_2(1 + C_2P_2)$ such that

$$\tilde{f}_1 = \frac{C_2P_2(r - \mu_1) - P_1C_2(\mu_1 + \gamma) - \beta\mu_2}{C_2P_2\left(\frac{1}{k} + \frac{P_1\beta_1}{P_2}\right) - C_1P_1\beta_1},$$

$$f_2 = r - \mu_2 - \left[\frac{2r}{k} + \frac{C_2P_2 + kP_1\beta_1(C_2 - C_1)}{k(C_2P_2 + kP_1C_2 - C_1P_1\beta_1)}\right]\tilde{f}_1 - \beta_1\left(\mu_2 - \frac{C_1P_1}{C_2P_2}\tilde{f}_1\right)(1 - P_1)$$

$$f_3 = \beta_1\tilde{f}_1 - P_2\left[\left(\frac{C_2P_2 + kP_1\beta_1(C_2 - C_1)}{k(C_2P_2 + kP_1C_2 - C_1P_1\beta_1)}\right)\tilde{f}_1 - \beta_1\left(\mu_2 - \frac{C_1P_1}{C_2P_2}\tilde{f}_1\right)\right] - (\mu_1 + \gamma).$$

$e_2 = f_1f_2 + f_3(f_1 + f_2) + \beta_1^2\tilde{f}_1\left(\mu_2 - \frac{C_1P_1}{C_2P_2}\tilde{f}_1\right) + P_1^2\frac{C_1}{P_2}\tilde{f}_1(f_3 + (\mu_1 + \gamma) - \beta_1\tilde{f}_1)$, $e_3 = \frac{1}{P_2}(f_3 + (\mu_1 + \gamma) - \beta_1\tilde{f}_1)\left[\left(\mu_2 - \frac{C_1P_1}{C_2P_2}\tilde{f}_1\right)(P_2(C_2[P_1\tilde{f}_1 - P_2] - C_1P_1)) - C_1P_1^2\tilde{f}_1(f_3 + f_1f_2)\right]$.

By Routh-Hurwitz's criterion, the roots of the characteristic equation (3.22) will have negative real parts if $e_1 > 0$ and $e_1e_2 > e_3$. This result is stated as follows.

Theorem 3.8:
The infected predator-free equilibrium point of the prey-predator system (2.1)*, given by* (3.12)*, is stable if* $e_1 > 0$ *and* $e_1e_2 > e_3$.

The result in Theorem 3.8 implies that coexistence of prey and predator species in the ecosystem is possible in the absence of infected predator, provided that the given parametric conditions are valid.

3.2.7. Stability of E_6

Recall from (3.19) that the basic reproduction number of the full system (2.1) is given by

$$\mathbf{R}_0^* = \max\left\{\frac{\beta_1\mu_2}{C_1P_1\left(\frac{P_2}{P_1}m_1 + \mu_1 + \gamma\right)}, \frac{\beta_2m_1}{P_1\mu_2}\right\}.$$

The following global stability result is explored.

Theorem 3.9:
The interior equilibrium point E_6 of the prey-predator model (2.1) *is globally asymptotically stable if* $\mathbf{R}_0^* > 1$.

Proof
Let $\mathbf{R}_0^* > 1$, such that the interior equilibrium point E_6 exists. Noting from (3.13) that $E_6 = (X_s^{**}, X_i^{**}, X_r^{**}, Y_s^{**}, Y_i^{**})$, then the following Lyapunov function of quadratic type [28] is defined:

$$\mathcal{V} = \frac{1}{2}[(X_s - X_s^{**}) + (X_i - X_i^{**}) + (X_r - X_r^{**}) + (Y_s - Y_s^{**}) + (Y_i - Y_i^{**})]^2 \quad (3.23)$$

The time derivative of $\mathcal{V}$ in (3.23) along the solution path of the full system (2.1) is given by

$$\dot{\mathcal{V}} = [(X_s - X_s^{**}) + (X_i - X_i^{**}) + (X_r - X_r^{**}) + (Y_s - Y_s^{**}) + (Y_i - Y_i^{**})]\frac{dT}{dt}, \quad (3.24)$$

where $T(t) = X_s + X_i + X_r + Y_s + Y_i$. Recall from (2.3) of the boundedness result (Theorem 2.1) that

$$\frac{dT}{dt} \leq rk - \mu T, \text{ and since } X_s^{**} + X_i^{**} + X_r^{**} + Y_s^{**} + Y_i^{**} = \frac{rk}{\mu}.$$

