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DYNAMIC ANALYSIS OF PREDATOR-PREY MODEL WITH CANNIBALISM INTERVENTION AND DISEASE INFECTION IN PREY USING HOLLING TYPE II RESPONSE FUNCTION

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ABSTRACT

This study discusses the intervention of cannibalism and disease spread with Holling Type II response function in the predator-prey model. It is assumed that disease infection is limited to the prey population and cannot be cured so that in this model there are three subpopulations namely susceptible prey, infected prey and predators. In addition, there is cannibalism in the predator population. The objectives of this study include constructing a predator-prey model with cannibalism intervention and disease infection in prey using Holling Type II response function, identifying the stability of the equilibrium point of the model and interpreting the model based on simulation results. Analysis of the stability of the equilibrium point is carried out with a linearization approach and the Routh-Hurwitz criterion was used to determine equilibrium stability. Based on the stability analysis, 5 (five) equilibrium points are obtained, namely population extinction, susceptible prey exists, predator extinction, infected prey extinction and population exists where the population extinction equilibrium point is unstable and the other equilibrium points are stable with the certain conditions. From the simulation, it is obtained that the numerical results are in accordance with the analytical results of the stability analysis of the equilibrium point of the model and for infinite time, there will be no population extinction while the state of susceptible prey exists, predator extinction, infected prey extinction and population exists can occur if the stability conditions are met. Based on the numerical simulations, it was found that changes in the parameter values of the rate of change of susceptible prey to infected prey and the coefficient of predator cannibalism in day^{-1} can cause changes in the type of stability of the equilibrium point. Thus, rate of change susceptible prey to infected prey and the coefficient of predator cannibalism affects the population of prey and predator.

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1. INTRODUCTION

Every organism must interact with each other which can have an impact on these organisms in the form of positive or negative impacts on one species or positive or negative on both [1]. One form of interaction between species is predation. Predation is an interaction between prey and predator where the predator preys for its survival and the predator acts as a regulator of the prey population [2]. Alfred Lotka and Vito Volterra first introduced a mathematical model in 1926 to describe the relationship between prey and predator which eventually became known as the Lotka-Volterra model. This model continues to evolve, one of which is in describing predator-prey interactions through a concept called the response function which describes the amount of prey consumed by predators as a function of prey population density [3]. This function was introduced by Holling in 1953 which consists of Holling type I, Holling type II and Holling type III response functions [4]. Some studies that use Holling response function are [5]-[10].

In the Lotka-Voltera model, it is assumed that there is no disease infection in the environment of prey and predator populations. While in reality what happens in the environment shows that prey and predator populations can be infected with a disease [8]. Therefore, attention is needed to the spread of disease in prey and predators because it can contribute to the decline in the number of species to extinction. One of the efforts made is by using a mathematical prey-predator model which involves the spread of disease. The research was first conducted by [11] or known as the eco-epidemiology problem. Research on this eco-epidemiology problem continues to grow, including by [12] examining the analysis of predator-prey systems involving the existence of a proportional harvesting factor in predators and assuming disease spread in prey. In the study, it is assumed that the disease is incurable and the predation function uses Holling type I. There are five equilibrium points obtained by using Holling type I. Five equilibrium points were obtained with the population extinction equilibrium point never reached. The eco-epidemiology model research was then developed in [13] by adding the assumption that the disease in the prey population can be cured and obtained the result that the population extinction equilibrium point is unstable from the five equilibrium points that exist. Furthermore, [14] discusses the predator-prey interaction model response function using Cowley Martin with the assumption that the disease only spreads in the prey population while the research [4] discusses the predator-prey interaction model with the assumption that the disease only infects the prey population and only infected prey are preyed upon by predators by involving harvesting factors on prey. From these studies, the conditions are determined so that prey and predator populations can exist.

In addition to disease infection, cannibalism is also a biological phenomenon that can affect the existence of prey and predators. Cannibalism can help regulate the number of predators, thus reducing the risk of extinction of prey, but cannibalism can also be a factor in predator extinction. Therefore, research is needed on the predator-prey model that involves the cannibalism factor. Research [15] examined the dynamic behavior of the Lotka-Volterra predator-prey model involving predator cannibalism and found that cannibalism has positive and negative effects on system stability, depending on the dynamic behavior of the original system. Research [16] extended the model by adding stage structure and cannibalism in predators. The predator population was divided into juvenile and adult predators where it was assumed that adult predators hunted prey and juvenile predators at a rate represented by Holling type I functional response. Furthermore, [17] combines the models [18] and [15] by taking the example of shrimp (prey) and crab (predator) interaction models. In the study, it was assumed that predators attacked susceptible and infected prey with predation functions using Holling type I responses and prey-prey interactions following cannibalism behavior in predators.

In contrast to previous studies, researchers formulated a mathematical model by modifying the model in research [17], namely replacing the predation function applied to the Holling type II response function but using the cannibalism function in research [16]. The assumption of Holling type II response function is based on the fact that crabs need time to prey on shrimp. The purpose of this study is to construct a predator-prey model due to the intervention of cannibalism and disease infection using the Holling type II response function, determine the equilibrium point of the model and identify the type of stability and interpret the model through numerical simulations.

