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A dynamic effect of infectious disease on prey predator system and harvesting policy

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ABSTRACT

The paper deals with a model that describes a prey predator system with disease in the prey population where we have investigated the effect of harvest on the disease when vaccination strategies fail to recover the infected prey population. Many infectious diseases like varicella, which is a highly transferable infection caused by the varicella zoster virus and causes even death if untreated. When the disease affected the prey species, prey species is divided into two categories: susceptible prey and infected prey. From infected prey, the disease is transmitted to the susceptible prey species. It is assumed that infection effect both prey and predator species, but the disease is debilitating and ultimately causing death for predators. Once a predator is infected, it can be considered to be dead and infected prey does not recover due to failure of vaccination strategies. The infected prey species are subjected to harvesting at low and high harvesting rates. It is shown that effective harvesting of infected prey can control the spread of disease and prevent predator species from extinction. Equilibrium points are obtained by linearization and Jacobian matrix. The local and global stability of the various equilibrium points of the system was investigated. It is observed that coexistence of both the prey and predator species is possible through non-periodic solution due to the Bendixson-Dulac criterion. With the help of Routh-Hurwitz criterion and Liapunov function, local and global stability of the non-periodic orbits are determined. Some numerical simulations have been carried out to justify the results obtained.

KEY WORDS: PREY-PREDATOR MODEL, EQUILIBRIUM POINTS, STABILITY ANALYSIS, HARVESTING ACTIVITY

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INTRODUCTION

Mathematical models have become important tools in analyzing the dynamical relationship between predator and their prey. The predator prey system is one of the well-known models which have been studied and discussed a lot. The Lotka-Volterra predator prey system has been proposed to describe the population dynamics of two interacting species of a predator and its prey (Lotka, 1925, Volterra, 1931, Arb Von et al., 2013), Lotka–Volterra equation are of form

$$\begin{cases} \frac{dx}{dt} = ax - bxy \\ \frac{dy}{dt} = cxy - dy \end{cases} \quad (1.1)$$

Where x and y are the prey and predator respectively; a is the growth rate of the prey (species) in the absence of interaction with the predator (species), b is the effect of the predation of species to species, c is the growth rate of species in perfect conditions: abundant prey and no negative environmental impact and d is the death rate of the species in perfect conditions: abundant prey and no negative environmental impact from natural cause. One of the unrealistic assumptions in the Lotka-Volterra model is that the growth of the prey populations is unbounded in the absence of the predator. Murray (Murray, 1989) modified the Lokta-Volterra model and the model were based on assumptions that the prey population exhibits logistic growth in the absence of predators, then the model obtained:

$$\begin{cases} \frac{dx}{dt} = ax\left[1 - \frac{x}{k_1} - b\frac{y}{k_2}\right] \\ \frac{dy}{dt} = cy\left[1 - \frac{y}{k_2} - d\frac{x}{k_1}\right] \end{cases} \quad (1.2)$$

where a, b, c, d, k_1, k_2 are all positive constants. This model was investigated and the conditions for stability obtained.Ecological populations suffer from various types of diseases. These diseases often play significant roles in balancing the population sizes. Most important models for the transmission of infectious diseases descend from the classical SIR model (Kermack and McKendrick,1927). In the past decades, several epidemic models with disease in prey have been extensively studied in various forms and contexts, for example, by Hethcote, (2000), Hethcote et al., (2004), Johri et al., (2012), Nandi et al., (2015), Sujatha et al., (2016), Mbava, (2017) and Yang, (2018).

In particular, a predator-prey model with disease in the prey and analyzed a model of a three species eco-epidemiological system, namely, susceptible prey, infected prey and predator (Chattopadhyay and Arino,1999). Another prey-predator model with harvesting activity of prey which has been observed is that when the harvesting activity of prey is taken into consideration, then the population size of predator decreases and the naturally stable equilibrium of the model becomes unstable (Singh and Bhatti, 2012). A mathematical model to study the response of a predator-prey model to a disease in both the populations and harvesting of each species (Das, 2014), the model with two-stage infection in prey, the early stage of infected prey is more vulnerable to predation by the predator and the later stage of infected pests is not eaten by the predator (Nandi el al., 2015), harvested prey – predator model with SIS epidemic disease in the prey population (Sujatha et al., 2016). The predator–prey model with disease in super-predator are investigated and obtained the results that in the absence of additional mortality on predator by a super - predator, the predator species survives extinction (Mbava, 2017). A diffusive predator-prey model with herd behavior has been developed and the local and global stability of the unique homogeneous positive steady state is obtained (Yang, 2018).

