Stability analysis of prey predator model Holling type II with infected prey

K Latipah\textsuperscript{1}, A R Putri\textsuperscript{1,*}, M Syafwan\textsuperscript{1}

\textsuperscript{1}Department of Mathematics, Andalas University, Padang, Indonesia

*Corresponding author: arrivalputri@gmail.com

Abstract. We describe a prey-predator model with infected prey. The model using Holling response function of type II is a nonlinear system of ordinary differential equations consisting of two distinct population. Critical points of the model was determined and stability of the system was analyzed by eigenvalues of Jacobian matrix. The behaviour of the dynamical system was analyzed through this stability. Furthermore, threshold number determining the system is free of disease or infected was computed. Numerical solutions are presented on phase plane to confirm the analytical solutions.

1. Introduction

A prey predator model is an interaction model between two population, prey and predator that affects the growth of both populations. Studying dynamic of the prey predator system is important to control prey and predator population in ecological theory. Numerous prey predator models have been studied to describe the complex interaction of two population [5,7].

Interaction between prey and predator need the specific form of the response function that shows predator population increasing or prey population decreasing. It depends on several factors, including the carrying capacity of the environment, the level of saturation of predator, and the level of competition between predators. Holling and Tanner has been adding different response functions to prey predator model. Those models is known as the Holling Tanner model [3].

Furthermore, the size of human and animal population can be affected by infectious diseases. Epidemic influence on the predation was first developed [1,2,4]. They modified the Lotka-Volterra prey predator model with high predation rate. The prey predator model with infected prey has been developed [11]. They assumed that predator can be infected by consuming infected prey. On the other hand, the prey predator model with infected predator has been studied [6,8]. In epidemic theory, threshold parameter is used to determine whether population free of disease or infected [10,12].

In this paper, we considered a prey predator model that was proposed [9]. We reformulated the model based on condition that there are infected prey in population. The predation is modeled by Holling type II functional response. These models are analyzed qualitatively and threshold number determining the system is free of disease or infected was computed. The results provide an overview of the spread of the disease between prey and predator.
The paper is organized as follows. The prey predator model with infected prey is given in section 2. The disease free model is briefly discussed in section 3. In section 4 we have presented stability of the system and determine threshold parameter corresponding with its stability. In section 5, we have provide numerical simulation to confirm the analytical solutions.

2. The Prey Predator Model

Mathematical model dynamic prey predator were reformulated from the model that proposed [11]. The model was developed under assumption:

- Susceptible prey follow logistical growth.
- Infected prey does not reproduce.
- Infected prey does not recover or become immune and die more easily.
- The response function is Holling type II.
- The average growth of susceptible predator is proportional to predation of susceptible prey.
- Infection occurs when predator hunting or consuming infected prey.

The model describing interaction between prey and predator with infected prey lead to the following system equations

\[
\begin{align*}
\dot{S} &= r S \left(1 - \frac{S + I}{K}\right) - \varphi_1 I S - \frac{a_1 S P}{1 + a_1 S}, \\
\dot{I} &= \varphi_1 I S - \frac{a_2 I P}{1 + a_2 I} - \mu I, \\
\dot{P} &= \frac{\beta S P}{1 + a_1 S} - \gamma P - \frac{a_2 I P}{1 + a_2 I},
\end{align*}
\]  

(1)

where \( S \) is susceptible prey population, \( I \) is infected prey population and \( P \) is predator population, \( r \) is prey growth rate, \( K \) is carrying capacity, \( \varphi_1 \) and \( \varphi_2 \) are rate of infection, \( \alpha_1 \) and \( \alpha_2 \) are interaction between prey and predator, respectively, \( \beta \) is predator growth rate, \( \mu \) is death rate of infected prey, and \( \gamma \) is predator mortality rate, \( a_1 \) and \( a_2 \) are the respective half saturation rates of \( S \) and \( I \). All parameters are positive constant.

Stability of the system (1) will be analyzed based on the eigenvalues of the Jacobian matrix for all its critical points. The stability of system (1) is also associated with the threshold number.

