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Research Article

Mathematical Modeling of Infectious Disease and Prey-Predator Interaction with Optimal Control

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In this paper, the impact of viral illnesses on the predator-prey relationship with an optimal control analysis is studied. An ecoepidemiological model of four compartments, namely, susceptible prey, susceptible predator, infected prey, and infected predator populations, in the interaction of the prey-predator system is formulated. The fundamental tenet of our ecoepidemiology model is that sick predators do not engage in predation. It is confirmed that the system's solution exists, is positive, and is bounded. The system's equilibrium points are determined and computed. Lyapunov functions and a linearizing form are used for local and global stability analysis, respectively. The next generation matrix approach is used to calculate the threshold value for diseased predators and prey at the disease-free equilibrium point. Optimal treatment options for vulnerable and infected populations are established by applying optimal control theory to the ecoepidemiology model of a prey-predator system. MATLAB software is utilized to obtain numerical simulations that validate the analytical outcomes. The optimal control problem simulations demonstrate that the number of infected populations in a given prey-predator system can be decreased by implementing control measures.

1. Introduction

Mathematical modeling is a vital process to understand and solve problems in diverse fields of study such as epidemiology, ecology, and ecoepidemiology [1, 2]. The foundation of many mathematical models used in population dynamics is the Malthus economic growth model and the Lotka–Volterra prey-predator models [3]. The prey-predator model then rose to fame and developed into a strong field of applied mathematics research [2, 4].

These days, mathematical ecology and mathematical epidemiology are integrated applied mathematics subfields that have combined to form a new field of study known as mathematical ecoepidemiology [2, 5, 6]. In a given ecosystem, regular cycles of prey and predator populations fluctuate due to external factors like infectious diseases, secondary predation, or climate changes [7–9]. Interaction of prey-predator systems is a nonlinear and complex phenomenon; because of its complexity, a number of ecoepidemiological studies are conducted by including different assumptions in their interaction [2, 7, 8]. A variety of infectious diseases have plagued prey-predator populations. These include *Pasteurella multocida*, avian pox, Newcastle disease, influenza, and *Sarcoptes* in foxes and coyotes, as well as *Yersinia pestis* in prairie dogs [10–12]. Studying the spread of infectious diseases plays an important role in regulating the number of predator-prey populations in a given ecosystem [4, 8, 13].

Anderson and May [14] were the first that combined mathematical ecology and epidemiology. Currently, more researchers are interested to conduct research in the area of mathematical ecoepidemiology. Many ecoepidemiological studies consider disease in prey [4–6]; others assumed predators consume infected preys [15]. There are also some

studies that assumed the disease in predators only [1, 16] and other studies assuming predators consume both susceptible and infected preys [8, 9].

A small number of research studies have examined the use of ideal controls in predator-prey disease models. In [17], the effect of hunting in both species is added as a control variable to create an optimum control issue in the Lotka–Volterra model. In a particular prey-predator system, certain mechanisms will be required to manage the infection of both prey and predators. One strategy involves preventing infection and harvesting prey to increase a healthy prey, as examined in [18, 19]. The authors in [20] took a farming case study by considering pests as preys and other species catching pests as a predator. In their study, to remove the number of both preys and predators, they have used prey and predator pesticide as controlling measures, and their result argues that the use of pesticide would be effective to minimize the number of both prey and predator with a minimum cost. Some studies are also applied optimal control analysis to prey-predator interaction by implementing controls such as, the separation and treatment of diseased prey populations [21], by separation of infected prey from predators [22], and providing medications to infected preys in a reaction-diffusion prey-predator model [23].

This research studies a mathematical ecoepidemiology model with disease infection in both the prey and predator populations, assuming that infected predators will not be able to catch any prey because of the disease. To reduce infections, control measures using prevention and treatment of infected prey and predator populations are also examined in the extension of the optimal control study.

2. Model Formulation

The whole population in the predator-prey system is subdivided into four classes. The subdivisions are as follows: $s(t)$ denote susceptible prey, $I(t)$ denote infected prey, $y(t)$ denotes susceptible predator, and $z(t)$ denotes infected predator. As a result, the number of prey and predator populations is given by $N(t) = s(t) + I(t) + y(t) + z(t)$. In formulating the present model, the following assumptions have been used:

(i) The prey population grows logistically in the absence of illnesses, with an intrinsic growth rate (r) and an environmental carrying capacity (k).

(ii) Prey that is vulnerable can only procreate up to the point of its carrying capacity.

(iii) Because of the illness infection, infected prey and predators suffer from reduced growth, recovery, and reproduction rates, with fatality rates of δ_1 and δ_2, respectively.

(iv) When susceptible prey comes into contact with infected prey, it is expected that basic mass action kinetics with a rate of β will govern the infection process.

(v) When an infected predator comes into contact with a susceptible predator, the contact process is thought to follow basic mass action kinetics with a convolution rate of α. The natural death rate of the susceptible predator is μ.

(vii) When a susceptible predator encounters susceptible and infected prey, the predation functional response will result in predation coefficients with efficiency p_1, p_2, and $q\ p_1$, $q\ p_2$, where q is the transformation of eaten prey to predation q

(viii) It is believed that infected predators are feeble and ill-suited to capture any prey. If a susceptible predator becomes infected, it either stays diseased or dies out.

The descriptions of the state variables and all the parameters used in our model are provided in Tables 1 and 2, respectively.

Using the assumptions and descriptions of the parameters, we have the following dynamical systems:

$$\frac{ds}{dt} = rs\left(1 - \frac{s+I}{k}\right) - \beta sI - p_1 sy, \tag{1}$$

$$\frac{dI}{dt} = \beta sI - p_2 Iy - \delta_1 I, \tag{2}$$

$$\frac{dy}{dt} = qp_1 sy + qp_2 Iy - \alpha yz - \mu y, \tag{3}$$

$$\frac{dz}{dt} = \alpha yz - \delta_2 z, \tag{4}$$

with initial conditions $s(0) \geq 0, I(0) \geq 0, y(0) \geq 0, z(0) \geq 0$, and $0 < q \leq 1$.

3. Qualitative Analysis of the Model

In order to better understand the dynamical aspects of the dynamic system (1)–(4), a qualitative analysis of it will be conducted. This will help to shed light on how infectious diseases affect the prey-predator system. We will discuss the model's qualitative analysis in this section, including its positivity, boundedness, and existence of solutions. Along with calculating the equilibrium points, we will also determine the equilibrium points' local and global stability analyses. The ensuing subsections cover and discuss each of these ideas.

