Mathematical Biology and Bioinformatics
2020. V. 15. № 2. P. 268–294. doi: 10.17537/2020.15.268

------------------------------- MATHEMATICAL MODELING -----------------------------

Modeling of Insect-Pathogen Dynamics with Biological Control
Sangeeta Saha*, Guruprasad Samanta**
Department of Mathematics, Indian Institute of Engineering Science and Technology, Shibpur, Howrah - 711103, India

Abstract. In this work, a model is proposed to analyze the effect of wild plant species on biologically-based technologies for pest control. It is assumed that the pest species have a second food source (wild host plants) except crops. Analytical results prove that the model is well-posed as the system variables are non-negative and uniformly bounded. The permanence of the system has been verified. Equilibrium points and corresponding stability analysis have also been performed. Numerical figures have supported the fact that the interior steady state if it exists, remains stable for any transmission rate. Henceforth biological control has a stabilizing effect. Furthermore, the results prove that biological control is beneficial not only for wild plants but for crops too.

Key words: eco-epidemic model, modified Leslie–Gower functional response, extinction scenarios, bifurcation, global stability.

INTRODUCTION

Theoretical and applied ecologists, these days, give attention to biologically-based technologies to control the pest population. It is assumed that these technologies do not have such harmful effects on human health or on the environment [1, 2]. Use of microbial pesticides is one of the important methods of bio-control. It is relatively stable formulations of micro-organisms which extinguish pests by producing poisons, causing disease, preventing the establishment of other microorganisms and etc. [3, 4, 5]

Many experimental and theoretical experiments have been done on biological control based on microbial pathogens of the pest population. Anderson and May [6, 7], in their work, have shown that pathogen can regulate insect population. Insect-pathogen interactions model is mainly classified on the basis of population dynamics of the host species as well as transmission dynamics of the disease. Some studies consider that the insect population grows exponentially in absence of any disease [5, 8, 9]. This assumption is quite justifiable in the agricultural system as one seeks to control the insect below the crop-damage threshold there. Some studies [10, 11, 12] reveal that, in the forestry systems and also in field populations, self-mechanism of the insect species affect the dynamics of the insect-pathogen interaction. Begon et al. in their work [12] indicates that inclusion of density dependence makes a situation of coexistence of host species. Other studies are based on the assumptions of stage-dependent susceptibility which occurs in the insect population [8]. Moerbeek and Van Den Bosch [13] comes to the conclusion (based on experiment) that the dynamics of insect-pathogen systems depend mainly on that stage which is preferable (susceptible) to the disease.

In disease transmission models, it is known that the susceptible population becomes infectious either through direct contact between susceptible and infective individuals [9, 10, 14]
or through encountering with the free-living infective stage in the environment \[15, 16, 17\]. All these models assume that the insect pest has only crop as its food resource and their birth rates depend on the amount of crop growth.

It is observed that many insect pest species do not feed only on crop but they also choose more wild host plant species as their food resource. This additional food resource effects the insect-pathogen models for biological control. This work, therefore, is formulated in such a way that the bio-control of a pest can be studied who live mainly on a crop species but have a second host plant species occurring in the environment. Here we have studied the effect of the wild host plant (using pathogen as biotic insecticides) on insect population in a bio-control program. Also, the effect of a bio-control program on the dynamics of a wild host plant species has been analyzed.

There are already some research papers published on eco-epidemiological systems \[18, 19, 20, 21, 22, 23\]. Xiao and Chen \[24, 25\] and others in their work consider only those systems where disease spreads only in the prey species and the predator has a unique food resource. On the other hand, Venturino \[23\] analyzes a prey-predator model where the predator is effected with a disease. Compare with that model, an eco-epidemiological model has been considered here and the herbivore pest species is affected by a disease. The pests usually live on crop and on wild plant species. So it is logical to assume that pest species declines with density dependence and hence an eco-epidemiological model can be proposed concerning biological control.

This work is categorized as follows: section 2 describes the mathematical formulation with positive initial conditions. Section 3 proves that the model is well-posed. Extinction conditions for all species have been analysed in section 4. Section 5 contributes to the nature of possible equilibria with corresponding feasibility conditions and the persistence of the system has been shown in Section 6. Section 7 gives local stability analysis of the equilibria. Locally stable equilibria change their stability behaviour through transcritical bifurcation under some restrictions which have been analysed in section 8. The subsequent section gives the global stability of the equilibrium points. In Section 10, numerical results are obtained by varying some of the vital system parameters. In the final discussion section, a brief conclusion has been provided where we have interpreted our results in terms of their ecological implications.

### 2. MATHEMATICAL MODEL: BASIC EQUATIONS

Formulating a biological system in mathematical terms is essential to analyse the dynamical nature of the system with time. But any ecological system is not so easy to describe as there are many controlling factors present in the system. So before presenting a system mathematically, we need to make some assumptions to make it simple.

