Optimal control of predator-prey mathematical model with infection and harvesting on prey

Diva Amalia R. U.¹, Fatmawati¹*, Windarto¹, Didik Khusnul Arif²

¹Department of Mathematics, Faculty of Science and Technology, Universitas Airlangga, Surabaya 60115, Indonesia
²Department of Mathematics, Faculty of Mathematics, Computing and Data Science, Institut Teknologi Sepuluh Nopember, Surabaya 60111, Indonesia.

E-mail: fatmawati@fst.unair.ac.id

Abstract. This paper presents a predator-prey mathematical model with infection and harvesting on prey. The infection and harvesting only occur on the prey population and it assumed that the prey infection would not infect predator population. We analysed the mathematical model of predator-prey with infection and harvesting in prey. Optimal control, which is a prevention of the prey infection, also applied in the model and denoted as \( U \). The purpose of the control is to increase the susceptible prey. The analytical result showed that the model has five equilibriums, namely the extinction equilibrium \( (E_0) \), the infection free and predator extinction equilibrium \( (E_1) \), the infection free equilibrium \( (E_2) \), the predator extinction equilibrium \( (E_3) \), and the coexistence equilibrium \( (E_d) \). The extinction equilibrium \( (E_0) \) is not stable. The infection free and predator extinction equilibrium \( (E_1) \), the infection free equilibrium \( (E_2) \), also the predator extinction equilibrium \( (E_3) \), are locally asymptotically stable with some certain conditions. The coexistence equilibrium \( (E_d) \) tends to be locally asymptotically stable. Afterwards, by using the Maximum Pontryagin Principle, we obtained the existence of optimal control \( U \). From numerical simulation, we can conclude that the control could increase the population of susceptible prey and decrease the infected prey.

1. Introduction
Ecosystem consists of two components, namely the components of biotic (alive) and abiotic (not alive). The ecosystem itself is divided into two, namely natural ecosystems and artificial ecosystems. Natural ecosystems are ecosystems that form naturally without human intervention. In the other hand, artificial ecosystems occur with human intervention. Within the ecosystem, there is a reciprocal relationship or interaction between the living and the environment. Interactions that occur can be mutualism, commensalism, parasitism, and predation. This will all be studied in ecology. Ecology is a branch of biological science that studies about living things and their habitats [1].

In ecology there is the term food chain. The food chain is the transfer of energy from the organism at a tropical level to the next tropical level in the eating and eaten event in a specific order. Food chains are arranged in tropical levels, where one tropical level includes all organisms or species that share the same position in the food chain. In a food chain there are at least two species: predators and prey [2]. The occurrence of this food chain event will affect the population of a species, because there is interaction in the form of predation in a food chain.
Another problem that might occur in a population is an epidemic. Epidemic is when an illness or an outbreak occurs at a higher-than-expected frequency [3]. Epidemic events can occur in any population of living creatures, including populations in the food chain. One of the examples of epidemics that occur in real life is the outbreak of White Spot Disease (WSD) which attacks the penaid shrimp (*Penaeidae sp*) in a pond. Pond is an example of artificial ecosystem. The prevalence of this disease is caused by White Spot Syndrome Virus (WSSV). This disease is caused by a lack of oxygen levels in water, bad water quality, and poor aquatic environments [4].

The predation interactions in the ecosystem and the presence of epidemics that occur in the population, affect the balance of the ecosystem. The balance can be achieved if the average number of populations of predator and prey populations that are interacting in proportion. But the epidemic in the population will affect the balance of predators and prey. Therefore, we should find a way to cope with the occurrence of epidemics, one of which is to provide control in the form of prevention of the occurrence of epidemics in the prey. As mentioned above, WSD disease greatly affects the development of penaid shrimp populations, while penaid shrimps (*Penaeidae sp*) they have birds as their natural predators such as the cangak birds (*Ardea cinera rectirostris*) and blekok birds (*Ardeola ralloides*) [5].

