Eco-Epidemiological Model in The Interaction of Pelecanidae Birds and Tilapia Fish

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Abstract. Here in this article, we construct an eco-epidemiological mathematical model which describe an interaction between Tilapia as prey, Pelecanidae as predator and Vibrio vulnivicus as a bacterium that spread disease among Tilapia. The disease might be transferred into Pelecanidae from predation interaction. The model constructed as a five-dimensional non-linear ordinary differential equation. We analyze the existence and local stability of the equilibrium points using linearization with a Jacobian matrix. Some numerical simulations are given to give some interpretation to understand how these parameters affect the dynamic of the model. We find that when the disease spread among Tilapia uncontrolled, then the extinction on Tilapia and Pelecanidae might appear.

Keywords : Tilapia, Pelecanidae, Vibrio Vulnivicus, Eco-Epidemiological.

1 Introduction

Ecological and epidemiological mathematical models are fields of mathematics and biology which discuss several biological phenomena such as competition and predation between two species. Predation interaction can say as an interaction between predator populations and prey populations. In predation among animals, predators are mostly larger than prey. The simplest predator and prey model involving two species, namely one predator species and prey species was first introduced by Lotka in 1910 [1] and Volterra in 1926 [2] which is now better known as the Lotka - Volterra model. Based on the Lotka - Volterra model, when both species exist, a decrease in the number of prey populations and an increase in the name of predatory populations occur at a rate proportional to the frequency of encounters between individuals in both species [3]. Until today, many mathematical models have constructed by many authors based on the Lotka - Volterra model, such as [4], [5], [6]. In the other hand, there are several models which also combine the predatory models with diseases among prey or predator populations, such as in [7], [8], [9], [10], [11].

The eco-epidemiology model is a model that studies the spread of infectious diseases in a population in a hierarchy in an environment. Disease factors in the predator-prey system were first introduced by Anderson and May [12] who examined the study of disease control factors by selecting parameter values determined by various biological properties of the relationship between individual parasites and their hosts and interactions between these populations. The phenomenon that describes where the prey population is affected by the disease is explained by Chattopadhyay and Bairagi that occur in the Salton Sea, California [13].

In this paper, we will discuss the eco-epidemiological model on the interaction of Pelecanidae birds and Tilapia fish that have been attacked by poisoning due to the bacteria

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Vibrio vulnivicus that occurs in the Salton Sea, California. The bacteria that attack Tilapia make the body of Tilapia dehydrated. Several studies have shown that bacteria that attack Tilapia can change the behaviour of fish so that infected Tilapia fish will swim to the surface of the water and become more easily predated by birds Pelecanidae [14]. Pelecanidae birds that eat infected Tilapia will get poisoned and die. In this case, the infection process that occurs in the population of the Pelecanidae bird is based on Holling type II's response function because Tilapia is challenging to capture so the Pelecanidae bird is active in searching for its prey.

In this paper, a predator-prey mathematical model will construct with disease factors in the prey population. In the next discussion, an analysis will be carried out regarding the equilibrium points and the stability of the equilibrium points obtained. The fourth discussion will explain numerical simulations of system dynamics, and finally, conclusions will give regarding the research that has done.

2 Construction of The Mathematical Model

In this section, we will construct a mathematical model that describes problems that already discussed in the previous section. The first step, let us consider three populations, i.e. Tilapia as prey, Pelecanidae as predator and bacteria. Because we divide each Tilapia and release another population based on their health status, i.e. susceptible and infected. To be specific, let us consider that B as a Vibrio vulnivicus bacterium populations, T_S and T_I as susceptible and infected Tilapia populations, respectively, and P_S and P_I present susceptible and infected Pelecanidae populations, respectively.