A compartmental mathematical model based on the dynamics of the infection and apply vaccination strategies with herd immunity to reduce the intensity of disease spread in the prey-predator ecosystem (Bakare et al., 2012). We considered the work proposed by E.A. Bakare, because sometimes vaccination strategies become ineffective, in that case dynamic changes developed in the system, which we were investigated in the present work. We are trying to demonstrate the effect of vaccination when it failed to recover from the disease One of the purposes of this article is to explore the complex effect of the prey predator model in epidemiological system due to failure of vaccination strategies. The proposed model is characterized by a pair of first order nonlinear differential equations and the existence of the possible equilibrium points along with their stability is discussed. And finally, some numerical examples are discussed.

MATERRIAL AND METHODS

We shall consider the following prey predator system for analyzing it mathematically,

$$\begin{cases} \frac{dx}{dt} = -ax + bxy - cxz(1-\phi) \\ \frac{dy}{dt} = hy - exy - fyz(1-\phi) \\ \frac{dz}{dt} = hz - exz + fyz(1-\phi) - gz \end{cases} \quad (2.1)$$

Where x, y and z stand for the density of susceptible predator, susceptible prey andinfected prey populations, respectively.And the parameters 'a' is the natural death of the healthy susceptible predator, 'b' is the number of contact between susceptible prey and healthy susceptible predator, 'c' is the number of contact between healthy susceptible predator and infected prey, 'e' is the number of contact between healthy susceptible predator with infected prey and susceptible prey, 'f' is the number of contact between healthy susceptible prey and infected prey, 'g' is the harvesting rate of infected prey , h is the per capita birth rate of susceptible prey (per time) and infected prey and ϕ is the proportion of those successively vaccinated at birth.

The model consists of basic assumptions that we have made in formulating the model are: The relative birth rate for infected prey and that of susceptible prey remains the same.The disease is severely weakened and ultimately causing death for the predators. Once a predator is infected, it can be assumed to be dead. We will therefore consider only susceptible predator andinfectious disease spreads among the prey population by contact, and the rate of infection is proportional to the infected and the susceptible prey.The predator makes no difference between susceptible and infected members of the prey population. The predator becomes infected by consuming the infected prey. The rate of predator infection is proportional to the product of infected prey and susceptible predators.The infected prey does not recover.

To begin with, let us find the equilibrium points of the system (2.1)

The system (2.1) has the following equilibrium points:

$$E_0(0,0,0),\ E_1\left(\frac{h-g}{e},0,\frac{-a}{c(1-\phi)}\right),\ E_2\left(0,\frac{g-h}{f(1-\phi)},\frac{h}{f(1-\phi)}\right),\ E_3\left(\frac{h}{e},\frac{a}{b},0\right),\ E_4(x^*,y^*,z^*)$$

Where x^*, y^*, z^* are given by $x^*=\frac{h+fy^*-g}{e}$, $y^*=\frac{fa+cg}{fb+cf(1-\phi)}$, $z^*=\left(\frac{-a+by^*}{c(1-\phi)}\right)$

In the next section, let us discuss the stability of the five equilibrium points in the next which are obtained above.

RESULTS AND DISCUSSION

Stability Analysis: In this section, we analyzed the local behavior of the system (2.1) around each equilibrium point. The Jacobian matrix of the system of state variables is as follows:

$$J(x,y,z)=\begin{bmatrix} -a+by-cz(1-\phi) & bx & -c(1-\phi)x \\ -ey & h-ex-fz(1-\phi) & -fy(1-\phi) \\ -ez & f(1-\phi)z & h-ex+fy(1-\phi)-g \end{bmatrix}$$

To determine the stability of the equilibrium points, we look at the most useful techniques for analyzing nonlinear system is the linearized stability technique by theorem1.