It follows from (3.24) that

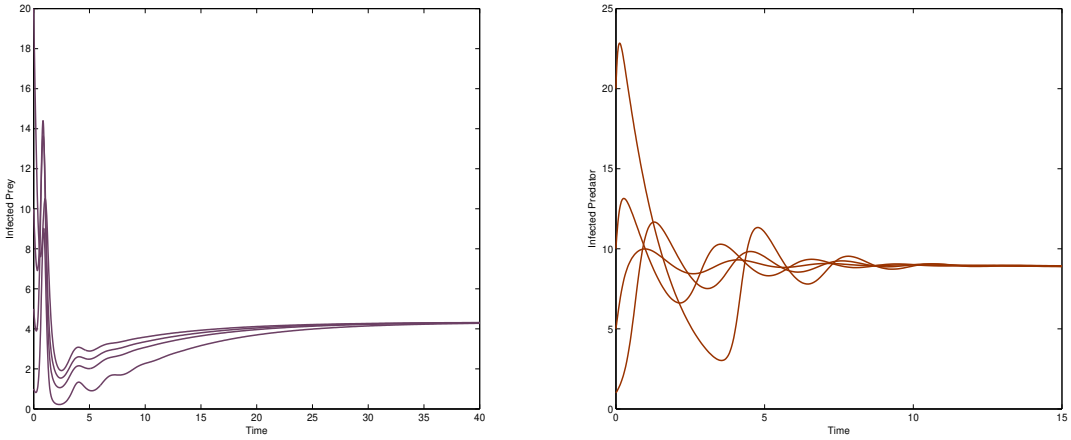
$$\begin{aligned}
\dot{\mathcal{V}} &\leq \left[T(t) - \frac{rk}{\mu}\right][rk - \mu T(t)] \\
&= \mu\left[T(t) - \frac{rk}{\mu}\right]\left[\frac{rk}{\mu} - T(t)\right] \\
&= -\mu\left[T(t) - \frac{rk}{\mu}\right]^2 \\
&= -\mu[(X_s - X_s^{**}) + (X_i - X_i^{**}) + (X_r - X_r^{**}) + (Y_s - Y_s^{**}) + (Y_i - Y_i^{**})]^2.
\end{aligned}$$

It can be seen that $\dot{\mathcal{V}} \leq 0$ with equality if and only if $X_s = X_s^{**}$, $X_i = X_i^{**}$, $X_r = X_r^{**}$, $Y_s = Y_s^{**}$ and $Y_i = Y_i^{**}$. Hence, by LaSalle's invariance principle [19], the largest invariant set in $\{(X_s, X_i, X_r, Y_s, Y_i) \in \mathbb{R}_+^5 : \dot{\mathcal{V}} = 0\}$ is the only set $\{E_6\}$. Thus, the interior equilibrium point $E_6 = (X_s^{**}, X_i^{**}, X_r^{**}, Y_s^{**}, Y_i^{**})$ is globally asymptotically stable. This completes the proof. □

The implication of Theorem 3.9 is that coexistence of prey and predator species in the ecosystem is possible while disease persists in the population. The presence of disease in the ecosystem, such that $\mathbf{R}_0^* > 1$, is independent of initial population sizes of both infected prey and predator species. This global stability result is depicted in Figure 3.3(a) for infected prey population and Figure 3.3(b) for infected predator population. The phase portrait which illustrates the stable coexistence of prey and predator populations is presented in Figure 3.4.

4. OPTIMAL CONTROL MODEL

In order to enhance the coexistence of both prey and predator species in the ecosystem, the prey-predator model (2.1) is extended by incorporating the following three time-dependent optimal control functions:



(a) (b)

Fig. 3.3. Convergence of solution trajectories of prey-predator model to the interior equilibrium point E_6 at different initial sizes. The parameter values used are: $mu_1 = 0.5, r = 11.2, \beta_1 = 0.4, P_1 = 1.0, \gamma = 0.35, P_2 = 0.2, P_3 = 1.0, \mu_2 = 0.65, k = 30, \beta_2 = 0.65, C_1 = 0.25, C_2 = 0.25, C_3 = 0.15$, so that $\mathbf{R}_0^* = 9.7293 > 1$.

