JTAM (Jurnal Teori dan Aplikasi Matematika)
http://journal.ummat.ac.id/index.php/jtam
p-ISSN 2597-7512 | e-ISSN 2614-1175
Vol. 8, No. 2, April 2024, pp. 364-377

Dynamical of Prey Refuge In a Diased Predator-Prey Model with Intraspecific Competition for Predator

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Article History:
Received : 29-10-2023
Revised : 15-03-2024
Accepted : 21-03-2024
Online : 01-04-2024

Keywords:
Prey refuge;
Intraspecific competition;
Predator-prey.

ABSTRACT

The predator-prey model described is a population growth model of an eco-epidemiological system with prey protection and predator intraspecific traits. Predation interactions in predator species use response functions. The aim of this research is to examine the local stable balance point and look at the characteristics of species resulting from mathematical modeling interventions. Review of balance point analysis, numerical simulation and analysis of given trajectories. The research results show the shape of the model which is arranged with a composition of 5 balance points. There is one rational balance point to be explained, using the Routh-Hurwitz criterion, $E(x^*, y^*, z^*)$. The characteristic equation and associated eigenvalues in the mathematical model are the local asymptotically stable balance points. In the trajectory analysis, local stability is also shown by the model formed. There are differences for each population to reach its point of stability. The role of prey protection behavior is very effective in suppressing the spread of disease. Meanwhile, intraspecific predator interactions are able to balance the decreasing growth of prey populations. If we increase the intraspecific interaction coefficient, we can be sure that the growth of the prey population will both increase significantly. When the number of prey populations increases significantly, of course disease transmission and prey protection become determining factors, the continuation of the model in exosite interactions. In prey populations and susceptible prey to infection, growth does not require a long time compared to the growth of predator populations. The time required to achieve stable growth is rapid for the prey species. Although prey species' growth is more fluctuating compared to predator populations. Predatory species are more likely to be stable from the start of their growth. The significance of predatory growth is only at the beginning of growth, while after that it increases slowly and reaches an ideal equilibrium point. Each species has its own characteristics, so extensive studies are needed on more complex forms of response functions in further research.

https://doi.org/10.31764/jtam.v8i1.19893

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A. INTRODUCTION

The eco-epidemiological concept has been widely discussed in recent years in the world of Indonesian research. On a broader scale, many world researchers have developed mathematical-ecological, mathematical-epidemiological, mathematical-economic and other approaches. All the links between mathematical modelling concepts in managing and understanding natural and social contexts. The concept of eco-epidemiology recognizes that biological and social environmental factors play an important role in shaping patterns of disease spread (S. Wang et al., 2018)(Liu et al., 2020). The population as a whole is greatly

influenced by the health of the population as a whole. An important study in eco-epidemiology is interactions between organisms. The interaction system between organisms shows how interactions occur between humans, animals, plants and microorganisms in an ecosystem(Convertino et al., 2021)(Chang et al., 2021). Diseases can move from one creature or species to another. Transmission can occur through interactions between species (Kumar et al., 2021). Intensive and frequent interactions will result in the accumulation of disease transmission more quickly. In general, interactions between animals and plants often carry or transport disease-causing agents from one subject to another.

Apart from interactions between organisms, there are environmental factors that support and influence, namely environmental factors. The eco-epidemiological system recognizes the importance of environmental factors in the spread of disease. Environmental factors containing the physical environment, climate, topography, water quality, soil quality, air quality, polluted areas, and sustainable environment can influence the existence and spread of causes of disease to grow and develop (Mukherjee, 2020). Naturally, in an ecosystem there will be diseases that continuously emerge. Therefore, many methods of transmission also continue to develop over time. Apart from environmental factors, there are also human behavioral factors which include social and human factors. Human behavioral habits, migration patterns, health services and social interactions can contribute greatly to patterns of disease spread. Nowadays, human mobility is very high, across regional, national and even cross-country areas. This event has occurred during the transmission of the SARS-CoV-2 or covid-19 virus (Jiang et al., 2020)(Qu et al., 2020). The movement of people and goods from one place to another is so fast, that the transmission of SARS-CoV-2 spreads very quickly throughout the world (Ibrahim, 2020)(Jiang et al., 2020). All interactions that occur are greatly influenced by the species or living creatures that live in the ecosystem. Therefore, ecological and epidemiological aspects allow researchers to form sustainable mathematical models. The interaction of predator and prey with disease in a population or both populations is an important tool in analysing the spread of disease. The research focus and novelty in this research is on infectious diseases carried by both populations, namely predator and prey. The SIR Kermac-McKendric model of disease spread has been widely developed and has received much attention (Al-salti et al., 2021)(Mukherjee, 2020). In the form of interaction, eco-epidemiological models discuss many studies of populations with disease in prey or both, namely predator and prey. Models of animal species that act as predators and plants as prey for infectious diseases were developed by (Otte & Pica-ciamarra, 2021). The results of his research show a scheme of invasion, persistence and spread of disease in a mathematical model.