Theorem 1:

Let $v(\lambda)=\lambda^3+A_1\lambda^2+A_2\lambda+A_3$ There are at most three roots of the equation $v(\lambda)=0$. Then the following statements are true:

a) If every root of the equation has absolute value less than one, then the equilibrium point of the system is locally asymptotically stable and equilibrium point is called a sink.

b) If at-least one of the roots of the equation has an absolute value greater than one, then the equilibrium point of the system is unstable and equilibrium point is called a saddle.

c) If every root of the equation has an absolute value greater than one, then the system is sourced.

d) The equilibrium point of the system is called hyperbolic if no root of the equation has absolute value equal to one. If there exists a root of the equation with absolute value equal to one, then the equilibrium point is called non-hyperbolic (i.e. one eigenvalue has a vanishing real part).

Let us preparefour propositions in order to discuss the local stability around each equilibrium point.

Preposition 1: For system (2.1),

The equilibrium point E_0 is locally asymptotically stable if h<1 and h<g.

Proof: The Jacobian matrix at E_0 (0,0,0) is given by

$$J(E_0)=\begin{bmatrix} -a & 0 & 0 \\ 0 & h & 0 \\ 0 & 0 & h-g \end{bmatrix}$$

The Eigenvalue corresponding to the equilibrium point E_0 (0,0,0) are –a, h, h-g. Only one Eigen value is negative and other two depends upon the value of h i.e. Birth rate of susceptible and infected prey. Then by theorem 1, we obtain $E_0$is locally asymptotically stable if h<1 and h<g.

Preposition 2: For system (2.1), The equilibrium point E1is locally asymptotically stable if $(1-\phi)\sqrt{ah-ag}<1$ and af+cg<1.

Proof: The Jacobian matrix at $E_1\left(\frac{h-g}{e},0,\frac{-a}{c(1-\phi)}\right)$ is given by

$$J(E_1)=\begin{bmatrix} 0 & \frac{b(h-g)}{e} & \frac{-c(1-\phi)(h-g)}{e} \\ 0 & g+\frac{fa}{c} & 0 \\ \frac{-ae}{c(1-\phi)} & \frac{-fa}{c} & 0 \end{bmatrix}$$

If the corresponding Eigenvalues are λ_1, λ_2, λ_3 then

$$\lambda_1 = -(1-\phi)\sqrt{ah-ag}$$
$$\lambda_2 = (1-\phi)\sqrt{ah-ag}$$
$$\lambda_3 = \frac{af+cg}{c}$$

Thenby theorem 1, we obtain $E_1\left(\frac{h-g}{e},0,\frac{-a}{c(1-\phi)}\right)$ is locally asymptoticallystable if $(1-\phi)\sqrt{ah-ag}<1$ and af+cg<1.

Preposition 3:For system (2.1),
The equilibrium point E_2 is locally asymptotically stable if $\sqrt{h^2-gh}<1$ and $bg+bh\phi<af+bg\phi+bh+ch$.

Proof: The Jacobian matrix at $E_2\left(0,\frac{g-h}{f(1-\phi)},\frac{h}{f(1-\phi)}\right)$ is given by

$$J(E_2)=\begin{bmatrix} -a+\frac{b(g-h)}{f(1-\phi)}-\frac{ch}{f} & 0 & 0 \\ \frac{e(h-g)}{f(1-\phi)} & 0 & -g+h \\ \frac{-eh}{f(1-\phi)} & h & 0 \end{bmatrix}$$

If the corresponding Eigenvalues are λ_1, λ_2, λ_3 then

$$\lambda_1 = -\sqrt{h^2-gh}$$
$$\lambda_2 = \sqrt{h^2-gh}$$
$$\lambda_3 = \frac{-af+bg-bh-ch-bg\phi+bh\phi}{f}$$

Then by theorem 1, we obtain $E_3\left(0,\frac{g-h}{(1-\phi)},\frac{h}{f(1-\phi)}\right)$ is locally asymptotically stable if $\sqrt{h^2-gh}<1$ and $bg+bh\phi<af+bg\phi+bh+ch$

Preposition 4:For system (2.1),
The equilibrium point E_3 is neutral if eigenvalue is imaginary.