3. Stability Analysis

3.1. Prey Predator Model with Free of Disease

First, we assume that there is no infected prey in the population. We consider system (1) with \( I = 0 \). The system (1) lead to

\[
\begin{align*}
\dot{S} &= r S \left(1 - \frac{S}{K}\right) - \frac{a_1 S P}{1 + a_1 S}, \\
\dot{P} &= \frac{\beta S P}{1 + a_1 S} - \gamma P.
\end{align*}
\]  

(2)

The are three critical points of the system (2)

\[
\begin{align*}
E_1 &= (0,0), \\
E_2 &= (K,0),
\end{align*}
\]

\[
E_3 = (S^*, P^*) = \left( \frac{\gamma}{\beta - \gamma a_1}, \frac{r}{\alpha_1} \left(1 - \frac{\gamma}{K(\beta - \gamma a_1)}\right) \left(1 + \frac{\gamma a_1}{\beta - \gamma a_1}\right) \right).
\]

2
Jacobian matrix for the critical points $E_1$ is

$$J_{E_1} = \begin{bmatrix} r & 0 \\ 0 & -\gamma \end{bmatrix}.$$  \hspace{1cm} (3)

There are two distinct real eigenvalues of matrix $J_{E_1}$. One is $r > 0$ and the other one is $-\gamma < 0$. It means that the critical point $E_1$ is a saddle point and unstable. Jacobian matrix for critical points $E_2$ is

$$J_{E_2} = \begin{bmatrix} -r - \frac{\alpha_1 K}{1 + a_1 K} & \frac{\beta K}{1 + a_1 K} - \gamma \\ 0 & \frac{\beta K}{1 + a_1 K} - \gamma \end{bmatrix}. \hspace{1cm} (4)$$

There are two distinct real eigenvalues of matrix $J_{E_2}$. One is $-r$ and other one is $\frac{\beta K}{1 + a_1 K} - \gamma$. It means that stability of critical points $E_2$ depend on value $\frac{\beta K}{1 + a_1 K} - \gamma$. Let

$$R_0 = \frac{\beta S}{\gamma (1 + a_1 K)}$$

is a threshold parameters. If $R_0 < 1$, then the critical point $E_2$ is node and asymptotically stable. On the other hand, if $R_0 > 1$, then the critical point $E_2$ is saddle point and unstable. Jacobian matrix for the critical points $E_3$ is

$$J_{E_3} = \begin{bmatrix} \frac{\gamma_1 (a_1 (K (\beta - a_1 y) - \gamma) - \beta)}{K \beta (\gamma - \gamma_1)} & -\frac{\alpha_1 y}{\beta} \\ \frac{\beta K}{K a_1} & 0 \end{bmatrix}. \hspace{1cm} (5)$$

Eigenvalues of matrix $J_{E_3}$ are

$$\lambda_{1,2} = -\frac{\gamma_1 (a_1 (K (\beta - a_1 y) - \gamma) - \beta)}{K \beta (\gamma - \gamma_1)} \pm \frac{\sqrt{\left(\frac{\gamma_1 (a_1 (K (\beta - a_1 y) - \gamma) - \beta)}{K \beta (\gamma - \gamma_1)}\right)^2 - 4 \frac{\gamma_1 (a_1 (K (\beta - a_1 y) - \gamma) - \beta)}{K a_1}}}{2}.$$

Stability of critical point $E_3$ depend on value $\frac{\gamma_1 (a_1 (K (\beta - a_1 y) - \gamma) - \beta)}{K \beta (\gamma - \gamma_1)}$, so that $\frac{\beta K}{\gamma (1 + a_1 K)} < 1 + \frac{\beta}{\gamma_1 (1 + a_1 K)}$.

(i) If $1 < R_0 < 1 + \frac{\beta}{\gamma_1 (1 + a_1 K)}$, then critical point $E_3$ is asymptotically stable.

(ii) If $R_0 > 1 + \frac{\beta}{\gamma_1 (1 + a_1 K)}$, then critical point $E_3$ is unstable.

3.2. Prey Predator Model with Infected Prey

Furthermore, we assume that there is infected prey in the population as show in the system (1). There are four critical points of the system (1).

$$e_1 = (0,0,0),$$

$$e_2 = (K,0,0),$$

$$e_3 = ((S^*,0,0^*),$$

$$e_4 = \left(\frac{\alpha}{\varphi_1}, \frac{r (K \varphi_1 - \mu)}{\varphi_1 (K \varphi_1 + r)}, 0\right).$$
Jacobian matrix for the critical point \( e_1 \) is

\[
J_{e_1} = \begin{bmatrix}
  r & 0 & 0 \\
  0 & -\mu & 0 \\
  0 & 0 & -\gamma
\end{bmatrix}.
\]

(6)

There are three distinct eigenvalues of matrix \( J_{e_1} \). One is \( r > 0 \), the other are \( -\mu < 0 \) and \( -\gamma < 0 \). It means that the critical point \( e_1 \) is a saddle point and unstable. Jacobian matrix for critical point \( e_2 \) is

\[
J_{e_2} = \begin{bmatrix}
  -r & -r - \phi_1 K & -\frac{a_1 K}{1+\alpha_1 K} \\
  0 & \phi_1 K - \mu & 0 \\
  0 & 0 & \frac{\beta K}{1+\alpha_1 K} - \gamma
\end{bmatrix}.
\]

(7)

There are three distinct eigenvalues of matrix \( J_{e_2} \) is \(-r\), \( \phi_1 K - \mu \), and \( \frac{\beta K}{1+\alpha_1 K} - \gamma \). It means that stability critical point \( e_2 \) depend on value \( \phi_1 K - \mu \), and \( \frac{\beta K}{1+\alpha_1 K} - \gamma \). Let

\[
R_3 = \frac{\phi_1 K}{\mu}
\]

is a threshold parameter.