To construct the model, let us consider the following assumptions:

1. In the absence of disease, the Tilapia population grows logistically with carrying capacity $K > 0$ and the intrinsic growth rate $r > 0$.
2. In the presence of disease, the Tilapia population divided into two separate classes, namely the susceptible Tilapia (T_S) and infected (T_I) populations with the total population was $N(t) = T_S + T_I$.
3. Infected Tilapia will more often surface due to dehydration. Therefore, the Pelecanidae population only preys on infected Tilapia.
4. The Pelecanidae consumption function follows the Holling Type II response function.

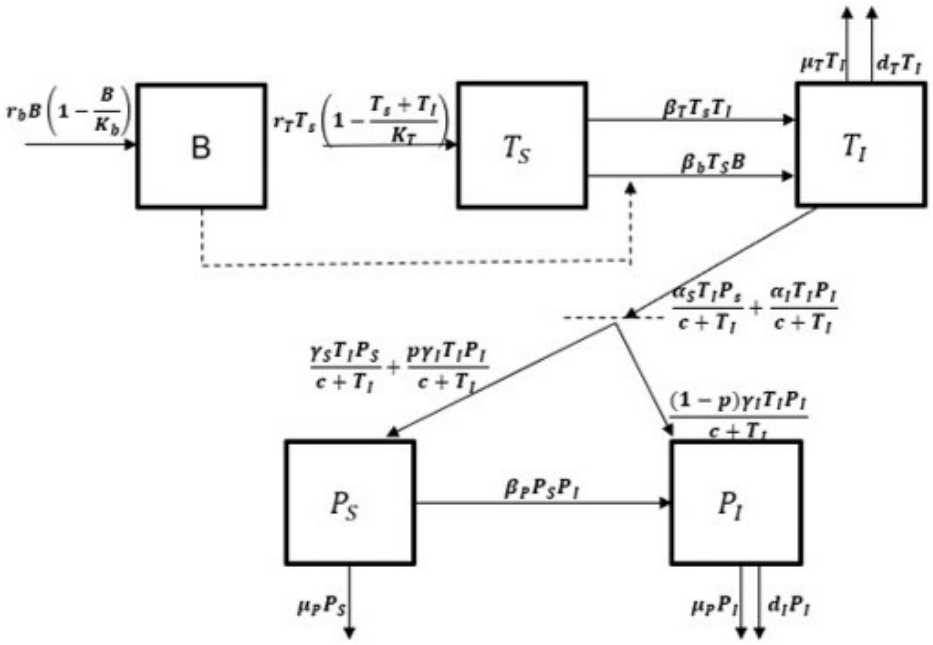

Figure 1. Transmission diagram for interaction between Tilapia, Pelecanidae and Vibrio vulnivicus bacterium.

Using transmission diagram in Figure 1 and previous assumptions, mathematical model for interaction between Tilapia, Pelecanidae, and Vibirio vulnivicus bacterium with disease among Tilapia and Pelecanidae now given by:

$$\frac{dB}{dt} = r_b B\left(1-\frac{B}{K_b}\right)$$

$$\frac{dTs}{dt} = r_T T_s\left(1-\frac{T_s+T_I}{K_T}\right) - \beta_T T_s T_I - \beta_b T_S B$$

$$\frac{dTi}{dt} = \beta_T T_S T_I + \beta_b T_S B - \mu_T T_I - d_T T_I - \frac{\alpha_S T_I P_S}{c+T_I} - \frac{\alpha_I T_I P_I}{c+T_I} \quad (1)$$

$$\frac{dPs}{dt} = \frac{\gamma_S T_I P_S}{c+T_I} + \frac{p\gamma_I T_I P_I}{c+T_I} - \mu_P P_S - \beta_P P_S P_I$$

$$\frac{dPi}{dt} = \frac{(1-p)\gamma_I T_I P_I}{c+T_I} + \beta_P P_S P_I - \mu_P P_I - d_I P_I$$

suplemented with initial conditions $B(0)=0, T_S(0)=0, T_I(0)=0, P_S(0)=0, P_I(0)=0$. Description for all parameters in system (1) is given in Table 1.