Proof: The Jacobian matrix at $E_3\left(\frac{h}{e},\frac{a}{b},0\right)$ is given by

$$J(E_3)=\begin{bmatrix} 0 & \frac{bh}{e} & \frac{c(\phi-1)h}{e} \\ \frac{-ae}{b} & 0 & \frac{af(\phi-1)}{b} \\ 0 & 0 & \frac{af(1-\phi)-bg}{b} \end{bmatrix}$$

If the corresponding Eigenvalues are λ_1, λ_2, λ_3 then

$$\lambda_1 = -\frac{(bg-af+af\phi)}{b}$$
$$\lambda_{2,3} = \pm i\sqrt{ah}$$

One Eigen value l1 is negative if af<bg+afϕ and the remaining two Eigen values l2 and l3 are imaginary. The Eigenvalues are purely imaginary, its real parts are exactly 0. The equilibrium point $E_3\left(\frac{h}{e},\frac{a}{b},0\right)$ is neutral.Then by theorem 1(d), we obtain this preposition.

Let us discuss the stability of the E_4 by Routh-Hurwitzcriterian.Local stability of the system (2.1) around the non-zero equilibrium point E_4.

The Jacobian matrix at $E_4(x^*,y^*,z^*)$ is given by

$$J(E_4)=\begin{bmatrix} -a+by^*-cz^*(1-\phi) & bx^* & -c(1-\phi)x^* \\ -ey^* & h-ex^*-fz^*(1-\phi) & -fy^*(1-\phi) \\ -ez^* & f(1-\phi)z^* & h-ex^*+fy^*(1-\phi)-g \end{bmatrix}$$

Where x*, y*, z* are given by

$$x^*=\frac{h+fy^*-g}{e},\quad y^*=\frac{fa+cg}{fb+cf(1-\phi)},\quad z^*=\left(\frac{-a+by^*}{c(1-\phi)}\right)$$

The characteristic polynomial for the Jacobian matrix J (E_4) is given by

$$\lambda^3+A_1\lambda^2+A_2\lambda+A_3=0$$

Where

$$A_1 = a-by^*+cz^*(1-\phi)-2h+2ex^*+fz^*(1-\phi)+g-fy^*(1-\phi)$$
$$A_2 = (-a+by^*-cz^*(1-\phi))(2h-2ex^*-fz^*(1-\phi)+fy*(1-\phi)-g)+$$
$$(h-ex^*-fz^*(1-\phi))(h-ex^*+fy^*(1-\phi)-g)+bex^*y^*-c(1-\phi)x^*ez^*$$
$$A_3 = (h-ex^*-fz^*(1-\phi))((a-by^*+cz^*(1-\phi))(h-ex^*+fy^*(1-\phi)-g)+c(1-\phi)x^*ez^*\}$$
$$-bex^*y^*(h-ex^*+fy^*(1-\phi)-g)-befx^*y^*z^*(1-\phi)-cf(1-\phi)^2x^*y^*ez^*$$

According to Routh-Hurwitzcriterian, $E_4(x^*,y^*,z^*)$ is asymptotically stable if and only if $A_1>0$, $A_3>0$ and $A_1A_2-A_3>0$.

Theorem 2. (E_0) is globally stable.

Proof. Let a Liapunov function be,

$$V(x,y,z)=x+y+z.$$
$$\frac{dV}{dt}=-ax-cxz(1-\phi)-exy-exz-gz+bxy+h(y+z)<0,\ if\ b,h<0.$$

The theorem above, then implies that (E_0) is globally asymptotically stable.

Now, let us find the global stability of the system (2.1) around all the equilibrium points for different 2-D planes by using Bendixson-Dulac criterion.

Theorem 3. E_2 is globally asymptotically stable in y-z plane.

Proof. Let,

$$H(y,z)=1$$

It is obvious that $H(y,z)>0$ if $y>0$ and $z>0$ if and.