Let \( Y(T) \) be the biomass of insect pest at time \( T \) which lives on the crop and also on wild plant species. First, it is assumed that the herbivore pest grows according to logistic curve with carrying capacity \( K_2 \) and growth rate \( r_2 \). So, we have

\[
\frac{dY}{dT} = r_2 \left( 1 - \frac{Y}{K_2} \right) Y. \tag{1}
\]

Insect population lives among a various range of potential food sources. Let, \( X(T) \) be the density of wild plant species at time \( T \). It can be assumed as the most preferred food sources to herbivore pest. In absence of pest population, let the wild plant grows logistically with carrying capacity \( K_1 \) and growth rate \( r_1 \). Moreover, most of the time, carrying capacity of pest population depends on plant’s biomass, i.e., the carrying capacity of herbivore species is proportional to the biomass of wild plants. In case of severe scarcity, the insect pests can choose other food source for surviving. But as the wild plants are taken as the most favourite food source and they are limited, so, the growth of pest population is also limited. In order to make the model more
realistic, let us assume that the herbivore pests grow with modified Leslie-Gower scheme \cite{26} and the plant-herbivore model takes the form:

\[
\begin{align*}
\frac{dX}{dT} &= r_1 X \left(1 - \frac{X}{K_1}\right) - B_1 XY, \\
\frac{dY}{dT} &= r_2 \left(1 - \frac{Y}{K_2 + AX}\right) Y.
\end{align*}
\] (2)

Here \( B_1 \) denotes the maximum per capita reduction rate of plant species. \( A \) is the conversion fraction of plants into pests and \( K_2 \) is the extent to which environment provides protection to the herbivores by providing alternative food source. Hence \( K_2 + AX \) represents the carrying capacity set by environmental resources and wild plant. It is noted that crop species has no dynamics of its own, i.e., it is always available in constant amounts. This assumption is considerable as crop number does not increase due to reproduction and it is completely under control of farmer. The farmer saws and harvests the crop according to his demand. So, there is no need to model the crop species explicitly, but its effect on insect biomass is explicit in the model which is considered in original carrying capacity of herbivores.

Sometimes pest pathogen is introduced in pest species which acts as a biotic insecticide. In this situation, the pest population is divided into two sub-population: susceptible pest population \((Y_1)\) and infected pest population \((Y_2)\). So, at time \( T \), overall pest population become \( Y(T) = Y_1(T) + Y_2(T) \).

In this work, it is assumed that only healthy pest species \((Y_1)\) can reproduce. However, the infective pest \( Y_2 \) still contributes with \( Y_1 \) to population growth toward the carrying capacity of healthy pest. \( M \) is the transmission rate where incidence is taken to be followed simple mass action rule. Let \( N \) be the mortality rate of infected pest population. Summing up all the assumptions, plant-insect-pathogen model is as follows:

\[
\begin{align*}
\frac{dX}{dT} &= r_1 X \left(1 - \frac{X}{K_1}\right) - B_1 XY_1 - B_2 XY_2, \quad X(0) = X_0 > 0, \\
\frac{dY_1}{dT} &= r_2 \left(1 - \frac{Y_1 + Y_2}{K_2 + AX}\right) Y_1 - MY_1 Y_2, \quad Y_1(0) = Y_{1,0} > 0, \\
\frac{dY_2}{dT} &= MY_1 Y_2 - NY_2, \quad Y_2(0) = Y_{2,0} > 0,
\end{align*}
\] (3)

where \( B_1, B_2 \) denote the attack rate of healthy and infected pest on plant species respectively. All the system parameters are assumed to be positive.

Let us take the following scaling: \( x = \frac{X}{K_1}, \ y_1 = \frac{Y_1}{AX}, \ y_2 = \frac{Y_2}{AX}, \ t = r_1 T \). Then the system becomes:

\[
\begin{align*}
\frac{dx(t)}{dt} &= x(1 - x) - b_1 xy_1 - b_2 xy_2, \quad x(0) = x_0 > 0, \\
\frac{dy_1(t)}{dt} &= r \left(1 - \frac{y_1 + y_2}{k + x}\right) y_1 - my_1 y_2, \quad y_1(0) = y_{1,0} > 0, \\
\frac{dy_2(t)}{dt} &= my_1 y_2 - ny_2, \quad y_2(0) = y_{2,0} > 0,
\end{align*}
\] (4)

where \( r = \frac{r_2}{r_1}, \ b_1 = \frac{AB_1 K_1}{r_1}, \ b_2 = \frac{AB_2 K_1}{r_1}, \ k = \frac{K_2}{AX}, \ m = \frac{AMK_1}{r_1} \) and \( n = \frac{N}{r_1} \).

\[3. \text{ NON-NEGATIVITY AND BOUNDEDNESS}\]

Non-negativity and boundedness of system \( (4) \) ensure whether the model is well-posed.
**Theorem 1.** All solutions of system (4) starting in $\mathbb{R}^3_+$ are non-negative for all time.

**Proof.** As the functions in right hand side of system (4) are continuous and locally Lipschitzian on $\mathbb{C}$ (space of continuous functions), so the unique solution $(x(t), y_1(t), y_2(t))$ of the system exists on $[0, \kappa)$, where $0 < \kappa \leq +\infty$ [27].

Let us show that, $x(t) \geq 0$, $\forall t \in [0, \kappa)$. If the statement does not hold, then $\exists t_1 \in [0, \kappa)$ such that $x(t_1) = 0$, $\dot{x}(t_1) < 0$ and $x(t) > 0$, $\forall t \in [0, t_1)$. Now the first equation of (4) gives

$$\frac{dx}{dt}\bigg|_{t=t_1} = x(t_1)[1 - x(t_1) - b_1y_1(t_1) - b_2y_2(t_1)] = 0,$$

which is a contradiction to $\dot{x}(t_1) < 0$. So, $x(t) \geq 0$, $\forall t \in [0, \kappa)$.

Similarly, we have $y_1(t) \geq 0$ and $y_2(t) \geq 0$, for all $t \in [0, \kappa)$, where $0 < \kappa \leq +\infty$. Hence the theorem.

