RESEARCH ARTICLE • OPEN ACCESS

The Dynamics of a Predator-Prey Model Involving Disease Spread In Prey and Predator Cannibalism

Nurul Imamah Ah et al.

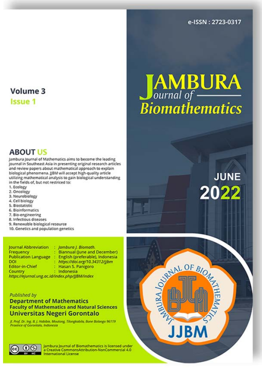
Volume 4, Issue 2, Pages 119–125, December 2023

Received 9 August 2023, Revised 23 October 2023, Accepted 5 December 2023, Published Online 31 December 2023

To Cite this Article : N. I. Ah et al.,"The Dynamics of a Predator-Prey Model Involving Disease Spread In Prey and Predator Cannibalism", *Jambura J. Biomath*, vol. 4, no. 2, pp. 119–125, 2023, *https://doi.org/10.37905/jjbm.v4i2.21495*

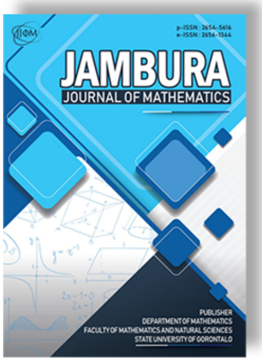
© 2023 by author(s)

JOURNAL INFO • JAMBURA JOURNAL OF BIOMATHEMATICS



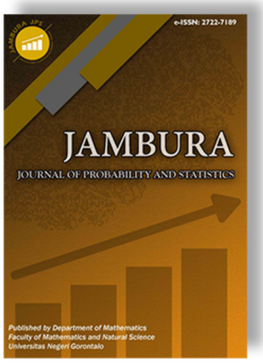
Homepage	:	http://ejurnal.ung.ac.id/index.php/JJBM/index
Journal Abbreviation	:	Jambura J. Biomath.
Frequency	:	Biannual (June and December)
Publication Language	:	English (preferable), Indonesia
DOI	:	https://doi.org/10.37905/jjbm
Online ISSN	:	2723-0317
Editor-in-Chief	:	Hasan S. Panigoro
Publisher	:	Department of Mathematics, Universitas Negeri Gorontalo
Country	:	Indonesia
OAI Address	:	http://ejurnal.ung.ac.id/index.php/jjbm/oai
Google Scholar ID	:	XzYgeKQAAAAJ
Email	:	editorial.jjbm@ung.ac.id

JAMBURA JOURNAL • FIND OUR OTHER JOURNALS

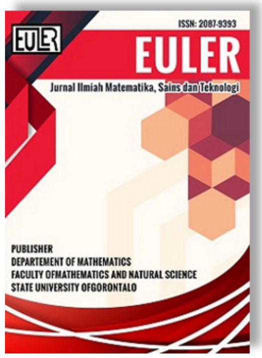


Jambura Journal of Mathematics

Jambura Journal of Mathematics Education



Jambura Journal of Probability and Statistics



EULER : Jurnal Ilmiah Matematika, Sains, dan Teknologi

Jambura Journal of Biomathematics, Volume 4, Issue 2, Pages 119–125, December 2023 https://doi.org/10.37905/jjbm.v4i2.21495

Research Article

Check for updates

The Dynamics of a Predator-Prey Model Involving Disease Spread In Prey and Predator Cannibalism

Nurul Imamah Ah[1,*], **Wuryansari Muharini Kusumawinahyu**[2], **Agus Suryanto**[3], **and Trisilowati** [4]

[1,2,3,4]Department of Mathematics, University of Brawijaya, Jl. Veteran, Ketawanggede, Lowokwaru, Malang, Jawa Timur 65145, Indonesia.
[1]Department of Mathematics Education, Muhammadiyah University of Jember, Jl. Karimata 49 Jember, Jawa Timur 68171, Indonesia.

ARTICLE HISTORY
Received 9 August 2023
Revised 23 October 2023
Accepted 5 December 2023
Published 31 December 2023

KEYWORDS
cannibalism
predator prey model
disease
dynamical analysis

ABSTRACT. *In this article, dynamics of predator prey model with infection spread in prey and cannibalism in predator is analyzed. The model has three populations, namely susceptible prey, infected prey, and predator. It is assumed that there is no migration in both prey and predator populations. The dynamical analysis shows that the model has six equilibria, namely the trivial equilibrium point, the prey extinction point, the disease free and predator extinction equilibrium point, the disease-free equilibrium point, the predator extinction equilibrium point, and the coexistence equilibrium point. The first equilibrium is unstable, and the other equilibria conditionally local asymptotically stable. The positivity and boundedness of the solution are also shown. The analytical result is supported by numerical simulation. It is shown that in such a high cannibalization the coexistence equilibrium is locally asymptotically stable.*

This article is an open access article distributed under the terms and conditions of the Creative Commons Attribution-NonComercial 4.0 International License. Editorial of JJBM: Department of Mathematics, Universitas Negeri Gorontalo, Jln. Prof. Dr. Ing. B. J. Habibie, Bone Bolango 96554, Indonesia.

1. Introduction

The relationship between predator and prey species, known as predation, is one of the interactions between species in an ecosystem. A mathematical model of predation was first proposed by Lotka and Volterra [1–3]. In the Lotka-Volterra model, it is assumed that both prey and predator are healthy. In fact, there is an interesting possibility of disease spreading among them and may influence the existence of prey and predators. As a result, Kermack et al [4] were among the first who used mathematical models to explore the spread of diseases or eco epidemiological models. Meanwhile, several researchers have discussed the eco-epidemiological model of predator prey with infected prey population [5–13]. Chattopadhyay et al [14] consider eco-epidemiological model which the transmission rate among the susceptible populations and the infected prey populations follows the simple law of mass action. The disease is spread among the prey population is not genetically inherited. And the infected populations do not become immune. The predator populations here use the type I Holling functional response. Furthermore, the ecoepidemiological model proposed by Biswas et al [15] implies that infected prey cannot become susceptible prey and that predators are harvested. Maisaroh et al [16] also analyzed the model of predator-prey with disease and proportional harvesting in predator.

