Research Article

Prey-Predator Model with Two-Stage Infection in Prey: Concerning Pest Control

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A prey-predator model system is developed; specifically the disease is considered into the prey population. Here the prey population is taken as pest and the predators consume the selected pest. Moreover, we assume that the prey species is infected with a viral disease forming into susceptible and two-stage infected classes, and the early stage of infected prey is more vulnerable to predation by the predator. Also, it is assumed that the later stage of infected pests is not eaten by the predator. Different equilibria of the system are investigated and their stability analysis and Hopf bifurcation of the system around the interior equilibria are discussed. A modified model has been constructed by considering some alternative source of food for the predator population and the dynamical behavior of the modified model has been investigated. We have demonstrated the analytical results by numerical analysis by taking some simulated set of parameter values.

1. Introduction

It is of great interest to consider the effects of pesticide on ecological communities from both an environmental and economical point of view with the rapid development of modern technology, industry, and agriculture. The main foods of mankind are different types of crops or vegetables. Due to the limitations of land for planting, our main interest is not to destroy even a single mole of food. But in nature many times the leaves or branches of those crop trees are consumed by so many small insects, animals, weeds, and so forth for their survival indicating the agriculture resource would severely be damaged from the environment. These insects or animals or weeds are known as pests. In a word an organism which harms agriculture by means of feeding on crops or parasitizing livestock is known to us as a pest. The term pest not only is used to refer to harmful animals but also relates to all other harmful organisms, namely, fungi, bacteria, virus parasites, and so forth. It is also possible that an animal

may be a pest in one case but beneficial or domesticated in another situation. But in this paper by the word pest we mean those livelihoods which are involved in the cause of the damage to the human populations directly or indirectly.

The control of pest is a worldwide problem. The most common technique of the pest control is use of chemical insecticides to control the pest population. The use of chemical insecticides is an attempt to control pest directly at low cost (see Uboh et al. [1]). But in many research works it is shown that these chemicals have many environmental effects that include chemical residing in crop and in the agricultural ecosystems. In nature, other than pest, there are some other types of population species known as natural enemy, which predate those pests. For an example, there are some birds, frog, and so forth which feed on agricultural pests and thus those species can be treated as predator species to the pests. So these populations can be used as one of the biological controls for the pest. A prey-predator model can be used to study pest control phenomenon (see the works of Tang et al.

[2], Ghosh et al. [3], Ghosh and Bhattacharya [4], Pei et al. [5], Liang et al. [6], Apreutesei [7], Kar et al. [8], Jana and Kar [9, 10], and Mondal et al. [11] and references therein). To control the pest species without using any pesticide control, use of natural enemy is one of the important ways. Again the pest population may be affected due to the infection of some bacterial or viral diseases. So another important natural way to control pest population is the consideration of the effect of some infectious diseases among the targeted pest population. Sun and Chen [12] and Tan and Chen [13] used infection to control the pest population. Some viral or bacterial infection caused the reduction of pest species significantly. Wang and Song [14] used mathematical model by infected pest to control the pest population. Haderl and Freedman [15], Venturino [16, 17], Kar and Mondal [18], Liu and Wang [19], Guo and Chen [20], Y. Zhang and Q. Zhang [21], Wang et al. [22], and Wang and Chen [23] also worked on prey-predator model with the disease in the prey populations. In our present paper we consider two stages of infected pest, namely, infected pest in the first stage and infected pest in the last stage. The two stages of the infection are considered on the assumption that the predator population would consume infected pest of the first stage but the predator would not consume the pest population at the second stage.

Again a prey-predator system plays an important role when an alternative source of food to the predator population is supplied (see Pahari and Kar [24], Srinivasu and Prasad [25], Srinivasu [26], Sabelis and van Rijn [27], Harwood and Obryeki [28], etc.). To control the pest, the effect of alternative food takes some attention compared to ordinary predator-prey dynamics in the context of biological conservation. For both the conservation of predator and reduction of the pest, an alternative source of food must be given to the natural enemy of the pest (see Jana and Kar [10]).

The paper is organized in the following schemes: In Section 2, we formulate the pest control model on the basis of some assumptions and in Section 3, we analyze the complex dynamics of that model. In Section 4, we analyze the model if predator populations would be provided with some supplementary food. In Section 5, we present some numerical simulations for validating our model and in the final section we give some outcomes from our whole paper.

2. Model Formulation and Description

The following assumptions are made to build the mathematical model for pest control.

P1. In this paper, we consider mainly two types of species: one is prey, that is, pest, denoted by N , and the other is predator, denoted by P . But in the presence of disease the pest population is divided into two classes, namely, susceptible (S) and infected (I). Again the infected pest population is classified into two stages: one is the early stage or first stage of infected pest (I_1) and the other is the later stage or last stage of infected pest (I_2). Therefore, at any time t the total pest population is $N(t) = S(t) + I_1(t) + I_2(t)$.

P2. We consider that the susceptible pest population grows logistically in the absence of the disease (see Das et al. [29]). So if r (> 0) is the intrinsic growth rate and K (> 0) is the carrying capacity, then the growth equation of susceptible pest is

$$\frac{dS}{dt} = rS \left(1 - \frac{S}{K} \right). \quad (1)$$

P3. Infected pest populations can be divided into two classes, namely, infected pest in the first stage and infected pest in the last stage. The susceptible pest becomes infected through direct contact with the early stage and later stage of infected pest at rates β_1 and β_2 , respectively, following the mass action law. After primary infection the pest population becomes more infected by the same disease because the infection takes some time to spread among the population. In this scenario, α is assumed as active rate of the disease. The infected pest dies linearly in two stages at rates σ and δ , respectively. Therefore the $S - I$ model for the pest population can be written as

$$\begin{aligned} \frac{dS}{dt} &= rS \left(1 - \frac{S}{K} \right) - (\beta_1 I_1 + \beta_2 I_2) S, \\ \frac{dI_1}{dt} &= (\beta_1 I_1 + \beta_2 I_2) S - (\alpha + \sigma) I_1, \\ \frac{dI_2}{dt} &= \alpha I_1 - \delta I_2. \end{aligned} \quad (2)$$

P4. The predator population consumes only the susceptible pest and the early stage of infected pest and consciously avoids the latter stage of infected pest for its better survival. We choose the predation function as Holling type II functional response $pS/(m + S)$, where p is the capturing rate and m is the half saturation constant, to predate the susceptible pest as some searching time is needed for capturing and handling the prey. But as the primary infected pest population is somewhat weaker than the susceptible pest, no more searching time is needed by the predators for catching the infected pest I_1 and thereby the predation function is assumed as Holling type I functional response in the form qI_1 , where q is the maximum capturing rate.

