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# Application of Mathematical Models Two Predators and Infected Prey by Pesticide Control in Nilaparvata Lugens Spreading in Bantul Regency

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**Abstract**

In this paper, we develop a mathematical model to analyze interactions between planthopper pests as prey and *menochilus sexmaculatus* and *mirid ladybug* as two predators where prey is controlled by pesticides. The interaction between predator and prey is modeled using the Holling type II response function. The predator and prey growth are modeled using a logistic function. From this model, we obtain eight equilibrium points. The three of these equilibrium points are analyzed using linearization and locally asymptotically stable. We simulate this model using data to predict the dynamics of planthopper population and its predators. Simulation result shows that all of these populations will survive because they are influenced by pesticide control and predation rates.
**Keywords: c**ontrol of pest, predator-prey model, the Holling type II, the logistic function.

**Abstrak**

PPada penelitian ini, kami membangun model matematika untuk menganalisis interaksi antara hama wereng sebagai mangsa (prey) dan menochilus sexmaculatus dan mirid ladybug sebagai dua pemangsa (predator) dimana mangsa dikontrol oleh pestisida. Interaksi antara predator dan prey dimodelkan menggunakan fungsi respon Holling tipe II sedangkan pertumbuhan predator dan prey dimodelkan menggunakan fungsi logistik. Dari model tersebut diperoleh delapan titik ekuilibrium. Tiga titik ekuilibrium dari titik-titik equilibrium tersebut dianalisis menggunakan metode linierisasi dan bersifat stabil asimtotik lokal. Kemudian model ini diaplikasikan pada data. Untuk memudahkan interpretasi antara mangsa dan dua pemangsa dilakukan simulasi numerik untuk memprediksikan dinamika populasi wereng dan predatornya. Hasil simulasi menunjukkan bahwa semua populasi tersebut akan bertahan hidup karena dipengaruhi oleh kontrol pestisida dan tingkat pemangsaan.
**Kata Kunci**: kontrol pestisida, model predator-prey, Holling tipe II, fungsi logistik.

## 1. INTRODUCTION

In Indonesia, the planthopper is paddy pest that can harm the plants directly by sucking the leaf midrib and plant cell liquid, so the plants become dry and the buds are reduced. The planthopper can transfer viruses that cause the color of leaves and stems of paddy to become yellow, brown straw, and finally, all paddy plants become dry like scalded [1]. There were five hectares of paddy farming in Baros, Tirtohargo Village, Bantul Regency, Special Region of Yogyakarta which were exposed to the brown planthopper pests in the rainy season in February 2018. This causes crop failure due to the planthopper pests and the high humidity factor. Farmers estimated the losses up to hundreds of

millions of rupiah due to the average of production costs were Rp. 30 million - Rp. 35 million per hectare [2]. Meanwhile, based on the recapitulation of brown planthopper attacks recorded by the Bantul Agriculture and Forestry Service in early February, paddy plants that were attacked by plant hopper were 280 hectares with a threat of 40 hectares.

According to [3], one of the efforts to control the planthopper pests is the spraying of pesticides with the appropriate dose. The inappropriate dose will cause the various undesirable effects, such as environmental pollution, pests become resistant to various types of pesticides, and pests become quickly adaptable to environmental changes [4]. This effort is not effective to control the growth of pests. Therefore, we need a system to control it using predators/natural enemies [5]. It should be supported by the availability of the predator population, for example, is an insect. The predator population are maintained when the pest populations are low

There are several kinds of Interactions between individuals, one of them is predation. Predation is the relationship between predators and prey in the interaction of two populations, for example, predation brown paddy planthopper (Nilaparvata lugens Stål.) which are preyed by their natural predators, such as the Menochilus sexmaculatus beetle and mirid ladybug (Cyrtorhinus lividipennis). The predation can be preyed by more than one natural enemy [6].

Mathematical models can be an important tool to determine the dynamic process between predators and pests and analyze the spread of pests at a certain time. This model was first proposed by Lotka Volterra. It consists of the rate of change in predator populations and pest populations as prey. The Lotka-Volterra model can be developed to model the interaction between two predators and one prey. Alebraheem and Abu-Hasan [7] developed the model of two predators and one prey with assuming that the growth of predators and prey follows logistical growth and competition occurs between the two predators. Besides, [8] and [9] have also reduced predatory prey models with prey infected with pesticide control. The combination between the model in Alebraheem and Abu-Hasan [7] and model in [8] and [6] is interesting research. Therefore, in this paper, we model the interaction between two predators and prey-infected with pesticide to control the spread of brown plant hopper pest in Bantul district. The mathematical model is formulated in the differential equations by considering the transfer diagram which illustrates the dynamics for predators, planthopper pests, and the effect of pesticides on the paddy plant ecosystems. The system analysis will be carried out by determination of the existence of the equilibrium point, the stability of the equilibrium point stability and simulations. We use data on leafhoppers and natural enemies recorded by the Department of Agriculture, Food, Maritime Affairs and Fisheries of Bantul Regency.

