Dynamics of the Transmission of Infectious Disease in a Pre-Predator Ecosystem

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***Abstract*-This paper is to study a mathematical model of the spread of infectious disease in a Predator-Prey Ecosystem. We discuss in detail the stability of the disease free and the endemic equilibrium by using the Routh-Hurwitz conditions to linearise the non-linear systems. We identify the threshold parameter R_0 (basic reproduction number) and that the disease free equilibrium always exists. Numerical Simulations are carried out .Implications of our analytical and numerical findings are discussed critically.**

Keywords: Routh-Hurwitz conditions, basic reproduction number, disease free equilibrium, infectious disease, Predator-Prey Ecosystem.

I. INTRODUCTION

When species interact, the population dynamics of each species is affected. In general there is a whole web of interacting species, called a trophic web, which makes for structurally complex communities. We consider here, systems involving two or more species, concentrating particularly on two species systems. There are three main types of interaction.(i) If the growth rate of one population is decreased and the other increased the populations are in a predator-prey situation.(ii) If the growth rate of each population is decreased, then it is competition.(iii) If each population's growth rate is enhanced, then it is called mutualism or symbiosis[8].Some mathematical models have been developed in this area. In 1926, Volterra [13] first proposed a simple model for the predation of one species by another to explain the oscillatory levels of certain fish catches in the Adriatic. This model was based on four assumptions. Firstly, the prey grows unboundedly in a Malthusian way in the absence of any predation. Secondly, the effect of the predation is to reduce the prey's per capita growth rate by a term proportional to the prey and the predator populations. Thirdly, in the absence of any prey for sustenance the predator's death rate results in exponential decay. Fourthly, the prey's contribution to the predator's growth is proportional to the available prey as well as the size of the predator population. The model is

$\frac{dN}{dt} = N(a - bp)$ and $\frac{dP}{dt} = P(cN - d)$ where

N is the prey population and P is the predator

population. This model also called Lokta-Volterra model was analyzed. Murray [8] modified the Lokta-Volterra model by changing of the assumptions made made by Volterra .In the model he obtained:

$$\frac{dN_1}{dt} = r_1 N_1 \left[1 - \frac{N_1}{K_1} - b_{12} \frac{N_2}{K_2} \right]$$

$$\frac{dN_2}{dt} = r_2 N_2 \left[1 - \frac{N_2}{K_2} - b_{21} \frac{N_1}{K_2} \right]$$ Where

$r_1, k_1, r_2, k_2, b_{12}, b_{21}$ are all positive constants. This model was analyzed and the conditions for stability established. Bedington et al [2] presented some results on the dynamic complexity of coupled predator-prey systems. Dunbar [3,4] studied in detail a modified Lokta-Volterra system with logistic growth of the prey and with both predator and prey dispersing by diffusion."Predator-Prey model are arguably the building blocks of the bio and ecosystems as biomasses are grown out of their resources to sustain their struggle for their very existence. Depending on their specific settings of applications, they can take the forms of resource-consumer, plant-herbivore, parasitic-host, tumor cells (virus)-immune system, susceptible-infectious interactions, etc. They deal with the general loss-win interactions and hence may have applications outside of ecosystems. When seemingly competitive interactions are carefully examined, they are often in fact some facts of predator-prey interaction in disguise"[5].

Another approach to modeling the interaction between prey and predators was developed to account as well for organisms (such as bacteria) taking up nutrients and this is called Jacob-Mond Model. This model was discovered independently in the several diverse applications. It is akin to the Haldane-Briggs Model and Michaelis-Menten Model in Biochemistry the Jacob-Mond Model in microbial ecology and the Beaverton-Holt model in fisheries. It serves as one of the important building blocks in studies of complex biochemical reactions and in ecology [12]. B.Dubey and R.K Upadhay, in their paper, a mathematical model is proposed and analyzed to study the dynamics of one-prey two-predator system with ratio-dependent predators' growth rate. Criteria for local stability, instability and global stability of the nonnegative equilibria are obtained. The permanent co-existence of the three species is also discussed. Finally, computer simulations are performed to investigate the dynamics of the system. S.Pathak et al in his work, we discovered that over the past hundred years, mathematics has been used to understand and predict the spread of disease, relating important public-health questions to basic transmission parameters. From prehistory to the present day, diseases have been a source of fear and superstition. A comprehensive picture of disease dynamics requires a variety of mathematical tools, from model creation to solving differential equations to statistical analysis. Although mathematics has been so far done quite well in dealing with epidemiology but there is no denying that there are certain factors which still lack proper mathematization.