2. RESEARCH METHODS

2.1 The Lotka-Volterra Model

This model was first introduced by Lotka (1925) and Volterra (1926) so it is often referred to as the Lotka-Volterra Model. The Lotka-Volterra model is the simplest Predator-Prey model that describes the interaction between prey and predator populations. It is assumed that the growth of predators depends on prey as the main source of food so that without predators, prey will grow rapidly in proportion to the current population size while without prey the predator population will become extinct. The interaction between prey and predator will contribute to the growth of predators and reduce the number of preys.

If x denotes the prey population at time t and y denotes the predator population at time t, then the Lotka-Volterra model can be written

$$\begin{aligned} \frac{dx}{dt} &= rx - \beta xy \\ \frac{dy}{dt} &= \beta xy - \varphi y \end{aligned} \tag{1}$$

with $r, \beta,$ and $\varphi > 0$. rx shows that prey grows exponentially in the absence of predation, $-\beta xy$ shows the effect of predation, namely to reduce the per capita growth rate of prey over a period of time proportional to the population of prey and predator, $-\varphi y$ shows the death rate of predators due to the absence of prey, resulting in exponential decay and βxy shows the contribution of prey to the growth rate of predators [3].

2.2 Holling Type II Response Function

The response function in ecology is expressed as a function of the predator's consumption rate against different prey densities. This function was introduced by Holling who categorized the response function into three types, one of which is the Holling type II response function. The response function illustrates that the consumption rate of predator increases as the prey population increases but will decrease when the predator approaches satiety. This is because when the prey population is small, some of the time of predator is spent searching for prey, while when the prey population is large, the predator spends the available time holding and digesting prey rather than searching for it, resulting in a lower consumption rate. This causes the consumption rate to reach half saturation. This type describes a predator that is actively moving to search for prey [19].

The type II response function is represented as follows.

$$F^{(II)}(x) = \frac{ax}{1+bx} \tag{2}$$

with a is predation rate on prey, b is handling time of prey by predators, and x is the prey population [20].

3. RESULTS AND DISCUSSION

3.1 Predator-Prey Model with Cannibalism Intervention and Disease Infection in Prey Using Holling type II Response Function

The assumptions that apply in the model include:

1. The disease spreads in the prey population and cannot be cured so that the prey population is divided into two sub-populations, namely susceptible prey and infected prey.
2. The prey population grows logistically.
3. Only susceptible prey can reproduce.
4. Predators perform predation following a Holling type II response function.
5. Diseases in prey cannot spread in predator populations.
6. Predators have a cannibalistic nature.

The compartment diagram of the predator-prey interaction corresponding to the applicable assumptions is given as in Figure 1 below:

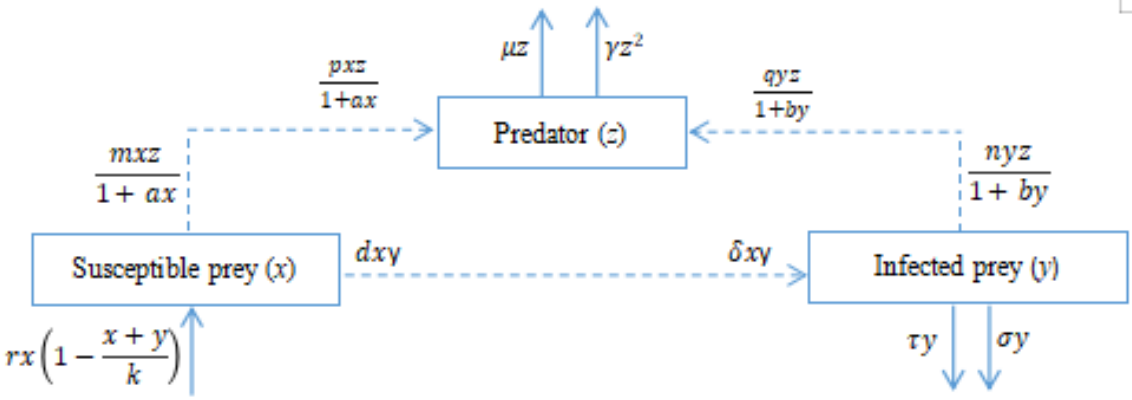


Figure 1. Diagram of the Compartment Model

The list of variables and parameters in the model is shown in Table 1 below:

Table 1. Variables and Parameters in the Model

Symbol	Description	Type	Unit
x	susceptible prey population	Variable	tail
y	infected prey population	Variable	tail
z	predator population	Variable	tail
r	intrinsic growth rate of prey	Parameter	day^{-1}
k	prey carrying capacity	Parameter	tail
d	rate of interaction between susceptible prey and infected prey	Parameter	$tail^{-1}day^{-1}$
δ	rate of change susceptible prey to infected prey	Parameter	$tail^{-1}day^{-1}$
m	predation rate on susceptible prey	Parameter	$tail^{-1}day^{-1}$
n	predation rate on infected prey	Parameter	$tail^{-1}day^{-1}$
σ	death rate due to disease	Parameter	day^{-1}
τ	natural mortality coefficient of infected prey	Parameter	day^{-1}
μ	predator natural mortality coefficient	Parameter	day^{-1}
a	handling time of susceptible prey by predators	Parameter	$tail^{-1}$
b	handling time of infected prey by predators	Parameter	$tail^{-1}$
p	prey conversion rate of susceptible prey	Parameter	$tail^{-1}day^{-1}$
q	prey conversion rate of infected prey	Parameter	$tail^{-1}day^{-1}$
γ	predator cannibalism coefficient	Parameter	$tail^{-1}day^{-1}$