**Theorem 2.** All solutions of system (4) which start in $\mathbb{R}^3_+$ are uniformly bounded for all time.

**Proof.** From the first equation of (4):

$$\frac{dx(t)}{dt} = x(1 - x) - b_1xy_1 - b_2xy_2 \leq x(1 - x) \Rightarrow \limsup_{t\to\infty} x(t) \leq 1.$$

Again

$$\frac{dy_1(t)}{dt} = r \left(1 - \frac{y_1 + y_2}{k + x}\right)y_1 - my_1y_2 \leq r \left(1 - \frac{y_1}{k + 1}\right)y_1 \Rightarrow \limsup_{t\to\infty} y_1(t) \leq (k + 1).$$

Let, $W(t) = y_1(t) + y_2(t)$. So,

$$\frac{dW}{dt} = \frac{dy_1}{dt} + \frac{dy_2}{dt} = r \left(1 - \frac{y_1 + y_2}{k + x}\right)y_1 - ny_1y_2 \leq r y_1 \left(1 - \frac{y_1}{k + 1}\right) - ny_1 \left(2 - \frac{y_1}{k + 1}\right) - \tau W,$$

where, $\tau = \min\{r, n\} \leq 2r(k + 1) - \tau W$. Then

$$W(t) \leq \frac{2r(k + 1)}{\tau} (1 - \exp(-\tau t)) + W(x_0, y_{1,0}, y_{2,0}) \exp(-\tau t),$$

as $t \to \infty$, $0 < W(t) \leq \frac{2r(1+k)}{\tau}$.

So, all the solutions of system (4) will enter into the region:

$$\Omega = \left\{(x, y_1, y_2) \in \mathbb{R}^3_+ : 0 < x(t) \leq 1; 0 < y_1(t) \leq (k + 1); 0 < W(t) < \frac{2r(k + 1)}{\tau} + \epsilon, \epsilon > 0\right\}.$$

**4. Extinction Scenarios**

It may happen that under some certain parametric restrictions, the population will tend to extinction with time. In this section we try to obtain those restrictions for which one or more species will be washed out from the system in long run.

Let us denote the following notations: $\bar{x} = \limsup_{t\to\infty} x(t)$; $\bar{y}_1 = \limsup_{t\to\infty} y_1(t)$; $\bar{y}_2 = \limsup_{t\to\infty} y_2(t)$. Similarly, $\underline{x} = \liminf_{t\to\infty} x(t)$; $\underline{y}_1 = \liminf_{t\to\infty} y_1(t)$; $\underline{y}_2 = \liminf_{t\to\infty} y_2(t)$.

Here we use the following facts:

(i) $\bar{x} \leq 1$;

(ii) $\bar{y}_1 \leq \frac{r}{2}$;

(iii) $\bar{y}_2 \leq \frac{r}{2}$;

(iv) $\underline{x} = 0$;

(v) $\underline{y}_1 = 0$;

(vi) $\underline{y}_2 \leq \frac{r}{2}$.

\[ \text{Mathematical Biology and Bioinformatics. 2020. V. 15.№ 2. doi: 10.17537/2020.15.268} \]
(ii) $y_1, y_2 \leq M$ (say), as all solutions are uniformly bounded.

The first two theorems will give us the extinction criterion of plant species while later two will show the extinction of susceptible pest population and the last theorem will give us the condition for extinction of infected pest population.

**Theorem 3.** If $b_1 y_1 > 1$, then $\lim_{t \to \infty} x = 0$.

**Proof.** Choose $0 < \epsilon < 1 - \frac{1}{b_1}$, $\exists T > 0$, s.t. $y_1 > y_1 - \epsilon$, $\forall t > T$.

For $t > T$:

$$
\frac{dx(t)}{dt} = x(1 - x) - b_1 x y_1 - b_2 x y_2,
$$

$$
< x(1 - x) - b_1 x (y_1 - \epsilon),
$$

$$
< x[1 - b_1 (y_1 - \epsilon)]
$$

$$
= -\mu x \quad \text{where,} \quad \mu = b_1 \left\{ y_1 - \frac{1}{b_1} - \epsilon \right\} > 0
$$

$\Rightarrow \lim_{t \to \infty} x(t) = 0$.

**Remark:** If the consumption of plant by healthy pest ($b_1 y_1$) become higher than growth rate of plant species, then automatically the plant species will be washed out from the system.

**Theorem 4.** If $b_2 y_2 > 1$, then $\lim_{t \to \infty} x = 0$.

**Proof.** Choose $0 < \epsilon < 1 - \frac{1}{b_2}$, $\exists T > 0$, s.t. $y_2 > y_2 - \epsilon$, $\forall t > T$.

For $t > T$:

$$
\frac{dx(t)}{dt} = x(1 - x) - b_1 x y_1 - b_2 x y_2,
$$

$$
< x(1 - x) - b_2 x (y_2 - \epsilon),
$$

$$
< x[1 - b_2 (y_2 - \epsilon)]
$$

$$
= -\mu x \quad \text{where,} \quad \mu = b_2 \left\{ y_2 - \frac{1}{b_2} - \epsilon \right\} > 0
$$

$\Rightarrow \lim_{t \to \infty} x(t) = 0$.

**Remark:** If the infected pest consumes the plant species at a higher rate, then plant population will be going to extinct from the system in long run.