(i) If \( R_0 < 1 \) and \( R_1 < 1 \), then the critical point \( e_2 \) is asymptotically stable. It means that the predator population will be vanished and the disease will be eradicated in prey population.

(ii) If \( R_0 > 1 \) and \( R_1 > 1 \), then the critical point \( e_2 \) is unstable

Jacobian matrix for critical point \( e_3 \) is

\[
J_{e_3} = \begin{bmatrix}
  \frac{r\gamma(a_1(K(\beta-a_1 \gamma)-\gamma)-\beta)}{K\beta(\beta-a_1 \gamma)} & \frac{\gamma}{\beta} & -\frac{a_1 \gamma}{\beta} \\
  0 & \frac{\phi_1 S^* - \alpha_2 P^* - \mu}{\alpha_1 K} & 0 \\
  \frac{r(K(\beta-a_1 \gamma)-\gamma)}{a_1 K} & \frac{\gamma}{\beta} & \frac{\phi_2 P^*}{\alpha_1 K}
\end{bmatrix}.
\]

(8)

Eigenvalues of \( J_{e_3} \) is

\[
\lambda_1 = \phi_1 S^* - \alpha_2 P^* - \mu,
\]

\[
\lambda_{2,3} = \frac{r\gamma(a_1(K(\beta-a_1 \gamma)-\gamma)-\beta)}{K\beta(\beta-a_1 \gamma)} \pm \sqrt{\left(\frac{\gamma}{\beta}\right)^2 - 4\frac{r(K(\beta-a_1 \gamma)-\gamma)}{K\alpha_1} \frac{1}{K\beta(\beta-a_1 \gamma)}},
\]

Stability of the critical point \( e_3 \) depend on value \( \phi_1 S^* - \alpha_2 P^* - \mu \) and \( \frac{r\gamma(a_1(K(\beta-a_1 \gamma)-\gamma)-\beta)}{K\beta(\beta-a_1 \gamma)} \). Let

\[
R_3 = \frac{\phi_1 S^*}{\alpha_2 P^* + \mu}
\]

is a threshold parameters.
(i) If \( 1 < R_0 < 1 + \frac{\beta}{\gamma a_1(1 + a_1 K)} \) and \( R_3 < 1 \), then the critical point \( e_3 \) is asymptotically stable.

(ii) If \( R_0 > 1 + \frac{\beta}{\gamma a_1(1 + a_1 K)} \) and \( R_3 > 1 \), then the critical point \( e_3 \) is unstable. It means that the disease will be endemic.

Jacobian for the critical point \( e_4 \) is

\[
J_{e_4} = \begin{bmatrix}
-\frac{\gamma}{\varphi_1 K} & -\frac{\gamma}{\varphi_1 K} & -\frac{\gamma}{\varphi_1 K} & -\frac{\alpha_2 \gamma}{\varphi_1 + \alpha_2 \gamma} \\
\frac{K \varphi_1}{\varphi_1 K + r} & 0 & 0 & -\frac{\alpha_2 \gamma}{\varphi_1 (K \varphi_1 - \mu)} \\
0 & 0 & 0 & M
\end{bmatrix}.
\] (9)

where

\[
M = \frac{\beta S^{***}}{1 + \alpha S^{***}} - \gamma - \frac{\varphi_2 I^{***}}{1 + \alpha I^{***}},
\]

\[
S^{***} = \frac{\mu}{\varphi_1},
\]

\[
I^{**} = \frac{\alpha \varphi_1 (K \varphi_1 - \mu)}{\varphi_1 (K \varphi_1 + r)}.
\]

Eigenvalues of \( J_{e_4} \) is

\[
\lambda_1 = M,
\]

\[
\lambda_2 = \frac{1}{2} \left( -\frac{\gamma}{\varphi_1 K} - \sqrt{\frac{\gamma^2}{\varphi_1 K} - 4 \frac{\gamma a_1 K^2 \varphi_1^2 \mu + 4 K \varphi_1 r \mu}{\varphi_1 K}} \right),
\]

\[
\lambda_2 = \frac{1}{2} \left( -\frac{\gamma}{\varphi_1 K} + \sqrt{\frac{\gamma^2}{\varphi_1 K} - 4 \frac{\gamma a_1 K^2 \varphi_1^2 \mu + 4 K \varphi_1 r \mu}{\varphi_1 K}} \right).
\]

Stability of critical point \( e_4 \) depend on value \( \frac{\beta S^{***}}{1 + \alpha S^{***}} - \gamma - \frac{\varphi_2 I^{***}}{1 + \alpha I^{***}} \). Let

\[
R_3 = \frac{\beta S^{***}}{\gamma (1 + \alpha S^{***})}
\]

is a threshold number.