P5. Conversion factors for the predator are assumed as h_1 and h_2 from the susceptible and primary infected pest, respectively. Also, to improve this model, we incorporate the density dependent factor ξP for predators, where ξ is the density dependent mortality rate. Let ε be the death rate of the predator.

Thus, keeping all the above assumptions in mind, we can obtain the following ecoepidemic model as follows:

$$\begin{aligned} \frac{dS}{dt} &= rS \left(1 - \frac{S}{K} \right) - (\beta_1 I_1 + \beta_2 I_2) S - \frac{pSP}{m + S}, \\ \frac{dI_1}{dt} &= (\beta_1 I_1 + \beta_2 I_2) S - qI_1P - (\alpha + \sigma) I_1, \end{aligned}$$

$$\begin{aligned}\frac{dI_2}{dt} &= \alpha I_1 - \delta I_2, \\ \frac{dP}{dt} &= h_1 \frac{pSP}{m+S} + h_2 q I_1 P - \varepsilon P - \xi P^2,\end{aligned}\quad (3)$$

with the initial conditions $S(0) > 0$, $I_1(0) > 0$, $I_2(0) > 0$, and $P(0) > 0$.

3. Analysis of the System

In this section we will study the different dynamical behavior like uniform boundedness, existence of different equilibria and their stability, and so forth.

3.1. Boundedness of the System

Lemma 1. *System (3) is uniformly bounded in the region $\Theta = \{(S, I_1, I_2, P) \in R_4^+ : 0 < B \leq \Omega/\eta\}$.*

Proof. See Appendix A. $\square$

3.2. Existence of Equilibria. System (3) has the following equilibria:

- (i) the trivial equilibrium $D_0 = (0, 0, 0, 0)$;
- (ii) the axial equilibrium $D_1 = (K, 0, 0, 0)$;
- (iii) the predator-free equilibrium point $D_2 = (\bar{S}, \bar{I}_1, \bar{I}_2, 0)$, where $\bar{S} = \delta(\alpha + \sigma)/(\delta\beta_1 + \alpha\beta_2)$, $\bar{I}_1 = r\delta[K(\delta\beta_1 + \alpha\beta_2) - \delta(\alpha + \sigma)]/(\delta\beta_1 + \alpha\beta_2)^2$, and $\bar{I}_2 = (\alpha/\delta)\bar{I}_1$;
- (iv) the infected pest-free equilibrium $D_3 = (S_3, 0, 0, P_3)$, where $P_3 = r(K - S_3)(m + S_3)/Kp$ and S_3 is a positive root of the following equation:

$$x_1 S^3 + x_2 S^2 + x_3 S + x_4 = 0, \quad (4)$$

where $x_1 = r\xi$, $x_2 = -(K - 2m)r\xi$, $x_3 = -mr\xi(2K - m) - Kp(\varepsilon - ph_1)$, and $x_4 = -Km(p\varepsilon + mr\xi)$;

- (v) the interior equilibrium point $D^* = (S^*, I_1^*, I_2^*, P^*)$, where $S^* = b(\gamma + qP^*)$, $I_1^* = [bq\xi(P^*)^2 + \{bq(\varepsilon - h_1 p) + \xi(m + b\gamma)\}P^* + m\varepsilon + b\gamma(\varepsilon - h_1 p)]/h_2 q(m + S^*)$, $I_2^* = (\alpha/\delta)I_1^*$, and P^* is a positive root of the following equation:

$$n_1 P^2 + n_2 P + n_3 = 0, \quad (5)$$

where $n_1 = bq(K\xi + b^2 q^2 r h_2)$, $n_2 = K(bq\varepsilon + m\xi + b\gamma\xi) - bKp q h_1 + bKp q h_2 + b^2 q^2 r h_2 (m + 2b\gamma - K)$, and $n_3 = (m + b\gamma)(K\varepsilon + bqr h_2 (b\gamma - K)) - bKp h_1 \gamma$, $b = \delta/(\delta\beta_1 + \alpha\beta_2)$, $\gamma = (\alpha + \sigma)$.

For positivity of D_2 , we assume $K > b\gamma$. The infection-free equilibrium D_3 will be feasible if $K > \max[S_3, 2m]$ and $\varepsilon > h_1 p$. In fact, (4) has exactly one root under these parametric conditions.

For (5), we can deduce the following cases.

Case 1. Let us assume that $K_0 = b^2 q r h_2 (m + b\gamma) \gamma / \{(bqr h_2 - \varepsilon)(m + b\gamma) + bh_1 p \gamma\}$. When $K > K_0$, then the constant coefficient n_3 is negative. So, by Descartes' rule of sign (5) has exactly one positive root; that is, unique positive interior equilibrium D^* exists.

Case 2. If $K_1 < K < K_0$, then $n_2 < 0$ and $n_3 > 0$, where $K_1 = b^2 q^2 r h_2 (m + 2b\gamma) / \{b p q h_1 + b^2 q^2 r h_2 - (b p q h_2 + b q \varepsilon + m \xi + b \gamma)\}$. Therefore, (5) will have two positive roots provided $n_2^2 > 4n_1 n_3$. In this case, the largest value of P^* will be considered and thus the unique interior equilibrium will be obtained.