**Theorem 5.** If $my_2 > r$, then $\lim_{t \to \infty} y_1(t) = 0$.

**Proof.** Choose $0 < \epsilon < y_2 - \frac{r}{m}$, $\exists T > 0$, s.t. $y_2 > y_2 - \epsilon$, $\forall t > T$.

For $t > T$:

$$
\frac{dy_1(t)}{dt} = r \left( 1 - \frac{y_1 + y_2}{k + x} \right) y_1 - m y_1 y_2,
$$

$$
< r y_1 - m y_1 y_2,
$$

$$
< \left\{ r - m(y_2 - \epsilon) \right\} y_1,
$$

$$
= -\mu y_1 \quad \text{where,} \quad \mu = m \left\{ y_2 - \frac{r}{m} - \epsilon \right\} > 0
$$

$\Rightarrow \lim_{t \to \infty} y_1(t) = 0$.

**Remark:** If the infected pest start to dominate the healthy pest population, then ultimately
susceptible pest will be washed out from the system.

**Theorem 6.** If \( y_1 > k + 2 \), then \( \lim_{t \to \infty} y_1 = 0 \).

**Proof.** Choose \( 0 < \epsilon < 1, \exists T_1 > 0 \), s.t. \( x(t) < 1 + \epsilon \), \( \forall t > T_1 \). Also, for \( 0 < \epsilon_1 < y_1 - k - 2 \), \( \exists T_2 > 0 \), s.t. \( y_1 > y_1 - \epsilon_1 \), \( \forall t > T_2 \).

For \( t > \max\{T_1, T_2\} \):

\[
\frac{dy_1(t)}{dt} = r \left( 1 - \frac{y_1 + y_2}{k + x} \right) y_1 - my_1 y_2.
\]

\[
< ry_1 \left( 1 - \frac{y_1}{k + x} \right),
\]

\[
< ry_1 \left( 1 - \frac{y_1 - \epsilon_1}{k + 1 + \epsilon} \right),
\]

\[
< ry_1 \left( 1 - \frac{y_1 - \epsilon_1}{k + 2} \right),
\]

\[
= -\mu y_1 \left[ \text{where, } \mu = \frac{r}{k + 2} \left\{ \frac{y_1 - k - 2 - \epsilon}{y_1} \right\} > 0 \right]
\]

\( \Rightarrow \lim_{t \to \infty} y_1(t) = 0. \)

**Remark:** If the amount of susceptible pest become high enough, then due to lack of food it will be going to extinct.

**Theorem 7.** If \( my_1 < n \), then \( \lim_{t \to \infty} y_2 = 0 \).

**Proof.** Choose \( 0 < \epsilon < \frac{n}{m} - \bar{y}_1 \), \( \exists T > 0 \), s.t. \( y_1 < \bar{y}_1 + \epsilon \), \( \forall t > T \).

For \( t > T \):

\[
\frac{dy_2(t)}{dt} = my_1 y_2 - ny_2,
\]

\[
< \left\{ m(\bar{y}_1 + \epsilon) - n \right\} y_2,
\]

\[
= -\mu y_2 \left[ \text{where, } \mu = m \left\{ \frac{n}{m} - \bar{y}_1 - \epsilon \right\} > 0 \right]
\]

\( \Rightarrow \lim_{t \to \infty} y_2(t) = 0. \)

**Remark:** If the transmission rate \( my_1 \) fails to overcome the mortality rate of infected pest, then there will be no more infected pest present in the system in long run.

### 5. EQUILIBRIUM POINTS

To obtain the equilibrium points we need to solve the nullclines of a model. System (4) gives the equilibrium points as follows:

1. **Trivial Equilibrium Point:** \( E_0(0, 0, 0) \).

2. **Axial Equilibrium Points:** \( E_1(1, 0, 0), E_2(0, k, 0) \).

3. **Planar Equilibrium Points:** \( E_3(\bar{x}, \bar{y}_1, 0) = \left( \frac{1 - kb_1}{1 + b_1}, \frac{1 + k}{1 + b_1}, 0 \right) \) and \( E_4(0, \bar{y}_1, \bar{y}_2) = \left( 0, \frac{n}{m}, \frac{r(km - n)}{m(km + r)} \right) \). \( E_3 \) is feasible when \( kb_1 < 1 \) and \( E_4 \) is feasible when \( km > n \).
4. Interior Equilibrium Point \( E^*(x^*, y^*_1, y^*_2) \) satisfies the equations:

\[
1 - x - b_1 y_1 - b_2 y_2 = 0,
\]
\[
r \left( 1 - \frac{y_1 + y_2}{k + x} \right) - m y_2 = 0,
\]
and \( my_1 - n = 0 \).

Solving: \( E^*(x^*, y^*_1, y^*_2) = \left( \frac{(1-k)m-b_1n-(b_2+1)r+\sqrt{\Delta}}{2m}, \frac{(1+k)m-b_1n+(b_2+1)r+\sqrt{\Delta}}{2b_2m} \right) \), where \( \Delta = \{ m(1 + k) + (b_2 + 1)r - b_1n \}^2 - 4rb_2\{ m(1 + k) - n(1 + b_1) \} \). So, we may get two interior equilibrium points: \( E^*_L(x^*_1, y^*_1, y^*_2) \) and \( E^*_R(x^*_R, y^*_1R, y^*_2R) \), where

\[
E^*_L = \left( \frac{(1-k)m-b_1n-(b_2+1)r-\sqrt{\Delta}}{2m}, \frac{(1+k)m-b_1n+(b_2+1)r-\sqrt{\Delta}}{2b_2m} \right)
\]
and

\[
E^*_R = \left( \frac{(1-k)m-b_1n-(b_2+1)r+\sqrt{\Delta}}{2m}, \frac{(1+k)m-b_1n+(b_2+1)r-\sqrt{\Delta}}{2b_2m} \right).
\]

Two feasible interior equilibrium points can be obtained only if \( (1-k)m-b_1n-(b_2+1)r-\sqrt{\Delta} > 0 \) hold simultaneously along with \( A > 0 \). Otherwise, the system have only one feasible interior equilibrium.