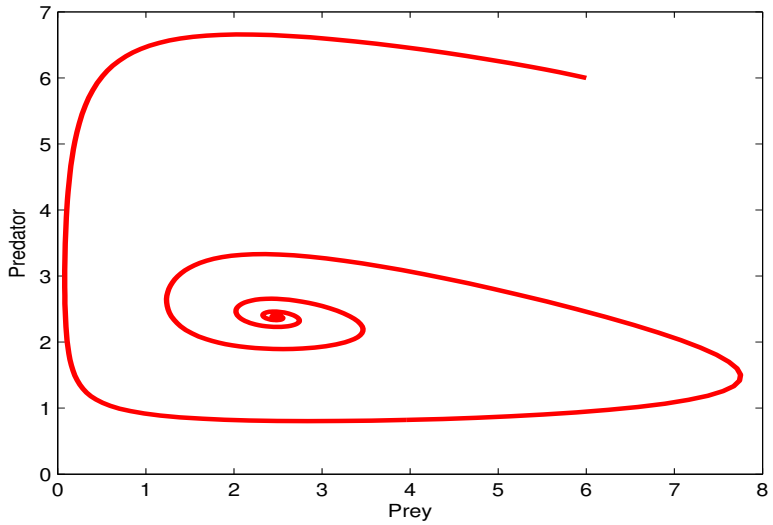


Fig. 3.4. Phase portrait of predator against prey showing stable coexistence of both populations around the interior (positive) equilibrium point.

1. The control function $u_1(t) \in [0, 1]$, which represents disease prevention measure for both prey and predator populations.
2. The function $u_2(t) \in [0, 1]$, which represents treatment control for prey population.
3. The control function $u_3(t) \in [0, 1]$ representing alternative source of food for predator survival.

Therefore, the prey-predator model (2.1) becomes a non-autonomous system of ordinary differential equations of the form:

$$\begin{aligned}
\frac{dX_s}{dt} &= rX_s\left(1-\frac{X_s}{k}\right)-(1-u_1(t))\beta_1 X_s X_i - P_1 X_s Y_s - \mu_1 X_s \\
\frac{dX_i}{dt} &= \beta_1 X_s X_i - P_2 X_i Y_s - P_3 X_i Y_i - (\mu_1+\gamma)X_i - u_2(t) r_0 X_i \\
\frac{dX_r}{dt} &= \gamma X_i + u_2(t) r_0 X_i - \mu_1 X_r \\
\frac{dY_s}{dt} &= C_1 P_1 X_s Y_s + C_2 P_2 X_i Y_s - (1-u_1(t))\beta_2 Y_s Y_i - \mu_2 Y_s + u_3(t) c_0 Y_s \\
\frac{dY_i}{dt} &= (1-u_1(t))\beta_2 Y_s Y_i + C_3 P_3 X_i Y_i - \mu_2 Y_i,
\end{aligned} \tag{4.25}$$

where r_0 and c_0 are the rate constants for the optimal controls $u_2(t)$ and $u_3(t)$, respectively. Since the goal is to reduce the risk of both prey and predator species going extinct, such that the populations of infected prey and predator are minimized at minimum costs possible, then the following objective functional is formed

$$\mathbf{J} = \int_0^{t_f} \left(A_1 X_i + A_2 Y_i + \frac{1}{2}[B_1 u_1^2 + B_2 u_2^2 + B_3 u_3^2] \right) dt, \tag{4.26}$$

where A_1, A_2, B_1, B_2 and B_3 are the positive weight constants for balancing the terms in the objective functional over the time interval $[0, t_f]$. Further, the cost function associated with the disease prevention measure for prey and predator populations is represented by the term $1/2B_1u_1^2$, while the treatment measure for prey population and the provision of alternative food source for predator are, respectively, represented by the terms $1/2B_2u_2^2$ and $1/2B_3u_3^2$. The objective functional is made of nonlinear cost on controls of quadratic type in line with the standards for optimal control problems (see, e.g. [1, 4, 14, 16, 24, 25]).