Cannibalism is also a biological phenomenon which may influence the existence of predator-prey. Kang et al [17] studied a single-species cannibalism model with stage structure. The model studied is a dynamical system of one population with an age structure that divides the population into two classes, namely eggs and adult class. Deng et al [18] considered the dynamic behaviors of Lotka–Volterra predator–prey model incorporating predator cannibalism and show that cannibalism has both positive and negative effects on the stability of the system, it depends on the dynamic behaviors of the original system. Biswas et al [19] also analyzed a predator-prey model with disease in both prey and predator populations. They consider that the predator population is cannibalistic in nature and the disease spread in the predator population through cannibalism. Rayungsari et al [20] also developed a cannibalism of eco-epidemiological models in predator population. They examine that cannibalism acts as a self-regulatory mechanism and controls the disease transmission among the predators by stabilizing the predator prey oscillations. Zhang et al [21] developed an eco-epidemiological model with stage structure and cannibalism in predators, resulting in a three-dimensional dynamical model. The predator population is separated into two subpopulations in Zhang's model, namely juvenile and adult predators. The juvenile predator birth rate is proportional to the number of adult predators, and it follows Malthus growth model. Adult predators hunt on prey and juvenile predators in the rate represented by the type I Holling functional response.

Different from previous research, the formulation of the model in this paper combines Chattopadhyay et al [15] and Deng et al [18] in which predators attack suceptible prey and infected prey. It is assumed that predatorprey interactions follow cannibalism behaviour in predator. Shrimp and crab are the example of this ecoepidemiological model. Shrimp disease including the white spot syndrome virus (WSSV) can be caused by poor environmental quality and condition of shrimp. The goals of this research

*Corresponding Author.

Email : nurulimamah@unmuhjember.ac.id (N. I. Ah), wmuharini@ub.ac.id (W. M. Kusumawinahyu), suryanto@ub.ac.id (A. Suryanto), and trisilowati@ub.ac.id (Trisilowati)
Homepage : http://ejurnal.ung.ac.id/index.php/JJBM/index / E-ISSN : 2723-0317
© 2023 by the Author(s).

are to recreate the predator-prey model which is accounting for the existence of disease spread in the prey population, to find the equilibrium point, to examine the stability of the equilibrium point, and to perform numerical simulations to illustrate analytical result.

2. Formulation of the Model

The formulation of the model in this paper is inspired by Chattopadhyay et al [15] who developed a predator prey model with disease in the prey as follows.

$$\begin{aligned}\frac{dx_s}{dt} &= r(x_s+x_i)\left(1-\frac{x_s+x_i}{k}\right)-cx_sx_i-a_ix_sy,\\ \frac{dx_i}{dt} &= cx_sx_i-a_2x_iy-\delta x_i,\\ \frac{dy}{dt} &= d_1x_sy+d_2x_iy-\mu y.\end{aligned} \tag{1}$$

In this paper, we introduce the simple predator prey model involving disease spread in prey and cannibalism predator. We are assuming that:

1. The formulation of this model includes susceptible and infected prey. The susceptible prey population with intrinsic growth rate r and environmental carrying capacity k.
2. The infected prey does not return to susceptible.
3. Predators are cannibalistic.
4. There is natural mortality in infected prey and predators.
5. There is no migration of both prey and predators.

Based on those assumptions, we formulate the model as follows.

$$\begin{aligned}\frac{dx_s}{dt} &= rx_s\left(1-\frac{x_s+x_i}{k}\right)-cx_sx_i-a_ix_sy=0,\\ \frac{dx_i}{dt} &= cx_sx_i-a_2x_iy-\delta x_i=0,\\ \frac{dy}{dt} &= d_1x_sy+d_2x_iy+\gamma y-\frac{\beta y^2}{q+y}-\mu y=0.\end{aligned} \tag{2}$$

where x_s, x_i, and y respectively represent susceptible, infected prey and predator, with the following initial conditions.

$$x_s(0)>0, x_i(0)>0, y(0)>0.$$

All parameters considered are positive, which is defined in Table 1.

2.1. Positivity and boundedness.

In this section, positivity, and boundedness of solutions of model (2) have been investigated.

> **Theorem 1.** *All solutions of model (2) with initial values $x_s(0)$, $x_i(0)$, $y(0)\in\mathbb{R}^3_+$ are non-negative [20].*

Proof. First, we proof that if $x_s(0)\geq 0$, $x_i(0)\geq 0$, and $y(0)\geq 0$, then $x_s(t)\geq 0$, $x_i(t)\geq 0$, and $y(t)\geq 0$ for $t>0$. If $x_s(0)=0$, then

$$\frac{x_s}{dt}=0,\ \text{at } t=0.$$

It means that the prey population density x_s does not change from the beginning to the next. Hence, it is assumed that $x_s(0)>0$. If $x_s(0)\geq 0$ for every $t\geq 0$ is not true, then there is $t_1>0$ such that $x_s(t)>0$ for $0<t<t_1$, $x_s(t)=0$, for $t=t_1$ and $x_s(t)<0$ for $t>t_1$, From model (2) we obtain:

$$\frac{x_s}{dt}=0,\ \text{at } t=t_1.$$

Thus, there is no change in the population density of x_s when $t=t_1$. This contradicts the statement that $x_s(t)<0$ for $t>t_1$. Therefore, the previous assumption is false, which means $x_s(0)\geq 0$ for every $t>0$. In the same way, it can be proof that $x_i(0)\geq 0$ and $y(0)\geq 0$ for every $t>0$. □

> **Theorem 2.** *All solutions of model (2) in the region $\Omega=(x_s+x_i+y)<\dfrac{\omega}{\rho}\in\mathbb{R}^3_+$ are uniformly bounded.*

Proof. Choose a function defined by $v(t)=x_s(t)+x_i(t)+y(t)$, where $x_s>0$, $x_i>0$, $y>0$.