6. PERSISTENCE

An ecological system is persistent (permanence) means all the populations in the system survive in long run, no matter what the initial populations are. Mathematically, it implies that strictly positive solutions do not have omega limit points on the boundary of the non-negative cone. Usually, average Lyapunov function is used to show the permanence of a system [28].

**Theorem 8.** System (4) is permanent if the following conditions hold

(i) \( 1 - b_1k > 0 \) and/or \( mk > n \);

(ii) \( m(1 + k) > n(1 + b_1) \);

(iii) \( m(km + r) > b_1n(km + r) + b_2r(km - n) \).

**Proof.** Let the average Lyapunov function is \( V(x, y_1, y_2) = x^\theta_1 y_1^{\theta_2} y_2^{\theta_3} \) where each \( \theta_i \) for \( i = 1, 2, 3 \) are assumed to be positive. In the interior of \( \mathbb{R}^3_+ \), we have

\[
\dot{V} = \phi(x, y_1, y_2) = \theta_1[1 - x - b_1 y_1 - b_2 y_2] + \theta_2 \left[ r \left( 1 - \frac{y_1 + y_2}{k + x} \right) - m y_2 \right] + \theta_3[my_1 - n].
\]

To prove the permanence, we need to show \( \phi(x, y_1, y_2) > 0 \) for all boundary equilibria of the system. The values of \( \phi(x, y_1, y_2) \) at the boundary equilibria \( E_0, E_1, E_2, E_3 \) and \( E_4 \) are as follows:

\[
E_0: \phi(0, 0, 0) = \theta_1 + \theta_2r - \theta_3n.
\]
\[
E_1: \phi(1, 0, 0) = \theta_2r - \theta_3n.
\]
\[
E_2: \phi(0, k, 0) = \theta_1(1 - kb_1) + \theta_3(mk - n).
\]
\[
E_3: \phi(\bar{x}, \bar{y}_1, 0) = \theta_3 \left[ \frac{m(1 + k)}{1 + b_1} - n \right].
\]
\[
E_4: \phi(0, \bar{y}_1, \bar{y}_2) = \theta_1 \left[ \frac{m(km + r) - b_1n(km + r) - b_2r(km - n)}{m(km + r)} \right].
\]

Now, \( \phi(0, 0, 0) \) and \( \phi(1, 0, 0) \) are positive for some positive \( \theta_i \) for \( i = 1, 2, 3 \). And if the inequalities stated in (i) – (iii) hold, then \( \phi \) is positive at \( E_0, E_1, E_2, E_3 \) and \( E_4 \) for some \( \theta_i > 0 \) for \( i = 1, 2, 3 \). So, system (4) is permanent [29] if the conditions (i) – (iii) are fulfilled.

274
7. LOCAL STABILITY ANALYSIS

Now we look for the local stability conditions of the equilibrium points with the help of corresponding Jacobian matrices and Routh-Hurwitz criterion. The Jacobian matrix of system (4) is

$$J = \begin{pmatrix} a_{11} & a_{12} & a_{13} \\ a_{21} & a_{22} & a_{23} \\ a_{31} & a_{32} & a_{33} \end{pmatrix},$$

where

$$a_{11} = 1 - 2x - b_1y_1 - b_2y_2; \quad a_{12} = -b_1x; \quad a_{13} = -b_2x; \quad a_{21} = \frac{ry_1(y_1+y_2)}{(k+x)^2}; \quad a_{22} = r - \frac{r(2y_1+y_2)}{k+x} - my_2; \quad a_{23} = -\left(\frac{r}{k+x} + m\right)y_1; \quad a_{31} = 0; \quad a_{32} = my_2; \quad a_{33} = my_1 - n.$$

For $E_0 = (0, 0, 0)$:

$$J|_{E_0} = \begin{pmatrix} 1 & 0 & 0 \\ 0 & r & 0 \\ 0 & 0 & -n \end{pmatrix}.$$

So, $\lambda_1 = 1, \quad \lambda_2 = r, \quad \lambda_3 = -n$. Here $\lambda_1, \lambda_2$ are always positive and hence we have the following theorem:

**Theorem 9.** $E_0$ is always an unstable equilibrium point.

For $E_1 = (1, 0, 0)$:

$$J|_{E_1} = \begin{pmatrix} -1 & -b_1 & -b_2 \\ 0 & r & 0 \\ 0 & 0 & -n \end{pmatrix}.$$

So, $\lambda_1 = -1, \quad \lambda_2 = r, \quad \lambda_3 = -n$. Here $\lambda_1, \lambda_3$ are always negative but $\lambda_2$ is positive. So we have the following theorem:

**Theorem 10.** $E_1$ is an unstable equilibrium point.

For $E_2 = (0, k, 0)$:

$$J|_{E_2} = \begin{pmatrix} 1 - b_1k & 0 & 0 \\ r & -r & -k(m + \frac{x}{k}) \\ 0 & 0 & mk - n \end{pmatrix}.$$

