Optimal Control for a Predator-Prey Model with Disease in the Prey Population

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ABSTRACT

In this study, an optimal control problem is formulated to a predator-prey model with disease in the prey population. This model is an adapted Lotka-Volterra model but with an applied SI epidemic dynamics on the prey population. Two controls are then applied to the system: first, a separating control, that is intended to separate the sound prey from the infected prey population, and the other serves as a treatment control that is to decrease the rate of death caused by the disease. We then formulate a finite-time horizon optimal control problem by minimizing the infected prey population at the final time and the cost induced from the application of the controls. Characterisation of the optimal controls is done using Pontryagin’s Optimality Principle by introducing the Hamiltonian with the associate adjoint variables. Using varied set of parameters, we analyse numerical simulations showing different scenarios from the system. The simulations are obtained using a forward-backward sweep method on the first order necessary conditions for the control problem.
Keywords: optimal control, predator-prey, Pontryagin optimality principle, forward-backward sweep method.
1. Introduction

Background of the Study. Population dynamics of different species has been of great interest among mathematicians for the past century. A prototype of a model describing the oscillations of populations of two species having a predator-prey relationship was independently developed by Lotka (1925) and Volterra (1926). The model is given as

\[ \dot{x} = x(a - bx - cy), \quad \dot{y} = y(-d + ex), \quad (1) \]

where \( x(t) \) and \( y(t) \) represent the prey and predator population at time \( t \), respectively, and \( \dot{x} \) denotes the time-derivative of the phase variable \( x \). Here, \( a \) represents the natural growth rate of the prey in the absence of predator, \( b \) is the carrying capacity of the prey population, \( c \) is the predation rate in the system, \( d \) is the natural death rate of predator in the absence of prey, and \( e \) is the effect of the presence of prey with the predator. Note that in a classical Lotka-Volterra, the model is represented where \( b = 0 \). However, in reference to Chauvet et al. (2002), it is necessary to have a logistic term on \( x \) whenever we consider a three species system, in order to maintain the boundedness of the solution to the model.

Based on the seminal work of Lotka and Volterra, different predator prey models are being developed, describing various physical interaction between prey and predator species. Li et al. (2016), for example, considered a predator-prey model wherein a non-smooth switched harvest on predators was introduced into the system. Also, researchers are intrigued on the effect of varying the functional responses of species. Classical types that have been of interest are due to Holling. For instance, a Holling type II functional response, given by \( f(S) = \frac{S}{m+S} \), has been considered by Liu and Chen (2003), taking into consideration at the same time a periodic constant impulsive immigration to the predator species in the system. Meanwhile, Ko and Ryu (2006) examined a predator prey system with Holling type II functional response and incorporated a prey refuge under Neumann boundary condition.

Aside from the dynamic of the population of species, another area of science that mathematicians have been attracted to is the area of epidemiology, particularly on epidemiological models. The most widely used epidemiological model is the classical SIR model developed by Kermack and McKendrick (1927). The model comprises of three main ingredients, referred to as compartments: the susceptible (\( S \)), infected (\( I \)), and recovered (\( R \)). One can also refer to Hethcote (1976) who investigated the asymptotic stabilities of several models, where temporary immunity, disease related fatalities, carriers, migration, dissimilar interacting groups, and transmission by vectors, are considered. Meanwhile,
Hethcote (2000), made a survey paper for infectious diseases. Moreover in the said paper by Hethcote (2000), the author presented a new approach by introducing a new compartment, that is, a compartment class for infants. Addawe and Pajimola (2016), studied a modified SIR model, intended to unravel the dynamics of Malaria transmission with an age-structured human population, moreover, the authors considered the effect of climate-change to the dynamics of the transmission of the said disease.

Now, since the goal of dynamics modeling is to describe the system in the most accurate way possible, models of predator-prey interaction are developed where communicable diseases are present. For example, Haque (2010) analysed a predator-prey system where disease is only present in the predator species. Li and Wang (2015) considered the same scenario, however, they used a stochastic predator prey model and considered the Beddington-DeAngelis functional response. Another predator-prey model with disease in the predator species was also studied by Venturino (2002). Hsieh and Hsiang (2008), on the other hand, investigated a predator prey model with disease in both the species.

