

Optimal Control Measures of Beddington De Angelis Response of Lotka Volterra Model

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ABSTRACT

This paper presents a mathematical model of the spread of disease in a prey population. Control methods include isolation control, i.e. separating the harvest from the infected crop through self-quarantine, and other medical control that reduces the mortality caused by the disease. To determine the impact of parameters that govern disease spread and species coexistence, a local study of the model is conducted around the disease-free equilibrium and the endemic equilibrium. The majority of the analytical findings are explained using some numerical solutions.

Keywords: Functional Response, Lotka Volterra, Optimal Control

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1. INTRODUCTION

A prey-predator system with two prey populations is mathematically modelled, one susceptible population, one diseased population, and one predator population is presented in this study. As a result of the lack of knowledge on the virus's unknown and untraceable pathophysiology, there are still many unanswered questions and no vaccine (Ahmad Bani Younes, Zeaid Hasan, 2020). The research also examines the dynamics and stability that emerge from one or more preys being unable to tell one predator from another (Inavamsi Enaganti, Bud Mishra, 2021). Our findings demonstrate the viability of using this model to forecast the spread of views as well as the effectiveness of various control measure, such as the best control strategy for minimizing the number of infected individuals. Making decisions about intervention programmes involves the optimal control theory is a significant way (Alfred Hugo, Emanuel Simanjilo, 2019). To provide an intervention plan for a particular disease, optimal control is used. It is an effective tool for starting the trade-off between prevention and treatment plans (Holly Gaff, Elsa Schaefer, 2009). As a result, we are able to analytically and numerically explain nonlinear differential equation from the perspective of stability analysis.

2. INFORMATION ABOUT THE MODEL

Our model equations will be derived from the well-known Lotka volterra model, which has been appropriately adapted to create a population of susceptible-infected prey and predators.

$$\left. \begin{aligned} \frac{dA}{dt} &= sA(t) \left(1 - \frac{A(t)}{k}\right) - xA(t)B(t) - \frac{bA(t)P(t)}{a+\alpha A(t)+\beta P(t)} \\ \frac{dB}{dt} &= xA(t)B(t) - yB(t)P(t) - fB(t) - \rho B(t) \\ \frac{dP}{dt} &= yB(t)P(t) + \frac{bA(t)P(t)}{a+\alpha A(t)+\beta P(t)} - eP(t) \end{aligned} \right\} \quad (1)$$

with $A, B, P \geq 0$ and all of the parameters are positive. The model is predicted on a number of assumptions. The system is separated into three groups; predators (P), infected preys (B) and susceptible preys (A). The variables s represent the intrinsic growth rate of the prey species, x represent the interference between susceptible and infected prey, b represent the rate at which prey are captured by predators, and y represent the force of infection between infected prey and predator, f represent the infection-related death rate, the rate of natural death of infected prey and predator is given by ρ and e .

3. ANALYSIS OF STABILITY IN NONLINEAR SYSTEMS

3.1 Equilibrium points

For the aim of examining the prey-predator model's local stability, the equilibrium points of the system are required. We shall discuss the vital points' stability characteristics.

TABLE.1 Stability of Equilibrium points

Equilibrium point	Explanation	Analysis
$e1(A = 0, B = 0, P = 0)$	Trivial	Saddle point
$e2(A = k, B = 0, P = 0)$	Infected prey & predator-free	Stable
$e3\left(A = -\frac{f+\rho}{x}, B = -\frac{s(-kx+f+\rho)}{kx(f+\rho)}, P = 0\right)$	predator-free	Stable
$e4(A = A^*, B = B^*, P = P^*)$	Coexistence	Stable

3.2 Existence and analysis of equilibrium points

The jacobian matrix of the system (1) at equilibrium point $E=(A(t), B(t), P(t))$ is given by

$$J = \begin{bmatrix} \mathfrak{N}_{11} & -A^2x & \mathfrak{N}_{13} \\ Bx & \mathfrak{N}_{22} & -By \\ \mathfrak{N}_{31} & Py & \mathfrak{N}_{33} \end{bmatrix}$$

Where $\aleph_{11} = s - \frac{sA}{k} - xAB - \frac{bAP}{a+\alpha A+\beta P} + A\left(-\frac{s}{k} - xB - \frac{bP}{a+\alpha A+\beta P} + \frac{bAP\alpha}{(a+\alpha A+\beta P)^2}\right)$,

$$\aleph_{13} = A\left(-\frac{bA}{a+\alpha A+\beta P} + \frac{bAP\beta}{(a+\alpha A+\beta P)^2}\right), \aleph_{22} = Ax - Py - f - \rho,$$

$$\aleph_{31} = P\left(\frac{bP}{a+\alpha A+\beta P} - \frac{bAP\alpha}{(a+\alpha A+\beta P)^2}\right), \aleph_{33} = By + \frac{bAP}{a+\alpha A+\beta P} - e + P\left(\frac{bA}{a+\alpha A+\beta P} - \frac{bAP\beta}{(a+\alpha A+\beta P)^2}\right)$$

According to the nature of Eigen values, the dynamical system (I) becomes stable when all three eigenvalues are negative for real roots or negative real portions for complex roots of the characteristic equation for the above jacobian matrix. In the absence of this, the dynamical system is unstable.