$$\begin{aligned}\frac{dv}{dt}+\rho v &= rx_s\left(1-\frac{x_s+x_i}{k}\right)-a_1x_sy-cx_sx_i+cx_sx_i\\ &\quad -a_2x_iy-\delta x_i-\mu y+d_1x_sy+d_2x_iy-\frac{\beta y^2}{q+y}\\ &\quad +\gamma y+\rho(x_s+x_i+y),\end{aligned}$$

if $d_1<a_1$, $d_2<a_2$, Then

$$\begin{aligned}\frac{dv}{dt}+\rho v &\leq rx_s\left(1-\frac{x_s+x_i}{k}\right)-\delta x_i-\mu y-\frac{\beta y^2}{q+y}+\gamma y\\ &\quad +\rho x_s+\rho x_i+\rho y,\\ &\leq rx_s\left(1-\frac{x_s+x_i}{k}\right)+(\rho-\delta)x_i+(\rho+\gamma-\mu)y\\ &\quad -\frac{\beta y^2}{q+y}+\rho x_s.\end{aligned}$$

choose $\rho<\min\{\delta,\mu-\gamma\}$, then.

$$\begin{aligned}\frac{dv}{dt}+\rho v &\leq rx_s\left(1-\frac{x_s+x_i}{k}\right)+\rho x_s,\\ &\leq rx_s-\frac{rx_s^2}{k}+\rho x_s,\\ &= (r+\rho)x_s-\frac{rx_s^2}{k},\\ &= -\frac{r}{k}\left[x_s^2-\frac{(r+\rho)k}{2r}x_s+\left(\frac{(r+\rho)k}{2r}\right)^2-\left(\frac{(r+\rho)k}{2r}\right)^2\right],\\ &= -\frac{r}{k}\left(x_s-\frac{(r+\rho)k}{2r}\right)^2+\frac{r}{k}\left(\frac{(r+\rho)^2k^2}{4r^2}\right),\\ &\leq \frac{(r+\rho)^2k}{4r}.\end{aligned}$$

We get.

$$\frac{dv}{dt}+\rho v(t)\leq w$$

with $w=\dfrac{(r+\rho)^2k}{4r}$.

$$\begin{aligned}e^{\rho t}\left(\frac{dv}{dt}+\rho v(t)\right) &\leq e^{\rho t}w,\\ \frac{d(e^{\rho t}v)}{dt} &\leq e^{\rho t}w,\\ e^{\rho t}v &\leq \int e^{\rho t}w dt,\\ v &\leq e^{-\rho t}w\left(\frac{e^{\rho t}}{\rho}+c\right),\\ v &\leq \left(\frac{w}{\rho}+ce^{-\rho t}w\right).\end{aligned}$$

Table 1. Definiton Parameter and Ecological Meaning

Parameter	Ecological Meaning
r	Intrinsic per capita growth rate of prey population
k	Carrying capacity of susceptible prey population
a_1	Maximum consumption rate of predator population
c	Disease transmission rate in prey population
a_2	Attack rate of infected prey
δ	Natural death rate of infected prey
μ	Natural death rate of predator population
d_1	Conversion rate of susceptible prey
d_2	Conversion rate of infected prey
γ	Conversion of cannibalism into predator birth
q	Half saturation constant of predator cannibalism
β	Predator cannibalism rate

with $C = cw$ by subtitution $t = 0$ to

$$v(t) \leq \frac{w}{\rho} + Ce^{-\rho t}.$$

We get.

$$C = v(0) - \frac{w}{\rho},$$

then

$$v(t) \leq \frac{w}{\rho} + \left(v(0) - \frac{w}{\rho}\right) e^{-\rho t},$$

if $v(0) \leq \frac{w}{\rho}$, then $v < \frac{w}{\rho}$, If $v(0) > \frac{w}{\rho}$, then $\frac{w}{\rho} < v(t) < v(0)$ because $\lim_{t\to\infty} v(t) = \frac{w}{\rho}$. Therefore all solution are uniformly bounded. □

3. Equilibrium Points and Stability Analysis

3.1. Equilibrium Points

We find an equilibrium points of equation by equating the derivatives on the left-hand side to zero, namely.

$$\begin{aligned} x_s\left[r\left(1 - \frac{x_s + x_i}{k}\right) - a_1 y - c x_i\right] &= 0, \\ x_i[c x_s - a_2 - \delta] &= 0,, \\ y\left[-\mu + d_1 x_s + d_2 x_i - \frac{\beta y}{q + y} + \gamma\right] &= 0 \end{aligned} \tag{3}$$

1. The trivial equilibrium point $E_0 = (0, 0, 0)$, that always exists $\mathbb{R}^3_+$.
2. The prey extinction equilibrium point
$$E_1 = (0, 0, \hat{y}).$$
where $\hat{y} = \frac{\mu q - \gamma q}{\gamma - \mu - \beta}$. Equilibrium point E_1 exists in $\mathbb{R}^3_+$, if $\gamma - \beta < \mu < \gamma$. This condition shows that even though suspectible and infected prey is extinct, predator still survives the rate of cannibalism greater than natural death rate of predator population.
3. The disease free and predator extinction equilibrium point
$$E_2 = (k, 0, 0).$$
Equilibrium points E_2 always exists in $\mathbb{R}^3_+$.
4. The disease-free equilibrium point.
$$E_3 = (\tilde{x}_s, 0, \tilde{y}).$$
where $\tilde{y} = \frac{rk - r\tilde{x}_s}{a_1 k}$. Equilibrium points of E_3 exists in $\mathbb{R}^3_+$, if $\tilde{x}_s < k$.
5. The predator extinction equilibrium point.
$$E_4 = \left(\frac{\delta}{c}, \frac{r(ck - \delta)}{c(ck + r)}, 0\right).$$
Equilibrium points E_4 exists in $\mathbb{R}^3_+$, if $ck > \delta$.
6. The coexistence equilibrium point $E_5 = (x_s^*, x_i^*, y^*)$ with
$$\begin{aligned} x_s^* &= \frac{\varphi_2 \pm \sqrt{\varphi_2^2 - 4\varphi_1\varphi_3}}{2\varphi_1}, \\ x_i^* &= \frac{a_2 kr + \delta a_1 k - (a_2 r + a_1 ck) x_s^*}{a_2(r + ck)}, \\ y^* &= \frac{c x_s^* - \delta}{a_2}. \end{aligned}$$
Where:
$$\begin{aligned} \varphi_1 &= d_2 P - d_1 Q, \\ \varphi_2 &= a_2 R - a_2 S + d_2 T, \\ \varphi_3 &= a_2 U - V + W, \\ P &= a_1 k c^2 + a_2^2 rc, \\ Q &= a_2 c^2 k + c a_2 r, \\ R &= \mu rc + \mu c^2 k + d_1 ck\delta + a_2 d_2 rq + a_1 d_2 ckq \\ &\quad + a_1 d_2 ckq + \beta cr + \beta c^2 k, \\ S &= a_2 d_1 qr + d_1 \delta r + a_2 d_1 ckq + \gamma c^2 k + d_2 ckr, \\ T &= a_1 \delta ck + a_1 \delta ck, \\ U &= a_2 \mu qr + \mu a_2 ckq + \delta\mu ck + \delta\gamma r + \delta\gamma ck + \delta d_2 kr \\ &\quad + \beta\delta ck, \\ V &= \delta\mu r + \gamma a_2 qr + \gamma cr + \gamma a_2 ckq + \delta a_2 d_2 kq \mp \delta^2 k \\ &\quad + a_2 d_2 kqr + \beta\delta r, \\ W &= \delta d_2 rk \end{aligned}$$
Let's $D = \varphi_2^2 - 4\varphi_1\varphi_3$ the following conditions are met.
   - if $D = 0$, then $x_s^* = \frac{-\varphi_2}{2\varphi_1}$, this equation has a positive root, when $\varphi_1 < 0$ and $\varphi_2 > 0$, or $\varphi_1 > 0$ and $\varphi_2 < 0$.
   - for $D > 0$:
     (a) if $\frac{\varphi_2}{\varphi_1} > 0$ and $\varphi_1\varphi_3 < 0$, then one fixed point is obtained.
     (b) if $\frac{\varphi_2}{\varphi_1} < 0$ and $\varphi_1\varphi_3 < 0$, then one fixed point is obtained.
     (c) if $\frac{\varphi_2}{\varphi_1} < 0$ and $0 < 4\varphi_1\varphi_3 < \varphi^2$, then two fixed point are obtained.