Thus, the positive value of P^* is given by

$$P^* = \frac{-n_2 + \sqrt{n_2^2 - 4n_1 n_3}}{2n_1}. \quad (6)$$

Furthermore, positivity of I_1^* implies that $\varepsilon > h_1 p$.

3.3. Stability Analysis

Theorem 2. *The trivial equilibrium D_0 is always unstable.*

Proof. All the eigenvalues at the trivial equilibrium are r , $-(\alpha + \sigma)$, $-\delta$, and $-\varepsilon$. Therefore, the trivial equilibrium is unstable. $\square$

Remark 3. It is concluded from the analysis of Theorem 2 that the trivial equilibrium is a saddle point with one-dimensional unstable and three-dimensional stable manifold. Biologically, it means that the predator population may become extinct from the environment in absence of pest population.

Theorem 4. *The axial equilibrium D_1 is locally asymptotically stable if $K < K_c = \min[m\varepsilon/(h_1 p - \varepsilon), b\gamma]$.*

Proof. The characteristic equation at D_1 is

$$(\lambda + r)(\lambda + a)(\lambda^2 + a_1 \lambda + a_2) = 0, \quad (7)$$

where $a = (m\varepsilon - K(h_1 p - \varepsilon))/(m + K)$, $a_1 = \delta + \alpha + \sigma - K\beta_1$, and $a_2 = \delta(\alpha + \sigma - K\beta_1) - K\alpha\beta_2$.

If $K < K_c$, then two eigenvalues are always real and negative, and another two eigenvalues will be negative or having negative real parts, hence the theorem. $\square$

Remark 5. The above stability analysis biologically indicates that the pest population increases rapidly towards its carrying capacity in absence of disease and predators while the environmental carrying capacity for susceptible pest population is below some threshold value K_c . Moreover, the stability of D_1 indicates that the predator-free equilibrium D_2 does not exist.

Now, we discuss the stability criteria of predator-free equilibrium. The characteristic equation at D_2 is

$$(\lambda + f)(\lambda^3 + b_1 \lambda^2 + b_2 \lambda + b_3) = 0, \quad (8)$$

where $f = (m + \bar{S})(\varepsilon - h_2 q \bar{I}_1) - h_1 p \bar{S}$, $b_1 = r\bar{S}/K + \delta + \alpha\beta_2 \bar{S}/\delta$, $b_2 = r\bar{S}[\delta^2 + (K - \bar{S})\delta\beta_1 + \alpha\beta_2 \bar{S}]/K\delta$, and $b_3 = r\delta(\alpha + \sigma)(K - \bar{S})/K$.

If $f < 0$, then one root of (8) will be real and negative. The other three roots are given by the second part of (8). Clearly, all b_i ($i = 1, 2, 3$) are positive and function of $\bar{S}$. So, we assume $\bar{S} = \rho$ as bifurcation parameter. Note that $\bar{S} \in (0, K)$.

Hence for any $\rho \in (0, K)$, the predator-free equilibrium is locally asymptotically stable if and only if $\Phi(\rho) = b_1(\rho)b_2(\rho) - b_3(\rho) > 0$. But the system enters into a Hopf bifurcation around D_2 if $\Phi(\rho) = 0$.

We shall discuss the Hopf bifurcation using the approach of Bhattacharyya and Bhattacharya [30].

As $b_2(0) = 0$ and $b_3(0) = r\delta(\alpha + \sigma)$, $\Phi(0) < 0$. Again, $b_1(K) > 0$, $b_2(K) > 0$, and $b_3(K) = 0$ indicate that $\Phi(K) > 0$. Since $\Phi(\rho)$ is a continuous function in $(0, K)$, by intermediate value theorem there exists $\rho^* \in (0, K)$ such that $\Phi(\rho^*) = 0$ and $b_3(\rho^*) > 0$. As $b_1(\rho)b_2(\rho)$ is a monotonic increasing function and $b_3(\rho)$ is a decreasing function in $[0, K]$, then the critical value ρ^* is unique. Now, we can easily calculate the first- and second-order derivative of $\Phi(\rho)$ with respect to ρ , and then the following two results are obtained:

$$\begin{aligned} \left[\frac{d\Phi(\rho)}{d\rho} \right]_{\rho=0} &> 0, \\ \left[\frac{d^2\Phi(\rho)}{d\rho^2} \right]_{\rho \in (0, K)} &> 0. \end{aligned} \quad (9)$$

The above results imply that

$$\left[\frac{d\Phi(\rho)}{d\rho} \right]_{\rho=\rho^*} > 0. \quad (10)$$

Thus, $\Phi(\rho) > 0$ for all $\rho \in (\rho^*, K)$. So, by Routh-Hurwitz criteria, the predator-free equilibrium is locally asymptotically stable in (ρ^*, K) and unstable in $(0, \rho^*)$. According to the definition of Beretta et al. [31], Guckenheimer and Holmes [32], and Bhattacharyya and Bhattacharya [30], a single Hopf bifurcation occurs at $\rho = \rho^*$ and for decreasing $\rho \in (0, \rho^*)$, it approaches a periodic solution. Therefore, this discussion provides the following theorem.

Theorem 6. *If $f < 0$, then the predator-free equilibrium D_2 is locally asymptotically stable for $\rho > \rho^*$ and unstable for $\rho < \rho^*$. Furthermore, the system enters into a single Hopf bifurcation around D_2 when $\rho = \rho^*$.*

Remark 7. Theorem 6 biologically indicates that the infected pest populations coexist with the susceptible pest population in absence of predator and exhibits oscillatory balance behavior, and for large value of the carrying capacity the susceptible pest population may persist for a long time without using pesticide. Also, if T is the period of the bifurcating periodic orbits close to $\rho = \rho^*$, then we get

$$T(\rho^*) = \frac{2\pi}{\omega_0}, \quad \omega_0 = \sqrt{b_2(\rho^*)} = \sqrt{\frac{b_3(\rho^*)}{b_1(\rho^*)}}. \quad (11)$$

Theorem 8. *The disease-free equilibrium $D_3 = (S_3, 0, 0, P_3)$ is locally asymptotically stable if $x_i > 0$, for $i = 1, 2, 3$ and $x_1 x_2 x_3 > x_3^2 + x_1^2 x_4$, where the symbols are defined in the proof.*

Proof. See Appendix B. $\square$

Now, the global stability analysis of the system around the disease-free equilibrium will be examined in the next theorem using the approach of reference name.