Table 1. Description of parameters in system (1).

Parameter	Definition	Value	Unit
$\boldsymbol{r_b, r_T}$	The intrinsic growth rate in bacterial and prey populations	$r_b, r_T \in (0, \infty)$	$\frac{1}{\text{time}}$
$\boldsymbol{K_b, K_T}$	Carrying capacity for bacterial and prey populations	$K_b, K_T \in (0, \infty)$	bacteria and prey
$\boldsymbol{\beta_T}$	Rate of disease transmission in prey	$\beta_T \in [0, \infty)$	$\frac{1}{\text{prey x time}}$
$\boldsymbol{\beta_B}$	Rate of disease transmission due to bacteria	$\beta_B \in [0, \infty)$	$\frac{1}{\text{bacteria x time}}$
$\boldsymbol{\mu_T}$	The natural mortality rate in prey	$\mu_T \in (0, \infty)$	$\frac{1}{\text{time}}$
$\boldsymbol{d_T}$	The death rate due to bacteria in prey	$d_T \in [0, \infty)$	$\frac{1}{\text{time}}$
$\boldsymbol{c}$	The saturation value of the response function	$c \in (0, \infty)$	prey
$\boldsymbol{\alpha_S, \alpha_I}$	Predation coefficient	$\alpha_S, \alpha_I \in [0, \infty)$	$\frac{\text{prey}}{\text{predator x time}}$
$\boldsymbol{\gamma_S, \gamma_I}$	Energy conversion from predation	$\gamma_S, \gamma_I \in [0, \infty)$	$\frac{1}{\text{time}}$
$\boldsymbol{p}$	Proportion	$p \in [0,1]$	-
$\boldsymbol{\mu_P}$	The natural death rate of the predator	$\mu_P \in (0, \infty)$	$\frac{1}{\text{time}}$
$\boldsymbol{\beta_P}$	Rate of transmission due to disease in predator	$\beta_P \in [0, \infty)$	$\frac{1}{\text{predator x time}}$
$\boldsymbol{d_I}$	Rate of death due to bacteria in predator	$d_I \in [0, \infty)$	$\frac{1}{\text{time}}$

3 Mathematical Model Analysis

In this section, we will analyze the existence and local stability of all equilibrium points of the system (1). The equilibrium points taken from taking the right-hand side of the system (1) equal to zero, and then solve them respect to all variables. For an example, if we take $P_I, P_S = 0$ substitute them into system (1) and solve it respect to variables B, T_S, T_I we have several possibilities of solution, i.e (1) $B = 0, T_S = 0, T_I = 0$, (2) $B = B, T_S = 0, T_I = 0$, (3) $B = 0, T_S = T_S, T_I = T_I$, and $B = 0, T_S = T_S, T_I = 0$. More detail for all equilibrium points will discuss in the next subsection.

After we analyze the existence of the equilibrium point, we also analyze the local stability criteria of these equilibrium points using the linearization of a system (1) with a Jacobian matrix. The Jacobian matrix of system (1) is given by:

$$J=\begin{bmatrix} r_b\left(1-\frac{B}{K_b}\right)-\frac{r_b B}{K_b} & 0 & 0 & 0 & 0 \\ -\beta_B T_S & r_T\left(1-\frac{T_S+T_I}{K_T}\right)-\frac{r_T T_S}{K_T}-\beta_T T_I-\beta_B B & -\frac{r_T T_S}{K_T}-\beta_T T_S & 0 & 0 \\ \beta_B T_S & \beta_B B+\beta_T T_I & \beta_T T_S-\mu_T-d_T-\frac{\alpha_S P_S}{c+T_I}+\frac{\alpha_S T_I P_S}{(c+T_I)^2}-\frac{\alpha_I P_I}{c+T_I}+\frac{\alpha_S T_I P_I}{(c+T_I)^2} & -\frac{\alpha_S T_I}{c+T_I} & -\frac{\alpha_I T_I}{c+T_I} \\ 0 & 0 & \frac{\gamma_S P_S}{c+T_I}-\frac{\gamma_S T_I P_S}{(c+T_I)^2}+\frac{p\gamma_I P_I}{c+T_I}-\frac{p\gamma_S T_I P_I}{(c+T_I)^2} & \frac{\gamma_S T_I}{c+T_I}-\beta_P P_I-\mu_P & \frac{p\gamma_I T_I}{c+T_I}-\beta_P P_S \\ 0 & 0 & \frac{(1-p)\gamma_I P_I}{c+T_I}-\frac{(1-p)\gamma_I T_I P_I}{(c+T_I)^2} & \beta_P P_I & \frac{(1-p)\gamma_I T_I}{c+T_I}-\beta_P P_S-\mu_P-d_I \end{bmatrix}$$