The size of the basic reproductive rate also influences the potential for disease transmission. Biological characteristics of the disease-carrying host also play a very important role in transmission. Meanwhile, other research concerns the predator prey system with disease in the prey (Al-salti et al., 2021). The research results show that prey plays an important role in transmission, because prey is still the main food for predators. Other studies that use prey as a model variable that transmits disease provide different characteristics (Das et al., 2022)(Arancibia-ibarra et al., 2021). The results of this research reveal that prey with disease do not always play a significant role in disease transmission. Prey with high survival and

environmental resistance do not transmit significant diseases. Even in certain cases, there are diseased insects that kill themselves so that their group's prey species are not infected.

Another form of mathematical model that is also introduced is an eco-epidemiological system with diseases in prey and weak allies in predators. The research results show the dynamics of population interactions in the stability and branching of the population model. Meanwhile, other research also states that the functional form of the response does not play an important role in determining the dynamic consequences of population interactions (Das et al., 2022; Li et al., 2021; Pratama et al., 2019; Qi & Meng, 2021). Functional response only determines the stability of the predator-prey dynamics model, consistently. In fact, many ecological effect variables intervene in the dynamics of population models. Some of these variables include the harvesting effect, allee effect, carrying capacity, and prey protection effect. There are research results that show the effect of prey protection in theoretical studies and field observations, providing an illustration that prey protection can stabilize the growth of the model population. Apart from being able to stabilize the population dynamics model, the prey protection effect also prevents the extinction of prey (Arancibia-ibarra et al., 2021; Barman et al., 2020; Z. Wang et al., 2020). The form of stabilization at the interior equilibrium point changes the unstable state to a stable state due to the increased concentration of prey protection. The results of other research propose a form of two predator systems, combining prey protection and observing the destabilization effect of the model. Another factor also shown by the research results is that the response function of predators to prey populations plays an important role in the dynamic consequences of interacting population species. There is also relevant research that substitutes the assumption of prey protection effects into the eco-epidemiology dynamic system. In diseased species, assuming a prey protection effect in the predator-prey interaction model is very possible. Starting from the basic assumptions above, this research presents a dynamic system of eco-epidemiology, as a research novelty. The predator-prey model of eco-epidemiology has been widely developed and richly analysed. The proposed basic population dynamics interaction model is as follows,

$$\begin{aligned} \frac{dx}{dt} &= rx\left(1-\frac{x}{k}\right)-\tau az\big((1-m)x\big), \\ \frac{dz}{dt} &= \sigma az\big((1-m)x\big)-\delta z, \end{aligned} \tag{1}$$

The model above consists of two populations, namely populations with density $x(t)$ and predators by density $y(t)$. The main objective of this research is to analyse the model for each population, by calculating the equilibrium point and testing the stability of the equilibrium point. Another objective is given by model analysis (1), in trajectories to see the survival of predator and prey species over a long period of time. In the case of ecosystem life, model (1) can involve species that are susceptible to disease. It is these vulnerable species that describe the mathematical model of the concept or model assumptions (1). Explicit examples in species include predatory fish infected with disease from their food source, or ferrets infected after eating diseased rabbits and many other examples. All parameter values in model (1) are non-negative, including r is the intrinsic growth rate of the prey population, k is the carrying

capacity of the prey population, a is the interaction predation coefficient on the prey population, δ is the natural death rate of predators, ρ is the conversion coefficient of the predator-prey interaction, m is a constant in the proportion of prey that uses shelter. In the interaction, the response function of predators meets the following assumptions:

$$\varphi(0) = 0 \qquad \varphi'(x) > 0 \qquad x > 0 .$$

B. METHODS

This type of research is literature study and analysis. Research development is carried out from research (S. Wang et al., 2018) with different models and analysis. The research stages go through several research steps: preparation of research model assumptions, balance point analysis, selection of local asymptotically stable balance points, trajectory analysis and drawing conclusions from research results. We assume that the prey population $(x(t))$ consists of two sub categories: vulnerable prey $(x(t))$ and prey infected with disease $(y(t))$. Several other assumptions that support the basic framework of research on predator-prey interaction models include: (1) susceptible prey and infected prey both have the ability to reproduce, (2) diseases are assumed to only spread among the prey population, not providing transmission. in predatory species. (3) the disease is assumed to not have a genetic nature or cannot be passed on through the population's genes, (4) the infected prey population does not become immune. Many disease models do not make the species immune. For example, disease *Ichthyopthiriasis* which attacks the body parts of fish, but this disease does not have a genetic nature and does not make the species immune to its transmission (Harris & Tavares, 2013). (5) prey that are susceptible to disease infection have saturation kinetics $\frac{\beta xy}{\alpha + y}$, where β measure the strength of disease infection and α is the effect of inhibiting the spread of disease. (6) Predators eat susceptible and infected prey with the respective predation coefficients being σ and τ.

The growth rate of prey consumed and becoming predators is ρ. (6) In predator species there are intraspecific interactions, which result in predator species competing with fellow species. Intraspecific interactions are considered because many predator species are particularly vicious in the predation process. Therefore, these characteristics are proposed and realistic to consider as confounders of species interactions. This intraspecific interaction behavior is also a novelty in the proposed research, because there are interactions and can disrupt the stability of the epidemiological population model. Combining the basic model in equation (1) and several assumptions above, the predator-prey model in an epidemiological system with prey protection and disease in prey is described in the following equation:

$$\begin{aligned}
\frac{dx}{dt} &= rx\left(1-\frac{x+y}{k}\right)-\frac{\beta xy}{\alpha+y}-\tau az\big((1-m)x\big),\\
\frac{dy}{dt} &= \frac{\beta xy}{\alpha+y}-\sigma az\big((1-m)y\big)-\delta_1 y,\\
\frac{dz}{dt} &= \rho\tau az\big((1-m)x\big)+\rho\sigma az\big((1-m)y\big)-\delta_2 z-cz^2,
\end{aligned} \tag{2}$$

with initial conditions,

$$x(0)=x_0>0,\ \ y(0)=y_0>0,\ z(0)=z_0>0.$$

Where all parameters in model (2) are considered with the following dimensional parameters, as shown in Table 1.

Table 1. Parameters, Biological Meanings and Unit

Parameters	Meaning	Unit
x	The susceptible prey population (time dependent),	$[N]$
z	The infected prey population (time dependent),	$[N]$
y	Predator population (time dependent),	$[N]$
r	Prey's one intrinsic growth rate,	$[T]^{-1}$
k	Prey's environmental carrying capacity,	$[T]^{-1}$
β	The infectious of the disease,	$[T]^{-1}$
α	The inhibition effect ,	$[N]$
τ	Predation coefficient susceptible prey population ,	$[N]^{-1}[T]^{-1}$
σ	Predation coefficient infected prey population,	$[N]^{-1}[T]^{-1}$
c	Intraspecific predation rate,	$[N]^{-1}[T]^{-1}$
δ_1	Natural death rate of prey,	$[T]^{-1}$
δ_2	Natural death rate of predators.	$[T]^{-1}$
a	Interaction predation coefficient on prey population,	-
m	Constant proportion of prey that uses shelter,	-
ρ	The growth rate of prey consumed and becoming predators,	-