## 2. THE PREDATOR PREY MODEL

The prey-predator model used to model the interaction between two predators, susceptible prey and infected prey assuming that the predator and prey growth follows the logistic function. The model is a system of non-linear differential equations that can be solved qualitatively by determining the stability of the equilibrium point. Furthermore, the dynamic behavior of the predatory prey model with two predators and infected prey can be determined through the analysis of the equilibrium point stability [10].

Let $X(t)$ be the number of individuals in the prey population is vulnerable at time $t$, $I(t)$ be the number of individuals in the prey population infected at time $t$, $Y(t)$ be the number of individuals in the first predator population at time $t$ and $Z(t)$ be the number of individuals in the second predator population at time $t$.

Assume that the predator population and the prey population are closed, i.e. no predators and

prey is migrating. The prey-predator model consists of two predators, susceptible prey and infected prey. There is no competition between two predators to get the prey. The growth of predators and prey follow the logistic function. Also assume that if there is no interaction between predators and prey, then prey growth follows the logistic model that is with a limited carrying capacity of $K$ and the intrinsic growth rate $r$, as a result, the prey will increase at a rate $rX\left(1-\frac{X}{K}\right)$. Then changing the population from $X$ to $I$ reduces the growth rate of $X$. This is influenced by the rate of interaction between susceptible and infected pests and the chance of transmitting infected pests to vulnerable pests $\beta$ and the population density of $X$ and infected pests produces an equation $\beta XI$. Predator predation in prey class uses the Holling type II response, namely $g(x)$. When there is an interaction between the first predator and the prey represented as $g_1(x)$, the growth of prey will be reduced by $g_1(x)Y$, i.e. the multiplication rate between the Holling type II response function and the predator population Y, that is $g_1(x)=\frac{\rho_1 X}{a+X}$, the predation rate by the first predator, thus $g_1(x)Y=\frac{\rho_1 XY}{(a+X)}$. When there is an interaction between the second predator and the prey $g_2(x)$, the growth of the prey will decrease by $g_2(x)Z$, i.e. the multiplication rate between the Holling type II response function and the predator population $Z$, obtained by $g_2(x)=\frac{\rho_2 X}{a+X}$. The $\rho_2$ the predation rate by the second predator such that $g_2(x)Z=\frac{\rho_2 XZ}{(a+X)}$.

The number of deaths of $X$ is caused by the pesticide $u$, i.e $uX$. Thus, the rate of change in the number of preys susceptible to time can be expressed by

$$\frac{dX}{dt}=rX\left(1-\frac{X}{K}\right)-\beta XI-\frac{\rho_1 XY}{a+X}-\frac{\rho_2 XZ}{a+X}-uX. \quad (1)$$

The prey class is infected, during the time interval for $t$, the rate of change is influenced by the migration of population $X$ to $I$, i.e $\beta XI$. The number of natural deaths $I$ is $mI$ the multiplication of the natural rate of death of pests m with the density of infected pests $I$. Then the number of deaths $I$ caused by pesticides $u$, namely $uI$. Then the reduced population of pests infected by predator $Y$ predation in infected pests I class in this predation uses the Holling type 1, $g_1(I)Y$, predation process which is the rate of multiplication between the function of the Holling type 1 response with predator Y density while the reduced population of pests infected due to predator $Y$ predation in the class of infected pests I in predation this predation process uses a Holling type 1 response $g_2(I)Z$ which is the rate of multiplication between the Holling type 1 response function with a predator density Z. with $g_1(I)=\mu_1 I$ and $g_2(I)=\mu_2 I$. From this description, obtained the equation of the rate of change in the population of infected pests into

$$\frac{dI}{dt}=\beta XI-\mu_1 IY-\mu_2 IZ-(m+u)I. \quad (2)$$

Then, if there are no pests, there will be a decrease in the population of the first predator with a natural mortality rate of p, but if there is a susceptible pest, there will be an interaction between the