An equilibrium point Ω_i is said to be locally stable if all eigenvalues of Jacobian matrix J evaluated in Ω_0 have a negative real part. If there are at least one eigenvalues of J is positive, then the equilibrium point is said to be unstable. Here is an example of a local stability calculation process for the equilibrium $\Omega_0 = (0,0,0,0,0)$.

$$\mathrm{J}\Omega_0=\begin{bmatrix} r_b & 0 & 0 & 0 & 0 \\ 0 & r_T & 0 & 0 & 0 \\ 0 & 0 & -\mu_T-d_T & 0 & 0 \\ 0 & 0 & 0 & -\mu_P & 0 \\ 0 & 0 & 0 & 0 & -\mu_P-d_I \end{bmatrix}$$

Based on the eigenvalues obtained, there are two positive eigenvalues so that the equilibrium point Ω_0 is unstable. The same explanation applies to other equilibrium points.

In the next two sections, we will show the existence and stability of all equilibrium points of the system (1).

3.1 Disease Free Equilibrium Point

Taking $P_I = 0$, system (1) has seven disease-free equilibrium points, which also completed with the existence and local stability criteria. The existence and local stability criteria are given in Table 2.

Table 2. Ekuilibrium points and stability of the ekuilibrium points

Ekuilibrium points	The condition of the ekuilibrium points appear	The condition for a stable ekuilibrium points
$\Omega_0 = (0,0,0,0,0)$	-	Unstable
$\Omega_1 = (B^1, 0,0,0,0)$	-	$\frac{r_T}{K_b\beta_b} < 1$
$\Omega_2 = (0, {T_S}^2, 0,0,0)$	-	Unstable
$\Omega_3 = (0, {T_S}^3, {T_I}^3, 0,0)$	$d_T + \mu_T > 0,\ K_T\beta_T > d_T + \mu_T$	Unstable
$\Omega_4 = (0, {T_S}^4, {T_I}^4, {P_S}^4, 0)$	$\gamma_S > \mu_P,$ $\frac{((c\beta_T+r_T)K_T+cr_T)\mu_P}{K_T\gamma_S r_T} < 1,$ $\frac{(c+K_T r_T+cK_T\beta_T)\beta_T\mu_P+r_T\gamma_S(d_T+\mu_T)}{(((d_T+\mu_T)\mu_P+K_T\beta_T\gamma_S)r_T} < 1$	Unstable
$\Omega_5 = \left(B^5, {T_S}^5, {T_I}^5, {P_S}^5, 0\right)$	$\gamma_S > \mu_P,$ $\frac{((c\beta_T+r_T)K_T+cr_T)\mu_P}{K_T\gamma_S r_T} < 1$	$-\frac{b}{a} < 0$ $a_3 > 0, a_1 > 0, (a_3 a_2 > 0)$