Almost all mathematical models of disease start from the base premise: that the population can be subdivided into a set of distinct classes dependent upon their experience with respect to the disease. One line of investigation classifies individuals as one susceptible, infectious or recovered. Such a model is termed as an SIR model. Disease transmission is a dynamical process driven by the interaction between the susceptible and the infective. Many models of epidemiology are based on the so called "mass action" assumption for transmission. In this work, we

have considered the case of the mathematical modeling of the spread of disease (infection) in Predator-Prey ecosystems. This paper is organized as follows. In the next section, we present the model assumptions. In the third section, we present the model equations and described various parameters and terms in the model. In the fourth section, we carry out the qualitative analysis of the model. Stability criteria's for the disease free equilibrium and the endemic equilibrium are derived. Basic reproductive number and Bifurcation theory were also discussed. The fifth section presents an illustrative example for the model. In the sixth section, we present different computer simulations of the system. In the last section, the biological significance of our analytical and numerical findings are discussed.

II. THE MODEL ASSUMPTIONS

The following examines the evolution of a predator-prey system, after an infectious disease has been introduced into the colony. We assume the following:

(a) The disease is benign to the prey; that is, the prey are carriers. The relative birth rate for Infected prey remains the same as that of the healthy susceptible prey.

(b) The disease is debilitating and ultimately fatal for the predators. Once a predator is Infected, it can be assumed to be dead. We will therefore consider only one population of predators, those that are susceptible.

(c) The disease is spread among the prey by contact, and the rate of infection is proportional to the infected and the susceptible population.

(d) The predators make no distinction between susceptible and infected members of the Prey population.

(e) The predator contract the disease by consuming the prey. The rate of predator infection is proportional to the product of infected prey and susceptible predators.

III. THE MODEL EQUATIONS

The model we analyzed in this paper is considered under the framework of the following nonlinear ordinary differential equations:

$$\frac{dR_2}{dt} = -a_1R_2 + b_1R_2R_{1,s} - c_1R_2R_{1,i}$$

$$\frac{dR_{1s}}{dt} = a_2R_{1,s} - b_2R_2R_{1,s} - c_2R_{1,s}R_{1,i} + d_2R_{1,i}$$

$$\frac{dR_{1i}}{dt} = a_2R_{1,i} - b_2R_2R_{1,i} + c_2R_{1,s}R_{1,i} - d_2R_{1,i}$$

(3.1)

At this points we will observe a qualitative change when a smooth small change is made to the parameter values(bifurcation parameters).

TABLE 1

Description of variables for transmission model

Variables	Descriptions
R_{1i}	Number of Infected Prey at time t
$R_{1,s}$	Number of Susceptible Prey at time t
R_2	Number of healthy Susceptible Predators at time t

TABLE 2

Description of constants for transmission model

Constant	Descriptions
a_1	natural death of the Healthy Susceptible Predator
a_2	per capita birth rate of Susceptible Prey (per time) and Infected Prey
b_1	number of contact between Healthy Susceptible Prey and Healthy Predator.
b_2	number of contact between Healthy Susceptible Prey and Healthy Predator for preying.
c_1	number of contact between Healthy Susceptible Predator and Infected Prey
d_1	rate at which infected Prey (carriers) are removed.

IV MODEL ANALYSIS

$\frac{dR_2}{dt}$= $-a_1R_2 + b_1R_2R_{1,s} - c_1R_2R_{1,i}$

$\frac{dR_{1s}}{dt}$= $a_2R_{1,s} - b_2R_2R_{1,s} - c_2R_{1,s}R_{1,i} + d_2R_{1,i}$

$\frac{dR_{1i}}{dt}$= $a_2R_{1,i} - b_2R_2R_{1,i} + c_2R_{1,s}R_{1,i} - d_2R_{1,i}$

From (3.1)

The equilibrium are obtained by setting the right-hand side of system (3.1) equal to zero, giving the following:

Solution

$-a_1R_2 + b_1R_2R_{1,s} - c_1R_2R_{1,i} = 0$

$a_2R_{1,s} - b_2R_2R_{1,s} - c_2R_{1,s}R_{1,i} + d_2R_{1,i}=0$

$a_2R_{1,i} - b_2R_2R_{1,i} + c_2R_{1,s}R_{1,i} - d_2R_{1,i} = 0$

(4.1)

The system in (3.1) has two equilibrium solutions

A disease-free equilibrium at (0, 0, 0) = $(R_2^*,R_{1,s}^*,R_{1,i}^*)$

An endemic equilibrium at $(\frac{a_2}{b_2},\frac{d_2}{c_2},\frac{-a_1c_2+b_1d_2}{c_1c_2})$= $(R_2^*,R_{1,s}^*,R_{1,i}^*)$

We determine the stability of the equilibrium points by computing the Jacobian Matrix of the system (3.1) at each equilibrium point

$$J(R_2,R_{1,s},R_{1,i}) = \begin{pmatrix} \frac{\partial f}{\partial R_2} & \frac{\partial f}{\partial R_{1,s}} & \frac{\partial f}{\partial R_{1,i}} \\ \frac{\partial g}{\partial R_2} & \frac{\partial g}{\partial R_{1,s}} & \frac{\partial g}{\partial R_{1,i}} \\ \frac{\partial h}{\partial R_2} & \frac{\partial h}{\partial R_{1,s}} & \frac{\partial h}{\partial R_{1,i}} \end{pmatrix}$$

$$J(R_2,R_{1,s},R_{1,i}) = \begin{pmatrix} -a_1+b_1R_{1,s}-c_1R_{1,i} & b_1R_2 & -c_1R_2 \\ -b_2R_{1,s} & a_2-b_2R_2-c_2R_{1,i} & -c_2R_{1,s}+d_2 \\ -b_2R_{1,i} & c_2R_{1,i} & a_2-b_2R_2+c_2R_{1,s}-d_2 \end{pmatrix}$$

At the equilibrium point $(R_2^*,R_{1,s}^*,R_{1,i}^*)$ = (0, 0, 0) the Jacobian is given by

$$J(0,0,0) = \begin{pmatrix} -a_1 & 0 & 0 \\ 0 & a_2 & d_2 \\ 0 & 0 & a_2-d_2 \end{pmatrix}$$

Trace J = $-a_1 - d_2$

Since a_1 *and* d_2 *are positive quantities* $Trace\ J <$ 0

Det (J) = $a_1a_2d_2 - a_1a_2^2$

Theorem 1.The nature of the equilibrium point (DFE):

Then the equilibrium point (DFE) (0,0,0) is

(i) Asymptotically stable if $-a_1 - d_2$<0 and $a_1a_2d_2 - a_1a_2^2$>0(ii) stable, but not asymptotically stable if $-a_1 - d_2$= 0 and $a_1a_2d_2 - a_1a_2^2$> 0 (iii) $-a_1 - d_2$> 0 or $a_1a_2d_2 - a_1a_2^2$ <0. (iv)stable node if

$-a_1 < 0, a_2 < 0 \; and \; a_1 - d_2 <$

$0 \; (v) unstable \; node \; if \; -a_1 > 0, a_2 >$

$0 \; and \; a_1 - d_2 > 0$ (vi) saddle point if $-a_1 <$

$0, a_2 < 0 \; or \; a_1 - d_2 > 0 \; , -a_1 > 0, a_2 >$

$0 \; and \; a_1 - d_2 < 0, -a_1 > 0, a_2 < 0 \; and \; a_1 - d_2 >$

0.(vii) stable focus if $-a_1, a_2 or \; a_1 - d_2$ are complex conjugate, not pure imaginary, and have negative real parts.(viii) unstable focus if $-a_1, a_2 or \; a_1 - d_2$ are complex conjugate, not pure imaginary, and have positive real parts.