3.1. Positivity of Solutions of the Model. It is important to demonstrate that all solutions of the system with positive initial data will remain positive for all $t \geq 0$ in order for the model to be well stated and epidemiologically significant. The following theorems will establish this.

Theorem 1 (positivity). *If all the initial conditions are nonnegative, then the solutions of system equations (1)–(4) $s(t)$, $I(t)$, $y(t)$, and $z(t)$ are all nonnegative for all $t \geq 0$.*

Table 1: Description of variables.

Variables	Descriptions
$s(t)$	The number of susceptible preys at time t
$I(t)$	Total number of infected preys at time t
$y(t)$	The number of susceptible predators at time t
$z(t)$	Infected predator population at time t

Table 2: The model parameter description.

Parameter	Description
r	Susceptible preys' growth rate
k	The susceptible prey carrying capacity
α	Convolution rate of a vulnerable predator that is prone to infection
β	The pace at which vulnerable prey becomes infected prey
p_1	The rate at which vulnerable prey is eaten by a vulnerable predator
p_2	The rate at which diseased prey is eaten by a vulnerable predator
q	The efficiency constant of predation
δ_1	Infected preys' infection-related death rate
δ_2	Natural and disease-induced death rate for infected predator
μ	The susceptible predators' natural death rate

Proof. Let us start from the positivity of equation (2).

Equation (2) given by $(dI/dt) = \beta sI - p_2 Iy - \delta_1 I$ can be separated as $(dI/dt) = (\beta s - p_2 y - \delta_1)I$. This equation can be written as $dI/I = (\beta s - p_2 y - \delta_1)dt$. Therefore, $I(t) = I(0)e^{(\beta s - p_2 y - \delta_1)t}$ is the solution to the differential inequality that can be derived by applying the variable separable technique and integration. Remember that an exponential function is always positive, regardless of the exponent's sign. In light of this, $I(t) \geq 0$ whenever $I(0) \geq 0$. Using the same process, the positivity of the other variables is likewise demonstrated. □

3.2. Boundedness of the Model. The dynamical system in the theoretical ecoepidemiology study is implied to be biologically valid by its boundedness. By demonstrating the boundedness and existence of the solution of the dynamical system (1)–(4) using the following theorem, we first demonstrate the biological validity of the model.

Theorem 2 (boundedness). *The solutions of the dynamical system (1)–(4) are all uniformly bounded.*

Proof. To show that each number of prey-predator population is bounded, we define a function N as $N(t) = s(t) + I(t) + y(t) + z(t)$ for all t. Differentiating the function N with time results, $(dI/dt) = (d(s + I + y + z))/(dt)$.

Hence, from equations (1)–(4), we obtain a simplified expression as $(dI/dt) \leq rs(1 - s + I/k) + qp_1 sy + qp_2 Iy$. For arbitrary choice of η, this can be expressed as $(dI/dt) + \eta N \leq rs(1 - s + I/k + \eta/r) + qp_1 sy + qp_2 Iy = \Lambda$. The foregoing equation can be expressed as $(dI/dt) \leq (\Lambda - \eta N(t))$. Here, $0 \leq N(t) \leq (\Lambda/\eta)(1 - \exp(-\eta t)) + N(0)\exp(-\eta t)$ is the general solution after solving the problem. Moreover, $N(t) \longrightarrow (\Lambda/\eta)$ can be seen as $t \longrightarrow \infty$. In other words, when time increases to infinity, the population size $N(t)$ rises from the value $N(0)$ at the beginning time and ends up with the bounded value Λ/η. Therefore, $0 \leq N(t) \leq \Lambda/\eta$ can be used to argue that N is bounded. Therefore, the positively invariant region $\Omega = \{(s, I, y, z) \in R_+^4; \quad N \leq (\Lambda/\eta)\}$, where the given dynamical system is both mathematically and biologically well posed. The solution of the dynamical system of model equations (1)–(4) remains in this feasible domain. □

Theorem 3 (existence). *Solutions of the dynamical system (1)–(4) together with the initial conditions exist in R_+^4, i.e., the model variables $s(t)$, $I(t)$, $y(t)$, and $z(t)$ exist for all t.*

Proposition 4. *Let the dynamical system (1)–(4) be denoted as follows:*

$$\begin{aligned} f_1 &= rs\left(1 - \frac{s + I}{k}\right) - \beta sI - p_1 sy, \\ f_2 &= \beta sI - p_2 Iy - \delta_1 I, \\ f_3 &= qp_1 sy + qp_2 Iy - \alpha yz - \mu y, \\ f_4 &= \alpha yz - \delta_2 z. \end{aligned} \tag{5}$$

All the partial derivatives of $((\partial f_i)/(\partial s_j)), \forall i, j$ for $1, 2, 3, 4$ with respect to the state variables exist, continuous, and are bounded in the feasible region Ω. Thus, a unique solution for the model (1)–(4) exists according to the Derrick and Grossman theorem.

3.3. Critical Points of the Model. The critical point of the dynamical system (1)–(4) is gained by solving $(ds/dt) = (dI/dt) = (dy/dt) = (dz/dt) = 0$. That is, we obtain the equilibrium points of the dynamical system by solving the equations

$$\begin{aligned} rs\left(1-\frac{s+I}{k}\right)-\beta sI-p_1sy&=0,\\ \beta sI-p_2Iy-\delta_1I&=0,\\ qp_1sy+qp_2Iy-\alpha yz-\mu y&=0,\\ \alpha yz-\delta_2z&=0. \end{aligned} \tag{6}$$

Hence, the dynamical system (1)–(4) has the following six critical points:

(i) Trivial equilibrium point $T_0 = (0, 0, 0, 0)$.

(ii) Axial equilibrium point $A_1 = (k, 0, 0, 0)$.

(iii) Infection-free equilibrium point $E_2 = (s^-, 0, y^-, 0)$, where $s^- = \mu/qp_1$ and $y^- = (r(kqp_1 - \mu)/kqp_1^2)$.

(iv) Predator-free equilibrium point $E_3 = (s, I, 0, 0)$, where $s = \delta_1/\beta$, $I = (r(\beta k - \delta_1))/(\beta(r + \beta k))$.

(v) Infected predator-free equilibrium point $E_4 = (s, I, y, 0)$, where $s = (qk(\beta\delta_1 + rp_2))/(\beta_2 qk + 2rqp_2)$, $I = (\mu(\beta_2 k + 2rp_2) - kqp_1(\beta\delta_1 + rp_2))/(p_2(\beta_2 qk + 2qrp_2))$, $y = (\beta kq(\beta\delta_1 + rp_2) + \beta r\mu - q\delta_1(\beta_2 k + 2rp_2))/(qp_2(\beta_2 k + 2rp_2))$.