(i) If \( R_2 < 1 + \frac{\varphi_2 I^{***}}{\gamma (1 + \alpha I^{***})} \), then the critical point \( e_4 \) is asymptotically stable.

(ii) If \( R_0 > 1 + \frac{\varphi_2 I^{***}}{\gamma (1 + \alpha I^{***})} \), then the critical point \( e_4 \) is unstable.

4. Numerical Solutions

Analytical result of the prey predator system (1) and (2) are confirmed by some numerical solutions showing phase portrait on phase plane with fix parameter [9]: \( r = 2, \varphi_3 = 0.0014, \varphi_2 = 0.1, \alpha_1 = 0.01, \alpha_2 = 0.5, \gamma = 1, \beta = 0.015, \mu = 0.9, \alpha_1 = 0.1, \alpha_2 = 0.5 \). Initial conditions set up at \((S(0), P(0)) = (200, 40) \) for \( K = 600 \), and \((S(0), P(0)) = (200, 150) \) for \( K = 490 \). Figure 1 and 3 show solutions of the system (2) and (1), respectively, Figure 2 show phase portrait of the system (2) that the critical points \( E_1 = (0, 0), E_2 = (K, 0), E_3 = (S^*, P^*) \) are unstable for \( K = 600 \). On the other hand, the critical points \( E_1 = (0, 0), \) and \( E_2 = (K, 0) \) are unstable, otherwise, the critical points \( E_3 = (S^*, P^*) \) asymptotically stable for \( K = 490 \).
Figure 1. Solution the system (2).

Figure 2. Phase portrait of the system (2).

Figure 3. Solution of the system (1).
5. Conclusion
Stability of prey predator system with free disease and infected prey has been analyzed. The result obtained state that the stability of the system depends on the value $K$, carrying capacity of prey population. The stability of the system is also related to threshold parameter. There are three critical points for prey predator system with free disease. Those critical points are unstable for value of $K = 600$ and stable for $K = 490$. Furthermore, solutions of the prey predator system with infected prey show that prey will die easily and predator will be infected caused by predation or consuming the infected prey.

Acknowledgments
Author would like acknowledge to the Directorate General of Higher Education, Republic of Indonesia for the funding of this research.

References
[1] Anderson RM and May RM 1979 Population biology of infectious diseases: Part-I Nature 280, 361–367
[2] Anderson RM and May RM 1986 The invasion, persistence, and spread of infectious diseases within animal and plant communities Philos. Trans. R. Soc. Lond B314 533–570
[3] Dawes JHP and Souza MO 2013 A derivation of Holling Type I, II, III Functional Response In Predator Prey Journal Of Theoretical Biology, 327 11-12
[4] Hadeler KP and Freedman HI 1989 Predator-prey populations with parasitic infection J.Math. Biol. 27 609–631
[5] Haque M 2009 Ratio-dependent predator-prey models of interacting populations Bull.Math. Biol. 71 430–452
[6] Haque M 2010 A predator-prey model with disease in the predator species only Nonlinear Anal. RWA 11 2224–2236
[7] Haque M 2011 A detailed study of the Beddington-DeAngelis predator-prey model. Math.Biosci. 234 1–16.
[8] Haque M, Sarwardi S, Preston S and Venturino E 2011 Effect of delay in a Lotka Volterra type predator-prey model with a transmissible disease in the predator species Math.Biosci. 234 47–57
[9] Hen-Ying H and Kue-Chin H 2008 Predator \-- prey model with disease infection in both population Math. Med. and Bio 25 217-266
[10] Heffernan JM, Smith RJ and Wahl LM 2005 Perspective on basic reproductive ratio J. The Royal Society Interface 2 281-293
[11] Mukhopadhyay B and Bhattacharyya B 2005 Dynamics od a delay-diffusion prey-predator model with disease in the prey J. Appl. Math. & Computing 17(1-2) 361-377.
[12] Becker NG, Bahramphour A and Dietz K 1994 Threshold parameter for epidemics in different community settings Mathematica Bioscience 129 189-200