Now, we denote

$$F_1(y,z)=hy-fyz(1-\phi),$$
$$F_2(y,z)=hz+fyz(1-\phi)-gz,$$
$$\Delta(y,z)=\frac{\partial}{\partial y}[F_1H]+\frac{\partial}{\partial z}[F_2H],$$

Then,

$$\Delta(y,z) = 2h - fz(1-\phi) + fy(1-\phi) - g.$$

Thus, $\Delta(y,z) < 0$ for all $y > 0$ and $z > 0$ if $h < 0$ and $f(1-\phi) < 0$ Therefore, by using Bendixson-Dulac criterion, there will be no periodic orbit in the y-z plane.

In the similar manner, we can show in the x-z plane for E1 with the condition $\Delta(x,z) < 0$ for all $x > 0$ and $z > 0$ if $h > 0$, in the x-y plane for E_3 with the condition $\Delta(x,z) < 0$ for all $x > 0$ and $y > 0$ if $h,b < 0$ and in the same way E_4 can be globally asymptotically stable in x-y,y-z and x-z planes.

We have performed some numerical simulation to study the role of harvesting on the prey predator system and we illustrate the dynamical and complex features of the model using MATLAB. In the starting, we fixed all parameters to ensure that the three classes of populations survive. Numerical simulations explain the effect of the parameters on the complex behavior of a given system (2.1).

(i) Let us consider following set of parameters,

a = 1.0; b = 1.5; c = 0.1; h = 0.5; e = 1.5;f=0.1;g=0.7,ϕ=0.91,

With initial condition x (0) =0. 8, y (0) =1. 70, z (0) =0. 75. For this set of parameter, we get the following variation of the population of the healthy predator, susceptible prey and infected prey with respect to time, which isillustrated below in figure 4 (a) and figure 4 (b).

(ii) Let us consider following set of parameters,

a = 1.0; b = 1.5; c = 0.1; h = 0.5; e = 1.5;f=0.1;g=0.1;ϕ=0.91;

With initial condition x (0) =0. 8, y (0) =1. 70, z (0) =0. 75. For this set of parameter, we get the following variation of the population of the healthy predator, susceptible prey and infected prey with respect to time, which is illustrated below in figure 4(c) and figure 4(d).

It is observed that effective harvesting of diseased prey, increase the growth rate of the susceptible predator population. If the value of harvesting rate g≥0. 7 then the infected prey population decreases more rapidly, but if the value of g<0.7 then infected prey population decreases slowly that shown in fig. 4(a), 4(b), 4(c) and 4(d) respectively. In this analysis, we have also observed that the whole population of the susceptible predators may be wiped out due to increase in the number of the susceptible and infected preys. This result shows that the system is biologically well behaved. In another case when the diseased prey can be washed out, a rational use of the stability criterion of non-zero equilibrium point may be useful for ecological balance. In this case, the parameters of the system should be regulated in such a way that stability criterion of non-zero equilibrium is satisfied but infected prey remains low enough. Some-

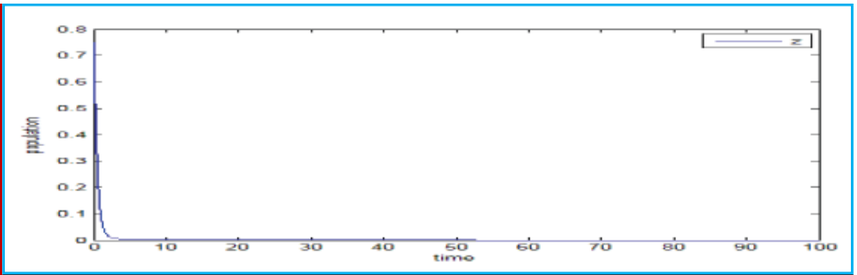

FIGURE 4a. Represents the effect of high harvesting on the population of the infected prey as time goes on.