(vi) The coexistence or positive equilibrium point $E^* = (s^*, I^*, y^*, z^*)$, where $s^* = (k\delta_2 + \alpha\delta_1)/(\alpha\beta)$, $I^* = (r[\alpha\beta k - \alpha\delta_1 - p_2\delta_2] - \beta p_1\delta_2)/(\alpha\beta(r + \beta))$, $y^* = \delta_2/\alpha$, $z^* = (qp_1(\alpha\delta_1 + \alpha\beta\delta_1 + rp_2\delta_2) + rqp_2(\alpha\beta k - \alpha\delta_1 - p_2\delta_2))/(\beta_3(r + \beta))$, and the equilibrium point is nonnegative for $\alpha\beta k - \alpha\delta_1 - p_2\delta_2 - \beta p_1\delta_2 > 0$.

3.3.1. Local Stability Analysis of the Critical Points. By employing the Jacobian matrix to linearize the dynamical system, the local stability can be achieved. The partial derivatives with respect to the state variables make up the components of the Jacobian matrix of the dynamical system $J(s, I, y, z)$, which is represented by the following matrix.

$$J(s,I,y,z)=\begin{bmatrix} r\left[1-\frac{s+I}{k}\right]-\frac{rs}{k}-\beta I-p_1s & -\frac{rs}{k} & -p_1y & 0\\ \beta I & \beta s-p_2y-\delta_1 & -p_2I & 0\\ qp_1y & qp_2y & qp_1s+qp_2I-\mu & -\alpha y,\\ 0 & 0 & \alpha z & \alpha y-\delta_2. \end{bmatrix}. \tag{7}$$

The local dynamics of all the critical points listed above are performed by substituting the values in the Jacobian matrix (7) and finding the eigenvalues of the matrix.

(i) The eigenvalues of the trivial equilibrium point T_0 are obtained to be $\lambda_1 = r$, $\lambda_2 = -\delta_1$, $\lambda_3 = -\mu$, and $\lambda_4 = -\delta_2$. Here three of the eigenvalues are negative and there is one positive eigenvalue; the trivial equilibrium point is an unstable saddle point.

(ii) The eigenvalues of the axial equilibrium point A_1 are obtained as $\lambda_1 = -r$, $\lambda_2 = \beta k - \delta_1$, $\lambda_3 = qk\,p_1 - \mu$, and $\lambda_4 = -\delta_2$. Therefore, the axial critical point A_1 is stable, if both $\beta k - \delta_1$ and $qk\,p_1 - \mu$ are negative, otherwise it is unstable.

(iii) The disease-free equilibrium point E_2 has two eigenvalues: $\lambda_1 = \alpha y^- - \delta_2$, $\lambda_2 = \beta s^- - p_2 y^- - \delta_1$. The other two eigenvalues are the solutions to the quadratic equation $(\lambda + a)(\lambda + b) + c = 0$, where $a = 2rs^- - rk/k + p_1 y^-$, $b = p_2 y + \delta_1 - \beta s^-$, and $c = qp_1^2 s^- y^-$. If $(a + b) > 0$ and $(ab) + c > 0$, then the solutions to the quadratic equation contain negative real portions according to the Routh–Hurwitz criterion. Therefore, if $\alpha y^- - \delta_2 > 0$, $\beta s^- - p_2 y^- - \delta_1 > 0$, $(a + b) > 0$, and $(ab) + c > 0$, then the

disease-free equilibrium point E_2 is stable; if one of the requirements fails, it is unstable.

(iv) Substituting the predator-free critical point $E_3 = (s, I, 0, 0) = (\delta_1/\beta, (r(\beta k - \delta_1))/(\beta(r + \beta k)), 0, 0)$ to the Jacobian matrix, we have:

$$J(E_3) = \begin{bmatrix} r\left[1 - \frac{s+I}{k}\right] - \frac{rs}{k} - \beta I - p_1 s & -\frac{rs}{k} & 0 & 0 \\ \beta I & \beta s - \delta_1 & -p_2 I & 0 \\ 0 & 0 & qp_1 s + qp_2 I - \mu & 0 \\ 0 & 0 & 0 & -\delta_2 \end{bmatrix}. \tag{8}$$

In this case, $\lambda_1 = -\delta_2$ and $\lambda_2 = qp_1 s + qp_2 I - \mu$ are two of the eigenvalues. The equation $(\lambda + a)(\lambda + b) + c = 0$ yields the other two eigenvalues where $a = 2rs + rI - rk/k$, $b = \delta_1 - \beta s$, and $c = r\beta sI/k$. If $qp_1 s + qp_2 I - \mu > 0$, $a + b > 0$, and $ab + c > 0$, then the predator-free critical point is stable using the two eigenvalues and the Routh–Hurwitz criterion.

3.3.2. Global Analysis of the Positive Critical Point

Theorem 5. *If the coexistence critical point $E^* = (s^*, I^*, y^*, z^*)$ exists and $P < N$, then the point of the dynamical systems is globally asymptotically stable.*

Proof. Constructing a Lyapunov function $L(s, I, y, z)$,

$$L = \frac{m_1(s - s^*)^2}{2} + \frac{m_2(I - I^*)^2}{2} + \frac{m_3(y - y^*)^2}{2} + \frac{m_4(z - z^*)^2}{2}, \tag{9}$$

where $L \geq 0$ for all s, I, y, z and $L = 0$ if and only if the state variables are at E^*. Differentiating the function L with time, we have

$$\frac{dL}{dt} = m_1(s - s^*)\frac{ds}{dt} + m_2(I - I^*)\frac{dI}{dt} + m_3(y - y^*)\frac{dy}{dt} + m_4(z - z^*)\frac{dz}{dt}. \tag{10}$$

Now by substituting the given dynamical system, we have

$$\begin{aligned} \frac{dL}{dt} &= m_1(s - s^*)\left[rs\left(1 - \frac{s+I}{k}\right) - \beta sI - p_1 sy\right] + m_2(I - I^*)[\beta sI - p_2 Iy - \delta_1 I] \\ &\quad + m_3(y - y^*)[qp_1 sy + qp_2 Iy - \alpha yz - \mu y] + m_4(z - z^*)[\alpha yz - \delta_2 z]. \end{aligned} \tag{11}$$