$\Omega_6 = (B^6, {T_S}^6, {T_I}^6, 0,0)$	$\frac{K_b\beta_b}{r_T} > 1,$ $\frac{K_T\beta_T r_T}{(K_b\beta_b + d_T + \mu_T)r_T + 2K_T K_b\beta_T\beta_b} > 1,$ $\frac{r_T K_T}{r_T {T_I}^6 + \beta_b K_T {T_I}^6 + \beta_b K_b K_T} > 1$	$\frac{T_I\gamma_S}{\mu_P(T_I + c)} < 1$ $\frac{T_I\gamma_I(1-p)}{\mu_P(T_I + c) + d_I(T_I + c)} < 1$ $h_1 > 0, h_2 > 0, (h_1 h_2 > 0)$

3.2 Coexistence Ekuilibrium Points

Especially for the coexistence ekuilibrium points, it cannot be done analytically, so the numerical approach is carried out as follows. The coexistence ekuilibrium point on this system is given by $\Omega_1 = (K_b, T_S, T_I, P_S, P_I)$ with

$$P_S = f_1(T_I)$$

$$T_S = f_2(T_I, P_I) \tag{2}$$

With T_I and P_I is the solution of two polynomials

$$F_1(T_I, P_1) = 0$$

$$F_2(T_I, P_1) = 0$$

To find the ekuilibrium point of the two polynomial functions above, it can be shown numerically using the Maple application by substituting the parameter values as follows: $r_b = 1,\ K_b = 500, r_T = 0.1, K_T = 30, \beta_T = 0.00002, \beta_B = 0.00005, \mu_T = \frac{1}{2X365}, d_T = 0.05, \alpha_S = 0.01, c = 1, \alpha_I = 0.1, \gamma_S = 0.01, p = 0.2, \gamma_I = 0.1, \mu_P = \frac{1}{20x365}, \beta_P = 0.035, d_I = 0.1$.The intersection point between F_1 and F_2 is given in the Figure 2.

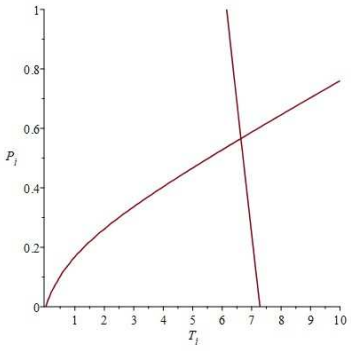

Figure 2. Intersection point between $F_1$and F_2

From the picture above, the intersection point between $F_1$and F_2occurs at P_I = 0.56 and T_I = 6.63. So substituting the value of P_I and T_I into equation (2) will get the balance point of coexistence as follows

$$\Omega_1 = (500,15.82,6.63,0.87,0.56)$$

and is stable. From following parameters above, polynomials $F_1$and F_2 are given by:

$$F_1 = 0.035P_IT_I + 0.0011797945205{T_I}^2 + 0.0035P_I + 0.003797260273{T_I}^3 + 7.04200\text{x}10^{-8}{T_I}^4 - 0.003571118493T_I + 0.00035P_I{T_I}^2 - 0.001968750000 + 0.00035P_IT_I$$

$$F_2 = 4.79452050\text{x}10^{-6}P_I{T_I}^2 + 0.003509589041P_IT_I - 0.0001986113718{T_I}^2 + 0.003504794520P_I - 0.0009848939764T_I + 0.00001371739539$$

4 Numerical Analysis

In this section, we will discuss numerical simulations of autonomous systems in systems (1). The parameter values used for autonomous simulation are $r_b = 1, r_T = 0.1, K_b = 500, K_T = 30, \beta_T = 0.00002, \beta_B = 0.00005, \mu_T = \frac{1}{2x365}, d_T = 0.05, c = 1, \alpha_S = 0.01, \alpha_I = 0.1, \gamma_S = 0.01, \gamma_I = 0.1, p = 0.2, \mu_P = \frac{1}{20x365}, \beta_P = 0.035, d_I = 0.1$ (except as stated differently) and the initial condition value of the model given by

$$B(0) = 100, T_S(0) = 1, T_I(0) = 3, T = P_S(0) = 2, P_I(0) = 2$$

4.1 Autonomous Simulation with Variations in the Value of α_I

This simulation aims to see how the severity of disease in birds Pelecanidae α_I affect system dynamics (1). The α_I parameter simulation is carried out with four variations of the different α_I values, namely α_I = 0.1,0.2,0.3,0.4 and sets the value of other parameters and initial values as given above. The results of the autonomous simulation are given in Table 3.