Therefore, the optimal control problem for the prey-predator system (4.25) is to seek a control triple $u^* = (u_1^*, u_2^*, u_3^*,)$, such that

$$\mathbf{J}(u^*) = \min\left\{ J(u_1, u_2, u_3) : u_1, u_2, u_3 \in \mathcal{U} \right\}, \tag{4.27}$$

where $\mathcal{U} = \{u_i(t) : 0 \leq u_i(t) \leq 1,$ Lebesgue measurable, $t \in [0, t_f],$ for $i = 1, 2, 3\}$ is a non-empty set of all admissible controls.

4.1. Analysis of the control model

The necessary conditions that must be satisfied by the optimal control triple u^* for the minimization problem are derivable from Pontryagin's Maximum Principle [26]. This principle converts the problem in (4.27) with (4.26) subject to the state system (4.25) into a problem of minimizing pointwise a Hamiltonian $\mathbf{H}$, with respect to $u_1(t)$, $u_2(t)$ and $u_3(t)$. Thus, the Hamiltonian for the control problem is given by

$$\begin{aligned}\mathbf{H} = \; & A_1X_i + A_2Y_i + \tfrac{1}{2}[B_1u_1^2 + B_2u_2^2 + B_3u_3^2] \\ & +\eta_1\left[rX_s\left(1-\frac{X_s}{k}\right) - (1-u_2(t))\beta_1X_sX_i - P_1X_sY_s - \mu_1X_s\right] \\ & +\eta_2\left[\beta_1X_sX_i - P_2X_iY_s - P_3X_iY_i - (\mu_1+\gamma)X_i - u_2(t)r_0X_i\right] \\ & +\eta_3[\gamma X_i + u_2(t)r_0X_i - \mu_1X_r] \\ & +\eta_4[C_1P_1X_sY_s + C_2P_2X_iY_s - (1-u_1(t))\beta_2Y_sY_i - \mu_2Y_s + u_3(t)c_0Y_s] \\ & +\eta_5[(1-u_1(t))\beta_2Y_sY_i + C_3P_3X_iY_i - \mu_2Y_i],\end{aligned} \tag{4.28}$$

where η_1, η_2, η_3, η_4 and η_5 are the adjoint or co-state variables corresponding to the state variables X_s, X_i, X_r, Y_S and Y_i, respectively. Applying the existence results as given in [2, 12], the following result is obtained.

Theorem 4.1:
For the optimal control triple u^* that minimizes the objective functional (4.26) *over $\mathcal{U}$ subject to* (4.25)*, there exist adjoint variables η_1, η_2, η_3, η_4 and η_5 satisfying the system governing the adjoint variables*

$$\begin{aligned}\tfrac{d\eta_1}{dt} = \; & \tfrac{2rX_s}{k}\eta_1 + (1-u_1)\beta_1X_i(\eta_1-\eta_2) + P_1Y_s(\eta_1 - C_1\eta_4) + (\mu_1 - r)\eta_1 \\ \tfrac{d\eta_2}{dt} = \; & (1-u_1)\beta_1X_s(\eta_1-\eta_2) + P_2Y_s(\eta_2 - C_2\eta_4) + P_3Y_i(\eta_2 - C_3\eta_5) \\ & +(\gamma + u_2r_0)(\eta_2-\eta_3) + \mu_1\eta_2 - A_1 \\ \tfrac{d\eta_3}{dt} = \; & \mu_1\eta_3 \\ \tfrac{d\eta_4}{dt} = \; & P_1X_s(\eta_1 - C_1\eta_4) + P_2X_i(\eta_2 - C_2\eta_4) + (1-u_1)\beta_2Y_i(\eta_4-\eta_5) \\ & +(\mu_2 - u_3c_0)\eta_4 \\ \tfrac{d\eta_5}{dt} = \; & P_3X_i(\eta_2 - C_3\eta_5) + (1-\mu_1)\beta_2Y_s(\eta_4-\eta_5) + \mu_2\eta_5 - A_2\end{aligned} \tag{4.29}$$

and with the transversality conditions

$$\eta_1(t_f) = \eta_2(t_f) = \eta_3(t_f) = \eta_4(t_f) = \eta_5(t_f) = 0. \tag{4.30}$$