Take out s, I, y, z, and we have

$$\frac{dL}{dt} = m_1(s-s^*)^2\left[r\left(1-\frac{s+I}{k}\right)-\beta I-p_1 y\right] + m_2(I-I^*)^2[\beta s - p_2 y - \delta_1] + m_3(y-y^*)^2[qp_1 s + qp_2 I - \alpha z - \mu] + m_4(z-z^*)^2[\alpha y - \delta_2]. \tag{12}$$

Grouping the positive and negative terms together in the equation, we obtain

$$\frac{dL}{dt} = P - N, \text{ where} \tag{13}$$

$P = m_1(s-s^*)^2 + m_2(I-I^*)^2\beta s + m_3(y-y^*)^2[qp_1 s + qp_2 I] + m_4(z-z^*)^2\alpha y$ and $N = m_1(s-s^*)^2[s + I/k + \beta I + p_1 y] + m_2(I-I^*)^2[p_2 y + \delta_1] + m_3(y-y^*)^2[\alpha z + \mu] + m_4(z-z^*)^2\delta_2$. □

Hence, $(dL/dt) < 0$ and the positive equilibrium point is stable whenever $P < N$.

3.4. Threshold Value R_0. The illness would eventually go extinct if the threshold value, $R_0 < 1$, which means that each infected individual produces, on average, less than one new affected individual. However, if $R_0 > 1$, then several infections are produced by infected individuals, and it is anticipated that the disease will continue to spread across the ecology.

Theorem 6 (infected prey threshold). *For an infected prey, the reproduction number for at the disease-free critical point is given by* $R_{01} = (\beta\mu k p_1)/(p_2 r(kqp_1 - \mu) + kqp_1^2\delta_1)$.

Proof. Consider infected prey equation $(dL/dt) = \beta s I - p_2 I y - \delta_1 I$. The appearance of new infections is given by $\mathcal{F} = \beta s I$, and transfer of individuals into and out of the infected compartments is given by $\mathcal{V} = p_2 I y + \delta_1 I$. Differentiating both $\mathcal{F}$ and $\mathcal{V}$ with the disease compartment I we get $F = \beta s$ and $V = p_2 y + \delta_1$. Evaluate F and V at disease-free equilibrium point $E_1 = (s^-, 0, y^-, 0)$, and we have $F(E_1) = (\beta\mu/qp_1)$ and $V(E_1) = p_2(r(kqp_1 - \mu)/kqp_1^2) + \delta_1$. By the next generation matrix method, it is known that $R_{01} = \rho(FV^{-1}) = (\beta\mu/qp_1)\ ((kqp_1^2)/(p_2 r(kqp_1 - \mu) + kqp_1^2\delta_1))$, where ρ is the spectral radius (largest eigenvalue). Hence, $R_{01} = (\beta\mu k p_1)/(p_2 r\ (kqp_1 - \mu) + kqp_1^2\delta_1)$ which proves the theorem. □

Theorem 7 (infected predator reproduction number). *The infected predator reproduction number at disease-free equilibrium point takes the form as* $R_{02} = (\alpha r(kqp_1 - \mu)/kq\delta_2 p_1^2)$.

Proof. From the model equation of infected predator $(dL/dt) = \alpha y z - \delta_2 z$, we have $\mathcal{F} = \alpha y z$ and $\mathcal{V} = \delta_2 z$. Then differentiate with respect to z and evaluate $\mathcal{F}$ and $\mathcal{V}$ at the disease-free equilibrium point $E_1 = (s^-, 0, y^-, 0)$, and we have $F = \alpha(r(kqp_1 - \mu)/kqp_1^2)$ and $V = \delta_2$. Using the next generation matrix, $R_{02} = \rho(FV^{-1}) = \alpha(r(kqp_1 - \mu)/kqp_1^2\delta_2)$, and hence $R_{02} = (\alpha r(kqp_1 - \mu)/kq\delta_2 p_1^2)$. □

4. Optimal Control Analysis

This section describes an optimal control issue that aims to reduce the number of infected predators and prey. In the given time interval $[0, t_f]$, the goal is to minimize the objective functional and find the optimal values of the controls $u = (u_1, u_2)$ such that the associated state trajectories s, I, y, z are solutions of the dynamical system (1)–(4) with initial conditions as given in the dynamical system (1)–(4). Treatment, patient tracing, and isolation are examples of preventive measures that the control function u_1 plays in lowering the rate of interaction with sick people. To improve the recovery rates of infected prey and predators, the control u_2 denotes providing all infected instances with intensive medical attention. Assumed to be bounded between 0 and 1 are the controls. We add the two control functions u_1 and u_2 to the constructed dynamical system (1)–(4) as follows:

$$\begin{aligned}
\frac{ds}{dt} &= rs\left(1-\frac{s+I}{K}\right) - (1-u_1)\beta s I - p_1 s y + u_2 I,\\
\frac{dI}{dt} &= (1-u_1)\beta s I - p_2 I y - (u_2 + \delta_1) I,\\
\frac{dy}{dt} &= qp_1 s y + qp_2 I y - (1-u_1)\alpha y z - \mu y + u_2 z,\\
\frac{dz}{dt} &= (1-u_1)\alpha y z - (u_2 + \delta_2) z.
\end{aligned} \tag{14}$$

4.1. The Objective Functional in the Control Problem. Our goal is to identify the best set of controls $(u_1(t), u_2(t))$ that will reduce the number of infected preys and predators $(I(t) + z(t))$ as well as treatment costs over a predetermined amount of time. We take into consideration the optimization issue of minimizing the objective functional:

$$J(u_1, u_2) = \int_0^{t_f}\left(c_1 I(t) + c_2 z(t) + \frac{1}{2}\sum_{i=1}^{2} b_i u_i^2\right)dt, \tag{15}$$

where $b_i, i = 1, 2$ are measures of the necessary relative cost of the interventions related to the controls u_1 and u_2 and t_f is the final time of the treatment, subject to control system (14). The weight constants of the infected prey and predator populations are represented by the coefficients, $c_i, i = 1, 2$, which can be selected to balance cost factors resulting from

population size. It is assumed that both $u_1(t)$ and $u_2(t)$ are bounded and Lebesgue integrable control functions. Within the cost functional, the cost associated with infected prey is denoted by the phrase $c_1 I(t)$, whereas the cost resulting from the infected predator population is indicated by the term $c_2 z(t)$. Consequently, we will identify the ideal control pair (u_1^*, u_2^*) so that

$$J(u_1^*, u_2^*) = \min J(u_1, u_2) \mid (u_1, u_2) \in \Gamma, \tag{16}$$

where the associated control set Γ is defined by

$$\Gamma = \left\{ (u_1, u_2) \mid u_i \text{ is measurable with } 0 \le u_i(t) \le 1, t \in \left[0, t_f\right], \quad i = 1, 2 \right\}. \tag{17}$$

The upper bounds for u_1 and u_2 can be seen as the utmost sums that an infected person may afford, while the lower bounds of the controls equate to no implementations.