Several previous studies have developed predator-prey mathematical model with infected prey [6] and the application of optimal controls in predator-prey system [7]. Furthermore, there is also the addition of harvesting factor in the predator-prey system with infected prey in attempt to balance the predator-prey populations [8]. But in real life, harvesting somehow is not an easy thing to do because it will be less efficient, especially if the prey population is a lot of small creatures such as shrimp. Therefore, another effort besides harvesting is needed to handle the infection. One of the efforts is by giving prevention to the infection. For the example, providing prevention to WSD infection by giving the prey population a liquid extract of mangrove tree to attempt to boost the shrimp’s immune against the WSD. Also, by giving a natural based liquid extract to a pond will be safer to environment compared to other chemical fluid drugs [9]. This effort is able to do in artificial ecosystem because the area is still accessible to human. In this paper, the mathematical model that developed by authors in [8] is modified by inserting an optimal control that is the prevention of infection in prey population.

2. The Predator-prey mathematical model with infection and harvesting on prey
Below are the assumptions that are used in formation of predator-prey mathematical model with infection and harvesting on prey:

a. The predator-prey mathematical model with infection and harvesting in prey involves three sub populations:
   i. \( x_1(t) \) is the number of susceptible prey population at \( t \).
   ii. \( x_2(t) \) is the number of infected prey population at \( t \).
   iii. \( y(t) \) is the number of predator population at \( t \).

b. The infection occurs because of a kind of virus and it is spread among prey population according to the S-I-S (Susceptible-Infected-Susceptible) model.

c. The susceptible prey population growth is following logistic model.

d. The infected prey population is harvested.

e. The predator population eats both type of prey.

f. The Holling type II response function is applied in the predation of susceptible prey and Holling type I response function is applied on infected prey.

g. Predator could not be infected.

h. When the prey population extinct, predator will experience natural death.

The parameter description of the model is given in Table 1.
Table 1. The parameter of predator-prey mathematical model

| Parameter | Explanation |
|-----------|-------------|
| $a$       | Logistic growth rate of susceptible prey |
| $k$       | Environmental carrying capacity |
| $\alpha$  | Rate of contact between susceptible prey and infected prey |
| $\beta$   | Rate of transformation from infected prey to susceptible prey |
| $p_1$     | Predation rate on susceptible prey |
| $p_2$     | Predation rate on infected prey |
| $h$       | Rate of harvesting of infected prey |
| $c_1$     | Conversion efficiency on susceptible prey |
| $c_2$     | Conversion efficiency on infected prey |
| $s$       | Half saturation constant |
| $m$       | Natural death rate of predator |

The predator-prey model with infection and harvesting on prey is presented as follows:

\[
\begin{align*}
\frac{dx_1}{dt} &= ax_1 \left(1 - \frac{x_1}{k}\right) - ax_1x_2 + \beta x_2 - \frac{p_1x_1y}{1+sx_1} \\
\frac{dx_2}{dt} &= ax_1x_2 - \beta x_2 - p_2x_2y - hx_2 \\
\frac{dy}{dt} &= \frac{c_1p_1x_1y}{1+sx_1} + c_2p_2x_2y - my
\end{align*}
\]

with $x_1, x_2, y \geq 0$ and all of the parameters are non-negative.

3. Analysis of the model

The Predator-prey mathematical model with infection and harvesting in prey has five equilibrium points, namely the extinction equilibrium ($E_0$), the infection free and predator extinction equilibrium ($E_1$), the infection free equilibrium ($E_2$), the predator extinction equilibrium ($E_3$), and the coexistence equilibrium ($E_4$) which will be mentioned as follows.