Description of the model. In this work, we are interested in a model where diseases are persistent only in the prey population. Such mathematical model has been examined by Sani et al. (2014). The model is assumed to obey specific assumptions (cf. Sani et al. (2014)):

- the system is divided into three groups; healthy preys \((x_s)\), infected preys \((x_l)\), and predators \((y)\);
- the growth of prey population follows a logistic function with intrinsic growth rate \(r\) having carrying capacity \(\kappa\);
- once a healthy prey gets infected due to contact with an infected prey, it moves to the infected compartment with the rate \(\alpha x_l\);
- recovery from the disease is possible, due, for instance, to its immune system with a constant rate \(\theta\);
- the rate of natural death for all preys is given by \(\mu\),
- and an infection-related death rate, given by \(\sigma\);
- the interaction of the predator with both preys is assumed to be of Holling type-II, given by the functions \(\frac{a_1y}{1+\eta_1x_s}\) and \(\frac{a_2y}{1+\eta_2x_l}\) for the sound and infected prey, respectively;
- in the absence of prey, predators will experience natural death at rate \(c.\)
Optimal Control for a Predator-Prey Model

From these assumptions, the mathematical model we consider here is given as follows:

\[
\dot{x}_s = r(x_s + x_l) \left( 1 - \frac{x_s + x_l}{\kappa} \right) - \alpha x_s x_l + \theta x_l - \mu x_s - \frac{a_1 x_s y}{1 + b_1 x_s}
\]

\[
\dot{x}_l = \alpha x_s x_l - (\theta + \mu + \sigma)x_l - \frac{a_2 x_l y}{1 + b_2 x_l}
\]

\[
\dot{y} = \beta_1 \left( \frac{a_1 x_s y}{1 + b_1 x_s} \right) + \beta_2 \left( \frac{a_2 x_l y}{1 + b_2 x_l} \right) - cy.
\]

**Objective.** The goal of this paper is to investigate an optimal control problem that aims to minimize the population of the infected prey at the end of a finite time interval \([0, T]\), and at the same time minimize the cost incurred from implementing this control. In particular, two controls are applied to the model investigated by Sani et al. (2014), the first will be separating the sound and the infected prey population, and the other one is a treatment for the disease that is persistent in the system. Yusuf and Benyah (2012) studied an optimal control problem on an SIR model where controls are considered to be vaccination and treatment from the disease. In this problem, a different treatment method will be used as that from Yusuf and Benyah (2012). More precisely, a scheme that will decrease the rate of death caused by the disease will be used. On the other hand, for the separation scheme, the method used by Apreutesei (2006) where species are intended to be separated and maximised at the end of the observational interval will be used. The only difference is that this paper aims to minimize a certain population at the final time of observation.

The rest of the paper is organized as follows. In Section 2, we discuss the formulation of the optimal control problem, and the optimal solutions will be characterised together with the necessary conditions. Moreover, in Section 3, numerical experiments will be presented and the results will be discussed. Lastly, in Section 4, we conclude the results and the possible modifications of the problem.
2. The Optimal Control Problem

2.1 Formulation of the Problem

Consider the following model (cf. [Sani et al. (2014)])

\[
\begin{align*}
\dot{x}_s &= r(x_s + x_l) \left(1 - \frac{x_s + x_l}{\kappa}\right) - \alpha x_s x_l + \theta x_l - \mu x_s - \frac{a_1 x_s y}{1 + b_1 x_s} \\
\dot{x}_l &= \alpha x_s x_l - (\theta + \mu + \sigma) x_l - \frac{a_2 x_l y}{1 + b_2 x_l} \\
\dot{y} &= \beta_1 \left(\frac{a_1 x_s y}{1 + b_1 x_s}\right) + \beta_2 \left(\frac{a_2 x_l y}{1 + b_2 x_l}\right) - cy,
\end{align*}
\]

where \(x_s := x_s(t), x_l := x_l(t), y := y(t)\) are the population density of the healthy prey, infected prey, and the predator at time \(t\), respectively. The parameters are described as in the paper of [Sani et al. (2014)]. Boundedness of solutions for the system was shown by [Sani et al. (2014)], enabling us to apply possible controls to this system.