C. RESULT AND DISCUSSION

1. Equilibrium Analysis

Equilibrium analysis is carried out using the concept of a linearized differential model. The proposed model (2) uses linearization, so that model (2) becomes;

$$
\begin{aligned}
&rx\left(1-\frac{x+y}{k}\right)-\frac{\beta xy}{\alpha+y}-\tau az\left((1-m)x\right)=0\\
&\frac{\beta xy}{\alpha+y}-\sigma az\left((1-m)y\right)-\delta_1 y=0\\
&\rho\tau az\left((1-m)x\right)+\rho\sigma az\left((1-m)y\right)-\delta_2 z-cz^2=0,
\end{aligned}
\tag{3}
$$

All equilibrium points that appear are five realistic equilibrium points. The equilibrium points associated with model (3) are expressed as follows:

a. The trivial equilibrium point $E_0(0,0,0)$,
b. Equilibrium point with no infected prey or predators $E_1(k,0,0)$. This condition can occur in model (2) with prey and predator species that are not involved in the spread of disease. However, this condition does not meet the stability of model (2), so it does not support research results and ideal ecosystem life.
c. Balance point without predatory species $E_2(x_2, y_2,0)$, this condition is very likely to occur in model (2). This condition is characterized by the absence of predators in the species ecosystem. However, this condition is the same as in the previous point in that this condition is not stable in the long term, or does not meet the ideal stability of a population dynamics model.
d. Disease-free balance point $E_3(x_3,0, z_3)$, this condition clearly occurs in species without the presence of disease. This means that the species that exist are predators and prey without disease. This condition can occur in an ecosystem that does not involve diseased species. An example can occur in the migratory behavior of species, because diseased species are unable to migrate, so new species that live tend to be without diseased species.
e. A point of balance that coexists $E_4(x_4, y_4, z_4)$, this condition occurs under ideal conditions of interacting species. Vulnerable prey species, diseased prey and predator species have stable conditions over a very long period of time and the three populations continue to interact. Equilibrium point $E_4(x_4, y_4, z_4)$ we symbolize it in a more general form, namely $E(x^*, y^*, z^*)$. It is clear that for the equilibrium point $E(x^*, y^*, z^*)$ positive value, otherwise the point has no meaning whatsoever for ecological knowledge.

Equilibrium points actually support the sustainability of the model in an ecosystem, but there are only a few equilibrium points that are stable and fulfill the form of ecosystem stability. Form an equilibrium point $E(x^*, y^*, z^*)$ becomes a point that is worth taking into account in terms of the sustainability of the species in the ecosystem. Equilibrium point $E(x^*, y^*, z^*)$ will be tested with criteria Routh-Hurwitz, to test the stability of its population growth. This point will be analysed further using the Jacobian matrix and eigenvalue criteria. Jacobian matrix associated with equilibrium point $E(x^*, y^*, z^*)$ are as follows,

$$J_{cob}(E)=\begin{bmatrix} j_{11} & j_{12} & j_{13} \\ j_{21} & j_{22} & j_{23} \\ j_{31} & j_{32} & j_{33} \end{bmatrix} \tag{4}$$

where,

$$\begin{aligned}
j_{11} &= r\left(1-\frac{x^*+y^*}{k}\right)-\frac{rx^*}{k}-\frac{\beta y^*}{\alpha+y^*}-\tau a z^*(1-m),\\
j_{12} &= -\frac{rx^*}{k}-\frac{\beta x^*}{\alpha+y^*}+\frac{\beta x^* y^*}{(\alpha+y^*)^2},\\
j_{13} &= -\tau a x^*(1-m),\\
j_{21} &= \frac{\beta y^*}{\alpha+y^*},\\
j_{22} &= \frac{\beta x^*}{\alpha+y^*}+\frac{\beta x^* y^*}{(\alpha+y^*)^2}-\sigma a z^*(1-m)-\delta_1,\\
j_{23} &= -\sigma a y^*(1-m),\\
j_{31} &= \rho\tau a z^*(1-m),\\
j_{32} &= \rho\sigma a z^*(1-m),\\
j_{33} &= \rho\tau a x^*(1-m)+\rho\sigma a y^*(1-m)-\delta_2-2cz^*.
\end{aligned}$$