Moreover, the optimal control triple (u_1^*, u_2^*, u_3^*) is characterized as follows

$$\begin{aligned}u_1^* = \; & \max\left\{0, \min\left\{1, \tfrac{1}{B_1}\left[\beta_1X_sX_i(\eta_2-\eta_1) + \beta_2Y_sY_i(\eta_5-\eta_4)\right]\right\}\right\}, \\ u_2^* = \; & \max\left\{0, \min\left\{1, \tfrac{1}{B_2}[r_0X_i(\eta_2-\eta_3)]\right\}\right\}, \\ u_3^* = \; & \max\left\{0, \min\left\{1, \tfrac{-c_0Y_s\eta_4}{B_3}\right\}\right\}.\end{aligned} \tag{4.31}$$

Proof
The system (4.29) governing the adjoint variables is obtained by finding the partial derivative of the Hamiltonian $\mathbf{H}$ (4.28) as follows:

$$\tfrac{d\eta_1}{dt} = -\tfrac{\partial\mathbf{H}}{\partial X_s}, \quad \tfrac{d\eta_2}{dt} = -\tfrac{\partial\mathbf{H}}{\partial X_i}, \quad \tfrac{d\eta_3}{dt} = -\tfrac{\partial\mathbf{H}}{\partial X_r}, \quad \tfrac{d\eta_4}{dt} = -\tfrac{\partial\mathbf{H}}{\partial Y_s}, \quad \tfrac{d\eta_5}{dt} = -\tfrac{\partial\mathbf{H}}{\partial Y_i}$$

with the transversality conditions:

$$\eta_1(t_f) = \eta_2(t_f) = \eta_3(t_f) = \eta_4(t_f) = \eta_5(t_f) = 0.$$

Finally, differentiating the Hamiltonian $\mathbf{H}$ partially with respect to each of the three controls u_1, u_2 and u_3, so that

$$\frac{\partial \mathbf{H}}{\partial u_1} \equiv B_1 u_1 + \beta_1 X_s X_i(\eta_1 - \eta_2) + \beta_2 Y_s Y_i(\eta_4 - \eta_5) = 0,$$

$$\frac{\partial \mathbf{H}}{\partial u_2} \equiv B_2 u_2 + r_0 X_i(\eta_3 - \eta_2) = 0,$$

$$\frac{\partial \mathbf{H}}{\partial u_3} \equiv B_3 u_3 + c_0 Y_s \eta_4 = 0,$$

which when solved on the interior of the control set $\mathcal{U}$ gives the required characterization (4.31). Hence, the proof. □

4.2. Simulations of the control model

Here, the numerical simulations of the coupled system of state equations (4.25) with adjoint equations (4.29) are conducted. The standard iterative fourth order forward-backward Runge-Kutta method is used to solve the optimality system due to difference in time orientations of the state and adjoint equations [18, 23]. The optimal control problem is simulated over the time interval [0, 15] in years using these parameter values: $\mu_1 = 0.5$, $r = 11.2$, $\beta_1 = 0.4$, $P_1 = 1.0$, $\gamma = 0.35$, $P_2 = 0.2$, $P_3 = 1.0$, $\mu_2 = 0.65$, $k = 30$, $\beta_2 = 0.65$, $C_1 = 0.25$, $C_2 = 0.25$, $C_3 = 0.15$. In addition, the balancing weight constants are given as $A_1 = 1$, $A_2 = 0.1$, $B_1 = 0.01$, $B_2 = 0.0001$ and $B_3 = 0.008$. The rate constants for the controls are chosen as $r_0 = 0.4$ and $c_0 = 0.15$.

Thus, to minimize the objective functional (4.26) with disease prevention control only at low cost of implementation, the control u_1 should be maintained at maximum (100%) effort for 8 years before being dropped to zero in final time as shown in the Figure 4.5(a). The control profile shown in Figure 4.5(b) indicates that single implementation of treatment control u_2 requires maximum (100%) effort throughout the period of the control intervention. It is observed in Figure 4.5(c) that alternative food source control u_3 should be at the upper bound for 14 years before coming to the lower bound. The combined implementation of the three controls for minimizing the objective functional is depicted in Figure 4.5(d), where it is can be observed that prevention control u_1 should be maintained at the upper bound for 8 years before declining to the lower bound, treatment control u_2 and provision of alternative food source u_3 should be maintained at maximum (100%) for about 2 years and almost 1 year, respectively.