a. The extinction equilibrium ($E_0$)

\[ E_0 = (x_{10}, x_{20}, y_0) = (0,0,0). \]

b. The infection free and predator extinction equilibrium ($E_1$)

\[ E_1 = (x_{11}, 0, 0) = (k,0,0). \]

c. The infection free equilibrium ($E_2$)

\[ E_2 = (x_{12}, 0, y_2) = \left(\frac{m}{c_1p_1-ms}, 0, \frac{ac_1(kc_1p_1-kms-m)}{k(c_1p_1-m)s^2}\right), \]

which exists if

i. $c_1p_1 > ms$

ii. $kc_1p_1 > m(ks + 1)$.

d. The predator extinction equilibrium ($E_3$)

\[ E_3 = (x_{13}, x_{23}, 0) = \left(\frac{\beta + h}{a}, \frac{a(k\alpha + kah - \beta^2 - 2\beta h - h^2)}{kh^2}, 0\right), \]

which exists if

\[ k(\alpha \beta + ah) > \beta^2 + 2\beta h + h^2. \]

e. The coexistence equilibrium ($E_4$)

\[ E_4 = (x_{14}^*, x_{24}^*, y^*) = \left(\frac{\beta + p_2y^* + h}{a}, \left(m - \frac{c_1p_1\beta + c_2p_2y^* + c_1p_1y^*}{a + s(\beta + p_2y^* + h)}\right) \frac{1}{c_2p_2}, \frac{1}{p_2x_2^* + \frac{p_1y^*}{1+sx_1^*}}\right). \]
which exists if
i. \( m > \frac{c_1p_1\beta + c_2p_2y^* + c_4p_3y^*}{a + s(\beta + p_2y^* + h)} \)
ii. \( \frac{ax_1^*}{k} > x_2^*h + ax_1^* \).

To determine the local stability of the equilibrium points, it is necessary to linearize the predator-prey mathematical model in the presence of infection and harvesting in the prey using the Jacobian matrix.

a. Stability of the extinction equilibrium \((E_0)\)
Linearizing the model (1)-(3) near the equilibrium \( E_0 \) gives eigenvalues: \( a, -\beta - h, \) and \( -m \). Since there is a positive eigenvalue, the equilibrium is unstable.

b. Stability of the infection free and predator extinction equilibrium \((E_1)\)
Linearizing the model (1)-(3) near the equilibrium \( E_1 \) gives eigenvalues: \(-a, ak - \beta - h, \) and \( \frac{c_1p_1k}{1 + sk} - m \). The \( E_1 \) equilibrium will be locally asymptotically stable if all of eigenvalues are negatives. Therefore, we have these following conditions:
   i. \( \frac{ak}{\beta + h} < 1 \)
   ii. \( \frac{c_1p_1k}{1 + sk} < m \).

c. Stability of the disease free equilibrium \((E_2)\)
Linearizing the model (1)-(3) near the equilibrium \( E_2 \) gives eigenvalues: \( K_2 \), and the roots of this following quadratic equation:
\[ \lambda^2 + (-K_1)\lambda + \frac{K_3m}{c_1} = 0, \]
where
\[ K_1 = a \left( 1 - \frac{2m}{k(c_1p_1 - ms)} \right) - \frac{a(kc_1p_1 - kms - m)}{kc_1p_1} \]
\[ K_2 = \frac{am}{(c_1p_1 - ms)} - \beta - \frac{ac_2p_2(kc_1p_1 - kms - m)}{k(c_1p_1 - ms)^2} - h \]
\[ K_3 = \frac{K_3m}{K_1}. \]
Based on the Routh-Hurwitz criteria, the roots of equation will be negatives, or the real parts will be negatives if only if \( -K_1, \frac{K_3m}{K_1} > 0 \).
It is observed that the equilibrium \( E_2 \) is locally asymptotically stable if
i. \( \frac{2am}{k(c_1p_1 - ms)} + \frac{a(kc_1p_1 - kms - m)}{kc_1p_1} > a \)
ii. \( kc_1p_1 > m(ks + 1) \)
iii. \( \frac{am}{(c_1p_1 - ms)} < \beta + \frac{ac_2p_2(kc_1p_1 - kms - m)}{k(c_1p_1 - ms)^2} + h \).