Table 3. Results of autonomous simulation with variations in the value of α_I

Case	Stable Equilibrium					Colour
	B	T_S	T_I	P_S	P_I	
$\alpha_I = 0.1$	500	15.82	6.63	0.87	0.56	Red
$\alpha_I = 0.2$	500	16.38	6.08	0.89	0.53	Magenta
$\alpha_I = 0.3$	500	16.86	5.59	0.92	0.5	Blue
$\alpha_I = 0.4$	500	17.29	5.17	0.94	0.47	Green

Based on the study of equilibrium point and equilibrium point stability in Table 3, from the eigenvalues, only the coexistence equilibrium point is stable. From Table 3, it can see that the smaller the value of α_I, the higher the population size of the infected Tilapia fish in the equilibrium is equal to 6.63. Besides, decreasing the amount of α_I also make the population of infected Pelecanidae birds in equilibrium is 0.56. The following is a picture of the simulation that has done in Figure 3.

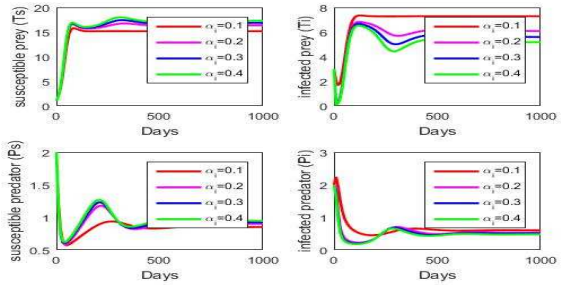

Figure 3. System dynamics with variation in the value of α_I

Predation in the Tilapia fish population decreases (it can interpret that poisoning due to the bacteria Vibrio vulnivicus weakens so that Tilapia fish do not have to surface), the population of Tilapia fish will increase. With the increase of Tilapia fish population makes the Pelecanidae bird population also abundant due to the abundant availability of food.

4.2 Autonomous Simulation with Variations in the Value of p

This simulation aims to see how the consumption level of predation by Pelecanidae birds can affect system dynamics (1). The p parameter simulation is carried out with five different p-value variations, namely p = 0.0.25,0.5,0.75,1 and sets the value of other parameters and initial values as given above. The results of the autonomous simulation are given in Table 4.

Table 4. Results of autonomous simulation with variations in the amount of p

Case	Stable Equilibrium					Colour
	B	T_S	T_I	P_S	P_I	
$p = 0$	500	15.40	7.05	0.35	0.24	Red
$p = 0.25$	500	15.92	6.54	1.00	0.63	Magenta
$p = 0.5$	500	16.36	6.10	1.63	0.97	Black
$p = 0.75$	500	16.73	5.72	2.25	1.25	Blue
$p = 1$	500	17.06	5.39	2.86	1.5	Green

Based on a review of the equilibrium point and equilibrium point in Table 4, from the eigenvalues, only the coexistence equilibrium point is stable. From Table 4, it can see that the smaller the value of p, the larger the size of Tilapia in the equilibrium is equal to 7.05. On the other hand, population values make Pelecanidae birds decrease in equilibrium decreases, both Pelecanidae bird populations are also vulnerable. The following is a picture of the simulation that has done in Figure 4.