4.2. Existence of Optimal Controls. We investigate the necessary and sufficient circumstances for the optimum control problem's solution to exist.

Proposition 8. *The model system (7) has an optimum control pair (u_1^*, u_2^*) that minimizes the objective functional $J(u_1, u_2)$ over the domain Γ with a corresponding solution of (s^*, I^*, y^*, z).*

Proof. Proposition 8 Fleming and Rishel [24–26] is the foundation for the proof of Proposition 8. The following criteria are listed and confirmed as necessary for existence:

(1) In a region Γ, the set of all solutions to the control system (7), along with the related control functions and beginning conditions, is nonempty.

(2) With coefficients based on time and state variables, the control system is expressed as a linear function with respect to the control variables.

(3) The integrand $\mathscr{L}$,

$$\mathscr{L}(s, I, y, z, u_1, u_2) = c_1 I(t) + c_2 z(t) + \frac{b_1}{2} u_1^2 + \frac{b_2}{2} u_2^2, \tag{18}$$

of the objective functional is convex on Γ.

The solutions to the state equations are bounded since the right-hand sides of the control system (7) are $\mathbb{C}^1$ and bounded above and below (we have demonstrated this in the boundedness of the model). The system is therefore Lipschitz with respect to the state variables, which is inferred from the Picard–Lindelöf theorem [27]. Therefore, condition (1) is met. The control system (7) shows that the right-hand sides rely linearly with respect to u_1 and u_2. Therefore, condition (2) is also true. The integrand $\mathscr{L}$ in the objective functional (7) is convex since it is quadratic in the controls, and the result comes from this, which helps us show condition (3). □

4.3. Characterization of the Controls. The optimal controls $u^* = (u_1^*, u_2^*)$, which provide the ideal levels for the various control measures and the corresponding states (s^*, I^*, y^*, z^*), are characterized in this section. Using Pontryagin's minimum principle, the required conditions for the optimal controls are found [28]. According to the details below, this approach transforms the model system (7) into a problem of minimizing a Hamiltonian, $\mathscr{H}$, point-wise with respect to u_1 and u_2.

Proposition 9. *Let $V = (s, I, y, z)$ and $u = (u_1, u_2)$. If $(V^*(t), u^*(t))$ is an optimal control pair, then a nontrivial vector $\lambda(t) = (\lambda_1(t), \lambda_2(t), \lambda_3(t), \lambda_4(t))$ exists which satisfies the following:*

$$\begin{aligned}
\lambda_1'(t) &= -\left(\lambda_1\left(r\left(1 - \frac{2s + I}{k}\right) - (1 - u_1)\beta I - p_1 y\right) + \lambda_2\left((1 - u_1)\beta I\right) + \lambda_3 q p_1 y\right),\\
\lambda_2'(t) &= -\left(c_1 + \lambda_1\left(\frac{-rs}{k} - (1 - u_1)\beta s\right) + \lambda_2\left((1 - u_1)\beta s - p_2 y - (u_2 + \delta_1)\right) + \lambda_3(q p_2 y)\right),\\
\lambda_3'(t) &= -(\lambda_1(-p_1 s) + \lambda_2(-p_2 I) + \lambda_3(q p_1 s + q p_2 I - (1 - u_1)\alpha z - \mu) + \lambda_4(1 - u_1)\alpha z),\\
\lambda_4'(t) &= -(c_2 + \lambda_3(-(1 - u_1)\alpha y) + \lambda_4\left((1 - u_1)\alpha y - (u_2 + \delta_2)\right)).
\end{aligned} \tag{19}$$

The transversality conditions are

$$\lambda_i\left(t_f\right) = 0, \tag{20}$$

and optimal controls are characterized as

$$u_1^* = \min\left\{1, \max\left\{0, \frac{(\lambda_2 - \lambda_1)\beta sI + (\lambda_4 - \lambda_3)\alpha yz}{b_1}\right\}\right\},$$
$$u_2^* = \min\left\{1, \max\left\{0, \frac{(\lambda_2 - \lambda_1)I + (\lambda_4 - \lambda_3)z}{b_2}\right\}\right\}. \tag{21}$$

Proof. The first part of our argument starts with writing the Hamiltonian and Lagrangian for optimum control system (7), which are provided by

$$\mathscr{L}(s, I, y, z, u_1, u_2) = c_1 I(t) + c_2 z(t) + \frac{b_1}{2}u_1^2 + \frac{b_2}{2}u_2^2, \tag{22}$$

and

$$\begin{aligned}\mathscr{H} = {} & c_1 I(t) + c_2 z(t) + \frac{b_1}{2}u_1^2 + \frac{b_2}{2}u_2^2 + \lambda_1\left(rs\left(1 - \frac{(s+I)}{k}\right) - (1-u_1)\beta sI - p_1 sy + u_2 I\right) \\ & + \lambda_2\left((1-u_1)\beta sI - p_2 Iy - (u_2 + \delta_1)I\right) + \lambda_3\left(qp_1 sy + qp_2 Iy - (1-u_1)\alpha yz - \mu y + u_2 z\right) \\ & + \lambda_4\left((1-u_1)\alpha yz - (u_2 + \delta_2)z\right).\end{aligned} \tag{23}$$

Applying Pontryagin's minimum principle, we thus obtain (19) from

$$\lambda'(t) = -\frac{\partial \mathscr{H}}{\partial V}\left(t, V^*(t), u^*(t), \lambda(t)\right). \tag{24}$$