first predator and the susceptible prey $\frac{\gamma_1\rho_1 XY}{a+X}$. Whereas if there is a susceptible pest, there will be an interaction between the first predator and the infected prey $\gamma_2\mu_1 IY$. A large number of $Y$ deaths are caused by the pesticide $u$ as $uY$. Thus, the rate of change in the number of first predators by the time can be expressed by

$$\frac{dY}{dt}=\frac{\gamma_1\rho_1 XY}{a+X}+\gamma_2\mu_1 IY-(p+u)Y. \tag{3}$$

Then, if there are no pests, there will be a decrease in the population of the second predator with a natural mortality rate of q, but if there is a susceptible pest, there will be an interaction between the second predator and the vulnerable prey by $\frac{\theta_1\rho_2 XZ}{a+X}$. Whereas if there is an infected pest, an interaction occurs between the second predator and the infected prey $\theta_2\mu_2 IZ$. The number of $Z$ deaths is caused by the pesticide $u$ as $uZ$. Thus, the rate of change in the number of second predators with respect to time can be expressed by

$$\frac{dZ}{dt}=\frac{\theta_1\rho_2 XZ}{a+X}+\theta_2\mu_2 IZ-(q+u)Z. \tag{4}$$

Based on Equations (1), (2), (3), and (4) obtained a mathematical model of two predators and prey infected with pesticide control in the form of a non-linear differential equation system as follows:

$$\begin{aligned}
\frac{dX}{dt}&=rX\left(1-\frac{X}{K}\right)-\beta XI-\frac{\rho_1 XY}{a+X}-\frac{\rho_2 XZ}{a+X}-uX\\
\frac{dI}{dt}&=\beta XI-\mu_1 IY-\mu_2 IZ-(m+u)I\\
\frac{dY}{dt}&=\frac{\gamma_1\rho_1 XY}{a+X}+\gamma_2\mu_1 IY-(p+u)Y\\
\frac{dZ}{dt}&=\frac{\theta_1\rho_2 XZ}{a+X}+\theta_2\mu_2 IZ-(q+u)Z
\end{aligned} \tag{5}$$

with the initial value: $X(0)=X_0$, $I(0)=I_0$, $\mathrm{Y}(0)=Y_0$, and $\mathrm{Z}(0)=Z_0$.

The mathematical models of two predators and prey were infected with pesticide control in the form of a nonlinear differential equation system. Qualitative Completion of the system (5) by looking at the behavior of the system around the equilibrium point. The equilibrium point for the two predators and prey mathematical models infected with pesticide control in the system (5) is obtained if $\frac{dX}{dt}=0$, $\frac{dI}{dt}=0$, $\frac{dY}{dt}=0$ and $\frac{dZ}{dt}=0$. If $\frac{dX}{dt}=0$, then

$$rX\left(1-\frac{X}{K}\right)-\beta XI-\frac{\rho_1 XY}{a+X}-\frac{\rho_2 XZ}{a+X}-uX=0\Leftrightarrow X\left[r\left(1-\frac{X}{K}\right)-\beta I-\frac{\rho_1 Y}{a+X}-\frac{\rho_2 Z}{a+X}-u\right]=0.$$

So, we obtain

$$X=0 \tag{6}$$

or

$$r\left(1-\frac{X}{K}\right)-\beta I-\frac{\rho_1 Y}{a+X}-\frac{\rho_2 Z}{a+X}-u=0. \tag{7}$$

Furthermore, if $\frac{dI}{dt}=0$, then

$$\beta XI-\mu_1 IY-\mu_2 IZ-(m+u)I=0 \Leftrightarrow I\left[\beta X-\mu_1 Y-\mu_2 Z-(m+u)\right]=0.$$

We obtain

$$I=0, \tag{8}$$

or

$$\beta X-\mu_1 Y-\mu_2 Z-(m+u)=0. \tag{9}$$

Furthermore, if $\frac{dY}{dt}=0$, then

$$\frac{\gamma_1\rho_1 XY}{a+X}+\gamma_2\mu_1 IY-(p+u)Y=0 \Leftrightarrow Y\left[\frac{\gamma_1\rho_1 X}{a+X}+\gamma_2\mu_1 I-(p+u)\right]=0.$$