In this work the controls that will be used are time-dependent and will be taken from the set of piecewise continuous functions from \([0, T]\), the set will be denoted as \(PC[0, T]\). Firstly, we consider the separating control. Denote this control by \(u(t)\). Our aim is to separate the sound and the infected prey by some factor, it may be in full separation or otherwise. To do this, we let the term \(\alpha u(t)x_s x_l\) be replaced by \(\alpha x_s x_l\) in the model. Observe that if \(u = 1\), the control \(u\) has no effect on the system at all, i.e., the equation will be just the same. However, if \(u\) is zero, it is easy to see that the infected and the healthy prey populations are totally separated. So, the quantity \(1 - u(t)\) exactly describes the strength of separation between the two prey populations.

For the application of a treatment control, as have been said earlier, the goal is to reduce the rate of death caused by the disease, so instead of \(\sigma\) we put \((1 - v(t))\sigma\), where \(v(t)\) is considered as the rate of treatment applied at time \(t\). Obviously, the controls are bounded, i.e., \(0 \leq u(t), v(t) \leq 1\), and so we take the admissible set \(U = \{u \in PC[0, T] : 0 \leq u(t) \leq 1, \forall t \in [0, T]\}\). The controlled
model is now formulated as follows:

\[
\begin{align*}
\dot{x}_s &= r(x_s + x_l)(1 - \frac{x_s + x_l}{\kappa}) - \alpha u(t)x_s x_l + \theta x_l - \mu x_s - \frac{a_1 x_s y}{1 + b_1 x_s} \\
\dot{x}_l &= \alpha u(t)x_s x_l - (\theta + \mu + (1 - v(t))\sigma) x_l - \frac{a_2 x_l y}{1 + b_2 x_l} \\
\dot{y} &= \beta_1 \left( \frac{a_1 x_s y}{1 + b_1 x_s} \right) + \beta_2 \left( \frac{a_2 x_l y}{1 + b_2 x_l} \right) - cy,
\end{align*}
\]  

(2)

with initial state \((x_s(0), x_l(0), y(0)) = (x_0^s, x_0^l, y_0^s)\), and \(u, v \in U\).  

(3)

Now, the objectives - which are to minimize the infected prey population at the end of the interval \([0, T]\), and to minimize the cost incurred over the application of the controls - will be achieved by minimizing the cost functional

\[
J(u, v) := x_l(T) + \frac{1}{2} \int_0^T \{A(1 - u(t))^2 + Bv(t)^2\} dt,
\]

where \(A\) and \(B\) are weight parameters that depends on the cost of application of each control. To summarize, we have the following optimal control problem:

\[
\min_{u, v \in U} J(u, v) \quad \text{subject to (2) and (3)}.
\]

(P)

2.2 Characterisation of the Optimal Controls

The Pontryagin’s Optimality Principle (POP) helps us to devise the necessary conditions for optimal controls \(u^*, v^* \in U\) presented by Lenhart and Workman (2007). But before we show these conditions, we have the following existence theorem for the said controls \(u^*, v^*\).

**Theorem 2.1.** There exists \(u^*, v^* \in U\), with the corresponding state variables \(x_s^*, x_l^*\) and \(y^*\), such that \(J(u^*, v^*) \leq J(u, v)\) for all \(u, v \in U\).

The proof of Theorem 2.1 follows directly from the existence theorem for the solutions of such problem (cf. Flemming and Rishel (1975)).