That is,

$$\begin{aligned}\lambda_1'(t) &= -\frac{\partial \mathscr{H}}{\partial s} = -\left(\lambda_1\left(r\left(1 - \frac{2s+I}{k}\right) - (1-u_1)\beta I - p_1 y\right) + \lambda_2\left((1-u_1)\beta I\right) + \lambda_3 qp_1 y\right), \\ \lambda_2'(t) &= -\frac{\partial \mathscr{H}}{\partial I} = -\left(c_1 + \lambda_1\left(\frac{-rs}{k} - (1-u_1)\beta s + u_2\right) + \lambda_2\left((1-u_1)\beta s - p_2 y - (u_2 + \delta_1)\right) + \lambda_3\left(qp_2 y\right)\right), \\ \lambda_3'(t) &= -\frac{\partial \mathscr{H}}{\partial y} = -\left(\lambda_1(-p_1 s) + \lambda_2(-p_2 I) + \lambda_3\left(qp_1 s + qp_2 I - (1-u_1)\alpha z - \mu\right) + \lambda_4(1-u_1)\alpha z\right), \\ \lambda_4'(t) &= -\frac{\partial \mathscr{H}}{\partial z} = -\left(c_2 + \lambda_3\left(-(1-u_1)\alpha y + u_2\right) + \lambda_4\left((1-u_1)\alpha y - (u_2 + \delta_2)\right)\right).\end{aligned} \tag{25}$$

Equation (20) provides the transversality conditions since all states are free at t_f. In terms of the controls, the Hamiltonian is minimized at the optimal control $u^* = (u_1^*, u_2^*)$. Consequently, on Γ, $\mathscr{H}$ is differentiated with regard to u_1 and u_2, respectively, to produce

$$\begin{aligned}0 &= \frac{\partial \mathscr{H}}{\partial u_1} = b_1 u_1 + (\lambda_1 - \lambda_2)\beta sI + (\lambda_3 - \lambda_4)\alpha yz, \\ 0 &= \frac{\partial \mathscr{H}}{\partial u_2} = b_2 u_2 - \lambda_2 I - \lambda_4 z.\end{aligned} \tag{26}$$

Therefore, on the interior sets, solving for u_1^* and u_2^* yields

$$u_1^* = \frac{(\lambda_2 - \lambda_1)\beta sI + (\lambda_4 - \lambda_3)\alpha yz}{b_1}, u_2^* = \frac{(\lambda_2 - \lambda_1)I + (\lambda_4 - \lambda_3)z}{b_2}. \tag{27}$$

We arrive to the following conclusions using typical control arguments using the boundaries on the controls: $0 \le u_1(t) \le 1, 0 \le u_2(t) \le 1$.

$$u_1^* = \min\left\{1, \max\left\{0, \frac{(\lambda_2 - \lambda_1)\beta s I + (\lambda_4 - \lambda_3)\alpha y z}{b_1}\right\}\right\},$$
$$u_2^* = \min\left\{1, \max\left\{0, \frac{(\lambda_2 - \lambda_1) I + (\lambda_4 - \lambda_3) z}{b_2}\right\}\right\}. \tag{28}$$

□

To summarise, the optimality system is comprised of the adjoint system (19) and the control system (14), along with the control characterizations (21) and their transversality conditions (20). That is, the optimality systems are

$$\begin{cases} \dfrac{ds}{dt} = rs\left(1 - \dfrac{(s+I)}{k}\right) - (1-u_1)\beta s I - p_1 s y + u_2 I, \\ \dfrac{dI}{dt} = (1-u_1)\beta s I - p_2 I y - (u_2 + \delta_1) I, \\ \dfrac{dy}{dt} = q p_1 s y + q p_2 I y - (1-u_1)\alpha y z - \mu y + u_2 z, \\ \dfrac{dz}{dt} = (1-u_1)\alpha y z - (u_2 + \delta_2) z, \\ \lambda_1'(t) = -\left(\lambda_1\left(r\left(1 - \dfrac{2s+I}{k}\right) - (1-u_1)\beta I - p_1 y\right) + \lambda_2\left((1-u_1)\beta I\right) + \lambda_3 q p_1 y\right), \\ \lambda_2'(t) = -\left(c_1 + \lambda_1\left(\dfrac{-rs}{k} - (1-u_1)\beta s\right) + \lambda_2\left((1-u_1)\beta s - p_2 y - (u_2 + \delta_1)\right) + \lambda_3\left(q p_2 y\right)\right), \\ \lambda_3'(t) = -(\lambda_1(-p_1 s) + \lambda_2(-p_2 I) + \lambda_3(q p_1 s + q p_2 I - (1-u_1)\alpha z - \mu) + \lambda_4(1-u_1)\alpha z), \\ \lambda_4'(t) = -(c_2 + (1-u_1)\alpha y(\lambda_4 - \lambda_3) - (u_2 + \delta_2))), \end{cases} \tag{29}$$

with

$$\lambda_1(t_f) = 0, \lambda_2(t_f) = 0, \lambda_3(t_f) = 0 \quad \text{and} \quad \lambda_4(t_f) = 0; \tag{30}$$

and

$$u_1 = \min\left\{1, \max\left\{0, \frac{(\lambda_2 - \lambda_1)\beta s I + (\lambda_4 - \lambda_3)\alpha y z}{b_1}\right\}\right\},$$
$$u_2 = \min\left\{1, \max\left\{0, \frac{(\lambda_2 - \lambda_1) I + (\lambda_4 - \lambda_3) z}{b_2}\right\}\right\}. \tag{31}$$

We then go ahead and solve the optimal control issue with the suggested model numerically.

5. Numerical Simulations

This section covers the numerical simulations of both the optimum control problem outlined in Section 4 and the model (1)–(4). We take into account the parameter selections provided in Table 3 for our simulation studies, and we also list the initial conditions in Table 4.

Using the MATLAB built-in routine of ODE45, the numerical solutions of the suggested model equations along with the initial conditions of the state system without control are solved numerically.

It is evident from Figure 1 that susceptible and infected prey populations are steadily declining, suggesting that susceptible predators are having an impact on prey. Then, as food becomes scarce, there is a gradual decline in the number of vulnerable predators. In a prey-predator system,