Remark 9. From Theorem 8, it is evident that the disease may become extinct from pest population and in this situation predator population can not consume pest easily. Therefore, the interaction between susceptible pest and predator will be ensured during long time for their survival.

Theorem 10. *The interior equilibrium D^* is locally asymptotically stable if all $y_i > 0$ for $i = 1, 2, 3, 4$ and $\Gamma(\xi) > 0$. But the system enters into the Hopf bifurcation around D^* when ξ passes through ξ^* .*

Proof. The details of the proof are given in Appendix C. $\square$

Remark 11. Theorem 10 provides us with a threshold parameter ξ^* such that, for $\xi < \xi^*$, the interior equilibrium is stable and the system bifurcates towards a periodic solution of small amplitude when ξ is increased. In this scenario, we can observe an oscillatory nature of the system. The period T of the bifurcating periodic orbits close to $\xi = \xi^*$ is given by

$$T(\xi^*) = \frac{2\pi}{\omega_0}, \quad \omega_0 = \sqrt{\frac{y_3(\xi^*)}{y_1(\xi^*)}}. \quad (12)$$

4. Modified Model

Here we study the system when some additional food is to be supplied from some alternating source to the predator populations and assume that the maximum growth rate due to this food is d . So here predator populations get their food from two sources, namely, from the prey population and from the alternative source of food. Therefore only the final differential equation of the predator population will be changed and other equations will remain in the same form. So combining these with the previous assumptions we construct our final revised model as follows:

$$\begin{aligned} \frac{dS}{dt} &= rS \left(1 - \frac{S}{K} \right) - (\beta_1 I_1 + \beta_2 I_2) S - \frac{pSP}{m + S}, \\ \frac{dI_1}{dt} &= (\beta_1 I_1 + \beta_2 I_2) S - qI_1 P - (\alpha + \sigma) I_1, \\ \frac{dI_2}{dt} &= \alpha I_1 - \delta I_2, \\ \frac{dP}{dt} &= h_1 \frac{pSP}{m + S} + h_2 qI_1 P + dP \left(1 - \frac{S}{K} \right) - \varepsilon P - \xi P^2. \end{aligned} \quad (13)$$

The initial conditions are $S(0) > 0$, $I_1(0) > 0$, $I_2(0) > 0$, and $P(0) > 0$.

4.1. Equilibria of Modified System. System (13) has six possible feasible equilibria as follows:

- (i) trivial equilibrium D_0 ,
- (ii) the axial equilibrium D_1 ,
- (iii) the predator-free equilibrium D_2 ,
- (iv) the infected pest-free equilibrium D_3 ,
- (v) the pest-free equilibrium $D_P(0, 0, P_2)$, (where $P_2 = (d - \epsilon)/\xi$),
- (vi) the interior equilibrium $D_*(S_*, I_{1*}, I_{2*}, P_*)$, where $S_* = b(\gamma + qP_*)$, $I_{1*} = [bq(K\xi + bdq)(P_*)^2 + \{bqK(\epsilon - h_1p) + K\xi(m + b\gamma) + bdq(m + 2b\gamma - K)\}P_* + (m + b\gamma)(K\epsilon + bd\gamma - dK) - h_1Kpb\gamma]/h_2q(m + S_*)$, $I_{2*} = (\alpha/\delta)I_{1*}$, and P_* is a positive root of the following equation:

$$n_1P^2 + n_2P + n_3 = 0, \quad (14)$$

where $n_1 = bq(K\xi + bq d + b^2q^2rh_2)$, $n_2 = K(bq\epsilon + m\xi + b\gamma\xi) - bKpqh_1 + bq(d + bqrh_2)(m + 2b\gamma - K)$, and $n_3 = (m + b\gamma)(K\epsilon + (bqrh_2 + d)(b\gamma - K)) - bKph_1\gamma$, $b = d/(\delta\beta_1 + \alpha\beta_2)$, $\gamma = (\alpha + \sigma)$.

Among all these equilibria the coordinates of the first four equilibria are the same as system (3). The main difference between system (3) and system (13) is the fact that in the later system there is one more equilibrium point which does not exist in previous system (3). The equilibrium point $D_P(0, 0, P_2)$ is the new equilibrium point and around this equilibrium the system will be totally pest-free. Further this equilibrium point exists if the maximum growth rate of the predator population due to the application of additional food (d) is of higher value than the death rate of the predator population. In the next theorem, we discuss the local asymptotic stability of the pest-free equilibrium E_P .

Theorem 12. *System (13) is locally asymptotically stable around the pest-free equilibrium $D_P(0, 0, 0, P_2)$ if $d > mr\xi/p + \epsilon$.*

Proof. The characteristic equation of the system around D_P is as follows:

$$\left(\lambda - r + p\frac{d - \epsilon}{m\xi}\right)\left(\lambda + \alpha + \sigma + q\frac{d - \epsilon}{\xi}\right)(\lambda + \delta) \cdot (\lambda + d - \epsilon) = 0. \quad (15)$$

Therefore it is clear that if $d > \epsilon$ and $mr\xi < p(d - \epsilon)$, that is, if $d > mr\xi/p + \epsilon$ holds, then all the eigenvalues are negative, hence the theorem. $\square$

5. Numerical Simulation

In this section we give some numerical simulations regarding the stability and bifurcation of system (3) around the interior equilibrium D^* . For simulation let us take $r = 1.5$, $K = 100$, $\beta_1 = 0.24$, $\beta_2 = 0.26$, $p = 0.5$, $m = 0.62$, $q = 0.6$, $\alpha = 0.25$, $\sigma = 0.1$, $\delta = 0.12$, $h_1 = 0.8$, $h_2 = 0.5$, $\epsilon = 0.3$,

and $\xi = 0.05$. We see that all the conditions for existence of interior equilibrium hold and the interior equilibrium point is $D^* = (6.45366, 1.08759, 2.26581, 7.82435)$. Now, all the eigenvalues at D^* are $-2.75187, -0.538162, -0.154566 \pm 0.353358i$. Therefore, according to Routh-Hurwitz criterion the interior equilibrium is locally asymptotically stable, depicted in Figure 1. This figure implies that all the trajectories oscillate initially and after some times they all converge their equilibrium points.