At the Endemic Equilibrium point (EDE) the non zero equilibrium point $(\frac{a_2}{b_2}, \frac{d_2}{c_2}, \frac{b_1d_2 - a_1c_2}{c_1c_2})$

$J\left(\frac{a_2}{b_2}, \frac{d_2}{c_2}, \frac{b_1d_2 - a_1c_2}{c_1c_2}\right)$ = Therefore Trace (J) = $\frac{a_1c_2 - b_1d_2}{c_1}$

$+ d_2$ and

Det (J) =($\frac{a_2b_1b_2d_2^2}{c_1}$

$- \frac{c_1a_2}{b_2}\left(\frac{a_1b_2d_2c_2 - b_2b_1d_2^2}{c_2^2} - \frac{(a_1b_2c_2 - b_1d_2b_2)(a_1c_2 - b_1d_2)}{c_1^2c_2}\right)$

$Let \Delta = T^2 - 4d$

□

*Theorem 2***.** The nature of the equilibrium point (EDE):

Then the equilibrium point (EDE) $(\frac{a_2}{b_2}, \frac{d_2}{c_2}, \frac{b_1d_2 - a_1c_2}{c_1c_2})$ is

asymptotically stable if $\frac{a_1c_2 - b_1d_2}{c_1} + d_2$ <0 and

$\frac{a_2b_1b_2d_2^2}{c_1} - \frac{c_1a_2}{b_2}\left(\frac{a_1b_2d_2c_2 - b_2b_1d_2^2}{c_2^2} - \frac{(a_1b_2c_2 - b_1d_2b_2)(a_1c_2 - b_1d_2)}{c_1^2c_2}\right)$

>0(ii) stable, but not asymptotically stable if

$\frac{a_1c_2 - b_1d_2}{c_1} + d_2 = 0$ and $\frac{a_2b_1b_2d_2^2}{c_1} - \frac{c_1a_2}{b_2}$

$\left(\frac{a_1b_2d_2c_2 - b_2b_1d_2^2}{c_2^2} - \frac{(a_1b_2c_2 - b_1d_2b_2)(a_1c_2 - b_1d_2)}{c_1^2c_2}\right)$ > 0 (iii)

$\frac{a_1c_2 - b_1d_2}{c_1} + d_2$ > 0 or $\frac{a_2b_1b_2d_2^2}{c_1} - \frac{c_1a_2}{b_2}$

$\left(\frac{a_1b_2d_2c_2 - b_2b_1d_2^2}{c_2^2} - \frac{(a_1b_2c_2 - b_1d_2b_2)(a_1c_2 - b_1d_2)}{c_1^2c_2}\right)$<0. (iv)

node if $\frac{a_2b_1b_2d_2^2}{c_1} - \frac{c_1a_2}{b_2}$

$\left(\frac{a_1b_2d_2c_2 - b_2b_1d_2^2}{c_2^2} - \frac{(a_1b_2c_2 - b_1d_2b_2)(a_1c_2 - b_1d_2)}{c_1^2c_2}\right) > 0$ and

$(\frac{a_1c_2 - b_1d_2}{c_1} + d_2)^2 - 4(\frac{a_2b_1b_2d_2^2}{c_1} - \frac{c_1a_2}{b_2}$

$\left(\frac{a_1b_2d_2c_2 - b_2b_1d_2^2}{c_2^2} - \frac{(a_1b_2c_2 - b_1d_2b_2)(a_1c_2 - b_1d_2)}{c_1^2c_2}\right) \geq 0$ (v)

Spiral point if $(\frac{a_1c_2 - b_1d_2}{c_1} + d_2)^2 - 4(\frac{a_2b_1b_2d_2^2}{c_1} - \frac{c_1a_2}{b_2}$

$\left(\frac{a_1b_2d_2c_2 - b_2b_1d_2^2}{c_2^2} - \frac{(a_1b_2c_2 - b_1d_2b_2)(a_1c_2 - b_1d_2)}{c_1^2c_2}\right) \geq 0$

$\frac{a_1c_2 - b_1d_2}{c_1} + d_2 \neq 0$

□

A. The Basic Reproduction Number in a Nutshell

The Basic Reproduction number R_0 is defined as the expected number of secondary case produced by a single (typical) infection in a completely susceptible population. R_0 is a dimensionless number and not a rate. It has units of time^{-1}.In this work, we define our Basic reproduction number as follows

$$R_0 = \frac{c_2}{d_2} = \frac{1}{S^*}$$

If R_0 < 1,the infection(disease) dies out, on the other hand, If R_0 >1,the infection (disease) spreads in the population.