Based on the compartment drawing, the predator-prey interaction model with the intervention of cannibalism and disease infection in prey using Holling type II response function is presented in the following system:

$$\begin{aligned}\frac{dx}{dt} &= rx\left(1-\frac{x+y}{k}\right)-\frac{mxz}{1+ax}-dxy\\ \frac{dy}{dt} &= \delta xy-\frac{nyz}{1+by}-\sigma y-\tau y\\ \frac{dz}{dt} &= \frac{pxz}{1+ax}+\frac{qyz}{1+by}-\mu z-\gamma z^2\end{aligned} \tag{3}$$

with $r, k, a, b, m, n, \delta, \gamma, \mu, d, e, \alpha, \tau, \gamma$ dan $\beta \in \mathbb{R}^+$.

3.2 Equilibrium Point of Model

Given the set $\mathbb{R}^3_+ = \{(x,y,z)|x \geq 0, y \geq 0, z \geq 0, x, y, z \in \mathbb{R}\}$. The equilibrium point of Equation (3) is the solution of Equation (3) that satisfies $\frac{dx}{dt}=0, \frac{dy}{dt}=0$, and $\frac{dz}{dt}=0$. Thus the following system of equations is obtained:

$$rx\left(1-\frac{x+y}{k}\right)-\frac{mxz}{1+ax}-dxy=0 \tag{4}$$

$$\delta xy-\frac{nyz}{1+by}-\sigma y-\tau y=0 \tag{5}$$

$$\frac{pxz}{1+ax}+\frac{qyz}{1+by}-\mu z-\gamma z^2=0 \tag{6}$$

Thus, the equilibrium point of **Equation (3)** is obtained, namely:

1. Equilibrium point of population extinction $E_0(0,0,0)$.
2. Susceptible prey equilibrium point exists $E_1(k,0,0)$.
3. Predator extinction equilibrium point $E_2\left(\frac{\sigma+\tau}{\delta},\frac{r(\delta k-(\sigma+\tau))}{\delta(r+dk)},0\right)$ with conditions $\frac{\delta k}{\sigma+\mu}>1$.
4. Prey extinction equilibrium point of infected $E_3(\hat{x},0,\hat{z})$ with $\hat{x}$ is a positive solution of
$$a^2r\gamma x^3+\gamma ra(2-ak)x^2+(\gamma r-2ak+pmk-\mu ma)x-k(\gamma r+\mu m)=0$$
and
$$\hat{z}=\frac{r\left(1-\frac{\hat{x}}{k}\right)(1+a\hat{x})}{m}$$
under the condition that $\frac{\hat{x}}{k}<1$.
5. The population equilibrium point exists $E_4(x^*,y^*,z^*)$ where x^* is the positive root of
$$Kx^6+Lx^5+Mx^4+Nx^3+Ox^2+Px+Q=0,$$
$$y^*=\frac{narx^{*2}+(mk\delta+rn-aknr)x^*-\big(rkn+mk(\sigma+\tau)\big)}{-(nar+nkda+mkb\delta)x^*+(mkb(\sigma+\tau)-rn-dkn)}$$
and
$$z^*=\frac{\big(\delta x^*-(\sigma+\tau)\big)(1+by^*)}{n}$$
with:

$A=(nar)^2$

$B=-nar(aknr-rn-mk\delta)$

$C=(aknr-rn-mk\delta)^2-2nar\big(rkn+mk(\sigma+\tau)\big)$

$D=2\big(rkn+mk(\sigma+\tau)\big)(aknr-rn-mk\delta)$

$E=\big(rkn+mk(\sigma+\tau)\big)^2$

$F=(nar+nkda+mkb\delta)^2$

$G=2(nar+nkda+mkb\delta)\big(rn+dkn-mkb(\sigma+\tau)\big)$

$H=\big(rn+dkn-mkb(\sigma+\tau)\big)^2$

$K=ab^2\delta\gamma A$

$L=b^2\delta\gamma(aB+aA-A)$

$M=b^2\delta\gamma B+b^2(\sigma+\tau)\gamma A-ab^2\delta\gamma C-ab^2\delta\gamma B-a\delta\gamma F-2a^2n\gamma\delta br$

$N=b^2\delta\gamma C+b^2(\sigma+\tau)\gamma B+ab^2\delta\gamma D-ab^2\delta\gamma C-(np-a\mu n+a\gamma\sigma-\gamma\delta)F+a\delta\gamma G$
$+(pbn+aqn-ab\mu n)nar+2a\gamma\delta b(aknr-rn-mk\delta)+(a\gamma rb-\gamma rb)2nar$