**Notation.** Hereinafter, we denote \(x = (x_s, x_l, y)\), \(w = (x_s, x_l)\), \(u = (u, v)\), \(f = (f_{x_s} f_{x_l} f_y)^T\) where \(f_{x_s}\), \(f_{x_l}\) and \(f_y\) denote the right-hand side of system (2), respectively. Also, we let \(\lambda = (\lambda_1 \lambda_2 \lambda_3)^T\) be the adjoint state.

Now, from POP, we define the Hamiltonian \(H(u, \lambda, x, t)\) as

\[
H(u, \lambda, x, t) = \frac{1}{2} \left[ A(1 - u)^2 + Bv^2 \right] + \lambda \cdot f.
\]
Theorem 2.2 (Necessary Conditions). If $u^* \in U^2$ are the optimal control for (P), with corresponding optimal state $x^*$, then the adjoint variable $\lambda$ exists and satisfies $\dot{\lambda} = -\partial_x H$, with transversality conditions $\lambda_1(T) = \lambda_3(T) = 0$ and $\lambda_2(T) = 1$. Furthermore, the optimal controls $u^*$ are characterized as

$$
\begin{cases}
    u^* = 0 & \text{if } \partial_u H > 0, \\
    u^* \in [0, 1]^2 & \text{if } \partial_u H = 0, \\
    u^* = 1 & \text{if } \partial_u H < 0.
\end{cases}
$$

and the state variables $x^*$ satisfy (2) subject to (3).

The derivation of these conditions is standard (cf. Lenhart and Workman (2007)), and so is omitted.

To determine the conditions above, we proceed as follows. First, we compute for the Jacobian $J = (\partial_{x_s} f \; \partial_{x_l} f \; \partial_y f)$. Then, we have

$$
\partial_{x_s} f = \begin{pmatrix}
    r - \frac{2x}{\kappa} (x_s + x_l) - \alpha x_l A_1 y (B_1 x_s - 1) \\
    \alpha x_l \\
    \beta_1 A_1 y (1 - B_1 x_s)
\end{pmatrix},
$$

$$
\partial_{x_l} f = \begin{pmatrix}
    r - \frac{2x}{\kappa} (x_s + x_l) - \alpha u x_s + \theta \\
    \alpha u x_s - (\theta + \mu + (1 - v)\sigma + A_2 y (B_2 x_l - 1)) \\
    \beta_2 A_2 (1 - B_2 x_l)
\end{pmatrix}
$$

and

$$
\partial_{y} f = \begin{pmatrix}
    -A_2 x_l \\
    -A_2 x_l \\
    -c
\end{pmatrix}
$$

where $A_1 = \frac{a_1}{1 + b_1 x_s}$, $A_2 = \frac{a_2}{1 + b_2 x_l}$, $B_1 = \frac{b_1}{1 + b_1 x_s}$, and $B_2 = \frac{b_2}{1 + b_2 x_l}$.

Thus,

$$
\dot{\lambda} = -\lambda^T \cdot J.
$$

Now, we characterize the expression for the optimal control $u^*$ in terms of the adjoint state and state variables. If $\partial_u H = 0$, then $u^* \in [0, 1]^2$, and

$$
\partial_u H = \begin{pmatrix}
    (A(u - 1) - \lambda_1 \alpha x_s x_l + \lambda_2 \alpha x_s x_l) \\
    B v + \lambda_2 \sigma x_l
\end{pmatrix}^T
$$

giving us

$$
u^* = \begin{pmatrix}
    (\frac{\lambda_1 - \lambda_2}{A} \alpha x_s x_l + 1) \\
    -\frac{\lambda_2 \sigma x_l}{B}
\end{pmatrix}^T.
$$
Therefore,
\[ u^* = \begin{cases} 
(0, 0) & \text{if } \partial_u H > 0, \\
\left(\frac{(\lambda_1 - \lambda_2)}{A} \alpha x s x l + 1, -\frac{\lambda_2 \sigma x l}{B}\right) & \text{if } \partial_u H = 0, \\
(1, 1) & \text{if } \partial_u H < 0.
\end{cases} \]