   Equilibrium point E_5 exists if $\frac{\delta}{c} < x_s^* < \frac{k(a_2 r + \delta a_1)}{a_2 r + a_1 ck}$.

3.2. *Local Stability*

Here we examine the eigen value by Jacobian matrix.

$$J = \begin{bmatrix} j_{11} & j_{12} & j_{13} \\ j_{21} & j_{22} & j_{23} \\ j_{31} & j_{32} & j_{33} \end{bmatrix},$$

where

$$\begin{aligned} j_{11} &= r - \frac{2rx_s}{k} - \frac{rx_i}{k} - a_1y - cx_i, \\ j_{12} &= \frac{2rx_s}{k} - cx_s, \\ j_{13} &= -a_1x_s, \\ j_{21} &= cx_i, \\ j_{22} &= cx_s - a_2y - \delta, \\ j_{23} &= -a_2x_i, \\ j_{31} &= d_1y, \\ j_{32} &= d_2y, \\ j_{33} &= -\mu + d_1x_s + d_2x_i + \gamma - \frac{2\beta(q+y) - \beta y}{(q+y)^2}. \end{aligned}$$

The stability of the equilibrium points of the model (2) are determined by the eigenvalues of the Jacobian matrix and the result is obtained in the following theorem.

Theorem 3. *The local stability of the equilibrium points of the model is as follows.*

i. The equilibrium $E_0 = (0,0,0)$ is always unstable.
ii. $E_1 = (0,0,\hat{y})$ is locally asymptotically stable if $r < a_1\hat{y}$ and unstable if $r > a_1\hat{y}$.
iii. $E_2 = (k,0,0)$ is locally asymptotically stable if $k < \min\{\frac{\delta}{c}, \frac{\mu-\gamma}{d_1}\}$ and unstable if $k > \min\{\frac{\delta}{c}, \frac{\mu-\gamma}{d_1}\}$.
iv. $E_3 = (\tilde{x}_s, 0, \tilde{y})$ is locally asymptotically stable if $c\tilde{x}_s < a_2\tilde{y} + \delta$.
v. $E_4 = \left(\frac{\delta}{c}, \frac{r(ck-\delta)}{c(ck+r)}, 0\right)$ is locally asymptotically stable if $\mu > \gamma + d_1\left(\frac{\delta}{c}\right) + d_2\left(\frac{ckr-\delta r}{c^2k+r}\right)$.
vi. $E_5 = (x_s^*, x_i^*, y^*)$ is locally asymptotically stable if $\rho_1 > 0$, $\rho_3 > 0$, and $\rho_1\rho_2 - \rho_3 > 0$.

Proof. 1. By substituting $E_0 = (0,0,0)$ to the model (1), we have

$$J(E_0) = \begin{bmatrix} r & 0 & 0 \\ 0 & -\delta & 0 \\ 0 & 0 & -\mu+\gamma \end{bmatrix},$$

Then we get eigen values $\lambda_1 = r$, $\lambda_2 = -\delta$, and $\lambda_3 = -\mu + \gamma$. Since λ_1 positive, equilibrium point $E_0 = (0,0,0)$ is always unstable.

2. From eq. (3), $E_1 = (0,0,\hat{y})$ complete the equation $-\mu + d_1x_s + d_2x_i + \gamma - \frac{\beta y}{q+y} = 0$, then we have the Jacobian matrix for E_1 is

$$J(E_1) = \begin{bmatrix} r - a_1\hat{y} & 0 & 0 \\ 0 & -a_2\hat{y} - \delta & 0 \\ d_1\hat{y} & d_2\hat{y} & \frac{\beta\hat{y}^2(q+\hat{y}) - 2\beta q\hat{y} - \beta\hat{y}^2}{(q+\hat{y})^2} \end{bmatrix},$$

The eigen values for $J(E_1)$ are $\lambda_1 = r - a_1\hat{y}$, $\lambda_2 = -a_2\hat{y} - \delta$, and $\lambda_3 = \frac{-\beta q\hat{y}}{(q+\hat{y})^2}$. E_1 is locally asymptotically stable if $r < a_1\hat{y}$.