$O=(pbn+aqn-ab\mu n)(aknr-rn-mk\delta)-(qn-b\mu n+2\gamma rb)nar-\mu n^2ar$
$-2a\gamma\delta b\big(rkn+mk(\sigma+\tau)\big)+2\gamma(\sigma+\tau)b(a-1)(aknr-rn-mk\delta)-b\gamma\delta D$

$$+b^2(\sigma+\tau)\gamma C - ab^2\delta\gamma E + ab^2\delta\gamma D + (np - arn + a\gamma + b^2(\sigma+\tau)\gamma C - \gamma\delta)G - a\delta\gamma H$$
$$+(\gamma + b^2(\sigma+\tau)\gamma C - \mu n)F$$
$$P = (pbn + aqn - ab\mu n)(krn + mk(\sigma+\tau)) + (qn - b\mu n + 2\gamma rb)(aknr - rn - mk\delta)$$
$$-\mu n(aknr - rn - mk\delta) + 2\gamma(\sigma+\tau)b(a-1)(krn + mk(\sigma+\tau)) - b^2\delta\gamma E + b^2\sigma\gamma D$$
$$+ab^2\delta\gamma E + (np - a\mu n + a\gamma(\sigma+\tau) - \gamma\delta)H + (\gamma(\sigma+\tau) - \mu n)G$$
$$Q = b^2(\sigma+\tau)\gamma E + (\gamma(\sigma+\tau) - \mu n)H + (qn - b\mu n + 2\gamma rb)(knr + mkr)$$
$$+\mu n\big(rkn + mk(\sigma+\tau)\big) + (\gamma(\sigma+\tau) - \mu n)H$$

The equilibrium point E_4 exists under the conditions $narx^{*2} + (mk\delta + rn - aknr)x^* - \big(rkn + mk(\sigma+\tau)\big) > 0$ and $\frac{(\sigma+\tau)}{\delta} < x^* < \frac{(mkb(\sigma+\tau) - rn - dkn)}{(nar + nkda + mkb\delta)}$

3.3 Stability Analysis

To determine the type of stability of the equilibrium point, it is necessary to linearize the **Equation (3)** by determining the Jacobian matrix,

$$J = \begin{bmatrix} \frac{\partial\left(\frac{dx}{dt}\right)}{\partial x} & \frac{\partial\left(\frac{dx}{dt}\right)}{\partial y} & \frac{\partial\left(\frac{dx}{dt}\right)}{\partial z} \\ \frac{\partial\left(\frac{dy}{dt}\right)}{\partial x} & \frac{\partial\left(\frac{dy}{dt}\right)}{\partial y} & \frac{\partial\left(\frac{dy}{dt}\right)}{\partial z} \\ \frac{\partial\left(\frac{dz}{dt}\right)}{\partial x} & \frac{\partial\left(\frac{dz}{dt}\right)}{\partial y} & \frac{\partial\left(\frac{dz}{dt}\right)}{\partial z} \end{bmatrix}. \tag{7}$$

Thus the Jacobian matrix of **Equation (3)** is obtained as follows:

$$\mathrm{J} = \begin{bmatrix} \mathrm{J}_{11} & \mathrm{J}_{12} & \mathrm{J}_{13} \\ \mathrm{J}_{21} & \mathrm{J}_{22} & \mathrm{J}_{23} \\ \mathrm{J}_{31} & \mathrm{J}_{32} & \mathrm{J}_{33} \end{bmatrix}. \tag{8}$$

with:

$$\mathrm{J}_{11} = r\left(1 - \frac{2x+y}{k}\right) - \frac{mz}{(1+ax)^2} - dy$$
$$\mathrm{J}_{12} = -\left(\frac{r}{k} + d\right)x$$
$$\mathrm{J}_{13} = -\frac{mx}{1+ax}$$
$$\mathrm{J}_{21} = \delta y$$
$$\mathrm{J}_{22} = \delta x - \frac{nz}{(1+by)^2} - (\sigma+\tau)$$
$$\mathrm{J}_{23} = -\frac{ny}{1+by}$$
$$\mathrm{J}_{31} = \frac{pz}{(1+ax)^2}$$
$$\mathrm{J}_{32} = \frac{qz}{(1+by)^2}$$
$$\mathrm{J}_{33} = \frac{px}{1+ax} + \frac{qy}{1+by} - 2\gamma z - \mu$$

Theorem 1. *The equilibrium point* $E_0(0,0,0)$ *is unstable.*

Proof. Substituting the equilibrium point $\mathrm{E}_0(0,0,0)$ into **Equation (8)** is obtained:

$$\mathrm{J}_{\mathrm{E}_0} = \begin{bmatrix} r & 0 & 0 \\ 0 & -(\sigma+\tau) & 0 \\ 0 & 0 & -\mu \end{bmatrix}. \tag{9}$$

By using the equation $|\lambda I - \mathrm{J}_{\mathrm{E}_0}| = 0$, from **Equation (9)** is obtained the eigenvalues $\lambda_1 = r, \lambda_2 = -(\sigma+\tau)$ and $\lambda_3 = -\mu$. Because there is an eigenvalue $\lambda_1 = r > 0$ as a result the equilibrium point E_0 is unstable.

Theorem 2. *The equilibrium point* $E_1(k,0,0)$ *is asymptotically stable under the conditions* $\frac{\delta k}{\sigma} < 1$ and $\frac{pk}{\mu(1+ak)} < 1$.