We obtain

$$Y=0 \tag{10}$$

or

$$\frac{\gamma_1\rho_1 X}{a+X}+\gamma_2\mu_1 I-(p+u)=0 \tag{11}$$

Furthermore, if $\frac{dZ}{dt}=0$, then

$$\frac{\theta_1\rho_2 XZ}{a+X}+\theta_2\mu_2 IZ-(q+u)Z=0 \Leftrightarrow Z\left[\frac{\theta_1\rho_2 X}{a+X}+\theta_2\mu_2 I-(q+u)\right]=0.$$

We obtain

$$Z=0 \tag{12}$$

or

$$\frac{\theta_1\rho_2 X}{a+X}+\theta_2\mu_2 I-(q+u)=0 \tag{13}$$

Based on the description, from Equations (6), (8), (10), and (12) the equilibrium point is $TE_1=(0,0,0,0)$. Then from Equations (7), (8), (10), and (12) the equilibrium point is $TE_2=(X^*,0,0,0)$. Then Equation (8), (10), and (12) are substituted into Equation (7),

$$r\left(1-\frac{X}{K}\right)-\beta(0)-\frac{\rho_1(0)}{a+X}-\frac{\rho_2(0)}{a+X}-u=0 \Leftrightarrow r\left(1-\frac{X}{K}\right)-u=0 \Leftrightarrow X=\frac{Kr-Ku}{r}.$$

So, we obtain

$$TE_2 = \left( \frac{Kr - Ku}{r}, 0, 0, 0 \right).$$

Next, from Equations (7), (8), (10), and (12) the equilibrium point is $TE_3 = \left( X^*, I^*, 0, 0 \right)$. Substitute equations (10) and (12) into equations (7) and (8):

$$r\left(1 - \frac{X}{K}\right) - \beta I - \frac{\rho_1(0)}{a+X} - \frac{\rho_2(0)}{a+X} - u = 0 \Leftrightarrow r\left(1 - \frac{X}{K}\right) - \beta I - u = 0\,, \tag{13}$$

$$\beta X - \mu_1(0) - \mu_2(0) - (m+u) = 0 \Leftrightarrow \beta X - (m+u) = 0 \Leftrightarrow X = \frac{(m+u)}{\beta}. \tag{14}$$

Substitute equation (14) into equation (13), we obtain $r\left(1 - \frac{\left[\frac{(m+u)}{\beta}\right]}{K}\right) - \beta I - u = 0,$

$I = \frac{r\left(1 - \frac{(m+u)}{\beta K}\right) - u}{\beta}$, and $TE_3 = \left( \frac{(m+u)}{\beta}, \frac{r\left(1 - \frac{(m+u)}{\beta K}\right) - u}{\beta}, 0, 0 \right)$.

Next, from Equations (7), (8), (11), and (12) an equilibrium point is $TE_4 = \left( X^*, 0, Y^*, 0 \right)$. Then, from Equations (7), (8), (10), and (13) an equilibrium point is $TE_5 = \left( X^*, 0, 0, Z^* \right)$. Then, from Equations (7), (8), (11), and (12) an equilibrium point is $TE_6 = \left( X^*, I^*, Y^*, 0 \right)$. Then, from Equations (7), (8), (10), and (13) an equilibrium point is $TE_7 = \left( X^*, I^*, 0, Z^* \right)$. Then, from Equations (7), (8), (11), and (13) an equilibrium point is $TE_8 = \left( X^*, I^*, Y^*, Z^* \right)$.

**Theorem 1.** If $r < u$, then the equilibrium point $TE_1 = (0,0,0,0)$ locally asymptotically stable.

**Proof:** Stability analysis of $TE_1$ can be determined by analysis of eigenvalue of the Jacobian matrix of linearized system (5) in around of equilibrium points $TE_1$. Then $TE_1$ is substituted into equations:

$$\begin{aligned}
&f_{1X}(TE_1) = r - u, \quad f_{1I}(TE_1) = 0, \quad f_{1Y}(TE_1) = 0, \quad f_{1Z}(TE_1) = 0,\\
&f_{2X}(TE_1) = 0, \quad f_{2I}(TE_1) = -m - u, \quad f_{2Y}(TE_1) = 0, \quad f_{2Z}(TE_1) = 0,\\
&f_{3X}(TE_1) = 0, \quad f_{3I}(TE_1) = 0, \quad f_{3Y}(TE_1) = -p - u, \quad f_{3Z}(TE_1) = 0,\\
&f_{4X}(TE_1) = 0, \quad f_{4I}(TE_1) = 0, \quad f_{4Y}(TE_1) = 0, \quad f_{4Z}(TE_1) = -q - u,
\end{aligned}$$