d. Stability of the predator extinction equilibrium \((E_3)\)
Linearizing the model (1)-(3) near the equilibrium \( E_3 \) gives eigenvalues: \( F_3 \) and the roots of this following quadratic equation:
\[ \lambda^2 - F_3\lambda + hF_3 = 0 \]
where
\( F_3 = a \left( 1 - \frac{2(\beta + h)}{ak} \right) - \left( \frac{a(ka\beta + kah - \beta^2 - 2\beta h - h^2)}{kha} \right) \)
Based on the Routh-Hurwitz criteria, the roots of equation will be negative or the real parts will be negatives if only if \( F_3, F_5 > 0 \).

So the equilibrium \( E_3 \) is locally asymptotically stable if

\[
\begin{align*}
\text{i.} & \quad \frac{2a(\beta + h)}{ak} + \left( \frac{a(ka\beta + kah - \beta^2 - 2\beta h - h^2)}{kha} \right) > a, \\
\text{ii.} & \quad ka\beta + kah > \beta^2 + 2\beta h + h^2 \\
\text{iii.} & \quad \frac{c_1p_1(\beta + h)}{a + s(\beta + h)} + \frac{c_2p_2}{kh^2} \left( \frac{a(ka\beta + kah - \beta^2 - 2\beta h - h^2)}{kha^2} \right) < m.
\end{align*}
\]

e. Stability of the coexistence equilibrium \( (E_4) \)

The stability of coexistence equilibrium \( (E_4) \) is difficult to be determined analytically because the equilibrium point does not appear explicitly and it depends on the many variables. Therefore, a numerical approach is needed to determine the stability of the coexistence equilibrium point \( (E_4) \) by using phase portrait. The parameter values of the model are given in Table 2 and 3, respectively.

**Table 2.** The initial values for phase portrait.

| Initial values | \( x_1 \) | \( x_2 \) | \( y \) | Colors |
|---------------|----------|----------|-------|--------|
| 1             | 4        | 2        | 2     | Blue   |
| 2             | 60       | 50       | 40    | Red    |
| 3             | 100      | 60       | 50    | Green  |

**Table 3.** Parameter values.

| Parameter | Value | Reference |
|-----------|-------|-----------|
| \( a \)   | 16    | Assumed   |
| \( k \)   | 100   | [8]       |
| \( \alpha \) | 0.8   | [8]       |
| \( \beta \) | 0.7   | [8]       |
| \( p_1 \) | 0.33  | [8]       |
| \( p_2 \) | 0.44  | [8]       |
| \( h \)   | 0.7   | [8]       |
| \( c_1 \) | 0.04  | [8]       |
| \( c_2 \) | 0.04  | [8]       |
| \( s \)   | 0.5   | [8]       |
| \( m \)   | 0.2   | [8]       |

Figure 1 is the numerical simulation result of phase portrait of the model (1)-(3). Those orbits tend to a same point as time evolves. Thus, based on the numerical simulation we can conclude that the coexistence equilibrium \( E_4 \) tends to be asymptotically stable.
4. Optimal control problem

The control of predator-prey system is possible to do if there is a certain limit that still can be reached by human. Control that applied in this model is in the form of prevention of infection. Based on that, we can form the predator-prey mathematical model with infection and harvesting on prey that has been modified by control variable as follows:

\[
\begin{align*}
\frac{dx_1}{dt} &= ax_1 \left(1 - \frac{x_1}{k}\right) - (1 - U)ax_1x_2 + \beta x_2 - \frac{p_1x_1y}{1 + sx_1}, \\
\frac{dx_2}{dt} &= a(1 - U)x_1x_2 - \beta x_2 - p_2x_2y - hx_2, \\
\frac{dy}{dt} &= c_1p_1x_1y + c_2p_2x_2y - my.
\end{align*}
\]

(4)  
(5)  
(6)

The purpose of the optimal control is to maximize the number of susceptible prey, also to minimize the cost of the control. The Maximum Pontryagin Principle [10] is used in this problem.