Proof. Substitution of the equilibrium point $E_1(k,0,0)$ into **Equation (8)** is obtained:

$$J_{E_1} = \begin{bmatrix} -r & -(r+dk) & \frac{-mk}{1+ak} \\ 0 & \delta k-(\sigma+\tau) & 0 \\ 0 & 0 & \frac{pk}{1+ak}-\mu \end{bmatrix}. \quad (10)$$

By using the equation $|\lambda I - J_{E_1}| = 0$, from **Equation (10)** is obtained the eigenvalues $\lambda_1 = -r, \lambda_2 = \delta k - (\sigma+\tau)$ and $\lambda_3 = \frac{\alpha k}{1+ak} - \varphi$. Thus, the equilibrium point E_1 is asymptotically stable under the conditions $\frac{\delta k}{\sigma} < 1$ and $\frac{pk}{\mu(1+ak)} < 1$.

Theorem 3. *The equilibrium point* $E_2(\hat{x},\hat{y},0)$ *is asymptotically stable under the condition that* $a_{33} < 0, a_{11} < -a_{22},$ and $a_{11}a_{22} < a_{12}a_{21}$.

Proof. Substituting the equilibrium points $E_2(\hat{x},\hat{y},0)$ with $\hat{x} = \frac{\sigma+\tau}{\delta}$ and $\hat{y} = \frac{r(\delta k-(\sigma+\tau))}{\delta(r+dk)}$ into **Equation (8)** is obtained:

$$J_{E_2} = \begin{bmatrix} r\left(1-\frac{2\hat{x}+\hat{y}}{k}\right)-d\hat{y} & -\left(\frac{r}{k}+d\right)\hat{x} & -\frac{m\hat{x}}{1+a\hat{x}} \\ \delta\hat{y} & \delta\hat{x}-(\sigma+\tau) & -\frac{n\hat{y}}{1+b\hat{y}} \\ 0 & 0 & \frac{p\hat{x}}{1+a\hat{x}}+\frac{q\hat{y}}{1+b\hat{y}}-\mu \end{bmatrix}. \quad (11)$$

By using the equation $|\lambda I - J_{E_2}| = 0$ is obtained characteristic equation $(\lambda - a_{33})[\lambda^2 - (a_{11}+a_{22})\lambda + (a_{11}a_{22} - a_{12}a_{21})] = 0$ with $a_{11} = r\left(1-\frac{2\hat{x}+\hat{y}}{k}\right) - d\hat{y}, a_{12} = -\left(\frac{r}{k}+d\right)\hat{x}, a_{21} = \delta\hat{y} a_{22} = \delta\hat{x} - (\sigma+\tau)$, and $a_{33} = \frac{p\hat{x}}{1+a\hat{x}} + \frac{q\hat{y}}{1+b\hat{y}} - \mu$. Thus obtained $\lambda = a_{33}$ or $\lambda^2 - (a_{11}+a_{22})\lambda + (a_{11}a_{22} - a_{12}a_{21}) = 0$. By using the concept of the Routh-Hurwitz criterion, the equilibrium point E_2 is asymptotically stable under the condition that $a_{33} < 0, a_{11} < -a_{22},$ and $a_{11}a_{22} < a_{12}a_{21}$.

Theorem 4. *The equilibrium point* $E_3(\tilde{x},0,\tilde{z})$ *is asymptotically stable under the conditions* $h_{22} < 0, h_{11} < -h_{33},$ and $h_{11}h_{33} > h_{13}h_{31}$.

Proof. Substitution of the equilibrium point $E_3(\tilde{x},0,\tilde{z})$ into **Equation (8)** is obtained:

$$J_{E_3} = \begin{bmatrix} r\left(1-\frac{2\tilde{x}}{k}\right)-\frac{m\tilde{z}}{(1+a\tilde{x})^2} & -\left(\frac{r}{k}+d\right)\tilde{x} & -\frac{m\tilde{x}}{1+a\tilde{x}} \\ 0 & \delta\tilde{x}-n\tilde{z}-(\sigma+\tau) & 0 \\ \frac{p\tilde{z}}{(1+a\tilde{x})^2} & q\tilde{z} & \frac{p\tilde{x}}{1+a\tilde{x}}-2\gamma\tilde{z}-\mu \end{bmatrix}. \quad (12)$$

Using the equation $|\lambda I - J_{E_3}| = 0$, the characteristic equation $(\lambda - h_{22})[\lambda^2 - (h_{11}+h_{33})\lambda + (h_{11}h_{33} - h_{13}h_{31})] = 0$ is obtained with $h_{11} = r\left(1-\frac{2\tilde{x}}{k}\right) - \frac{m\tilde{z}}{(1+a\tilde{x})^2}, h_{13} = -\frac{m\tilde{x}}{1+a\tilde{x}}, h_{22} = \delta\tilde{x} - n\tilde{z} - (\sigma+\tau), h_{31} = \frac{p\tilde{z}}{(1+a\tilde{x})^2}$ and $h_{33} = \frac{p\tilde{x}}{1+a\tilde{x}} - 2\gamma\tilde{z} - \mu$. So that $\lambda = h_{22}$ or $\lambda^2 - (h_{11}+h_{33})\lambda + (h_{11}h_{33} - h_{13}h_{31}) = 0$. Using the concept of Routh-Hurwitz, the equilibrium point E_3 is asymptotically stable under the conditions $h_{22} < 0, h_{11} < -h_{33},$ and $h_{11}h_{33} > h_{13}h_{31}$.