Theorem 3.1. At its equilibrium point $\{A=0, B=0, P=0\}$, the dynamical system (1) is saddle

Proof The matrix of Jacobians $e_1 = \begin{bmatrix} S & 0 & 0 \\ 0 & -f - \rho & 0 \\ 0 & 0 & -e \end{bmatrix}$

Now the eigenvalues are $\lambda_1 = s > 0$, $\lambda_2 = -f - \rho < 0$, $\lambda_3 = -e < 0$

hence the above equilibrium point is saddle.

Theorem 3.2 At its equilibrium point $\{A=k, B=0, P=0\}$, the dynamical system (1) is stable provided that $kx < f + \rho$.

Proof The jacobian matrix $e_2 = \begin{bmatrix} -s & -k^2x & \frac{-k^2b}{k\alpha+a} \\ 0 & kx - f - \rho & 0 \\ 0 & 0 & -e \end{bmatrix}$

Now the eigenvalues are $\lambda_1 = -s < 0$, $\lambda_2 = kx - f - \rho < 0$ with the condition $f + \rho > kx$,

$\lambda_3 = -e < 0$. As a result, the above equilibrium point is stable.

Theorem 3.3 At its equilibrium point $\{A = -\frac{f+\rho}{x}, B = -\frac{s(-kx+f+\rho)}{kx(f+\rho)}, P=0\}$, the dynamical system (1) is stable provided that $efkx + ekpx + fsy + \rho sy > kxxy$.

Proof The jacobian matrix $e_3 = \begin{bmatrix} Q & \frac{-(f+\rho)^2}{x} & \frac{-b(f+\rho)^2}{x^2\left(\frac{\alpha(f+\rho)}{x}+a\right)} \\ -\frac{s(-kx+f+\rho)}{k(f+\rho)} & 0 & \frac{sy(-kx+f+\rho)}{kx(f+\rho)} \\ 0 & 0 & -\frac{sy(-kx+f+\rho)}{kx(f+\rho)} - e \end{bmatrix}$

Where $Q = s - \frac{s(f+\rho)}{kx} + \frac{s(-kx+f+\rho)}{kx} + \frac{(f+\rho)\left(-\frac{s}{k} + \frac{s(-kx+f+\rho)}{k(f+\rho)}\right)}{x}$

Now the eigenvalues are $\lambda_1 = \frac{-ksx + i\sqrt{R}}{2kx}$, $\lambda_2 = \frac{-ksx - i\sqrt{R}}{2kx}$ & $\lambda_3 = \frac{-efkx - ekpx + kxxy - fsy - \rho sy}{(f+\rho)kx} < 0$ with the condition $efkx + ekpx + fsy + \rho sy > kxxy$, where $R = 4fk^2sx^2 + 4k^2psx^2 - k^2s^2x^2 - 4kxsf^2 - 8fkpsx - 4ksxp^2$. As a result, the above equilibrium point is locally stable.

Theorem 3.4 The interior equilibrium point $(A = A^*, B = B^*, P = P^*)$ is locally asymptotically stable.

Proof The Variation of the Jacobian matrix $J_4 = \begin{bmatrix} \aleph_{11} & -A^{2*}x & \aleph_{13} \\ B^*x & \aleph_{22} & -B^*y \\ \aleph_{31} & P^*y & \aleph_{33} \end{bmatrix}$

Where $\aleph_{11} = s - \frac{sA^*}{k} - xA^*B^* - \frac{bA^*P^*}{a+\alpha A^*+\beta P^*} + A^* \left(-\frac{s}{k} - xB^* - \frac{bP^*}{a+\alpha A^*+\beta P^*} + \frac{bA^*P^*\alpha}{(a+\alpha A^*+\beta P^*)^2} \right),$

$\aleph_{13} = A^* \left(-\frac{bA^*}{a+\alpha A^*+\beta P^*} + \frac{bA^*P^*\beta}{(a+\alpha A^*+\beta P^*)^2} \right), \aleph_{22} = A^*x - P^*y - f - \rho,$