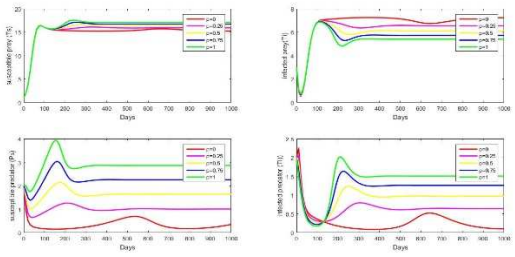

Figure 4. System dynamics with variation in the value of p

Based on the results in Table 4 and Figure 4, it concluded that when the proportion of energy conversion from predators to interactions between Pelecanidae and Tilapia fish decreases (it can interpret that a small Pelecanidae bird can consume tilapia). The results in birth in the Pelecanidae bird population little so that the population of the Pelecanidae bird will decrease and continue to decrease towards its equilibrium point.

4.3 Autonomous Simulation with Variations in the Value of β_T

For the last simulation aims to see how the rate of disease transmission in the Tilapia fish influences the dynamics of the system (1). The parameter simulation β_T is done with three different variations of the value of β_T, namely $\beta_T = 0.01,0.02,0.03$ and set the number of other parameters and initial values as given above. The results of the autonomous simulation are given in Table 5.

Table 5. Results of autonomous simulation with variations in the amount of β_T

Case	Stable Equilibrium				
	B	T_S	T_I	P_S	P_I
$\beta_T = 0.01$	500	6.01	5.99	0.90	0.52
$\beta_T = 0.02$	500	3.08	3.84	1.04	0.39
$\beta_T = 0.03$	500	2.09	2.79	1.17	0.32

From eigenvalues analysis for all above equilibrium points, we find that they all unstable. Further, we perform the autonomous simulation for this all scenario using the same initial condition, i.e. $B(0) = 100, T_S(0) = 2.5, T_I(0) = 3, P_S(0) = 1, P_I(0) = 0.2$. The following is a picture of the simulation that has done in Figure 6.

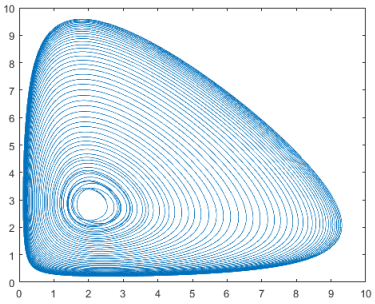

Figure 5. Phase portrait of Tilapia fish

Further, we continue our numerical experiment using three different initial conditions when $\beta_T = 0.01,0.02,0.03$. The initial condition gives in Table 6.

Table 6. Different initial condition

$B(0)$	$T_S(0)$	$T_I(0)$	$P_S(0)$	$P_I(0)$	Colour
100	2.5	3	1	0.2	Red
100	1.2	1.3	1.6	1.4	Blue
100	10	1	1	0.2	Black

Our numerical simulation give in Figure 6 (a), (b)

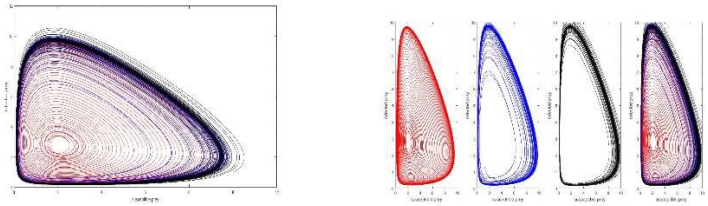

Figure 6 (a), (b). Numerical simulation of a system (1) with three different initial conditions when $\beta_T = 0.01,0.02,0.03$.

Our result in Figure 6 (a), (b) show a dependency of results with the initial condition. This result suggests the possibility of the appearance of chaos. The three dimension result of our previous simulation given in Figure 7 and Figure 8.

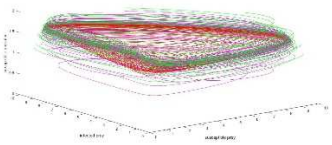

Figure 7. The three-dimensional phase of Tilapia and susceptible Pelecanidae

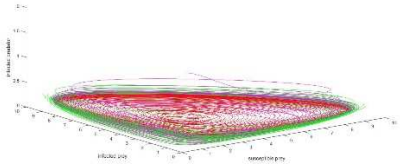

Figure 8. The three-dimensional phase of Tilapia and infected Pelecanidae

From the simulation that has done with several differential initial conditions, it can see that the results of the periodic solutions lead to different solutions. This indicates a Chaos phenomenon that occurs in the system.