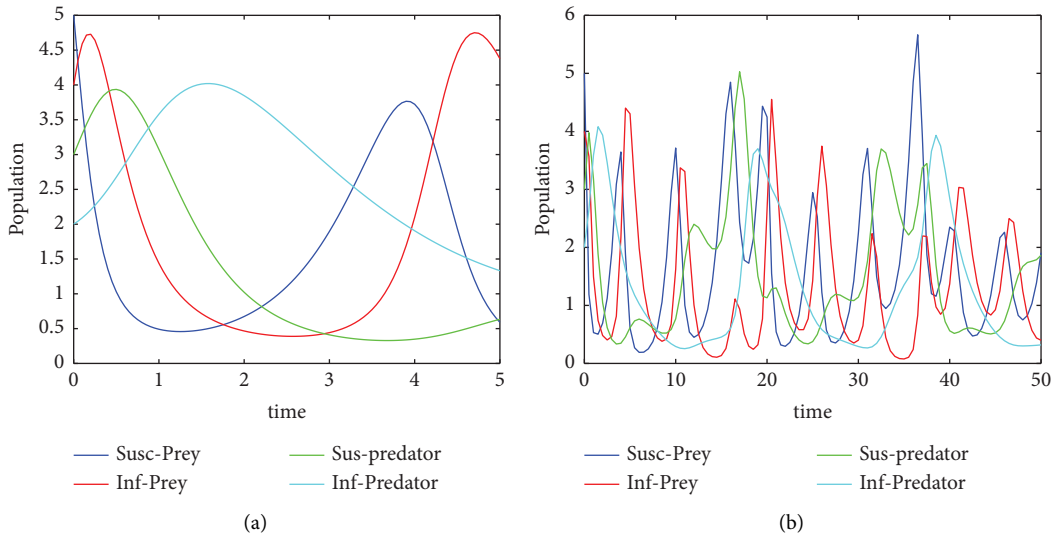

FIGURE 1: The dynamics of the state variables s, I, y, and z without applying the optimal control in time intervals [0, 5] and [0, 50]. (a) Solution with $t \in (0, 5)$. (b) Solution with $t \in (0, 50)$.

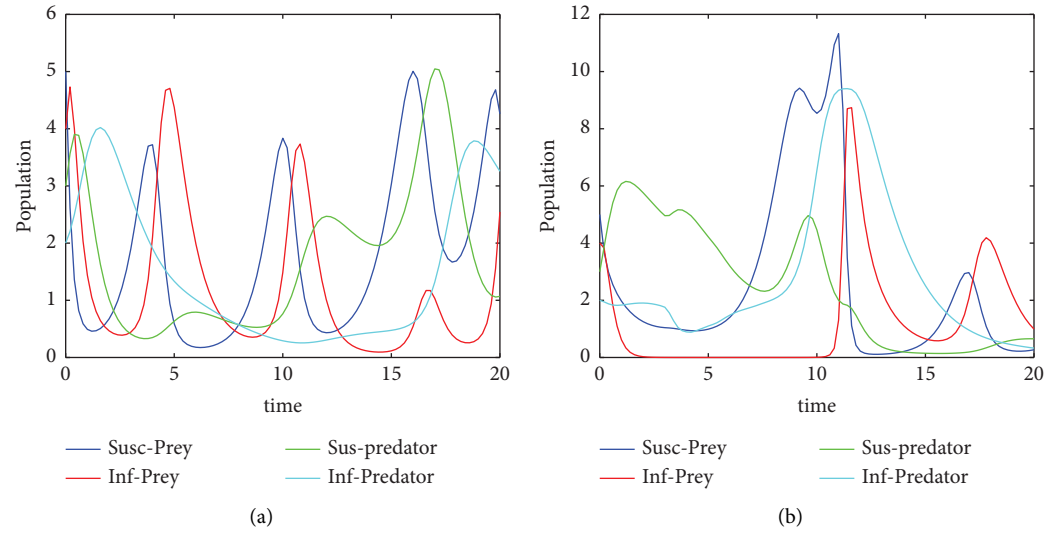

FIGURE 2: Dynamics of s, I, y, and z with parameter values from Table 3 in the interval [0, 20] with and without applying the control measures. (a) Without control. (b) With control.

population fluctuations occur continuously and are represented by varying amplitude graph oscillations.

Figure 1(b) illustrates how the predator populations affect preys. Here is an explanation for this. In the long run, both the prey and predator graphs show fast oscillation, a decrease in amplitude, and a population decline due to predation or infectious disease. If the population of susceptible predators increases, the population of infected prey decreases, meaning that all populations of susceptible predators will grow as a result of eating the prey population.

The forward-backward sweep numerical approach was utilized to solve the optimality system numerically [30]. An initial estimate of values for the ideal control pair (u_1, u_2) is necessary for the procedure to work. Using the Runge–Kutta technique of order four procedure, the state system is solved forward in time using the initial condition for the state variables. The adjoint system is then solved backward in time using the backward Runge–Kutta method of order four, along with the transversality condition (11) and the approximated solution of the state system. Next, the control pairs (u_1, u_2) are updated by combining the prior and current values of the characterization. The control variables' values are then calculated using the control characterization in (21).

When we apply the control measures for the prey and predator population, the number of infected ones is observed less than that without control. The comparisons of the solutions with control and without control are given in

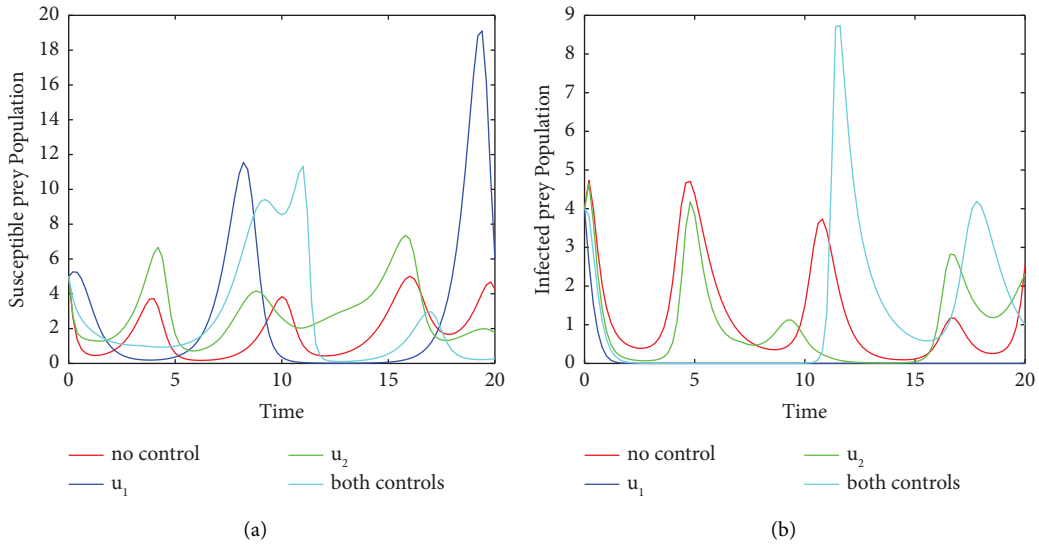

Figure 3: The solutions of susceptible and infected prey populations for the cost functional (7) without any control and applying each control successively with $c_1 = 0.04$, $c_2 = 0.04$, $b_1 = 500$, $b_2 = 200$, and parameter values from Table 3 in the time interval [0, 20]. (a) Susceptible prey. (b) Infected prey.