From the Jacobian matrix form, the characteristic equation associated with model (3) will be obtained as follows;

$$\lambda^3-a\lambda^2+b\lambda-c=0, \tag{5}$$

where,

$$\begin{aligned}
a &= j_{11}+j_{22}+j_{33},\\
b &= j_{11}j_{22}-j_{12}j_{21}-j_{13}j_{31}-j_{23}j_{32}+j_{11}j_{33}+j_{22}j_{33},\\
c &= j_{11}j_{23}j_{32}+j_{12}j_{21}j_{33}+j_{13}j_{31}j_{22}-j_{21}j_{13}j_{32}-j_{12}j_{23}j_{31}-j_{11}j_{22}j_{33},
\end{aligned}$$

The characteristic equation (5), it is clear that it is proven for the Routh–Hurwitz criterion. Necessary and sufficient conditions for the equilibrium point $E(x^*,y^*,z^*)$ to meet the local stability of the positive point is as follows, $a<0$ and $ab<c<0$. Ecologically, it is said that the predator and prey population that lives in an ecosystem survives for a long period of time. Even though there are diseased species, they can still live together for a long time. From the point of view of ecological science, of course this is desirable, because extinction of species is undesirable.

2. Numerical Simulation

Model (2) will be simulated with parameters to see the basic scheme of population growth for each species. Numerical simulation is also used to see the growth trajectories of predator-prey populations. The parameters for each model form are given from several valid, relevant and updated references. Several parameters are also taken from basic assumptions in ecosystem life. The parameters given mathematically are as follows,

$r = 1.52$, $k = 100$, $\alpha = 0.5$, $\rho = 0.08$, $a = 0.05$, $m = 0.4$, $\delta_1 = 0.0045$, $\delta_2 = 0.002$, $\tau = 0.04$, $\sigma = 0.06$, $\beta = 0.08$, and $c = 0.001$.

Model (2) is simulated with the parameters being;

$$\begin{aligned}
\frac{dx}{dt} &= 1.52x\left(1 - \frac{1}{100}x - \frac{1}{100}y\right) - \frac{0.08xy}{0.5+y} - 0.001860zx \\
\frac{dy}{dt} &= \frac{0.08xy}{0.5+y} - 0.002790zy - 0.0045y, \\
\frac{dz}{dt} &= 0.00014880zx + 0.00022320zy - 0.002z - 0.001z^2,
\end{aligned} \tag{6}$$

From model equation (6), a non-negative equilibrium point is obtained, which is as follows:

$$E_0 = (0,0,0) \tag{7}$$

$$E_1 = (100,0,0) \tag{8}$$

$$E_2 = (5.07333831\ 2, 89.6926810\ 9, 0) \tag{9}$$

$$E_3 = (98.4520799\ 2, 0, 12.6496694\ 9) \tag{10}$$

$$E_4 = (35.5937083\ 8, 57.2224595\ 9, 16.0683967\ 9) \tag{11}$$

Of the several equilibrium points above, the one that fulfills and is the most realistic to consider for stability analysis is the equilibrium point $E_4(x^*, y^*, z^*)$. Testing and analysis of equilibrium point stability is provided to see the long-term growth of the species. The analysis will be carried out using the Jacobian matrix stages and the Routh-Hurwitz criteria. At the equilibrium point $E_4(x^*, y^*, z^*)$ we obtain the Jacobian matrix as follows;

$$J_{cob}(E_4) = \begin{bmatrix} -0.541024367\ 6 & -0.541451677\ 9 & -0.066204297\ 59 \\ 0.079307028\ 84 & -0.048903516\ 53 & -0.159650662\ 3 \\ 0.002390977\ 442 & 0.003586466\ 164 & -0.016068396\ 79 \end{bmatrix} \tag{12}$$

From the Jacobian matrix (12), we get the form of the characteristic equation at the equilibrium point, namely;

$$\lambda^3 + 0.605996280\,9\,\lambda^2 + 0.079608987\,9\,\lambda + 0.001244798156 \qquad (13)$$

The necessary and sufficient conditions, which are important for the Routh-Hurwitz characteristic equation, are the fulfillment of the eigenfactors $\lambda < 0$, as follows;

$\lambda_1 = -0.425966337578987$, $\lambda_2 = -0.161989986817703$, and $\lambda_3 = -0.018039956523311 2.$