3. The Jacobian matrix for $E_2 = (k,0,0)$

$$J(E_2) = \begin{bmatrix} r & kr - ck & -a_1k \\ 0 & ck - \delta & 0 \\ 0 & 0 & -\mu + d_1k + \gamma \end{bmatrix},$$

has $\lambda_1 = -r$, $\lambda_2 = ck - \delta$, and $\lambda_3 = d_1k + \gamma - \mu < 0$ then $k < \frac{\mu-\gamma}{d_1}$. E_2 locally asymptotically stable if $k < \min\left\{\frac{\delta}{c}, \frac{\mu-\gamma}{d_1}\right\}$, otherwise, if $k > \min\left\{\frac{\delta}{c}, \frac{\mu-\gamma}{d_1}\right\}$, E_2 becomes unstable.

4. From eq. (3) we get $r - \frac{rx_s}{k} - \frac{rx_i}{k} - a_1y - cx_i = 0$, and $-\mu + d_1x_s + d_2x_i - \frac{\beta y}{q+y} + \gamma = 0$, then the Jacobian matrix for infected prey extinction point is

$$J(E_3) = \begin{bmatrix} \tilde{j}_{11} & \tilde{j}_{12} & \tilde{j}_{13} \\ \tilde{j}_{21} & \tilde{j}_{22} & \tilde{j}_{23} \\ \tilde{j}_{31} & \tilde{j}_{32} & \tilde{j}_{33} \end{bmatrix}.$$

where

$$\begin{aligned} &\tilde{j}_{11} = -\frac{r\tilde{x}_s}{k}, && \tilde{j}_{22} = c\tilde{x}_s - a_2\tilde{y} - \delta, \\ &\tilde{j}_{12} = \frac{r\tilde{x}_s}{k} - c\tilde{x}_s, && \tilde{j}_{23} = 0, \\ &\tilde{j}_{13} = -a_1\tilde{x}_s, && \tilde{j}_{31} = d_1\tilde{y}, \\ &\tilde{j}_{21} = 0, && \tilde{j}_{32} = d_2\tilde{y}, \\ &\tilde{j}_{33} = \frac{-\beta q\tilde{y}}{(q+\tilde{y})^2}. \end{aligned}$$

So that the eigen values are $\lambda_1 = c\tilde{x}_s - a_2\tilde{y} - \delta$, and λ_2, λ_3 is the eigen values of

$$J_1(E_3) = \begin{bmatrix} \tilde{j}_{11} & \tilde{j}_{13} \\ \tilde{j}_{31} & \tilde{j}_{33} \end{bmatrix}.$$

E_3 is locally asimtotically stable if $\det J_1(E_3) = \tilde{j}_{11}\tilde{j}_{33} - \tilde{j}_{13}\tilde{j}_{31} > 0$ and $\mathrm{trace} J_1(E_3) = \tilde{j}_{11} + \tilde{j}_{33} < 0$. The determinant and the trace of the matrix $J_1(E_3)$ are respectively, given by

$$\begin{aligned} \det J_1(E_3) &= \tilde{j}_{11}\tilde{j}_{33} - \tilde{j}_{13}\tilde{j}_{31}, \\ &= \left(\frac{\beta qr\tilde{x}_s\tilde{y}}{k(q+\tilde{y})^2}\right) + (d_1\tilde{y}a_1\tilde{x}_s) > 0, \text{ and} \\ \mathrm{trace} J_1(E_3) &= \tilde{j}_{11} + \tilde{j}_{33}, \\ &= -\frac{r\tilde{x}_s}{k} - \frac{\beta q\tilde{y}}{(q+\tilde{y})^2} < 0. \end{aligned}$$

Then E_3 is locally asimtotically stable if $c\tilde{x}_s < a_2\tilde{y} + \delta$.

5. Based on eq. (3), E_4 complete $r - \frac{rx_s}{k} - \frac{rx_i}{k} - a_1y - cx_i = 0$, then by substituting $E_4 = \left(\frac{\delta}{c}, \frac{r(ck-\delta)}{c(ck+r)}, 0\right)$ to the Jacobian matrix, obtained

$$J(E_4) = \begin{bmatrix} \bar{j}_{11} & \bar{j}_{12} & \bar{j}_{13} \\ \bar{j}_{21} & \bar{j}_{22} & \bar{j}_{23} \\ \bar{j}_{31} & \bar{j}_{32} & \bar{j}_{33} \end{bmatrix}.$$

Table 2. Parameter Value

Parameter	Simulation	Simulation 2	Simulation 3	Simulation 4	Simulation 5
r	0.2	0.2	5	0.2	2
k	1	0.5	0.5	0.5	1
a_1	0.5	0.5	5	0.5	0.5
c	0.5	0.1	1	0.5	0.4
a_2	0.2	0.2	5	0.2	0.5
δ	0.1	0.5	0.5	0.1	0.01
d_1	0.3	0.1	3	0.3	1
d_2	1	1	1	1	1
β	0.2	0.3	0.3	0.3	1
q	1	1	0.5	1	1
γ	0.2	0.5	0.5	0.5	0.5
μ	0.1	1	0.5	1	1

where

$$\begin{aligned} \bar{j}_{11} &= -\frac{r\delta}{ck}, & \bar{j}_{22} &= 0, \\ \bar{j}_{12} &= \frac{\delta(r-ck)}{ck}, & \bar{j}_{23} &= -a_2\left(\frac{r(ck-\delta)}{c(ck+r)}\right), \\ \bar{j}_{13} &= \frac{-a_1\delta}{c}, & \bar{j}_{31} &= 0, \\ \bar{j}_{21} &= -\frac{r(ck-\delta)}{(ck+r)}, & \bar{j}_{32} &= 0, \\ \bar{j}_{33} &= -\mu + d_1x_s + d_2x_i + \gamma. \end{aligned}$$

So that the eigen values $\lambda_1 = \gamma - \mu + d_1\left(\frac{\delta}{c}\right) + d_2\left(\frac{ckr-\delta r}{c^2k+r}\right)$, and $\lambda_{2,3}$ fulfill

$$J_1(E_4) = \begin{bmatrix} \bar{j}_{11} & \bar{j}_{12} \\ \bar{j}_{21} & \bar{j}_{22} \end{bmatrix},$$

E_4 asymptotically local stable if det $J_1(E_4) = \bar{j}_{11}\bar{j}_{22} - \bar{j}_{12}\bar{j}_{21} > 0$ and trace $J_1(E_4) = \bar{j}_{11} + \bar{j}_{22} < 0$. Respectively det $J(E_4) = -\left(\frac{\delta(r-ck)}{ck}\right)\left(-\frac{r(ck-\delta)}{(ck+r)}\right) > 0$ and trace $J_1(E_4) = -\dfrac{r\delta}{ck} < 0$, then E_4 is locally asimtotically stable if $\gamma + d_1\left(\frac{\delta}{c}\right) + d_2\left(\frac{ckr-\delta r}{c^2k+r}\right) < \mu$.