Table 3: Parameter values of the ecoepidemiology model.

Parameter	r	k	α	β	p_1	p_2	q	δ_1	δ_2	μ
Value	1.6	100	0.8	0.7	0.33	0.44	0.525	0.7	0.5	0.2
Source	[29]	[29]	[29]	[29]	[29]	[29]	Assumed	[29]	[29]	Assumed

Table 4: Initial values for the state variables of the model.

$s(0)$	$I(0)$	$y(0)$	$z(0)$
5	4	3	2

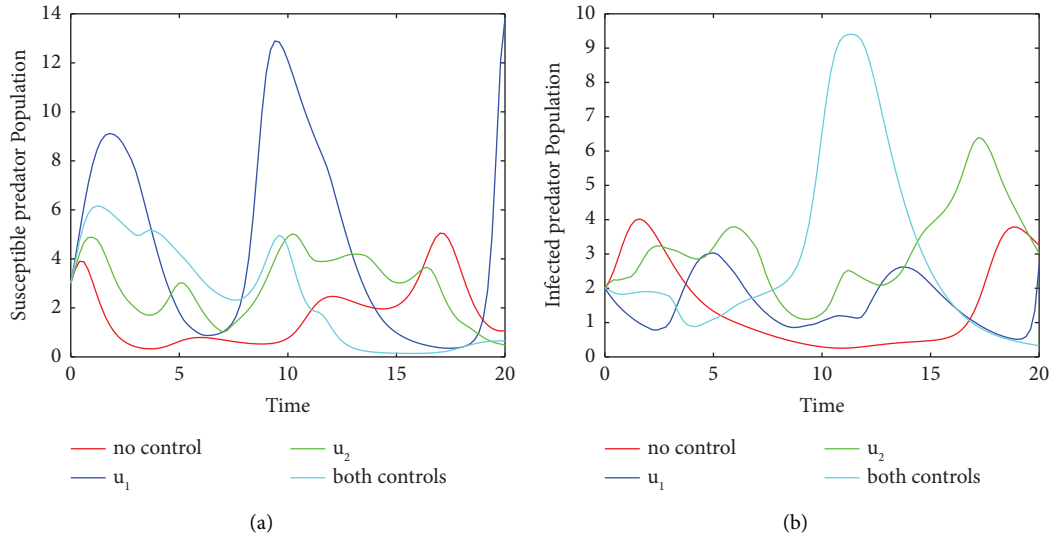

Figure 4: The dynamics of susceptible and infected predator populations for the cost functional (7) without any control and applying each control successively with $c_1 = 0.04$, $c_2 = 0.04$, $b_1 = 500$, $b_2 = 200$, and parameter values from Table 3 in the time interval [0, 20]. (a) Susceptible predator. (b) Infected predator.

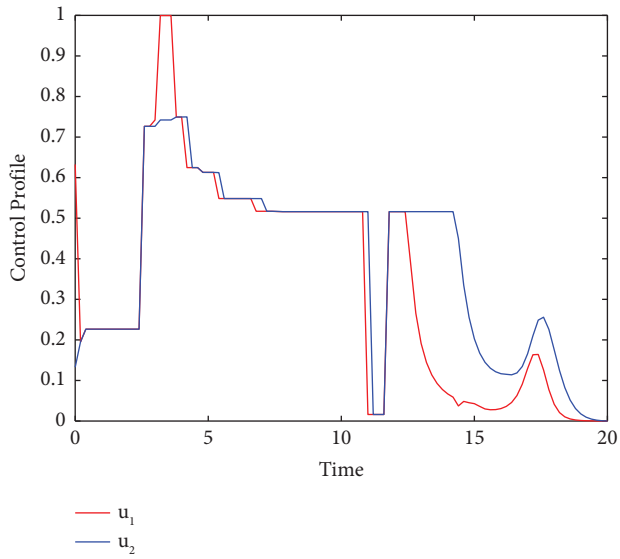

Figure 5: The control profiles.

Figure 2. The values of the associated costs are assumed to be $b_1 = 500$, $b_2 = 200$, and the values of the weight constants take $c_1 = 0.04$ and $c_2 = 0.04$ [18].

For both the susceptible and infected preys, we have comparatively simulated the impacts of applying control measures and the results are displayed in Figure 3. We can observe that reducing the number of infected preys will be effective by applying the prevention control measures. If no control intervention is applied, we obtain high number of infections and low number of susceptible population. On the other hand, applying only prevention method is more effective compared with that of applying both controls (see Figure 3(b)).

For both the susceptible and infected predators, we have comparatively simulated the impacts of applying control measures and the results are displayed in Figure 4. We can observe that reducing the number of infected preys will be effective by applying the prevention control measures. If no control intervention is applied, we obtain high number of infections and low number of susceptible population. On the other hand, applying only prevention method is more effective compared with that of applying both controls.

The control profiles for both the prevention and treatment effects of the prey-predator system are displayed in Figure 5. If there is high number of infections, the effort of implementing the control measures becomes high, while for small number of infections, there would be relatively less control implementations.

6. Conclusions

It is demonstrated that the created dynamical system's positivity, boundedness, and existence of solutions suggest that the system is meaningful and exhibits biologically sound behavior. Both endemic and disease-free equilibrium points are calculated. The Routh–Hurwitz criterion and the concept of the next generation matrix have been used for local stability analysis. By using the proper Lyapunov function, the global stability analysis of the endemic equilibrium point is demonstrated.

With the exception of diseased predators, whose amplitude is constant since we assume that they are not active in predation, the population of the prey-predator system is constantly changing, as seen by the oscillation of the graph with varying amplitudes. The number of susceptible predators increases in response to the number of prey, and over time, the graphs of prey and predators exhibit rapid oscillation, amplitude decrease, and population decline overall as a result of infectious diseases and predation.

The optimal control study has been applied in our ecoepidemiology model to protect the prey-predator population from infection and apply treatments to both the infected prey and infected predator populations. This study will put a new impact on studying diseases that occurred in the prey-predator population. The optimal control theory is characterized analytically by means of Pontryagin's maximum principle. The solution of the optimal control and the corresponding state solution are then studied numerically, and the solutions agree with our theoretical results.

This study can be extended by adding further assumptions, such as that the predator grows logistically or that disease-infected predator-prey heal, or by incorporating more variables, such as immunization, immigration, and movement on the relationship between prey and predator.

Data Availability

The data used to support the findings of this study are included within the article.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

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