Consequently, the optimality conditions are simply given by
\[ u^*(t) = \min\left\{1, \max\left\{0, \frac{(\lambda_1 - \lambda_2)}{A} \alpha x s x l + 1\right\}\right\}, \]
\[ v^*(t) = \min\left\{1, \max\left\{0, -\frac{\lambda_2 \sigma x l}{B}\right\}\right\}. \]

3. Numerical Results and Discussion

3.1 Numerical Results

In this section, we provide numerical examples to illustrate our result in the previous section. For this purpose, we consider the interval \([0, 30]\) employing the forward-backward sweep method on the necessary conditions established in Section 2. The numerical algorithm follows a similar procedure presented in Lenhart and Workman (2007).

The case with no controls (i.e., \((u,v) = (1,0)\)) is shown in Figure 1. In the case of no separation between the infected and healthy preys, but with treatment control (i.e., \(u = 1\) and \(v\) depends on the characterisation), a numerical illustration is shown in Figure 2 here we consider \(A = B = 1\) as values for both the weight parameters. As one can see in Figure 2, the application of the treatment policy clearly affects the population density of the infected prey population over the time interval \([20, 28]\), and is in fact lesser compared with the case with no controls (cf. Figure 1). This change was primarily due to the treatment applied to the infected prey over the interval \([3.33, 17.34]\), (whose definition is to lessen the disease infested population) and as a consequence, a decrease in population density after some time is observed for the infected prey population. Now we examine the case of taking higher value for the treatment weight \(B\), particularly, we take \(B = 20\). Referring to Figure 3, it can be observed that on the point where we apply treatment to the system, the application is more gradual compared with the case when \(B = 1\). This behaviour follows the fact that the increase in \(B\) implies that the ‘cost’ for the application...
of the treatment control is also increased. In turn, this made the rate of the application for the treatment control slower, making the quantity $\frac{1}{2} \int_0^T B v(t)^2 dt$ lesser. Next, we increase $B$ so that the change in population density would be significant. Taking $B = 1000$ (see Figure 1), we see that effect of the control is discernible. However, the change is insignificant. It seems that the application of the control is quite ineffective, and hence not a good choice to achieve the desired result for the minimization problem.

We now turn our interest in the effect of the other control to the system. We let $v \equiv 0$ on the interval $[0, 30]$, and assume that the separating control follow the optimality condition $u(t) = \min \left\{ 1, \max \left\{ 0, \frac{(\lambda_1 - \lambda_2) Ax_2 x_1 + 1}{A} \right\} \right\}$. Looking at Figure 5, we see that the infected prey population rapidly increases and the sound prey population decreases simultaneously with the full application of the separating control. That is of course due to fact that the only cause for the persistence of the infected prey species is its interaction with the sound prey species. Comparing this result with the system with no controlled applied (see Figure 1), it appears that the scheme illustrated in Figure 5 has satisfied our objective to minimize the number of infected preys at the final time.

Now, we show the effect of the weight parameter $A$ on the strategy of applying the separating control, noting that the condition $v \equiv 0$ over all $[0, 30]$ is still imposed. In view of Figure 6, the span for the application of the separating control is decreased compared to that in Figure 5. This is because the cost for application of control $u$, which is basically the weight parameter $A$, is increased. Note also that the population at the final time of the infected prey became larger compared to the infected prey population in Figure 5. Apparently, minimizing the integral $\frac{1}{2} \int_0^T A u(t)^2 dt$ gives a more desirable effect than minimizing the quantity $x(T)$ in the cost.

If we look at the effect of simultaneous application of both controls on the system, the instance $A = B = 1$ (refer to Figure 7) shows that treatment control is irrelevant in the current scheme. Of course it is relatively similar with the simulation in Figure 5.