*Theorem 3***.** From the system (3.1) it follows that

(i) If R_0 < 1,then there is no positive equilibrium(i.e. the disease or infection dies out)

(ii) If $R_0 > 1$, then there is a unique positive equilibrium (R^*_2, $R^*_{1,s}$, $R^*_{1,i}$) of the system (3.1)

$$R_2^* = \frac{a_2}{b_2}, R_{1,s}^* = \frac{d_2}{c_2}, R_{1,i}^* = \frac{-a_1c_2 + b_1d_2}{c_1c_2}$$

□

B. Bifurcation Theory

Bifurcation generally refers to something splitting apart. With regard to differential equations or systems involving a parameter. It refers to abrupt changes in the character of the solutions as the parameter is changed continuously. It is the mathematical study of changes in the qualitative or topological structure of a given family, such as the integral curves of a family of vector fields, and the solutions of a family of differential equations. Most commonly applied to the mathematical study of dynamical systems, a bifurcation occurs when a small smooth change made to the parameter values (the bifurcation parameters) of a system causes a sudden qualitative or topological change in its behavior. Bifurcation occurs in both continuous systems (described by ODEs, DDEs, and PDEs) and discrete systems. In this work, the bifurcation parameter values are

$$R_2^* = \frac{a_2}{b_2}, R_{1,s}^* = \frac{d_2}{c_2}, R_{1,i}^* = \frac{-a_1c_2 + b_1d_2}{c_1c_2}$$

IV. ILLUSTRATIVE EXAMPLE

Assuming that the variables R_2 ,$R_{1,s}$,$R_{1,i}$ have been scaled so that one unit of population represents a large number of Individuals. Given the following values for the constants. $a_1 = 1$, $a_2=1$, $b_1=1$, $b_2=1$, $c_1=1$, $c_2=1/2$, $d_2 = 1$.

We have the following system of equations

$$\frac{dr_2}{dt} = -r_2 + r_2r_{1,s} - r_2r_{1,i}$$

$$\frac{dr_{1,s}}{dt} = r_{1,s} - r_2r_{1,s} - \frac{r_{1,s}r_{1,i}}{2} + r_{1,i}$$

$$\frac{dr_{1,i}}{dt} = -r_2r_{1,i} - \frac{r_{1,s}r_{1,i}}{2}$$

(5.1)

$$-r_2 + r_2r_{1,s} - r_2r_{1,i} = 0$$

(5.2)

$$r_{1,s} - r_2r_{1,s} - \frac{r_{1,s}r_{1,i}}{2} + r_{1,i} = 0$$

(5.3)

$$-r_2r_{1,i} - \frac{r_{1,s}r_{1,i}}{2} = 0$$

(5.4)

The system (1.2) has two equilibrium solutions

(a)A disease free equilibrium at (0,0,0) = $\left(r_2^*, r_{1,s}^*, r_{1,i}^*\right)$

(b) An endemic equilibrium at (1,2,1) = $\left(r_2^*, r_{1,s}^*, r_{1,i}^*\right)$

We determine the stability of the equilibrium points by computing the Jacobian matrix of the system (3.1) at each equilibrium point.

The nature of the equilibrium points:

At the equilibrium point $\left(r_2^*, r_{1,s}^*, r_{1,i}^*\right)$= (0, 0, 0) the Jacobian matrix is given by

$$J(0,0,0) = \begin{pmatrix} -1 & 0 & 0 \\ 0 & 1 & 1 \\ 0 & 0 & 0 \end{pmatrix}$$

Trace (J) = 0

Det (J) =0

Hence since Trace (J) = 0 and Det (J) = 0.We say that the equilibrium points (DFE) (0, 0, 0) is stable but not asymptotically stable.

At the EDE (the non-zero equilibrium point

(1, 2, 1)

$$J(1,2,1)=\begin{pmatrix} 0 & 1 & -1 \\ -2 & -\frac{1}{2} & 0 \\ -1 & \frac{1}{2} & 0 \end{pmatrix}$$

Trace (J) = 1/2< 0 and Det (J) = 3/2 > 0

Hence, since Trace (J) < 0 and Det (J)>0 , we say that the equilibrium point (EDE) (1,2,1) is asymptotically stable.

VI. NUMERICAL SIMULATION

In this section we present computer simulation of some solutions of the system (5.1).From practical point of view, numerical solutions are very important beside analytical study.

We take parameters of the system as $r_2(0)$= 0.9, $r_{1,s}(0)$= 1.90,$r_{1,i}(0)$=0.80 at time t = 0,over the time interval [0,20].