6. From eq. (3), E_5 complete the equation $r - \frac{rx_s}{k} - \frac{rx_i}{k} - a_1y - cx_i = 0$, and $-\mu + d_1x_s + d_2x_i - \dfrac{\beta y}{q+y} + \gamma = 0$, then by substituting $E_5 = (x_s^*, x_i^*, y^*)$ to the Jacobian matrix, we get

$$\begin{aligned} J(E_5) &= \begin{bmatrix} j_{11}^* & j_{12}^* & j_{13}^* \\ j_{21}^* & j_{22}^* & j_{23}^* \\ j_{31}^* & j_{32}^* & j_{33}^* \end{bmatrix}, \text{ or} \\ |J(E_5) - \lambda I| &= \begin{bmatrix} j_{11}^* - \lambda & j_{12}^* & j_{13}^* \\ j_{21}^* & j_{22}^* - \lambda & j_{23}^* \\ j_{31}^* & j_{32}^* & j_{33}^* - \lambda \end{bmatrix}, \\ \det|J(E_5) - \lambda I| &= (j_{11}^* - \lambda)(j_{22}^* - \lambda)(j_{33}^* - \lambda) + A_8 \\ &\quad + A_9 - (j_{11}^* - \lambda)A_3 - (j_{22}^* - \lambda)A_1 \\ &\quad - (j_{33}^* - \lambda)A_2, \\ &= -\lambda^3 + (j_{11}^* + j_{22}^* + j_{33}^*)\lambda^2 \\ &\quad + (A_1 + A_2 + A_3 - A_4 - A_5 - A_6)\lambda \\ &\quad + A_7 + A_8 + A_9 - A_{10} - A_{11} - A_{12}, \\ &= -\lambda^3 + \tilde{\rho}_1\lambda^2 + \tilde{\rho}_2\lambda + \tilde{\rho}_3. \end{aligned}$$

where

$$\begin{aligned} j_{11}^* &= -\frac{rx_s^*}{k}, & j_{22}^* &= cx_s^* - a_2y^* - \delta, \\ j_{12}^* &= \frac{rx_s^*}{k} - cx_s^*, & j_{23}^* &= -a_2x_i^*, \\ j_{13}^* &= -a_1x_s^*, & j_{31}^* &= d_1y^*, \\ j_{21}^* &= cx_i^*, & j_{32}^* &= d_2y^*, \\ j_{33}^* &= \frac{-\beta qy^*}{(q+y^*)^2}, & \rho_1 &= j_{11}^* + j_{22}^* + j_{33}^*, \\ A_1 &= j_{13}^*j_{31}^*, & \rho_2 &= A_1 + A_2 + A_3 - A_4 - A_5 - A_6, \\ A_2 &= j_{12}^*j_{21}^*, & \rho_3 &= A_7 + A_8 + A_9 - A_{10} - A_{11} - A_{12}, \\ A_3 &= j_{23}^*j_{32}^*, & A_8 &= j_{12}^*j_{23}^*j_{31}^*, \\ A_4 &= j_{11}^*j_{22}^*, & A_9 &= j_{13}^*j_{21}^*j_{32}^*, \\ A_5 &= j_{22}^*j_{33}^*, & A_{10} &= A_3j_{11}^*, \\ A_6 &= j_{11}^*j_{33}^*, & A_{11} &= A_1j_{22}^*, \\ A_7 &= A_4j_{33}^*, & A_{12} &= A_2j_{33}^*. \end{aligned}$$

The characteristic equation from $J(E_5)$ is $\lambda^3 + \rho_1\lambda^2 + \rho_2\lambda + \rho_3 = 0$, with $\rho_1 = -\tilde{\rho}_1$, $\rho_2 = -\tilde{\rho}_2$, and $\rho_3 = \tilde{\rho}_3$.
To find the local stability of eq. (2) we use Routh Hurwitz criterion. E_5 is locally asymptotically stable if:

- $\rho_1 > 0$,
- $\rho_1\rho_2 - \rho_3 > 0$, and
- $\rho_3 > 0$.

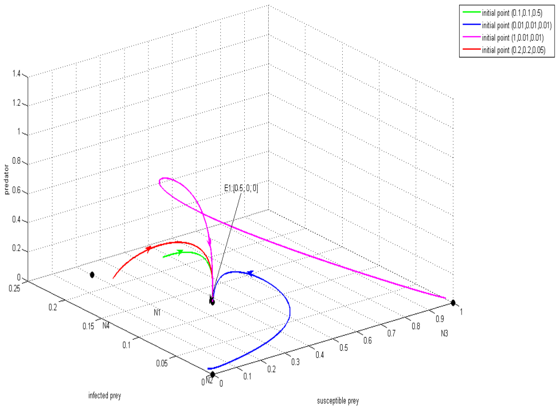
Because $\rho_1\rho_2 - \rho_3$ is too complex and difficult, so the stability of E_5 is evaluated numerically.

□

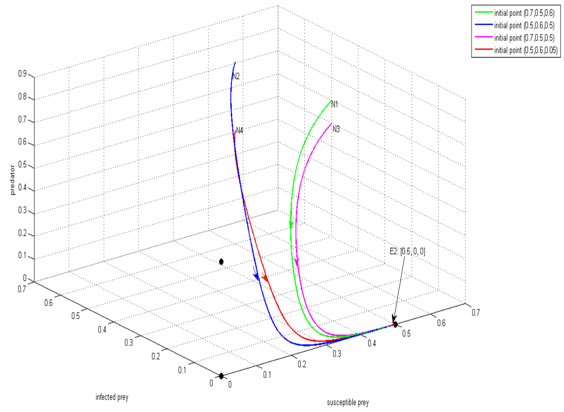
4. Numerical Simulation

In this section, we give some flow of solutions to demonstrate the stability around the equilibrium points that are associated with the previous theoretical result. We use Runge-Kutta 4^{th} order as the numerical methods, the numerical simulations of the model (2) are illustrated with a various condition based on [15–19] as given in Table 2 as follows.