Then we obtained $J(\mathrm{f}(TE_1))$ i.e:

$$J(\mathrm{f}(TE_1)) = \begin{pmatrix} r-u & 0 & 0 & 0 \\ 0 & -m-u & 0 & 0 \\ 0 & 0 & -p-u & 0 \\ 0 & 0 & 0 & -q-u \end{pmatrix}$$

Polynomial characteristics of $J(\mathrm{f}(TE_1))$ is

$$\mathrm{P}(\lambda) = \det\left(\lambda I_4 - J(\mathrm{f}(TE_1))\right)$$

$$= \left| \lambda \begin{pmatrix} 1 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 \\ 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 1 \end{pmatrix} - \begin{pmatrix} r-u & 0 & 0 & 0 \\ 0 & -m-u & 0 & 0 \\ 0 & 0 & -p-u & 0 \\ 0 & 0 & 0 & -q-u \end{pmatrix} \right|$$

$$= \begin{vmatrix} \lambda-(r-u) & 0 & 0 & 0 \\ 0 & \lambda+(m+u) & 0 & 0 \\ 0 & 0 & \lambda+(p+u) & 0 \\ 0 & 0 & 0 & \lambda+(q+u) \end{vmatrix}$$

$$= \left(\lambda-(r-u)\right)\left(\lambda+(m+u)\right)\left(\lambda+(p+u)\right)\left(\lambda+(q+u)\right)$$

where $I_4$ is an identity $4\times 4$ matrix. Then it is obtained characteristic equation

$$P(\lambda) = \det\left(\lambda I_4 - J(\mathrm{f}(TE_1))\right) = 0$$

$$\Leftrightarrow \left(\lambda-(r-u)\right)\left(\lambda+(m+u)\right)\left(\lambda+(p+u)\right)\left(\lambda+(q+u)\right) = 0$$

$\lambda_1 = r-u,\ \lambda_2 = -(m+u),\ \lambda_3 = -(p+u),$ and $\lambda_4 = -(q+u)$. because $m, p, q, u > 0$, so $\lambda_2, \lambda_3, \lambda_4 < 0,$. $TE_1$ locally asymptotically stable if $\lambda_1 < 0,\ r-u<0 \Leftrightarrow r<u$.

**Theorem 2.** If $u < r$ and $f_{22}, f_{33}, f_{44} < 0$ then the equilibrium point $TE_2 = \left(\frac{Kr-Ku}{r}, 0, 0, 0\right)$ locally asymptotically stable, where $f_{22} = \beta\left(\frac{Kr-Ku}{r}\right) - m - u$, $f_{33} = \frac{\gamma_1\rho_1\left(\frac{Kr-Ku}{r}\right)}{a+\left(\frac{Kr-Ku}{r}\right)} - p - u$, and

$$f_{44} = \frac{\theta_1\rho_2\left(\frac{Kr-Ku}{r}\right)}{a+\left(\frac{Kr-Ku}{r}\right)} - q - u.$$

**Theorem 3.** If $g_{44}<0,\ g_{33}<0,\ \ g_{11}+g_{22}<0$ and $g_{11}g_{22}-g_{12}g_{21}>0$ then the equilibrium point $TE_3=\left(X^*,I^*,0,0\right)$ locally asymptotically stable, where $X^*=\frac{(m+u)}{\beta}$, $I^*=\frac{r\left(1-\frac{(m+u)}{\beta K}\right)-u}{\beta}$, $g_{11}=r-\frac{2rX^*}{K}-\beta I^*-u$, $g_{12}=-(m+u)$, $g_{21}=\beta I^*$, $g_{22}=\beta X^*-(m+u)$, $g_{33}=\frac{\gamma_1\rho_1X^*}{a+X^*}+\gamma_2\mu_1I^*-(p+u)$ and $g_{44}=\frac{\theta_1\rho_2X^*}{a+X^*}+\theta_2\mu_2I^*-(q+u)$.

## 3. SIMULATION

In this section, the simulation of an equilibrium point is performed to find out the dynamic behavior of the completion of the system (5) over a long period around the equilibrium point. In this simulation, the brown planthopper is prey in the model, while menochilus sexmaculatus was the first predator and mirid ladybug as the second predator. To simulate the system (5), we use parameter values based on data from the agriculture, food, fisheries and marine services in the districts of Bantul, [6], [1], and [9], i.e. $r=1, K=10, m=0.1, p=0.3, q=0.2, u=0.5, \beta=0.7, \rho_1=0.5,\ \rho_2=0.45, \gamma_1=0.15, \gamma_2=0.2, \theta_1=0.25, \theta_2=0.1, \mu_1=0.2, \mu_2=0.1, X(0)=3, I(0)=2, Y(0)=2,$ and $Z(0)=2$.