So, $\lambda_1 = 1 - b_1k, \quad \lambda_2 = -r, \quad \lambda_3 = mk - n$. Here $\lambda_2$ is always negative. So we have the following theorem:

**Theorem 11.** $E_2$ is a stable equilibrium point if $b_1k > 1$ along with $mk < n$ hold.

For $E_3 = (\tilde{x}, \tilde{y}_1, 0)$:

$$J|_{E_3} = \begin{pmatrix} a_{11} & a_{12} & a_{13} \\ a_{21} & a_{22} & a_{23} \\ 0 & 0 & a_{33} \end{pmatrix},$$

where $a_{11} = -\tilde{x}; \quad a_{12} = -b_1\tilde{x}; \quad a_{13} = -b_2\tilde{x}; \quad a_{21} = \frac{r\tilde{y}_1^2}{(k+x)^2}; \quad a_{22} = -\frac{r\tilde{y}_1}{k+x}; \quad a_{23} = -\left(\frac{r}{k+x} + m\right)\tilde{y}_1; \quad a_{33} = m\tilde{y}_1 - n$. One eigenvalue will be

$$\lambda_1 = m\tilde{y}_1 - n$$

(6)
and other two will be the roots of the quadratic equation:

$$\lambda^2 + P_1\lambda + P_2 = 0,$$

where $P_1 = -(a_{11} + a_{22}) > 0, P_2 = -a_{21}a_{12} > 0$. Therefore, $E_3$ is stable if $\lambda_1 < 0$, i.e., $m(1 + k) < n(1 + b_1)$. So, we have the following theorem:

**Theorem 12.** $E_3$ is locally asymptotically stable (LAS) if $m(1 + k) < n(1 + b_1)$ holds.

For $E_4 = (0, \bar{y}_1, \bar{y}_2)$:

$$J|_{E_4} = \begin{pmatrix}
    a_{11} & 0 & 0 \\
    a_{21} & a_{22} & a_{23} \\
    0 & a_{32} & 0
\end{pmatrix},$$

where $a_{11} = 1 - b_1\bar{y}_1 - b_2\bar{y}_2$; $a_{21} = \frac{r\eta_1(\eta_1 + \eta_2)}{k^2}$; $a_{22} = -\frac{r\eta_1}{k}$; $a_{23} = -\left(\frac{x}{k} + m\right)\bar{y}_1$; $a_{32} = m\bar{y}_2$.

One eigenvalue will be

$$\lambda_1 = 1 - b_1\bar{y}_1 - b_2\bar{y}_2$$

(7)

and other two will be the roots of the quadratic equation:

$$\lambda^2 + Q_1\lambda + Q_2 = 0,$$

where $Q_1 = -a_{22} > 0, Q_2 = -a_{23}a_{32} > 0$. Therefore, $E_4$ is stable if $\lambda_1 < 0$, i.e., $(m - b_1n)(km + r) < b_2r(km - n)$. So, we have the following theorem:

**Theorem 13.** $E_4$ is locally asymptotically stable (LAS) if $(m - b_1n)(km + r) < b_2r(km - n)$ holds.

Now, for $E^*(x^*, y_1^*, y_2^*)$:

$$J|_{E^*} = \begin{pmatrix}
    a_{11} & a_{12} & a_{13} \\
    a_{21} & a_{22} & a_{23} \\
    a_{31} & a_{32} & a_{33}
\end{pmatrix},$$

where $a_{11} = -x^*$; $a_{12} = -b_1x^*$; $a_{13} = -b_2x^*$; $a_{21} = \frac{r\eta_1^*(\eta_1^* + \eta_2^*)}{(k + \sigma)^2}$; $a_{22} = -\frac{r\eta_1^*}{k + \sigma}$; $a_{23} = -\left(\frac{x}{k + \sigma} + m\right)y_1^*$; $a_{31} = 0$; $a_{32} = my_2^*$; $a_{33} = 0$. Characteristic equation for $E^*(x^*, y_1^*, y_2^*)$ will be

$$\lambda^3 + B_1\lambda^2 + B_2\lambda + B_3 = 0, \quad (8)$$

where $B_1 = -(a_{11} + a_{22})$ $B_2 = a_{11}a_{22} - a_{12}a_{21} - a_{23}a_{32}$, $B_3 = -a_{32}(a_{13}a_{21} - a_{11}a_{23})$. Here $B_1, B_2, B_3 > 0$. Let, $\Delta = B_1B_2 - B_3$.

According to Routh-Hurwitz criterion, all roots of equation (8) have negative real parts if $B_1 > 0$, $B_3 > 0$ and $B_3(B_1B_2 - B_3) > 0$. So, we have the following theorem:

**Theorem 14.** $E^*(x^*, y_1^*, y_2^*)$ will be LAS if $\Delta = B_1B_2 - B_3 > 0$.

8. BIFURCATION ANALYSIS

Here we have analysed under which conditions the equilibrium points will change their stability and for this we have used Sotomayor’s Theorem [30] and the Hopf Bifurcation Theorem [31]. In order to apply Sotomayor’s Theorem, one of the eigenvalues of the Jacobian matrix at the equilibrium point needs to be zero.

Let $V = (v_1, v_2, v_3)^T$ and $W = (w_1, w_2, w_3)^T$ be the eigenvectors of $J|_{(eq.\ point)}$ and $J|_{(eq.\ point)}^T$ corresponding to zero eigenvalue of the equilibrium point respectively.