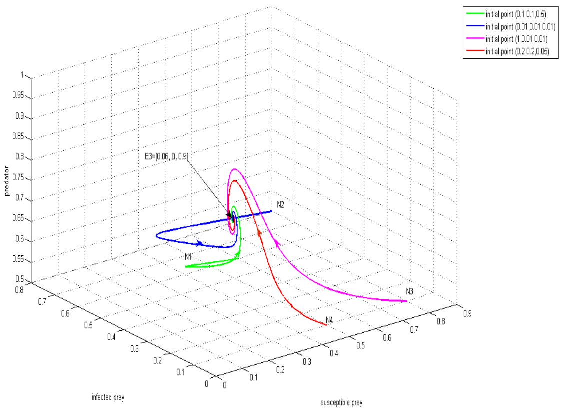
For parameter value in Table 2 for simulation 1, E_1 exists i.e. $[0, 0, 0.5]$ and it is asymptotically local stable. Since it is satisfying stability condition in Theorem 3 that $r < a_1\hat{y}$. The density of susceptible and infected prey goes to extinction, and the density of predator population exists. Its condition was shown in Figure 1a. E_1 shows that there are no shrimps, but crabs are exist. By using the parameter value in in Table 2 for simulation 2, E_2 exists i.e. $[0.5, 0, 0]$ and it is asymtotically local stable, Since Theorem 3 is fulfilled $k < \min\{\frac{\delta}{c}, \frac{\mu-\gamma}{d_1}\}$. This is consistent with the analytical result since the Jacobian matrix eigenvalues are negative numbers. The density of suceptible prey population



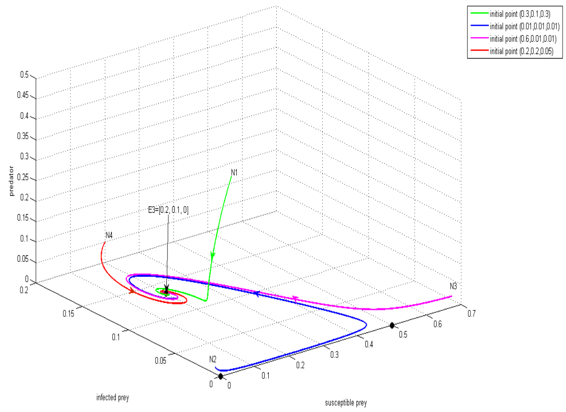
(a) E_1



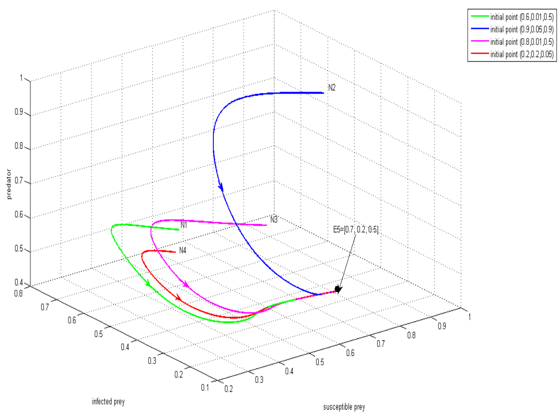
(b) E_2



(c) E_3



(d) E_4



(e) E_5

Figure 1. The figure depics the solution of the model (1) for equilibrium point:

is existed, but the density of infected prey and predator tends to extinction. Figure 1b depicts its current condition. E_2 demonstrates that shrimp are present but crabs are not.Numerical simulation around the equilibrium point E_3 using a parameter value in Table 2 simulation 3, E_3 is exists i.e., $[0.06, 0, 0.9]$ and it is asymtotically local stable, the stability conditions is fulfilled according to the Theorem 3 with the stability condition $c\tilde{x}_s = 0,0048 < a_2\tilde{y} + \delta = 5.5421$. The density of susceptible prey and predator population exists, but the density of infected prey is heading to extinction. This condition was shown in Figure 1c. E_3 shows that there isn't a healthy shrimp, but both sick shrimps and crabs exist. Figure 1d use the parameter value in Table 2 simulation 4, E_4 exists i.e., $[0.2, 0.1, 0]$ and it is asymtotically local stable, this is consistent with the analytical result in Theorem 3 and satisfy the condition $\gamma + d_1\left(\frac{\delta}{c}\right) + d_2\left(\frac{rck - r\delta}{c^2k + r}\right) = 0.7378 < \mu = 1$. The density of predators is heading to extinction, but the density of susceptible prey and infected prey exists. E_4 illustrates that there are both healthy and sick shrimp, but no crabs. Figure 1e use the selected parameter value in Table 2 simulation 5, E_5 exists, i.e., $[0.7, 0.2, 0.5]$ and it is asymtotically local stable, and satisfy stability condition using Routh Hourwitz criterion, $\rho_1 = 1.485 > 0$, $\rho_3 = 0.2048 > 0$, and $\rho_1\rho_2 - \rho_3 = 0.3391 > 0$. This is consistent with the analytical result in Theorem 3, so the density of all spesies shrimps and crabs exists.

5. Conclusion

We have formulated a model to describe an interaction of prey species and predator cannibalism. we show the dynamics of the system, especially the behaviour of solutions around the equilibrium point. There are six equilibria in this model, namely the trivial equilibrium point, the prey extinction point, the disease free and predator extinction equilibrium point, the disease-free equilibrium point, the predator extinction equilibrium point, and the coexistence equilibrium point. The first equilibrium is unstable, and the other equilibria is asymptotically stable with conditionally stable. All the result are based on numerical simulations by Runge Kutta method.

Author Contributions. **Ah, N.I.**: Conceptualization, methodology, software, formal analysis, investigation, resources, data curation, writing—original draft preparation. **Kusumawinahyu, W.M.**: Conceptualization, methodology, validation, writing—review and editing, visualization, supervision. **Suryanto, A.**: Conceptualization, methodology, validation, writing—review and editing, visualization, supervision. **Trisilowati**: Conceptualization, methodology, validation, writing—review and editing, visualization, supervision.

Acknowledgement. The authors are thankful the editors and reviewers who have supported us in improving this manuscript.