Now if the numerical value of the parameter β_2 is increased then the oscillation in the solution curve increases. In Figure 2, we see that for $\beta_2 = 0.281$ oscillation increases but system (3) remains asymptotically stable around D^* . Again for $\beta_2 = 0.282$ and the other parameter value the same as previous we have an unstable interior equilibrium D^* (see Figure 3). Further increment of β_2 , say, $\beta_2 = 2$, and the other parameter value the same as that before make the system extinction of predator population and system is unstable around D_2 (see Figure 4).

Next to compare systems (3) and (13), taking all the other parameters fixed if we take the value of $\beta_2 = 0.276$ and $d = 0.01$ make the increment in oscillation but system (14) remains asymptotically stable at the interior equilibrium D_* (see Figure 5) but further increment of β_2 , say, $\beta_2 = 0.281$, and all other parameters having the same values make the system unstable around D_* (see Figure 6). For further increment of d , say, for $d = 0.375$, the new pest-free equilibrium is created and it becomes asymptotically stable and this phenomenon is depicted in Figure 7. Therefore the major important observation of the modified model is that if the additional food is supplied as a sufficient amount then the system may be pest-free.

6. Conclusions

In the present paper, we have formulated and discussed a prey-predator model by dividing the prey compartment into three classes, where the disease spreads and developed in two stages. Five equilibria are obtained, namely, trivial, axial, predator-free, infection-free, and interior equilibrium, and the dynamical behavior of the system is investigated. The important critical value K_c of the carrying capacity is obtained and it has been found that when the environmental carrying capacity for susceptible pest population is below K_c , the pest population sharply increases in absence of infection and predator. Furthermore, we have seen that the system enters into a single Hopf bifurcation around the predator-free equilibrium and for large value of the carrying capacity the susceptible pest population may persist for a long time without using pesticide.

Also, our global stability analysis of the system around the disease-free equilibrium indicates that the interaction between susceptible pest and predator will be ensured during long time for their survival. We proved that the interior equilibrium is locally asymptotically stable under some parametric conditions and the system enters into the Hopf bifurcation around the interior equilibrium while the density

FIGURE 1: Stable solution curves.

dependent mortality rate passes through the threshold value ξ^* .

Our main objective is to provide a pest-free system. In this regard, stability of the system at the pest-free equilibrium point is the most targeted job. But in the first model (i.e., system (3)), we observe that there is no pest-free equilibrium point whereas the modified system has a pest-free equilibrium point. In this regard, we may conclude that the additional food on the natural enemy of the pest population has a strong impact to remove the pest population.

Appendices

A. Uniform Boundedness of the System

Let us assume $h_1 > h_2$ and define a function

$$B = S + I_1 + I_2 + \frac{1}{h_1}P. \quad (\text{A.1})$$

Taking the time derivative of (A.1) along the solution of (3), we obtain

$$\begin{aligned} \frac{dB}{dt} = & rS \left(1 - \frac{S}{K}\right) - \left(\frac{h_1 - h_2}{h_1}\right) q I_1 P - \sigma I_1 - \delta I_2 \\ & - \epsilon P - \xi^2 P^2. \end{aligned} \quad (\text{A.2})$$

Therefore, for each $\eta > 0$,

$$\begin{aligned} \frac{dB}{dt} + \eta B \leq & S \left[r \left(1 - \frac{S}{K}\right) + \eta \right] - (\sigma - \eta) I_1 \\ & - (\delta - \eta) I_2 - \frac{\epsilon - \eta}{h_1} P. \end{aligned} \quad (\text{A.3})$$

Let $\eta < \min(\sigma, \delta, \epsilon)$. Then from (A.3), we obtain

$$\frac{dB}{dt} + \eta B \leq S \left[r \left(1 - \frac{S}{K}\right) + \eta \right]. \quad (\text{A.4})$$

FIGURE 2: Stable solution curves and oscillation increases for $\beta_2 = 0.281$.

Now, we assume $h_2 \geq h_1$ and in this case, we consider

$$B = S + I_1 + I_2 + \frac{1}{h_2}P,$$

$$\frac{dB}{dt} = rS \left(1 - \frac{S}{K}\right) - \left(\frac{h_2 - h_1}{h_2}\right) \frac{pSP}{m + S} - \sigma I_1 - \delta I_2 \quad (\text{A.5})$$

$$- \varepsilon P - \xi^2 P^2.$$

Then for $\eta < \min(\sigma, \delta, \varepsilon)$, we have

$$\frac{dB}{dt} + \eta B \leq S \left[r \left(1 - \frac{S}{K}\right) + \eta \right]. \quad (\text{A.6})$$

The maximum value of $rS(1 - S/K) + \eta S$ is $K(r + \eta)^2/4r = \Omega$ (say).

Then (A.6) can be written as

$$\frac{dB}{dt} + \eta B \leq \Omega. \quad (\text{A.7})$$

Applying the theorem of differential inequality [33], we obtain

$$0 < B(S, I_1, I_2, P)$$

$$\leq \frac{\Omega}{\eta} (1 - e^{-\eta t}) + B(S(0), I_1(0), I_2(0), P(0)). \quad (\text{A.8})$$

Thus, for $t \rightarrow \infty$,

$$0 < B \leq \frac{\Omega}{\eta}. \quad (\text{A.9})$$

Hence all the solutions of (3) initiating in R_+^4 are confined in the region

$$\Theta = \left\{ (S, I_1, I_2, P) \in R_+^4 : 0 < B \leq \frac{\Omega}{\eta} \right\}. \quad (\text{A.10})$$

FIGURE 3: Unstable solution curves for $\beta_2 = 0.282$.