It is clear that the fulfillment of the sufficient conditions and necessary conditions of Routh-Hurwitz has been fulfilled by the presence of these eigenvalues. This results in an equilibrium point $E_4\left(x^*, y^*, z^*\right)$ becomes a locally stable equilibrium point over a long period of time. In the discussion of our analysis, a global analysis for this equilibrium point has not been provided. We are aware of the shortcomings and for a global stable equilibrium point still in the analysis of our research team. In general, it can be said that vulnerable prey, diseased prey and predator species can each coexist in a sustainable ecosystem. This is of course very useful if the species in question is a species that is useful or can be used for the needs and life of the ecosystem. We also provide trajectory analysis in the research discussion analysis. The trajectories of the growth of all populations in equation (2) are as follows,

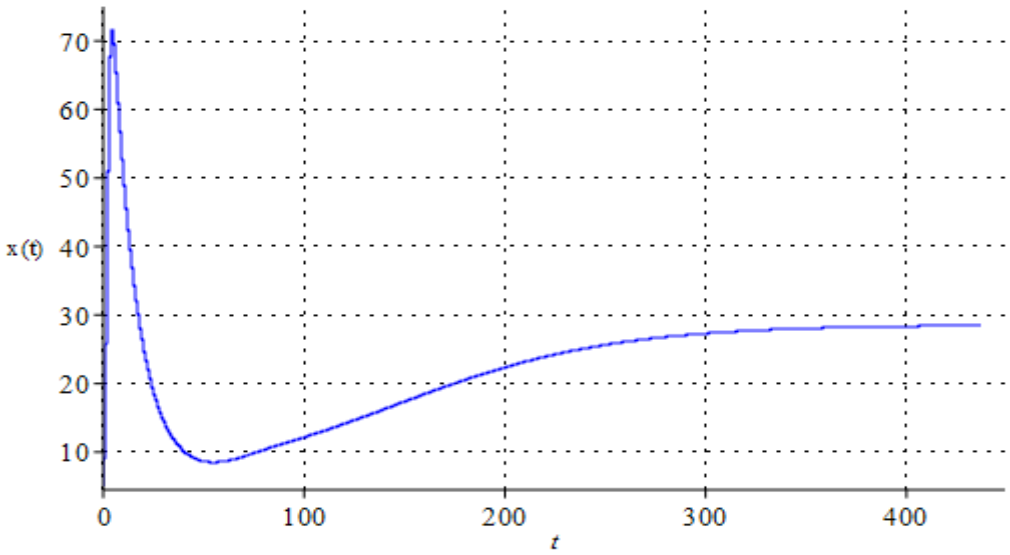

Figure 1. Trajectories species prey vulnerable

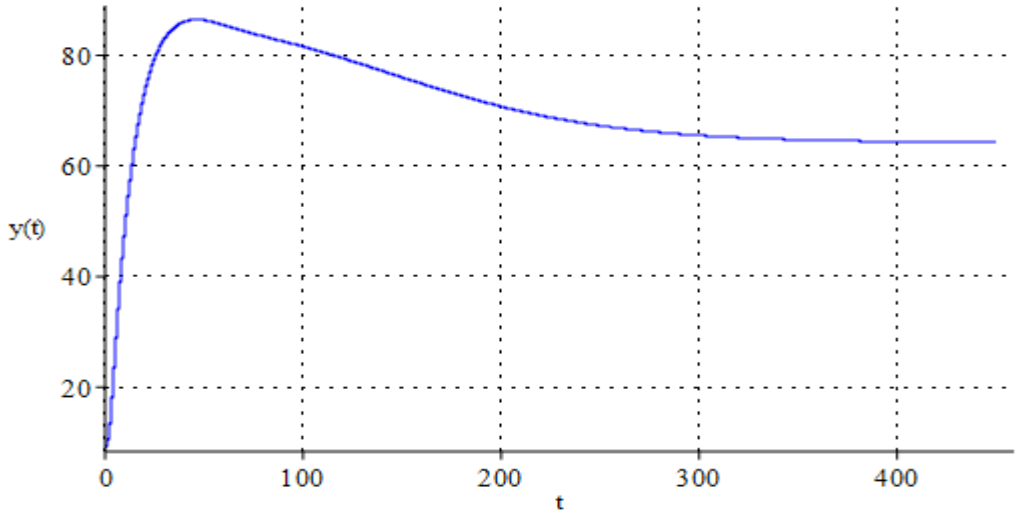

Figure 2. Trajectories of prey species infected with disease

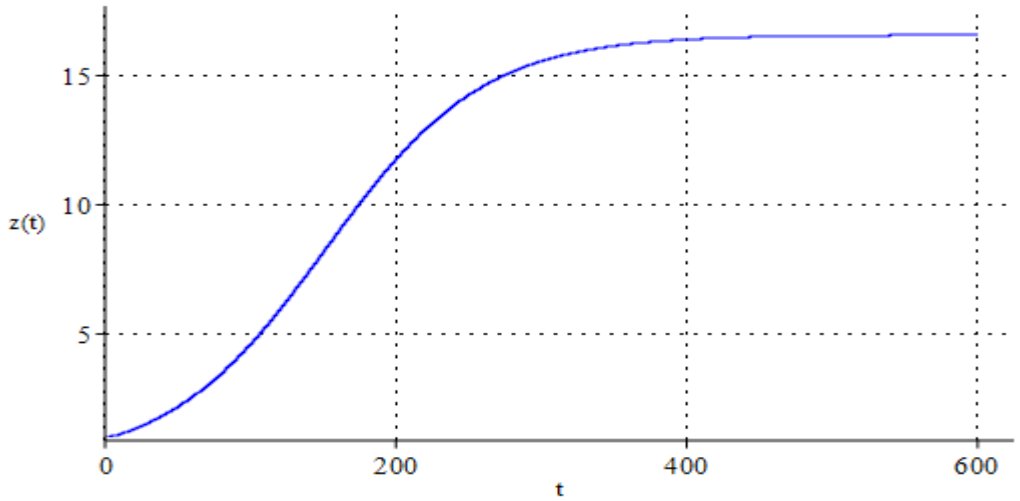

Figure 3. Trajectories species predator

Figure 1, Figure 2, and Figure 3 all show the movement of trajectories towards their point of stability. This is called the absence of extinction in the growth of the species, whether susceptible prey or infected prey. The unique characteristic of vulnerable prey species is that their growth trajectories show significant growth at the beginning of the interaction, but after passing the saturation point of the curve, the direction of population growth also decreases significantly. However, the decline in trajectories in vulnerable prey species reaches a point of stability. Fluctuations in trajectories in vulnerable prey species are very interesting to discuss further. Especially at the peak point of predation and the valley of predation trajectories, until it grows back to its stable point. Basically, all interactions have their own characteristics when interacting with predator species. In prey species infected with disease, growth also occurs significantly at the beginning of the interaction.