Funding. This research received no external funding.

Conflict of interest. The authors declare no conflict of interest.

Data availability. Not applicable.

References

[1] A. A. Berryman, "The origins and evolution of predator-prey theory," *Ecology*, vol. 73, no. 5, pp. 1530–1535, 1992. DOI:10.2307/1940005

[2] H. S. Panigoro and E. Rahmi, "Computational dynamics of a Lotka-Volterra Model with additive Allee effect based on Atangana-Baleanu fractional derivative," *Jambura Journal of Biomathematics (JJBM)*, vol. 2, no. 2, pp. 96–103, 2021. DOI:10.34312/jjbm.v2i2.11886

[3] D. Mukherjee, "Fear induced dynamics on Leslie-Gower predator-prey system with Holling-type IV functional response," *Jambura Journal of Biomathematics (JJBM)*, vol. 3, no. 2, pp. 49–57, 2022. DOI:10.34312/jjbm.v3i2.14348

[4] W. O. Kermack and A. Mckendrick, "Contributions to the mathematical theory of epidemics—I," *Bulletin of Mathematical Biology*, vol. 53, no. 1-2, pp. 33–55, 1991. DOI:10.1016/S0092-8240(05)80040-0

[5] Y. P. Zhang, M. J. Ma, P. Zuo, and X. Liang, "Analysis of a eco-epidemiological model with disease in the predator," *Applied Mechanics and Materials*, vol. 536-537, pp. 861–864, 2014. DOI:10.4028/www.scientific.net/AMM.536-537.861

[6] Y. Xiao and L. Chen, "Modeling and analysis of a predator-prey model with disease in the prey," *Mathematical Biosciences*, vol. 171, no. 1, pp. 59–82, 2001. DOI:10.1016/S0025-5564(01)00049-9

[7] D. Greenhalgh and M. Haque, "A predator-prey model with disease in the prey species only," *Mathematical Methods in the Applied Sciences*, vol. 30, no. 8, pp. 911–929, 2007. DOI:10.1002/mma.815

[8] L. Han, Z. Ma, and H. W. Hethcote, "Four predator prey models with infectious diseases," *Mathematical and Computer Modelling*, vol. 34, no. 7-8, pp. 849–858, 2001. DOI:10.1016/S0895-7177(01)00104-2

[9] H. W. Hethcote, W. Wang, L. Han, and Z. Ma, "A predator - Prey model with infected prey," *Theoretical Population Biology*, vol. 66, no. 3, pp. 259–268, 2004. DOI:10.1016/j.tpb.2004.06.010

[10] M. S. Rahman and S. Chakravarty, "A predator-prey model with disease in prey," *Nonlinear Analysis: Modelling and Control*, vol. 18, no. 2, pp. 191–209, 2013. DOI:10.15388/NA.18.2.14022

[11] X. Zhou, J. Cui, X. Shi, and X. Song, "A modified Leslie-Gower predator-prey model with prey infection," *Journal of Applied Mathematics and Computing*, vol. 33, no. 2010, pp. 471–487, 2009. DOI:10.1007/s12190-009-0298-6

[12] A. Lahay, M. R. F. Payu, S. L. Mahmud, H. S. Panigoro, and P. Zakaria, "Dynamics of a predator-prey model incorporating infectious disease and quarantine on prey," *Jambura Journal of Biomathematics (JJBM)*, vol. 3, no. 2, pp. 75–81, 2022. DOI:10.34312/jjbm.v3i2.17162

[13] R. Ibrahim, L. Yahya, E. Rahmi, and R. Resmawan, "Analisis dinamik model predator-prey tipe Gause dengan wabah penyakit pada prey," *Jambura Journal of Biomathematics (JJBM)*, vol. 2, no. 1, pp. 20–28, 2021. DOI:10.34312/jjbm.v2i1.10363

[14] J. Chattopadhyay and O. Arino, "A predator-prey model with disease in the prey," *Nonlinear Analysis: Theory, Methods & Applications*, vol. 36, no. 6, pp. 747–766, 1999. DOI:10.1016/S0362-546X(98)00126-6

[15] S. Biswas, S. Samanta, and J. Chattopadhyay, "A cannibalistic eco-epidemiological model with disease in predator population," *Journal of Applied Mathematics and Computing*, vol. 57, no. 2018, pp. 161–197, 2017. DOI:0.1007/s12190-017-1100-9

[16] S. Maisaroh, R. Resmawan, and E. Rahmi, "Analisis kestabilan model predator-prey dengan infeksi penyakit pada prey dan pemanenan proporsional pada predator," *Jambura Journal of Biomathematics (JJBM)*, vol. 1, no. 1, pp. 8–15, 2020. DOI:10.34312/jjbm.v1i1.5948

[17] Y. Kang, M. Rodriguez-Rodriguez, and S. Evilsizor, "Ecological and evolutionary dynamics of two-stage models of social insects with egg cannibalism," *Journal of Mathematical Analysis and Applications*, vol. 430, no. 1, pp. 324–353, 2015. DOI:10.1016/j.jmaa.2015.04.079

[18] H. Deng, F. Chen, Z. Zhu, and Z. Li, "Dynamic behaviors of Lotka–Volterra predator–prey model incorporating predator cannibalism," *Advances in Difference Equations*, vol. 2019, no. 2019, pp. 1–17, 2019. DOI:10.1186/s13662-019-2289-8

[19] S. Biswas, S. Samanta, and J. Chattopadhyay, "A model based theoretical study on cannibalistic prey–predator system with disease in both populations," *Differential Equations and Dynamical Systems*, vol. 23, no. 2015, pp. 327–370, 2014. DOI:10.1007/s12591-014-0211-0

[20] M. Rayungsari, A. Suryanto, W. M. Kusumawinahyu, and I. Darti, "Dynamics analysis of a predator–prey fractional-order model incorporating predator cannibalism and refuge," *Frontiers in Applied Mathematics and Statistics*, vol. 9, pp. 1–12, 2023. DOI:10.3389/fams.2023.1122330

[21] F. Zhang, Y. Chen, and J. Li, "Dynamical analysis of a stage-structured predator-prey model with cannibalism," *Mathematical Biosciences*, vol. 307, pp. 33–41, 2019. DOI:10.1016/j.mbs.2018.11.004