We consider an optimal control problem with the objective function given by

\[
J(U) = \int_0^T \left( -\omega_1 x_1(t) + \omega_2 U^2(t) \right) dt
\]

(7)

where \(\omega_1, \omega_2\) are weighted constants for the state \(x_1\) and the control variable. Our goal is to find an optimal control \(U\) such that

\[
J(U^*) = \min_{\Gamma} J(U)
\]

(8)

where \(\Gamma = \{U|0 \leq U \leq 1\}\).

The Maximum Pontryagin Principle converts the equations (4)-(6), (7) and (8) into a problem of minimizing pointwise a Hamiltonian \(H\), with respect to \(U\) [11], that is

\[
H = -\omega_1 x_1 + \omega_2 U^2 + \begin{pmatrix} \delta_1 \\ \delta_2 \\ \delta_1 \end{pmatrix}^T \begin{pmatrix} \alpha x_1 \left(1 - \frac{x_1}{k}\right) - (1 - U)ax_1x_2 + \beta x_2 - \frac{p_1x_1y}{1 + sx_1} \\ a(1 - U)x_1x_2 - \beta x_2 - p_2x_2y - hx_2 \\ c_1p_1x_1y + c_2p_2x_2y - my \end{pmatrix}
\]

\[
= -\omega_1 x_1 + \omega_2 U^2 + \begin{pmatrix} \delta_1 \\ \delta_2 \\ \delta_1 \end{pmatrix}^T \begin{pmatrix} \alpha x_1 \left(1 - \frac{x_1}{k}\right) - (1 - U)ax_1x_2 + \beta x_2 - \frac{p_1x_1y}{1 + sx_1} \\ a(1 - U)x_1x_2 - \beta x_2 - p_2x_2y - hx_2 \\ c_1p_1x_1y + c_2p_2x_2y - my \end{pmatrix}
\]

The variable \(\delta_i, i = 1, 2, 3\) are called adjoint variables satisfying the following co-state equations...
where the transversality conditions
\[ \delta_1(t_f) = \delta_3(t_f) = \delta_3(t_f) = 0. \]

The optimal control \( U \) can be solve from the optimally condition,
\[ \frac{\partial H}{\partial U} = 0. \]
Hence, we obtain
\[ U = \frac{(\delta_2 - \delta_1)ax_1x_2}{2\omega_2} \]
The value of \( U \) is in interval between 0 and 1, so that some possibilities are obtained below:
\[ U^* = \begin{cases} 
0, & \text{if } \frac{(\delta_2 - \delta_1)ax_1x_2}{2\omega_2} \leq 0 \\
\frac{(\delta_2 - \delta_1)ax_1x_2}{2\omega_2}, & \text{if } 0 < \frac{(\delta_2 - \delta_1)ax_1x_2}{2\omega_2} < 1 \\
1, & \text{if } \frac{(\delta_2 - \delta_1)ax_1x_2}{2\omega_2} \geq 1 
\end{cases} \]
Hence, we obtain the optimal control variable value as follows
\[ U^* = \min \left( \max \left( 0, \frac{(\delta_2 - \delta_1)ax_1x_2}{2\omega_2} \right), 1 \right) \] (9)

Next we discuss the numerical approach of the optimality system. The optimality system is the state and adjoint systems coupled with the optimal control characterization.

5. Numerical simulation
In this section, we present the numerical simulations of model (4)-(6) with and without optimal control. An iterative scheme is used for solving the optimality system. The state equations are solved by the forward Runge-Kutta method of order 4. Then the co-state equations with the transversality conditions are solved by the backward Runge Kutta method of order 4. Finally, the controls are updated by using a convex combination of the previous controls and the value from the characterizations for \( U^* \). This iterative process is stopped when current state, co-state and control values converge sufficiently [12].