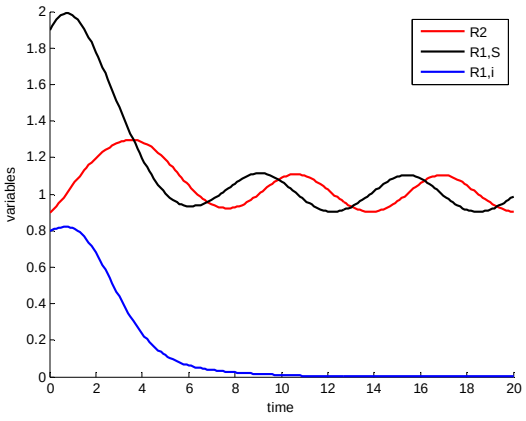

Figure 6a.We observed that the population of infected prey undergoes a decay or rather extinction.

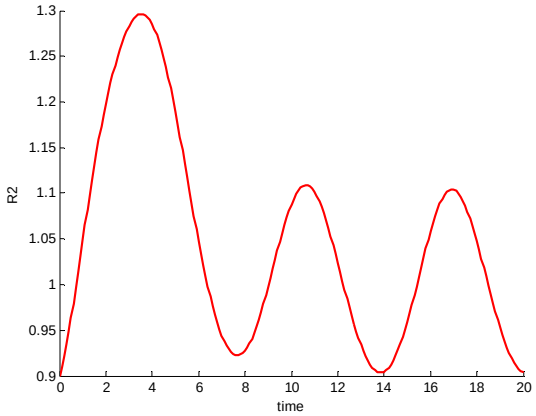

Fig 6b represents The population of the Susceptible predator as time goes on

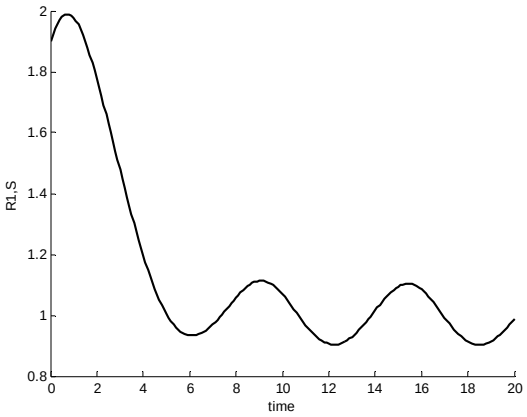

Fig 6c represents the population of the Susceptible Prey as time goes on.

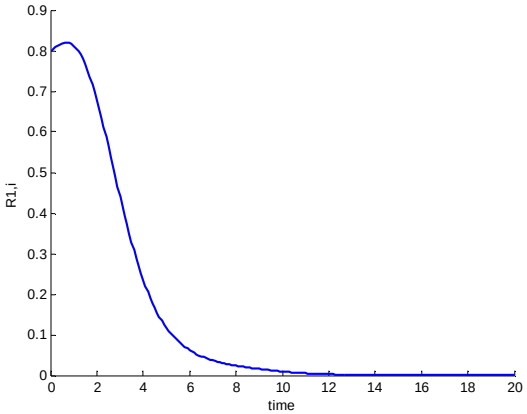

Fig 6d Shows the population of the Infected Prey as time goes on.

VII. CONCLUSION

A mathematical model of the spread of an infectious disease in a Prey-Predator ecosystem is designed and rigorously analyzed. The study which takes into cognizance the evolution of a Predator-Prey Ecosystem after an infectious disease has been introduced into the colony. We discuss in detail the stability of the disease free and endemic equilibrium by using the Routh-Hurwitz conditions to linearized the non-linear systems. We also try to identify the epidemiological thresholds parameter R_0 (The Basic reproduction number) and that the disease free equilibrium always exists. We discovered that as the population grows, the number of infected prey increases which makes the population of healthy susceptible predators to die out in the system over the time interval.There is possibility of the extinction of the population of healthy susceptible predator. Since the infected prey are carriers the population of the healthy susceptible and infected prey would increase as they interact in the ecosystem and thereby create adequate contact with the population of healthy susceptible predators. In the long run the whole population of the healthy susceptible predators may be wiped out. We believed that if vaccination or necessary campaign can be made to fight against the disease there could be hope for the survival of the population of healthy susceptible predators.

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