B. Local Stability of the Disease-Free Equilibrium

The characteristic equation of system (3) around the disease-free equilibrium is

$$\lambda^4 + x_1\lambda^3 + x_2\lambda^2 + x_3\lambda + x_4 = 0, \quad (\text{B.1})$$

where

$$\begin{aligned} x_1 &= \alpha + \delta + \sigma + S_3 \left(\frac{r}{K} - \beta_1 \right) + P_3 \left[q \right. \\ &\quad \left. - \frac{pS_3}{(m + S_3)^2} \right], \\ x_2 &= q\delta P_3 + \frac{S_3 P_3}{K(m + S_3)^3} \left[Kh_1 m p^2 + (m + S_3) \right. \\ &\quad \cdot (m^2 q r - Kp(\alpha + \delta + \sigma) + 2mqrS_3 + qrS_3^2) \\ &\quad \left. + \frac{p\beta_1 S_3^2 P_3}{(m + S_3)^2} - \frac{pqS_3 P_3^2}{(m + S_3)^2} + \frac{1}{K} [K\delta(\alpha + \sigma) \right. \\ &\quad \left. - r\beta_1 S_3^2 + S_3(r(\alpha + \delta + \sigma) - K\delta\beta_1 - K\alpha\beta_2)] \right], \end{aligned}$$

$$\begin{aligned} x_3 &= \frac{S_3}{K(m + S_3)^3} \left[KpqP_3^2 (h_1 m p - \delta(m + S_3)) \right. \\ &\quad \left. + r(m + S_3)^3 (\delta(\alpha + \sigma) - S_3(\delta\beta_1 + \alpha\beta_2)) \right. \\ &\quad \left. + Kh_1 m p^2 P_3 (\alpha + \delta + \sigma - \beta_1 S_3) + P_3 (m + S_3) \right. \\ &\quad \cdot \{ m^2 q r \delta - kp\delta(\alpha + \sigma) + qr\delta S_3^2 \\ &\quad \left. + S_3 (2mqr\delta + Kp(\delta\beta_1 + \alpha\beta_2)) \} \right], \\ x_4 &= \frac{h_1 m p^2 S_3 P_3}{(m + S_3)^3} (\delta(\alpha + \sigma) + q\delta P_3 - S_3(\delta\beta_1 \\ &\quad + \alpha\beta_2)). \end{aligned} \quad (\text{B.2})$$

Hence for local stability of the disease-free equilibrium D_3 , the following Routh-Hurwitz criterion must be satisfied:

- (i) $x_i > 0$ ($i = 1, 2, 3, 4$),
- (ii) $x_1 x_2 x_3 > x_3^2 + x_1^2 x_4$.

FIGURE 4: Predator extinct for $\beta_2 = 2$.

C. Local Stability and Hopf Bifurcation of the Interior Equilibrium

The characteristic equation of system (3) around the interior equilibrium is

$$\lambda^4 + y_1\lambda^3 + y_2\lambda^2 + y_3\lambda + y_4 = 0, \quad (\text{C.1})$$

where

$$y_1 = \delta + \xi P^* - A_{11} - A_{12},$$

$$y_2 = \delta \xi P^* - \alpha A_{13} - A_{11} (\delta + \xi P^* - A_{22}) - \delta A_{22} - \xi A_{22} P^* + A_{14} A_{41} + q A_{42} I_1^* + \beta_1 A_{21} S^*,$$

$$y_3 = \alpha A_{13} (A_{21} - \xi P^*) - \delta \xi A_{22} P^* + A_{14} A_{41} (\delta - A_{22}) + q \delta A_{42} I_1^* + A_{14} A_{21} A_{42} + A_{11} (\alpha A_{13} + A_{22} (\delta + \xi P^*) - \delta \xi P^* - q A_{42} I_1^*) + \delta \beta_1 A_{21} S^* + \xi \beta_1 A_{21} S^* P^* - q \beta_1 A_{41} S^* I_1^*,$$

$$y_4$$

$$= \alpha A_{13} (\xi A_{21} P^* - A_{41} (q I_1^* + A_{14})) + A_{11} (\xi (\alpha A_{13} + \delta A_{22}) P^* - q \delta A_{42} I_1^*) + \delta A_{14} (A_{21} A_{42} - A_{22} A_{41}) + \delta \beta_1 (\xi A_{21} P^* - q A_{41} I_1^*) S^*,$$

$$A_{11} = \frac{p S^* P^*}{(m + S^*)^2} - \frac{r S^*}{K},$$

$$A_{13} = \beta_2 S^*,$$

$$A_{14} = \frac{p S^*}{m + S^*},$$

$$A_{21} = \frac{I_1^*}{b},$$

$$A_{22} = \left(\beta_1 - \frac{1}{b} \right) S^*,$$

FIGURE 5: Stable solution curve when we supplied an alternating source of food (i.e., revised model) with $d = 0.01$ and $\beta_2 = 0.276$.

$$\begin{aligned} A_{41} &= \frac{h_1 m p P^*}{(m + S^*)^2}, \\ A_{42} &= h_2 q P^*. \end{aligned} \quad (C.2)$$

By Routh-Hurwitz criterion, the interior equilibrium D^* is locally asymptotically stable if $y_i > 0$ ($i = 1, 2, 3, 4$) and $y_1 y_2 y_3 > y_3^2 + y_4 y_1^2$.

Now, we shall find out the conditions for which the system enters into Hopf bifurcation around the interior equilibrium D^* .