Increasing the values of $A$ and $B$, letting $A = B = 20$, we obtain an illustrative result displayed in Figure 8. Just as in figure 7 no treatment is required to achieve minimization of the cost functional. However, due to the increase in the weight parameter $A$, the time for application of the control $u$, has been more immediate compared with that of 7. Figures 7 and 8 show that it suffices to consider only the separation control to achieve a desirable result for the minimization problem.
Optimal Control for a Predator-Prey Model

The last three figures show simulations where we used forward Euler method on the state equations, while backward Euler method for the adjoint equations. Also, we used a different convex combination for the characterisation of the controls, instead of using the update \( u = 0.5(u_{\text{old}} + u_{\text{new}}) \), we use what is described in [Lenhart and Workman, 2007, Chapter 8). These simulations are done to differentiate the control scheme achieved using different solving methods for ODEs.

As can be easily observed, the implementation of the controls is very much different from the simulations shown in Figures 1 to 8, where we used RK4 for solving the ODEs describing the state and adjoint equations. In Figure 10, the implementation of controls does not take extremal values, as opposed to the previous simulations. Also, it is noticeable that the treatment control assumes non-zero values on the interval \([0, T]\). With this control scheme, the infected prey, and predator populations take zero values on the final time \(T\). However, just like from the previous simulations, applying treatment control seems to be futile. This is because, as shown in 11, the characterisation of the treatment control applies treatment on the subinterval \([1, 7]\), which just causes penalisation on the term \( \frac{1}{2} \int_0^T Bv(t)^2 dt \), and gives not much of a difference on the term \(x_i(T)\), as compared with Figure 9.
3.2 Figures

Figure 1: The system with no controls.

Figure 2: \(A = 1, B = 1\), without separation.

Figure 3: \(A = 1, B = 20\), without separation.

Figure 4: \(A = 1, B = 1000\), without separation.
Figure 5: $A = 1$, $B = 1$, without treatment

Figure 6: $A = 20$, $B = 1$, without treatment

Figure 7: $A = 1$, $B = 1$, with both controls

Figure 8: $A = 20$, $B = 20$, with both controls
4. Conclusion

In this study, a deterministic dynamics model of a predator-prey system with disease in the prey population was considered and an optimal control.
problem of the system was studied. Two controls are considered, one is to separate the infected prey and the sound prey population, and the other one is a treatment control that lessens the chance of death due to the disease. The controls are characterised using Pontryagin’s Maximum(Minimum) Principle, where we used the Hamiltonian and the adjoint variables described by Lenhart and Workman (2007). The effect of the controls in the system were investigated one at a time, and then the system is simulated where both controls are allowed to have different values. For the initial set of simulations, where we used RK4 to solve the set of ODEs describing the state and adjoint equations, it is found out that the optimal values for the treatment control $v$ is zero over the whole finite time interval $[0, T]$, and that for the control $u$, we apply full separation at a neighborhood containing the final time $T$. The simulations showed that if we set the value of the separating control $u$ to zero, and let the control $v$ follow the computed characterisation, the treatment strategy suggests that we should apply full treatment at some interval in the middle of the duration $[0, T]$. However, since the aim to minimize the infected prey population at the final time is not satisfied, we have suggested that it is better not to apply treatment at all since the application penalises the cost of the application, and this remark was justified by the simulations on the latter part of our numerical treatment. The control $u$, when following the characterisation done, takes values equal to zero at the end of the observational interval. The duration of this application, however, depends on the weight parameter $A$, i.e., if we have a small value for $A$, we are allowed to have a longer duration of application, compared when its value is higher. Meanwhile, using forward and backward Euler for solving the state and adjoint equations, respectively, the remark that it is better to just apply separation control still applies, as justified by the numerical example (cf. 11). In conclusion, the most effective control to minimize the infected prey at time $T$ is the separating control applied in an interval which consists the terminal time, and the length of this interval depends on the value of the weight parameter $A$ and that the futility of applying treatment to the system is persistent, whichever method for solving ODEs we use. As for future work, one can investigate a different approach to the treatment control, aside from lessening the effect caused by the parameter describing death due to the disease.

References


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