The effects of combining all the three optimal controls on the dynamical behaviors of prey and predator populations are illustrated in Figure 4.6. The size of susceptible prey population with control reduces due to predation when compared with the size without control, but the population does not go extinct due to the presence of the combined optimal control as shown in Figure 4.6(a). This suggests the possibility of stable coexistence of predators with low density of prey population as indicated in [27]. The infected prey population with optimal control (Figure 4.6(b)) decreases sharply when compared with the case without control. Further, in Figure 4.6(c), it can be seen that the size of susceptible predator population with combined optimal control is higher than the size without control. Whereas, the presence of the combined optimal control reduces the population size of the infected predator in the ecosystem.

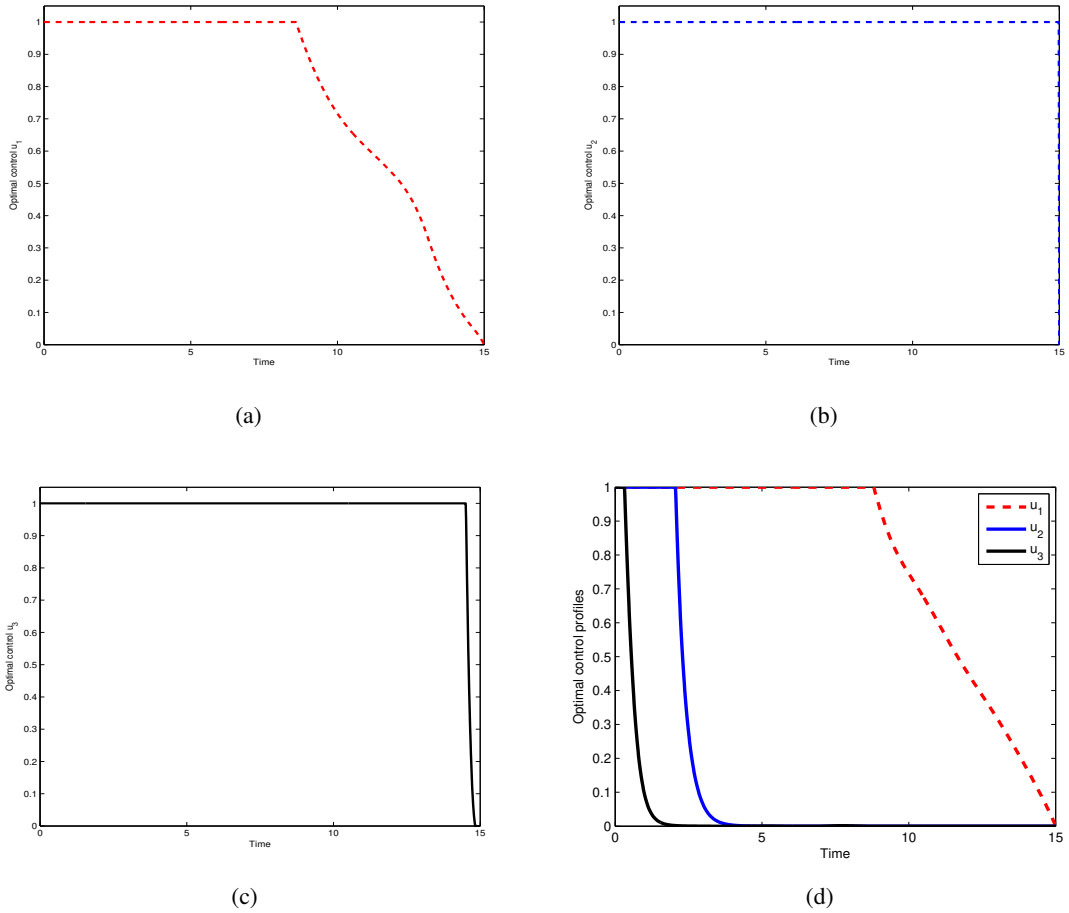


Fig. 4.5. Control profiles for single and combined implementations of u_1, u_2 and u_3.