Let us define a function $\Gamma : (0, \infty) \rightarrow \mathbb{R}$ of ξ as follows:

$$\Gamma(\xi) = y_1(\xi) y_2(\xi) y_3(\xi) - y_3^2(\xi) - y_4(\xi) y_1^2(\xi). \quad (C.3)$$

The necessary and sufficient conditions for Hopf bifurcation to occur are that there exists $\xi = \xi^*$ such that

- (i) $\Gamma(\xi^*) = 0$,
- (ii) $d\text{Re}(\lambda(\xi))/d\xi \neq 0$.

Note that the critical value ξ^* is obtained from the equation $\Gamma(\xi) = 0$. For $\xi = \xi^*$, characteristic equation (C.1) becomes

$$\left(\lambda^2 + \frac{y_3}{y_1}\right) \left(\lambda^2 + y_1 \lambda + \frac{y_1 y_4}{y_3}\right) = 0. \quad (C.4)$$

Suppose (C.4) has four roots, say, λ_i ($i = 1, 2, 3, 4$), with λ_1, λ_2 being the pair of purely imaginary roots. Then we have

$$\begin{aligned} \lambda_3 + \lambda_4 &= -y_1, \\ \omega_0^2 + \lambda_3 \lambda_4 &= y_2, \\ \omega_0^2 (\lambda_3 + \lambda_4) &= y_3, \\ \omega_0^2 \lambda_3 \lambda_4 &= y_4, \end{aligned} \quad (C.5)$$

where

$$\omega_0 = \text{Im } \lambda_1(\xi^*). \quad (C.6)$$

From the above relations, it is obtained that

$$\omega_0^2 = \frac{y_3}{y_1}. \quad (C.7)$$

FIGURE 6: Unstable solution curves of the revised model when $\beta_2 = 0.281$ and $d = 0.01$.

If λ_3 and λ_4 are complex conjugate, then from above we have $\text{Re}(\lambda_3) = -y_1 < 0$, but if they are real roots, then we get $\lambda_3 < 0$ and $\lambda_4 < 0$. Further, since $\Gamma(\xi)$ is a continuous function of all its roots, then there exists an open interval $(\xi^* - \epsilon, \xi^* + \epsilon)$ containing ξ , where λ_1 and λ_2 are complex conjugate for ξ .

Suppose

$$\lambda_{1,2}(\xi) = \rho_1(\xi) \pm \rho_2(\xi). \quad (\text{C.8})$$

Now, we shall verify the transversality condition

$$\frac{d \text{Re}(\lambda_j(\xi))}{d\xi} \neq 0, \quad j = 1, 2. \quad (\text{C.9})$$

Substituting $\lambda_1(\xi) = \rho_1(\xi) + \rho_2(\xi)$ in (C.1) and calculating the derivative, we get

$$\begin{aligned} E(\xi) \rho_1'(\xi) - F(\xi) \rho_2'(\xi) + G(\xi) &= 0, \\ F(\xi) \rho_1'(\xi) + E(\xi) \rho_2'(\xi) + H(\xi) &= 0, \end{aligned} \quad (\text{C.10})$$

where

$$\begin{aligned} E(\xi) &= 4\rho_1^3 - 12\rho_1\rho_2^2 + 3y_1(\rho_1^2 - \rho_2^2) + 2y_2\rho_1 \\ &\quad + y_3, \\ F(\xi) &= 12\rho_1^2\rho_2 - 4\rho_2^3 + 6y_1\rho_1\rho_2 + 2y_2\rho_2, \\ G(\xi) &= (\rho_1^3 - 3\rho_1\rho_2^2)y_1' + (\rho_1^2 - \rho_2^2)y_2' + \rho_1y_3' \\ &\quad + y_4', \\ H(\xi) &= (3\rho_1^2\rho_2 - \rho_2^3)y_1' + 2\rho_1\rho_2y_2' + \rho_2y_3'. \end{aligned} \quad (\text{C.11})$$

Since $E(\xi)G(\xi) + F(\xi)H(\xi) \neq 0$ at $\xi = \xi^*$, we have

$$\left[\frac{d \text{Re}(\lambda_j(\xi))}{d\xi} \right]_{\xi=\xi^*} = \frac{E(\xi)G(\xi) + F(\xi)H(\xi)}{E^2(\xi) + F^2(\xi)} \neq 0. \quad (\text{C.12})$$

Thus, the Hopf bifurcation conditions are verified.

FIGURE 7: Pest-free solution curves of the revised model for $\beta_2 = 2$ and $d = 0.376$.

Conflict of Interests

The authors declare that there is no conflict of interests regarding the publication of this paper.

References

- [1] F. E. Uboh, E. N. Asuquo, M. U. Eteng, and E. O. Akpanyung, "Endosulfan-induces renal toxicity independent of the route of exposure in rats," *American Journal of Biochemistry and Molecular Biology*, vol. 1, no. 4, pp. 359–367, 2011.
- [2] S. Tang, Y. Xiao, L. Chen, and R. A. Cheke, "Integrated pest management models and their dynamical behaviour," *Bulletin of Mathematical Biology*, vol. 67, no. 1, pp. 115–135, 2005.
- [3] S. Ghosh, S. Bhattacharyya, and D. K. Bhattacharya, "The role of viral infection in pest control: a mathematical study," *Bulletin of Mathematical Biology*, vol. 69, no. 8, pp. 2649–2691, 2007.
- [4] S. Ghosh and D. K. Bhattacharya, "Optimization in microbial pest control: an integrated approach," *Applied Mathematical Modelling*, vol. 34, no. 5, pp. 1382–1395, 2010.
- [5] Y. Pei, X. Ji, and C. Li, "Pest regulation by means of continuous and impulsive nonlinear controls," *Mathematical and Computer Modelling*, vol. 51, no. 5-6, pp. 810–822, 2010.
- [6] J. Liang, S. Tang, and R. A. Cheke, "An integrated pest management model with delayed responses to pesticide applications and its threshold dynamics," *Nonlinear Analysis: Real World Applications*, vol. 13, no. 5, pp. 2352–2374, 2012.
- [7] N. C. Apreutesei, "An optimal control problem for a pest, predator, and plant system," *Nonlinear Analysis. Real World Applications*, vol. 13, no. 3, pp. 1391–1400, 2012.
- [8] T. K. Kar, A. Ghorai, and S. Jana, "Dynamics of pest and its predator model with disease in the pest and optimal use of pesticide," *Journal of Theoretical Biology*, vol. 310, pp. 187–198, 2012.
- [9] S. Jana and T. K. Kar, "Modeling and analysis of a prey-predator system with disease in the prey," *Chaos, Solitons & Fractals*, vol. 47, no. 1, pp. 42–53, 2013.
- [10] S. Jana and T. K. Kar, "A mathematical study of a prey-predator model in relevance to pest control," *Nonlinear Dynamics*, vol. 74, no. 3, pp. 667–683, 2013.
- [11] P. K. Mondal, S. Jana, and T. K. Kar, "A theoretical approach on controlling agricultural pest by biological controls," *Acta Biotheoretica*, vol. 62, no. 1, pp. 47–67, 2014.
- [12] S. Sun and L. Chen, "Mathematical modelling to control a pest population by infected pests," *Applied Mathematical Modelling*, vol. 33, no. 6, pp. 2864–2873, 2009.