Theorem 5. *The equilibrium point* $E_4(x^*,y^*,z^*)$ *is asymptotically stable under the conditions* $U < 0, W > 0$ and $\frac{W-UV}{U} > 0$

Proof. Substitution of the equilibrium point $E_4(x^*,y^*,z^*)$ into **Equation (8)** is obtained:

$$J_{E_4} = \begin{bmatrix} r\left(1-\frac{2x^*+y^*}{k}\right)-\frac{mz^*}{(1+ax^*)^2}-dy^* & -\left(\frac{r}{k}+d\right)x^* & -\frac{mx^*}{1+ax^*} \\ \delta y^* & \delta x^*-\frac{nz^*}{(1+by^*)^2}-(\sigma+\tau) & -\frac{ny^*}{1+by^*} \\ \frac{pz^*}{(1+ax^*)^2} & \frac{qz^*}{(1+by^*)^2} & \frac{px^*}{1+ax^*}+\frac{qy^*}{1+by^*}-2\gamma z^*-\mu \end{bmatrix}. \quad (13)$$

By using the equation $|\lambda I - J_{E_3}| = 0$, the characteristic equation $\lambda^3 + U\lambda^2 + V\lambda + W = 0$ is obtained. By using the concept of the Routh-Hurwitz criterion, the equilibrium point E_5 is asymptotically stable under the conditions $U < 0, W > 0$ and $\frac{W-UV}{U} > 0$ with:

$$\begin{aligned} U &= -(j_{11}+j_{12}+j_{13}) \\ V &= j_{11}j_{22}+j_{11}j_{33}+j_{22}j_{33}+j_{13}j_{31}-j_{23}j_{32}-j_{12}j_{21}+j_{12}j_{21}j_{33} \\ W &= j_{11}j_{22}j_{33}+j_{12}j_{23}j_{31}+j_{13}j_{21}j_{32}+j_{11}j_{23}j_{32}-j_{13}j_{22}j_{31} \\ j_{11} &= r\left(1-\frac{2x^*+y^*}{k}\right)-\frac{mz^*}{(1+ax^*)^2}-dy^*, \\ j_{12} &= -\left(\frac{r}{k}+d\right)x^*, \\ j_{13} &= -\frac{mx^*}{1+ax^*}, \\ j_{21} &= \delta y^*, \\ j_{22} &= \delta x^*-\frac{nz^*}{(1+ey^*)^2}-(\sigma+\tau), \\ j_{23} &= -\frac{ny^*}{1+by^*}, \\ j_{31} &= \frac{pz^*}{(1+ax^*)^2}, \\ j_{32} &= \frac{qz^*}{(1+by^*)^2}, \text{and} \\ j_{33} &= \frac{px^*}{1+ax^*}+\frac{qy^*}{1+by^*}-2\gamma z^*-\mu. \end{aligned}$$

3.4 Simulation and Interpretation of the Model

In this section, the solution graph is given as an illustration of the analysis of the stability of the equilibrium point that has been carried out previously by analytical means. The parameter values in this simulation are taken around the parameters of several studies of the predator-prey model, that is [13], [17], [21], and [22] as shown in Table 2 below:

Table 2. Parameter Values of Simulation Model

Parameters	Simulation E_1	Simulation E_2	Simulation E_3	Simulation E_4
r	0.2	0.2	0.2	0.2
k	10	10	50	50
δ	0.005	0.05	0.001	0.1
d	0.2	0.2	0.2	0.2
m	0.1	0.3	0.3	0.3
n	0.1	0.2	0.2	0.2
σ	0.4	0.2	0.2	0.05
τ	0.2	0.2	0.2	0.2
μ	0.2	0.2	0.2	0.2
a	0.02	0.2	0.2	0.2
b	0.02	0.1	0.1	0.1
p	0.001	0.02	0.2	0.2
q	1	0.02	0.2	0.2
γ	0.01	0.4	0.4	0.2

If the parameter values in Table 2 are substituted into Equation (3), Figure 2 is obtained as follows:

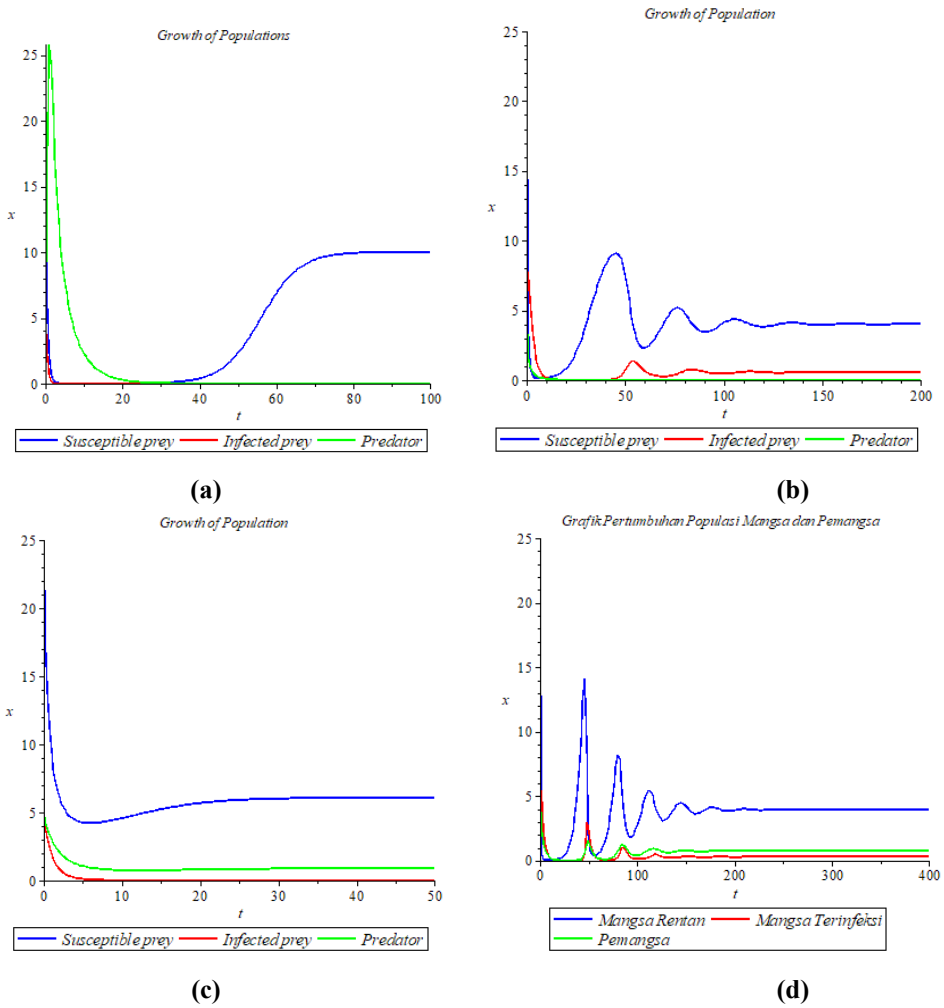
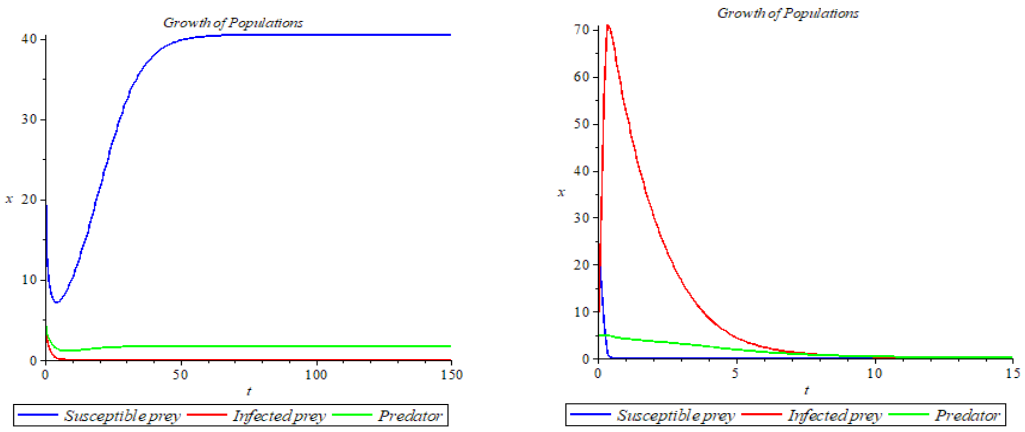


Figure 2. Growth of Prey and Predator Populations (a) Equilibrium Point E_1, (b) Equilibrium Point E_2, (c) Equilibrium Point E_3, (d) Equilibrium Point E_4

Based on the parameter values in **Table 2** for the simulation E_1, the equilibrium point E_1 exists, namely $(100,0,0)$ and is asymptotically stable because the stability conditions E_1, namely $\frac{\delta k}{\sigma} < 1$ and $\frac{pk}{\varphi(1+ak)} < 1$ are met. The simulation results E_1 as shown in **Figure 2** (a) show that the susceptible prey population exists while the infected prey and predator populations are heading towards extinction. Based on the parameter values in **Table 2** for simulation E_2,the equilibrium point E_2 exists, namely$(8,0.91,0)$ and is asymptotically stable because the stability conditions E_2,namely$a_{33} < 0, a_{11} < -a_{22},$ and$a_{11}a_{22} < a_{12}a_{21}$ are met. The simulation results E_2 as shown in **Figure 2** (b) show that the susceptible prey and infected prey populations exist while the predator population is heading towards extinction. Based on the parameter values in **Table 2** for simulation E_3, the equilibrium point E_3 exists, namely$(90.18,0,1.87)$ and is asymptotically stable because the stability conditions E_3 are met, namely$h_{22} < 0, h_{11} < -h_{33}$, and $h_{11}h_{33} > h_{13}h_{31}$.The simulation results E_3 as shown in **Figure 2** (c) show that the population of susceptible prey and predators exist while the population of infected prey is heading towards extinction. Based on the parameter values in **Table 2** for simulationE_4,the equilibrium pointE_4 exists, namely$(3.98,0.32,0.76)$and is asymptotically stable because the stability conditions$U < 0, W > 0$ and $\frac{W-UV}{U} > 0$ are met. The simulation results E_4 as shown in **Figure 2** (d) show that the population of susceptible prey, infected prey and predators exist.