Let $f = (f^{(1)}, f^{(2)}, f^{(3)})^T$, where

Mathematical Biology and Bioinformatics. 2020. V. 15. Nº 2. doi: 10.17537/2020.15.268
\( f^{(1)} = x(1 - x) - b_1xy_1 - b_2xy_2, \)
\( f^{(2)} = r \left( 1 - \frac{y_1 + y_2}{k + x} \right) y_1 - my_1y_2, \)
\( f^{(3)} = my_1y_2 - ny_2. \)

**Theorem 15.** The system undergoes a transcritical bifurcation with respect to the bifurcation parameter \( b_1 \) around \( E_2(0, k, 0) \) if \( kb_1 = 1 \) along with \( mk < n \).

**Proof.**

\[ J|_{E_2} = \begin{pmatrix} 1 - b_1k & 0 & 0 \\ r & -r & -k \left( m + \frac{r}{k} \right) \\ 0 & 0 & mk - n \end{pmatrix}. \]

Let, \( b_{1|TC_1} \) be the value of \( b_1 \) s.t \( J|_{E_2} \) has a simple zero eigenvalue at \( b_1 = b_{1|TC_1} \).

So, at \( b_1 = b_{1|TC_1} = k^{-1} : \)

\[ J|_{E_2} = \begin{pmatrix} 0 & 0 & 0 \\ r & -r & -(r + mk) \\ 0 & 0 & mk - n \end{pmatrix}. \]

Here, \( \lambda_1 = -r < 0 \) and \( \lambda_2 = mk - n < 0. \)

After some calculations: \( V = (1, 1, 0)^T \) and \( W = (1, 0, 0)^T \).

Therefore,

\[ \Omega_1 = W^T f_{b_1}(E_2, b_{1|TC_1}) = -(xy_1)|_{E_2} = 0, \]
\[ \Omega_2 = W^T [Df_{b_1}(E_2, b_{1|TC_1})]V = -k \neq 0 \]

and \( \Omega_3 = W^T [D^2f(E_2, b_{1|TC_1})(V, V)] = -2(1 + b_1) \neq 0. \)

By Sotomayor’s Theorem, the system undergoes a transcritical bifurcation [32, 33] around \( E_2 \) at \( b_1 = b_{1|TC_1} = k^{-1}. \)

**Theorem 16.** The system undergoes a transcritical bifurcation with respect to the bifurcation parameter \( m \) around \( E_2(0, k, 0) \) if \( mk = n \) along with \( kb_1 > 1. \)

**Proof.**

\[ J|_{E_2} = \begin{pmatrix} 1 - b_1k & 0 & 0 \\ r & -r & -k \left( m + \frac{r}{k} \right) \\ 0 & 0 & mk - n \end{pmatrix}. \]

Let, \( m_{TC_2} \) be the value of \( m \) s.t \( J|_{E_2} \) has a simple zero eigenvalue at \( m = m_{TC_2} \).

So, at \( m = m_{TC_2} = \frac{n}{k} : \)

\[ J|_{E_2} = \begin{pmatrix} 1 - b_1k & 0 & 0 \\ r & -r & -(r + mk) \\ 0 & 0 & 0 \end{pmatrix}. \]

Here, \( \lambda_1 = 1 - b_1k < 0 \) and \( \lambda_2 = -r < 0. \)

After some calculations: \( V = (0, (n + r), -r)^T \) and \( W = (0, 0, 1)^T \).

Therefore,

\[ \Omega_1 = W^T f_m(E_2, m_{TC_2}) = (y_1y_2)|_{E_2} = 0, \]
\[ \Omega_2 = W^T [Df_m(E_2, m_{TC_2})]V = -kr \neq 0. \]
where $a$ is a negative real part at $a$ and $b$.

By Sotomayor’s Theorem, the system undergoes a transcritical bifurcation around $E_2$ at $m = m_{[TC_2]} = \frac{n}{k}$.

**Theorem 17.** The system undergoes a transcritical bifurcation with respect to the bifurcation parameter $m$ around $E_3(x, y_1, 0)$ if $m(1 + k) = n(1 + b_1)$.

**Proof.**

$$J|_{E_3} = \begin{pmatrix} a_{11} & a_{12} & a_{13} \\ a_{21} & a_{22} & a_{23} \\ 0 & 0 & a_{33} \end{pmatrix},$$

where $a_{11} = -\frac{x}{x}; a_{12} = -b_1 \frac{x}{x}; a_{13} = -b_2 \frac{x}{x}; a_{21} = -\frac{y_1}{(k+x)}; a_{22} = -\frac{y_1}{(k+x)}; a_{23} = -\left(\frac{x}{x} + m\right) \frac{y_1}{x}; a_{33} = \frac{m}{y_1} - 1.$

Let $m_{[TC_3]}$ be the value of $m$ s.t. $J|_{E_3}$ has a simple zero eigenvalue and two eigenvalues with negative real parts at $m = m_{[TC_3]}$.

So, at $m = m_{[TC_3]} = \frac{n(1+b_3)}{1+k}$:

$$J|_{E_3} = \begin{pmatrix} -\frac{x}{x} & -b_1 \frac{x}{x} & -b_2 \frac{x}{x} \\ \frac{y_1}{(k+x)} & \frac{y_1}{(k+x)} & \left(\frac{x}{x} + m\right) \frac{y_1}{x} \\ 0 & 0 & 0 \end{pmatrix}.$$

Calculations give:

$V = (b_1 \{r(1+b_1)(1+b_2) + (1+k)\} - r b_2 (1+b_2)^2, -\{r(1+b_1)(1+b_2) + (1+k)\} - (1+b_1)^2)^T$ and $W = (0, 0, 1)^T$.