- [13] Y. Tan and L. Chen, "Modelling approach for biological control of insect pest by releasing infected pest," *Chaos, Solitons & Fractals*, vol. 39, no. 1, pp. 304–315, 2009.
- [14] X. Wang and X. Song, "Mathematical models for the control of a pest population by infected pest," *Computers & Mathematics with Applications*, vol. 56, no. 1, pp. 266–278, 2008.
- [15] K. P. Haderler and H. I. Freedman, "Predator-prey populations with parasitic infection," *Journal of Mathematical Biology*, vol. 27, no. 6, pp. 609–631, 1989.
- [16] E. Venturino, "Epidemics in predator models: disease among the prey," in *Mathematical Population Dynamics: Analysis of Heterogeneity, Theory of Epidemics*, O. Arino, D. Axelord, M. Kimmel, and M. Langlais, Eds., vol. 1, pp. 381–393, Wuerzt Publishing, Winnipeg, Canada, 1995.
- [17] E. Venturino, "Epidemics in predator-prey models: disease in the predators," *Journal of Mathematics Applied in Medicine and Biology*, vol. 19, no. 3, pp. 185–205, 2002.
- [18] T. K. Kar and P. K. Mondal, "A mathematical study on the dynamics of an eco-epidemiological model in the presence of delay," *Applications and Applied Mathematics*, vol. 7, no. 1, pp. 300–333, 2012.
- [19] X. Liu and C. Wang, "Bifurcation of a predator-prey model with disease in the prey," *Nonlinear Dynamics Systems*, vol. 62, no. 4, pp. 841–850, 2010.
- [20] H. Guo and L. Chen, "Time-limited pest control of a Lotka-Volterra model with impulsive harvest," *Nonlinear Analysis. Real World Applications*, vol. 10, no. 2, pp. 840–848, 2009.
- [21] Y. Zhang and Q. Zhang, "Dynamical behavior in a delayed stage-structure population model with stochastic fluctuation and harvesting," *Nonlinear Dynamics*, vol. 66, no. 1-2, pp. 231–245, 2011.
- [22] X. Wang, Y. Tao, and X. Song, "Analysis of pest-epidemic model by releasing diseased pest with impulsive transmission," *Nonlinear Dynamics*, vol. 65, no. 1-2, pp. 175–185, 2011.
- [23] T. Wang and L. Chen, "Nonlinear analysis of a microbial pesticide model with impulsive state feedback control," *Nonlinear Dynamics*, vol. 65, no. 1, pp. 1–10, 2011.
- [24] U. K. Pahari and T. K. Kar, "Conservation of a resource based fishery through optimal taxation," *Nonlinear Dynamics*, vol. 72, no. 3, pp. 591–603, 2013.
- [25] P. D. N. Srinivasu and B. S. R. V. Prasad, "Role of quantity of additional food to predators as a control in predator-prey systems with relevance to pest management and biological conservation," *Bulletin of Mathematical Biology*, vol. 73, no. 10, pp. 2249–2276, 2011.
- [26] P. D. N. Srinivasu, B. S. R. V. Prasad, and M. Venkatesulu, "Biological control through provision of additional food to predators: a theoretical study," *Theoretical Population Biology*, vol. 72, no. 1, pp. 111–120, 2007.
- [27] M. W. Sabelis and P. C. J. van Rijn, "When does alternative food promote biological pest control?" in *Proceedings Second International Symposium on Biological Control of Arthropods*, M. S. Hoddle, Ed., vol. 1, pp. 428–437, USDA Forest Service, 2005.
- [28] J. D. Harwood and J. J. Obryeki, "The role of alternating prey in sustaining predator populations," in *Proceedings Second International Symposium on Biological Control of Arthropods, II*, M. S. Hoddle, Ed., pp. 4253–4262, 2005.
- [29] K. P. Das, S. Roy, and J. Chattopadhyay, "Effect of disease-selective predation on prey infected by contact and external sources," *BioSystems*, vol. 95, no. 3, pp. 188–199, 2009.
- [30] S. Bhattacharyya and D. K. Bhattacharya, "Pest control through viral disease: mathematical modeling and analysis," *Journal of Theoretical Biology*, vol. 238, no. 1, pp. 177–196, 2006.
- [31] E. Beretta, G. I. Bischi, and F. Solimano, "Oscillations in a system with material cycling," *Journal of Mathematical Biology*, vol. 26, no. 2, pp. 143–167, 1988.
- [32] J. Guckenheimer and P. J. Holmes, *Nonlinear Oscillations, Dynamical Systems, and Bifurcations of Vector Fields*, Springer, New York, NY, USA, 1983.
- [33] G. Birkoff and G. C. Rota, *Ordinary Differential Equations*, Ginn, Boston, Mass, USA, 1982.