Based on data and [6], we obtained several parameters as follows: it will meet the conditions in the theorem. As a result, predators will become extinct while prey will survive and as shown in figure 1.

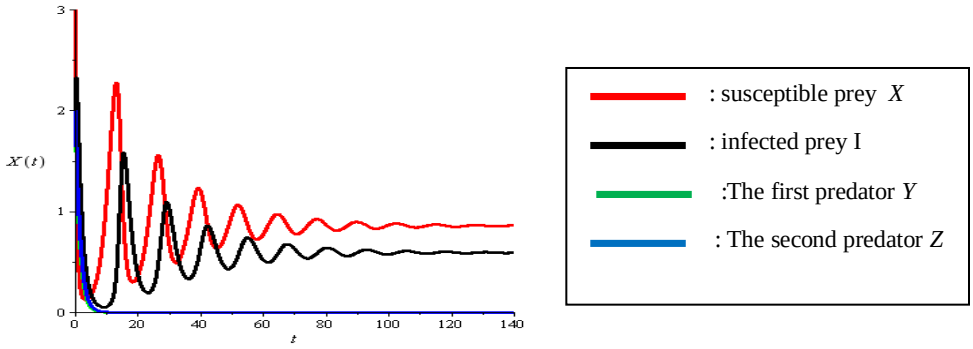


**Figure 1.** The behavior of time series in System (5) uses data and $u=0.5$.

From Figure 1, with given initial value, it can be seen that at a certain time $t$, the solution converges to TE3. In a sense when given initial values, at the beginning of the population of vulnerable and infected prey down then rose slightly to a certain value and the population of both predators fell to near extinction. If we change $u=0.01$, we get

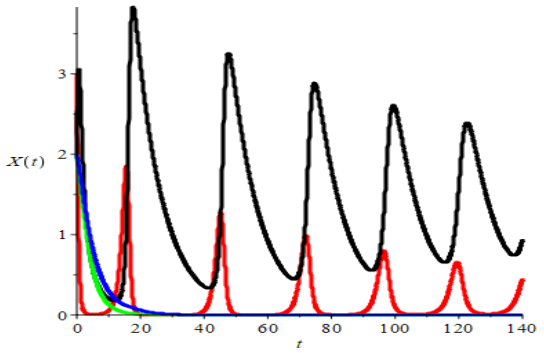


**Figure 2.** The behavior of time series in System (5) uses data and $u = 0.01$.

In Figure 2, given the initial value, it can be seen that at a certain time $t$ the population of susceptible prey and infected prey unstable and both of the predators die. But if we change $u = 1$, we get

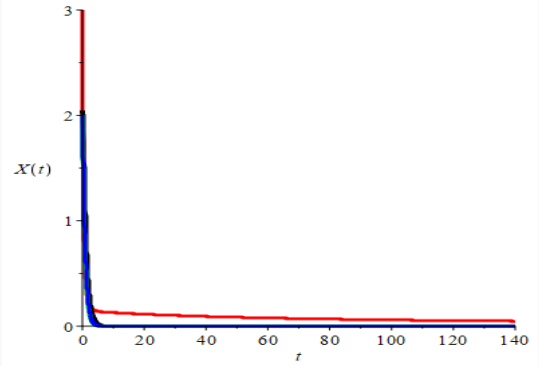


**Figure 3.** The behavior of time series in System (5) uses data and $u = 1$.

From Figure 3, given the initial value, it can be seen that at a certain time $t$ that the solution converges to TE1. Because all of predator and prey dies.

## 4. CONCLUSION

In this study, we derive a mathematical model to describe the interaction for two predators and prey was infected with pesticide control. System (5) has eight equilibrium points. The stability of the equilibrium point System (5) is analyzed only local stability by the linearization method. If the intrinsic growth rate of pests is less than the death rate due to the administration of pesticides then all predators and prey will become extinct and be locally asymptotically stable. The intrinsic growth rate of pests is more than the death rate due to the administration of pesticides so that only vulnerable pests will remain alive while others will become extinct and are asymptotically stable. All of these populations will survive because they are influenced by pesticide control and predation rates.

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