Similar to prey, because both prey species' trajectories are not much different from vulnerable prey species. In the early days of interaction, growth was very fast, and experienced fluctuations as time went by. Another unique characteristic of susceptible prey species and prey

infected with disease is that growth reaches a point of stability at the same time. Meanwhile, the difference is that when interactions between species have reached the saturation point, prey populations with disease do not form valley trajectories, but go straight to the point of stability. This is also confirmed in his research (Pratama et al., 2023) that is this characteristic is what makes prey species infected with disease have more stable growth compared to other species. Meanwhile, in predatory species the trajectory continues to grow, although at the beginning the growth is more sloping. If we look at the trajectory coefficient, it can be said that with the presence of susceptible prey and diseased prey, the growth of predators is very slow, but they are still growing positively this was also confirmed by (Arancibia-ibarra et al., 2021; Z. Wang et al., 2020). What is interesting is that the growth of predators to reach a point of stability takes twice as long compared to susceptible prey and prey infected with disease. In ecological cases, this can happen, because interactions between species in the ecosystem must reach a point of stability this was also confirmed by (Q et al., 2021; Qi & Meng, 2021). We are fully aware that population stability can still change. These changes can be in the form of natural factors or environmental factors that cannot be predicted. Stability analysis with local asymptotic stability does not guarantee the stability of all populations forever.