5. CONCLUSION

A nonlinear mathematical model of prey-predator interacting populations in an ecological system with disease spread is studied. The five-dimensional eco-epidemiological model is analyzed with a view to providing insights into the behavior of ecosystem in the presence of disease. Seven possible equilibrium points, namely trivial, axial, disease-free, predator-free, infected prey-free, infected predator-free and interior equilibrium points are analytically determined. Asymptotic stability of the eco-epidemiological system is analyzed to investigate the behavior of the system around each of the obtained equilibrium points. Some conditions that guarantee the existence of each prey and predator species, as well as coexistence of both prey and predator populations are given using local linearization and Lyapunov functions techniques.

In addition, the eco-epidemiological model is extended to include three time-dependent optimal controls, such as disease prevention in both species, treatment strategy for prey and provision of alternative food source for predator survival. Hence, the optimal control prey-predator model is analyzed using Pontryagin's maximum principle in order to enhance the coexistence of both prey and predator species by reducing the risks of their extinction in the ecosystem, such that the populations of infected prey and predator species are minimized at minimum costs possible.

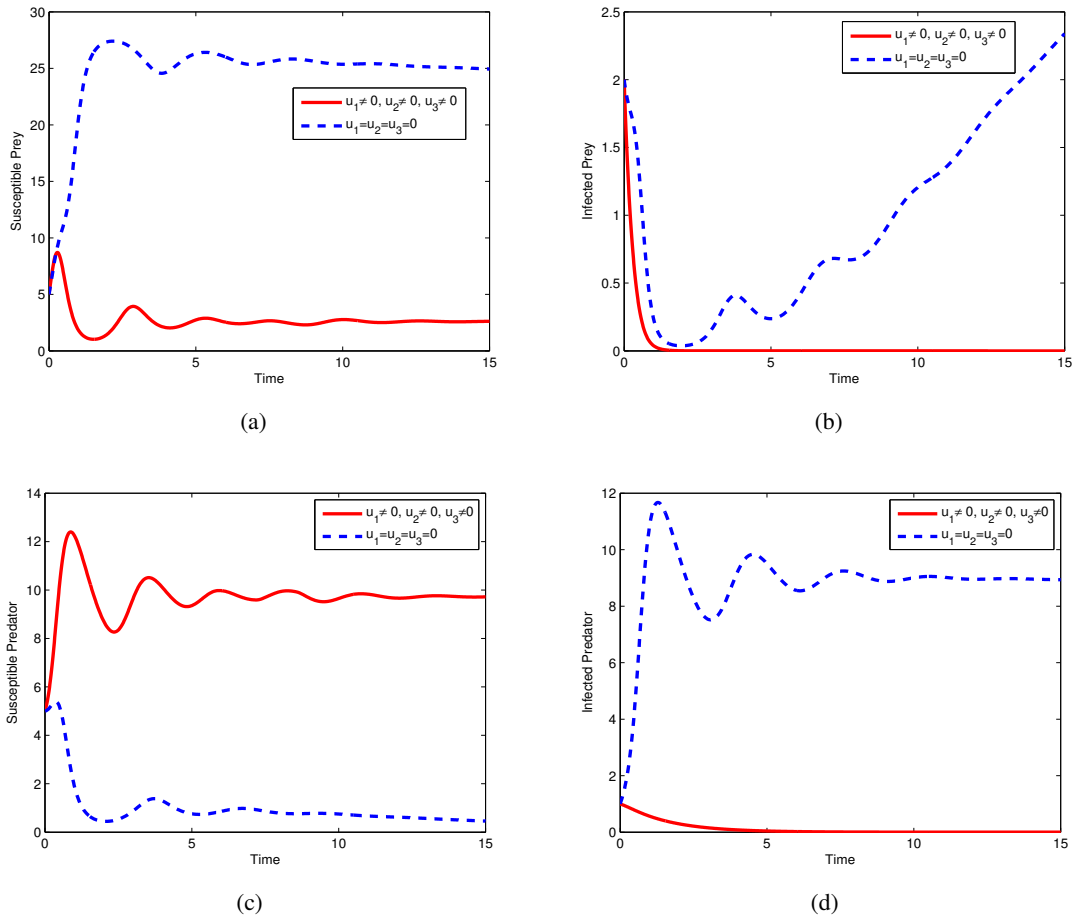


Fig. 4.6. Combined effects of optimal controls u_1, u_2 and u_3 on the dynamics of the prey-predator model.

The analysis of the eco-epidemiological model presented in this study can be applied to a wide spectrum of interactions between different kinds of living beings in the environment.

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