Next, a numerical simulation is given which shows that there is a change in the rate of change susceptible prey to infected prey. This simulation was carried out in two cases, namely when the rate of change susceptible prey to infected prey decreased to$\delta = 0.01$ and when the rate of change susceptible prey to infected prey increased to $\delta = 0.65$, previously based on **Table 2** (Simulation E_4) the value was $\delta = 0.2$. The dynamics of population growth based on the case of changes in the value of the rate of change susceptible prey to infected prey is as shown in **Figure 3** below:



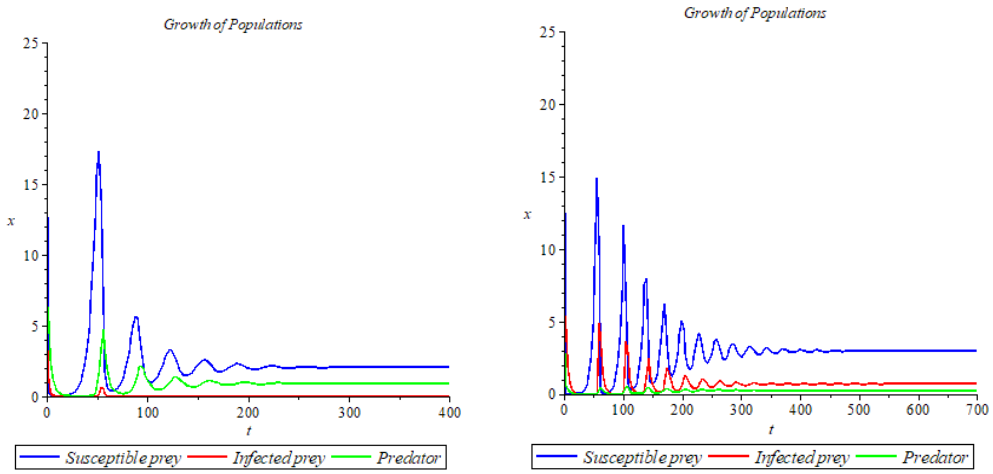
(a) **(b)**

Figure 3. Growth of Prey and Predator Populations (a) $\delta = 0.01$, (b) $\delta = 0.65$

Based on **Figure 3** (a), when the rate of change susceptible prey to infected prey decreases to $\delta = 0.01$, it can be seen that over time the population of infected prey experiences extinction while the populations of susceptible prey and predators continue to exist. Thus, it can be concluded that there is a change in the stability of the equilibrium point of the existing population, namely E_4, which was initially stable (**Figure 2** (d)) and becomes unstable when the value of the rate of change susceptible prey to infected prey decreases to $\delta = 0.01$.

Based on **Figure 3** (b), when the rate of change susceptible prey to infected prey increases to $\delta = 0.65$, it can be seen that over time all populations are heading towards extinction. Thus, it can be concluded that there is a change in the stability of the equilibrium point of the existing population, namely E_4, which was initially stable (**Figure 2** (d)) and becomes unstable when the value of rate of change susceptible prey to infected prey increases to $\delta = 0.65$.

Furthermore, numerical simulations were carried out which showed that there was a change in the predator cannibalism coefficient. This simulation was carried out in two cases, namely when the predator cannibalism coefficient decreased to $\gamma = 0.1$ and when the predator cannibalism coefficient increased to $\gamma = 1.2$, previously based on **Table 2** (Simulation E_4) the value was $\gamma = 0.2$. Population growth dynamics based on cases of changes in predator cannibalism coefficient values are as shown in **Figure 4** below:



(a) **(b)**

Figure 4. Growth of Prey and Predator Populations (a) $\gamma = 0.1$, (b) $\gamma = 1.2$

Based on **Figure 4** (a), when the predator cannibalism coefficient decreases to $\gamma = 0.1$, it can be seen that over time the infected prey population experiences extinction while the vulnerable prey and predator populations continue to exist. Thus, it can be concluded that there is a change in the stability of the equilibrium point of the existing population, namely E_4, which was initially stable (**Figure 2** (d)) and becomes unstable when the predator cannibalism coefficient decreases to $\gamma = 0.1$.

Based on **Figure 4** (b), when the predator cannibalism coefficient increases to $\gamma = 1.2$, it can be seen that over time all populations continue to exist. Thus, it can be concluded that there is no change in the stability of the equilibrium point of the existing population, namely E_4, when the predator cannibalism coefficient increases to $\gamma = 1.2$.

4. CONCLUSION

The predator-prey model with cannibalism intervention and disease infection in prey using Holling type II response function is in the form of a system of non-linear differential equations as in **Equation (3)**. The equilibrium points generated by the model are the equilibrium points of population extinction, susceptible prey exists, infected prey extinction, predator extinction and population exist. Based on the identification of the stability of the equilibrium point, it is obtained that the equilibrium point of population extinction is unstable, which means that over time, there will be no population extinction while the susceptible prey exists, the extinction of infected prey, the extinction of predators and the population exists are asymptotically stable with conditions, which means that these conditions can occur over time if the conditions are met. Based on the numerical simulations, it was found that changes in the parameter values of the rate of change of susceptible prey to infected prey and the coefficient of predator cannibalism can cause changes in the type of stability of the equilibrium point. Thus, rate of change susceptible prey to infected prey and the coefficient of predator cannibalism affects the population of prey and predator.

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