$\aleph_{31} = P^* \left(\frac{bP^*}{a+\alpha A^*+\beta P^*} - \frac{bA^*P^*\alpha}{(a+\alpha A^*+\beta P^*)^2} \right), \aleph_{33} = B^*y + \frac{bA^*P^*}{a+\alpha A^*+\beta P^*} - e + P^* \left(\frac{bA^*}{a+\alpha A^*+\beta P^*} - \frac{bA^*P^*\beta}{(a+\alpha A^*+\beta P^*)^2} \right)$

The characteristic equation is $\Delta(\lambda) = \lambda_3 + C_1\lambda_2 + C_2\lambda + C_3$

Where $C_1 = -(\aleph_{11} + \aleph_{22} + \aleph_{33}) = 3I - (\aleph_{11} + \aleph_{22} + \aleph_{33}) = 3I - L,$

$C_2 = \aleph_{11}\aleph_{22} + \aleph_{11}\aleph_{33} - \aleph_{12}\aleph_{21} - \aleph_{13}\aleph_{31} + \aleph_{22}\aleph_{33} - \aleph_{23}\aleph_{32}$

$= 3I^2 - (2\aleph_{11} + 2\aleph_{22} + 2\aleph_{33})I + \aleph_{11}\aleph_{22} + \aleph_{11}\aleph_{33} - \aleph_{13}\aleph_{31} + \aleph_{22}\aleph_{33} + A^{2*}B^*x^2 + B^*P^*y^2,$
 $= 3I^2 - 2LI + M$

$C_3 = \aleph_{11}\aleph_{32}\aleph_{23} - \aleph_{11}\aleph_{22}\aleph_{33} + \aleph_{12}\aleph_{21}\aleph_{33} - \aleph_{12}\aleph_{23}\aleph_{31} - \aleph_{13}\aleph_{21}\aleph_{32} + \aleph_{13}\aleph_{22}\aleph_{31}$

$= I^3 - (\aleph_{11} + \aleph_{22} + \aleph_{33})I^2 + (\aleph_{11}\aleph_{33} + \aleph_{22}\aleph_{33} + \aleph_{11}\aleph_{22} + A^{2*}B^*x^2 + B^*P^*y^2 - \aleph_{13}\aleph_{31})I -$

$\aleph_{11}\aleph_{22}\aleph_{33} - \aleph_{11}B^*P^*y^2 - A^{2*}B^*xy\aleph_{31} - A^{2*}B^*x^2\aleph_{33} + \aleph_{13}\aleph_{22}\aleph_{31} - B^*P^*xy\aleph_{13}$

$= I^3 - LI^2 + MI + N$

According to Routh Hurwitz's criterion, all of J_4 's eigenvalues have real components that are negative.

if $C_1 > 0, C_2 > 0, C_3 > 0, C_1C_2 > C_3$

Where $C_1C_2 = [3I - L][3I^2 - 2LI + M]$

$= 9I^3 - 9LI^2 + (2L^2 + 3M)I - ML$

$> I^3 - LI^2 + MI + N$

$= C_3$

Hence we proved $C_1C_2 > C_3$. The provided system of the nonlinear differential equation (I) is therefore locally asymptotically stable around a non-trivial equilibrium point ($A = A^*, B = B^*, P = P^*$) if the assumptions made in the theorem are true.

4. APPLICATION OF OPTIMAL CONTROL IN PREY-PREDATOR MODEL

Three different categories of optimal control exist: Dynamic programming, indirect approaches and direct methods (Francesco Biral, Enrico Bertolazzi and Paolo Bosetti, 2016). We present an indirect approach based on the maximum Pontryagin principle. The goal of the model equations (1) is to control the spread of disease among prey population. One of the optimal controls can be a time-dependent control, and the other is a remedy for a disease that persists in the system. The maximum rate of control will be drawn from the set of piecewise continuous functions from $[0, T]$, and the set will be referred to as $PC[0, T]$. In addition, we assume that the separation controls have limitations. The $u(t)AB$ rate of infection of susceptible prey populations is predicted on the following assumptions. The control rate achieved by separating infected and susceptible prey then varies over time and is always at its maximum when $u=1$. Since the control u has no impact on the system at all, the equation remains the same. On the other hand, if $u=0$, it is clear that the populations of infected and the healthy prey are entirely distinct. Thus, the quantity $1-u(t)$ perfectly captures the degree of separation between the two prey populations. As previously stated, the objective of the application of treatment control is to lower the rate of disease-related deaths, therefore instead of using f , we use $(1-v(t))f$, where $v(t)$ is the rate of treatment applied at time t . The controls must be bounded because they are $0 \leq u(t)$, $v(t) \leq 1$, thus we choose the acceptable set $U = \{u \in PC[0, T] : 0 \leq u(t) \leq 1, \forall t \in [0, T]\}$ (Alfred Hugo, Emanuel Simanjilo, 2019).