5 Conclusions

In this work, we have constructed the mathematical model to describe an interaction between Tilapia as a prey, Pelecanidae as Predator, and Vibrio vulnivicus bacterium as the caused of disease in Tilapia. Our model describes how disease in Tilapia may transfer to Pelecanidae through predation. We construct the model as a five-dimensional ordinary differential equation. Our model show seven equilibrium points, with only three of them that might stable with or without any condition. From numerical simulations, we find that there should be a control on the spread of disease among Tilapia to avoid the extinction of Pelecanidae of Tilapia itself. For further research, the reader may continue the analysis of our model related to the possibility of the appearance of Chaos phenomena.

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References

[1] A. J. Lotka, "Analytical Note on Certain Rhythmic Relations in Organic Systems," *Proc. Natl. Acad. Sci.*, 1920, doi: 10.1073/pnas.6.7.410.
[2] V. Volterra, "Fluctuations in the abundance of a species considered mathematically," *Nature*. 1926, doi: 10.1038/118558a0.
[3] M. C. Anisiu, "Lotka, Volterra and their model," *Didact. Math.*, 2014.
[4] S. Triharyuni and D. Aldila, "A mathematical model of predator-prey interaction between seal-herring and steelhead trout," in *AIP Conference Proceedings*, 2017, doi: 10.1063/1.4991256.
[5] I. M. Sayekti, M. Malik, and D. Aldila, "One-prey two-predator model with prey harvesting in a food chain interaction," in *AIP Conference Proceedings*, 2017, doi: 10.1063/1.4991228.
[6] D. Aldila, A. J. Hutchinson, M. Woolway, N. Owen-Smith, and E. Soewono, "A mathematical model of black rhino translocation strategy," *J. Math. Fundam. Sci.*, 2015, doi: 10.5614/j.math.fund.sci.2015.47.1.8.
[7] Y. Xio and L. Chen, "Modelling and analysis of a predator-prey model with disease in the prey," *Math Biosci 2001*, vol. 171, pp. 59–82, 2001.

[8] K. P. Hadeler and H. I. Freedman, “Predator-prey populations with parasitic infection,” *J. Math. Biol.*, 1989, doi: 10.1007/BF00276947.
[9] E. Venturino, “The influence of diseases on Lotka-Volterra systems,” *Rocky Mt. J. Math.*, 1993, doi: 10.1216/rmjm/1181072471.
[10] E. Venturino, “Epidemics in predator-prey models: Disease among the prey. In: Arino O, Axelrod D, Kimmel M, Langlais M, editors. Mathematical population dynamics: analysis of heterogeneity.,” *Math. Popul. Dyn. Anal. Heterog.*, vol. 1, pp. 381–93, 1995.
[11] A. Putri and D. Aldila, “Prey-predator interaction model between herring and seals with phocine distemper virus (PDV) infection in seals,” in *AIP Conference Proceedings*, 2018, doi: 10.1063/1.5064236.
[12] R. M. Anderson and R. M. May, “The invasion, persistence and spread of infectious diseases within animal and plant communities.,” *Philosophical transactions of the Royal Society of London. Series B, Biological sciences*. 1986, doi: 10.1098/rstb.1986.0072.
[13] J. Chattopadhyay and N. Bairagi, “Pelicans at risk in Salton sea - An eco-epidemiological model,” *Ecol. Modell.*, 2001, doi: 10.1016/S0304-3800(00)00350-1.
[14] A. Morris and K. Lafferty, “Altered behavior of parasitized killifish increases susceptibility to predation by bird final hosts,” *Ecology*, 1996.