The analysis part of this paper, the shape of the predator-prey model with disease in the prey has been described and the equilibrium point analysed. Ecologically and epidemiologically, relevant assumptions can be realized for the model being developed; interactions between vulnerable prey and predators, interactions between disease-infected prey and predators, and interactions between predators and each other. Conditional traits or characteristics such as prey protection properties, rate of spread of infection, and predator conversion efficiency play an important role in the proposed model (2). In the form of model equation (2), the following ecological and epidemiological logical assumptions can be given;

a. Infectious diseases in model assumption (2) can be prevented by controlling protective interactions in prey. This means that if the prey has a level of protective interaction, then the rate of disease spread can be controlled. This control is only limited to suppressing the growth rate of spread, not eliminating the disease.
b. Ways to control infectious diseases in prey can be considered simply, namely the effect of prey protection must be relatively small.
c. Intraspecific interactions that occur in predator species also greatly influence the growth of the species' ecosystem. If intraspecific interactions are very large, there will be extinction of predator species and prey infected with disease.

Based on the discussion above, we found several basic concepts based on research results, that the impact of prey protection and intraspecific interactions have a detrimental impact on the stability of population growth. In many cases these two factors seriously disrupt the stability of the eco-epidemiological system. This is in line with the opinion of relevant researchers (Arancibia-ibarra et al., 2021; Jiang et al., 2020; S. Wang et al., 2018) that stability effects are often seen in predator-prey systems without disease in the prey and/or predator. Based on the findings at the equilibrium point, an increase in the rate of transmission or spread of disease can cause a loss of stability. This also occurs in intraspecific interactions. Therefore, the stability effect of the predator-prey population model is largely determined by the

characteristics of predator interactions, such as conversion coefficients, functional types of predator responses to prey populations, intraspecific interactions. Analysis of research results using mathematical models, showing the effect of prey protection, can be applied to control the spread of disease. The level of prey protection required to control disease spread is primarily determined by the transmission rate and predator conversion ratio.

D. CONCLUSION AND SUGGESTIONS

This paper discusses the study of the predator-prey model with the protective nature of prey that spreads disease and intraspecific interactions that occur in predator species. The characteristics that emerge are the nature of prey that is susceptible and infected with disease. Both populations are represented by variables $x(t)$, while in the disease infected prey variable it is symbolized by $y(t)$. Variables for predator species that have intraspecific traits are represented by variables $z(t)$. The three species interact in an ecosystem that has carrying capacity. The assumptions given for the behavior of the three species are in accordance with the conditions of the species and are rational. The mathematical model formed from these assumptions occurs in model (2). Model (2) is where the equilibrium point is analysed, given numerical simulations and the population growth trajectories analysed. The rational equilibrium point considered is the point $E_4\left(x^*, y^*, z^*\right)$ from the five equilibrium points obtained in model (2). The research results consider the survival of all species, so the equilibrium point is taken. Analysis and testing of numerical simulations are also provided, to see the necessary and sufficient conditions for model stability (2). The Routh-Hurwitz criterion, has been given by model (2) on the stability of each population. The analysis provided is a local asymptotic stability analysis. In the numerical simulation, eigenvalues associated with model (2) appear, namely; $\lambda_1 = -0.4259663\,37578987$, $\lambda_2 = -0.1619899\,86817703$, and $\lambda_3 = -0.0180399565233112$. Meanwhile, the trajectory analysis also provides a simulation of the movement of each population growth.

The growth of each population shows fluctuating growth. In populations, susceptible prey and infected prey have the same characteristics. Even population growth over time is relatively the same. A significant difference occurred in the growth of the predator population, which turned out to be very slow. For predator species to achieve sustainable growth, it takes longer than for susceptible prey species or infected prey. Meanwhile, if we look at the fluctuations, the population of vulnerable prey species is very fluctuating, this happens because interactions between prey can occur at any time, so it is from this interaction that the infection process spreads. This of course results in vulnerable species, very fluctuating towards the point of stability. In infected prey species, there is no significant fluctuation in fitness, because growth slopes from a height curve towards a point of stability. Each population species has its growth characteristics. Based on the results of this research, the development of model (2) can be carried out by looking at harvesting or exploitation and harvesting behavior. It is very possible in model (2) to give shape, harvesting and optimum principles for calculating profits. Harvesting that will be carried out can occur in susceptible prey species, infected prey and predator species. In ecological ecosystems, it is very possible that these species are harvested.

After harvesting, the next follow-up is to calculate the optimum profit from the harvesting business using bio-economic principles.

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