The modified model (1) of time-dependent is given by:

$$\left. \begin{aligned} \frac{dA}{dt} &= sA \left(1 - \frac{A}{k}\right) - xu(t)AB - \frac{bAP}{a + \alpha A + \beta P} \\ \frac{dB}{dt} &= xu(t)AB - yBP - fv(t)B - \rho B \\ \frac{dP}{dt} &= yBP - \frac{bAP}{a + \alpha A + \beta P} - eP \end{aligned} \right\} \quad (2)$$

Now, the goals-which are to reduce the number of sick prey population at the end of the interval $[0, T]$ and to reduce the expense associated with applying controls-will be met by reducing the cost functional (Simon J.S.H., Rabago J.F.T., 2018).

$J(u, v) = B(T) + \frac{1}{2} \int_0^T \{X(1 - u(t))^2 + Yv(t)^2\} dt$ where T is the final time, X and Y are based on the expense of applying each control. We have the following optimal control issue, to sum it up: $\min_{f, g \in F} J(f, g)$ subject to (1) and (2).

We can create the required conditions for optimal controls $u^*, v^* \in U$ with the use of the Pontryagin's Optimality Principle (POP) presented by Lenhart and Workman (2007) (Suzanne Lenhart, John T Workman, 2007). Hereafter, we denote $k = k_A, k_B, k_P$ where k_A, k_B and k_P stand for the respective right-hand sides of system(2). We also allow $\omega = (\omega_1 \omega_2 \omega_3)$ to be the neighbouring state (Simon J.S.H., Rabago J.F.T., 2018).

Now, from POP, we define the Hamiltonian (H), with respect to (HUGO) $1-u(t)$, $v(t)$ as $H = \frac{1}{2} \{X(1 - u(t))^2 + Yv(t)^2\} + \omega \cdot k$

$$= \frac{1}{2} \{X(l - u(t))^2 + Yv(t)^2\} + \omega_1 \left\{ sA \left(1 - \frac{A}{k} \right) - xu(t)AB - \frac{bAP}{a + \alpha A + \beta P} \right\} + \omega_2 \{xu(t)AB - yBP - fv(t)B - \rho B\} + \omega_3 \{yBP - \frac{bAP}{a + \alpha A + \beta P} - eP\}$$

where ω_i , $i=1,2,3,4$ are the co-state variables linked with A,B,P. The neighbouring equations are obtained by $\frac{d\omega_i}{dt} = -\frac{\partial H}{\partial i}$, with transversality condition $\omega_i(T)=0$.

From the above Hamiltonian we obtain the following adjoint equations

$$\frac{\partial H}{\partial A} = -\omega_1 \left(s - \frac{2As}{k} - xuB - \frac{bP}{a + \alpha A + \beta P} + \frac{\alpha bAP}{(a + \alpha A + \beta P)^2} \right) - \omega_2 xuB - \omega_3 \left(\frac{\alpha bAP}{(a + \alpha A + \beta P)^2} - \frac{bP}{a + \alpha A + \beta P} \right)$$

$$\frac{\partial H}{\partial B} = -\omega_1 xuA - \omega_2 (xuA - yP - fv - \rho) - \omega_3 yP$$

$$\frac{\partial H}{\partial P} = -\omega_1 \left(\frac{bAP\beta}{(a + \alpha A + \beta P)^2} - \frac{bA}{a + \alpha A + \beta P} \right) - \omega_2 (-y\beta) - \omega_3 \left(y\beta + \frac{bAP\beta}{(a + \alpha A + \beta P)^2} - \frac{bA}{a + \alpha A + \beta P} - e \right)$$

The optimality of the control problem is obtained by

$$u^*(t) = \min\{l, \max\{0, \frac{(\omega_1 - \omega_2)xAB}{X} + 1\}\}$$

$$v^*(t) = \min\{l, \max\{0, -\frac{\omega_2 fB}{Y}\}\}$$

5. DISCUSSIONS AND CONCLUSIONS

In this study the dynamics of an eco-epidemiological model are investigated. Certain control measures, such as ideal control measure, are included in the development and analysis of a deterministic model for the dynamics of a disease transmission in a prey. Using the Maximum Pontryagin Principle, the methods for eradicating disease in the prey-predator system were assessed, and the numerical results show that, due to the optimal control measures can increase the number of susceptible prey population and decrease the population of infected prey. By separating infected from other populations and predators from prey populations tends to reduce the prevalence of disease in the population. So, in order to prevent the spread of the disease, we advise separating the infected population from the susceptible.

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