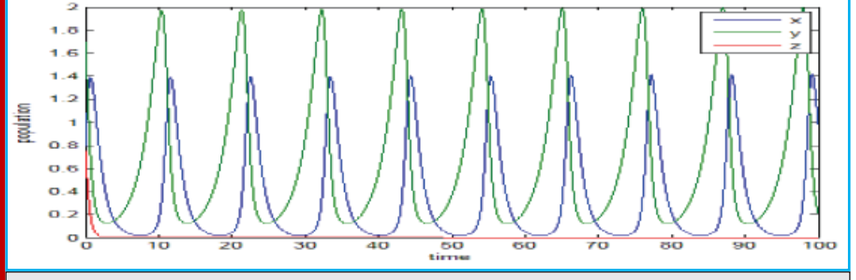

FIGURE 4b. Represents the effect of high harvesting on the population of the healthy predator, susceptible prey and infected prey as time goes on.

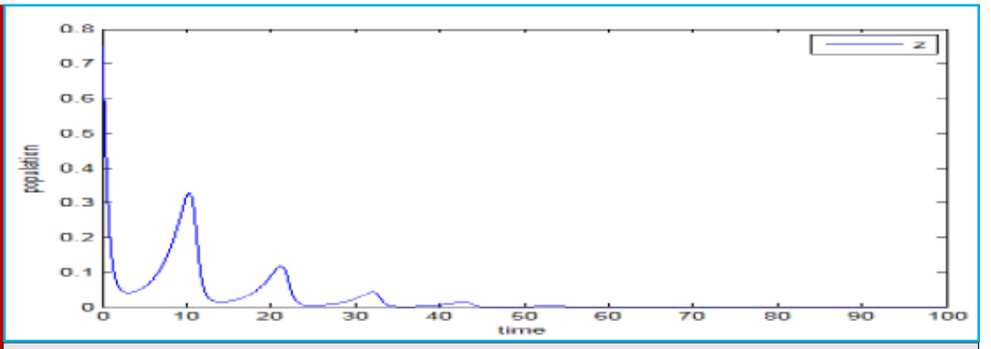

FIGURE 4c. Represents the effect of low harvesting on the population of the infected prey as time goes on.

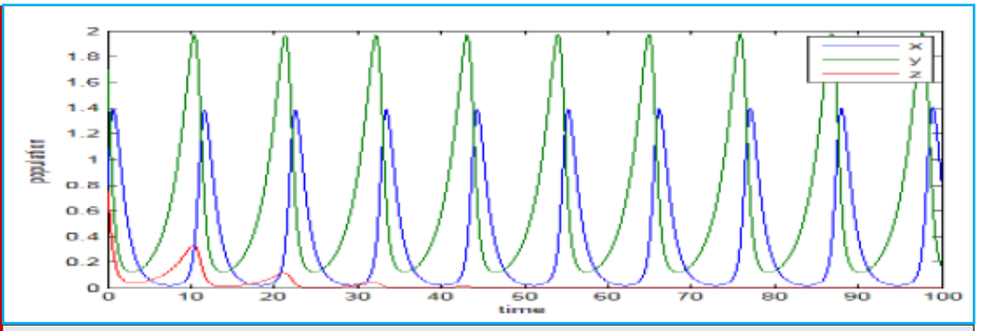

FIGURE 4d. Represents the effect of low harvesting on thepopulation of the healthy predator, susceptible prey and infected prey as time goes on.

times, harvesting became a suitable option for prevention of the population rather than the vaccination strategies. Therefore, effective harvesting became essential for the survival of the population.

CONCLUSION

A non-linear system based on the epidemic SIR model has been studied and discussed. Conditions for local and global stability at various equilibrium points were obtained. We have illustrated the effective harvesting of diseased prey in the whole system and reveal that the increases of predator population when the harvesting rate of infected prey population increases. We may conclude that effective harvesting of diseased prey may be used as a biological control for the spread of disease. And maintain balance in these species populations by preventing in the predator population to extinction. Finally, some numerical simulations illustrate and supplement our theoretical analysis by considering different parameter values. Low harvesting and high harvesting rates play an important role in this analysis. Global stability of equilibrium E_0 shows that disease free equilibrium always exists. In future other effecting condition can be used to save the predator population by introducing alternative food for predator rather than diseased prey.

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