The result of numerical simulation will be compared in healthy prey population \((x_1)\) and also in infected prey population \((x_2)\). The simulation will be done based on the initial values and parameter values that shown in Table 2 and Table 3. The numerical simulation results of the model are given as follows.
Figure 2 showed that there is a difference in susceptible prey population number between before and after being given control variable. On the 50 days of observation based on the result, it can be seen that there is an increase on susceptible prey population and it will constant after it reaches number of 100 until the end of observation. It indicates that giving control in the form of prevention of infection in prey is quite influential and can be used as an effort to increase the number of susceptible prey population.

Figure 3 shows that there is a difference in the number of prey populations infected before and after the addition of control variables. On observations made over 50 days, it showed that there was a decrease in the number of infected prey populations and would then be constant after approaching 0 until the end of the observation. This indicates that giving control in the form of prevention of infection in prey is quite influential and can be used as an effort to increase the number of infected prey population.
Figure 4 shows the profile of the optimal control $u^*$. From Figure 4, it can be seen that the control variable $u^*$ on prevention is in the range of 0.9 to 1. The business performed on the first day is 0.99 and continues to decrease until 0.96, until the 30th day. Then on the 31st day the business will rise steadily until it reaches 1 and then decrease again to 0.96 as the last day of observation.

6. Conclusion
Based on the analytical result of predator-prey mathematical model with infection and harvesting on prey, we obtained five equilibriums, namely the extinction equilibrium ($E_0$), the infection free and predator extinction equilibrium ($E_1$), the infection free equilibrium ($E_2$), the predator extinction equilibrium ($E_3$), and the coexistence equilibrium ($E_4$). The extinction equilibrium is unstable, whereas the other equilibriums have their condition to be locally asymptotically stable. By using the Pontryagin Maximum Principle, the optimal control theory is then derived analytically. From the numerical simulation result, it is shown that the prevention effort can increase the number of susceptible prey population and decrease the population of infected prey.

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References
[1] Logan D R 2015 *A First Course in Differential Equations* Third Edition (New York: Springer Science+Business Media Inc.)
[2] Jorgensen S E 2009 *Ecosystem Ecology* First Edition (Italy: Elsevier)
[3] Guerrant R L, Walker D H, and Weller P F 2011 *Tropical Infectious Diseases: Principles, Pathogens and Practice* Third Edition (China: Saunders Elseviers)
[4] Javier M J 2010 Dynamics of intensive production of shrimp *Litopenaeus vannamei* affected by white spot disease *Aquaculture* 300 113
[5] Chattopadhyay J and Bairagi N 2001 Pelicans at risk in salton sea-an eco-epidemiological model *Ecol. Model.* 136 103
[6] Chattopadhyay J, Bairagi N and Chaudhuri S 2007 Harvesting as disease control measure in an
eco-epidemiological system-atheoretical study *Math. Biosci.* 217 134

[7] Kar T K and Gosh B 2012 Sustainability and optimal control of an exploited prey predator system through provision of alternative food to predator *Biosystems* 109 220

[8] Sujatha K and Gunasekaran M 2016 Dynamic in a harvested prey-predator model with Susceptible-Infected-Susceptible (SIS) epidemic disease in the prey *Adv. Appl. Math. Biosci.* 7 23

[9] Wahjuningrum D, Sholeh S H and Nuryati S 2006 Pencegahan infeksi virus White Spot Syndrome Virus (WSSV) pada udang windu (*Penaeus monodon*) dengan Cairan Ekstrak Pohon Mangrove (CEPM) *Avicennia sp.* dan *Sonneratia sp.* *Jurnal Akuakultur Indonesia* 5 65

[10] Pontryagin L S, Boltyanskii V G, Gamkrelidze R V and Mishchenko E F 1962 *The Mathematical Theory of Optimal Processes* (New York: John Wiley & Sons)

[11] Naidu D S 2002 *Optimal Control Systems* (New York: CRC PRESS)

[12] Lenhart S and Workman J T 2007 *Optimal Control Applied to Biological Models* (New York: Chapman & Hall)