Therefore,

$$\Omega_1 = W^T f_m(E_3, m_{[TC_3]}) = -(y_1 y_2)|_{E_3} = 0,$$

$$\Omega_2 = W^T [Df_m(E_3, m_{[TC_3]})V] = r(1+k)(1+b_1) \neq 0$$

and $\Omega_3 = W^T [D^2 f(E_3, m_{[TC_3]})(V, V)] = -2mr(1+b_1)^2 \{r(1+b_1)(1+b_2) + (1+k)\} \neq 0$.

By Sotomayor’s Theorem, the system undergoes a transcritical bifurcation around $E_3$ at $m = m_{[TC_3]} = \frac{n(1+b_3)}{1+k}$.

**Theorem 18.** The system undergoes a transcritical bifurcation with respect to the bifurcation parameter $b_1$ around $E_4(0, y_1, y_2)$ if $b_1 n(km + r) + 2r km - n = m(km + r)$.

**Proof.**

$$J|_{E_4} = \begin{pmatrix} a_{11} & 0 & 0 \\ a_{21} & a_{22} & a_{23} \\ 0 & a_{32} & 0 \end{pmatrix},$$

where $a_{11} = 1 - b_1 y_1 - b_2 y_2; a_{21} = \frac{y_1 (y_1 + y_2)}{k}; a_{22} = -\frac{y_1}{k}; a_{23} = -\left(\frac{x}{x} + m\right) \frac{y_1}{x}; a_{32} = m y_2.$

Let $b_1_{[TC_3]}$ be the value of $b_1$ s.t. $J|_{E_4}$ has a simple zero eigenvalue and two eigenvalues with negative real parts at $b_1 = b_1_{[TC_3]}$.
So, at \( b_1 = b_{1[TC4]} \):

\[
J|_{E_4} = \begin{pmatrix}
0 & -\frac{r_1 (y_1 + y_2)}{x} & 0 & - \left( \frac{r}{x} + m \right) y_1 \\
\frac{r_1 (y_1 + y_2)}{x} & 0 & -\frac{r_1}{x} & \frac{r m y_2}{x} \\
0 & -\frac{r_1}{x} & 0 & \frac{r_1}{x} \\
-\left( \frac{r}{x} + m \right) y_1 & \frac{r m y_2}{x} & \frac{r_1}{x} & 0
\end{pmatrix}.
\]

Calculations give:

\[
V = \left( k(km + r), 0, \frac{rk(r+n)}{km+r} \right)^T \quad \text{and} \quad W = (1,0,0)^T.
\]

Therefore,

\[
\Omega_1 = W^T f_{b_1}(E_4, b_{1[TC4]}) = -(xy_1)|_{E_4} = 0,
\]

\[
\Omega_2 = W^T \left[ Df_{b_1}(E_4, b_{1[TC4]})V \right] = - \frac{rk(km - n)}{m} \neq 0
\]

and \( \Omega_3 = W^T \left[ D^2 f(E_4, b_{1[TC4]})(V, V) \right] = -2k \{ k(km + r)^2 + rk b_2(r + n) \} \neq 0. \)

By Sotomayor’s Theorem, the system undergoes a transcritical bifurcation around \( E_4 \) at \( b_1 = b_{1[TC3]} \).

9. GLOBAL STABILITY

Lyapunov function guarantees the global stability of those equilibrium points which are locally asymptotically stable (LAS) under some certain conditions. This section contains such conditions for which equilibrium points are globally asymptotically stable.

**Theorem 19.** The axial equilibrium \( E_2(0,k,0) \) is globally asymptotically stable if \( r - b_1 k < 0, k + 1 - rk < 0 \) and \( r + km < n \).

**Proof.** Consider a suitable Lyapunov function as;

\[
V_1(x, y_1, y_2) = x + \left[ y_1 - k - k \log \left( \frac{y_1}{k} \right) \right] + y_2.
\]

Here, \( V_1(x, y_1, y_2) \) is a positive definite function for all \( (x, y_1, y_2) \) other than \( (0, k, 0) \).

Now time derivative of \( V_1 \) computed along the solutions of system (4) is given by:

\[
\dot{V}_1 = \frac{dx}{dt} + \left( 1 - \frac{k}{y_1} \right) \frac{dy_1}{dt} + \frac{dy_2}{dt}
\]

\[
= x(1 - x) - b_1 xy_1 - b_2 x y_2 + (y_1 - k) \left\{ r \left( 1 - \frac{y_1 + y_2}{k + x} \right) - my_2 \right\} + my_1 y_2 - ny_2
\]

\[
= x(1 - x) - b_1 y_1 - b_2 y_2 - \frac{r y_2}{k + x} (y_1 - k) + m k y_2 + r (y_1 - k) \left[ 1 - \frac{y_1}{k + x} \right] - ny_2
\]

\[
< x(1 - x) - b_1 y_1 - b_2 y_2 - \frac{r y_1 y_2}{k + x} + (r + mk)y_2 + \frac{r x}{k + x} (y_1 - k) - ny_2
\]

\[
\leq x(1 - x) - b_2 y_2 + \left( \frac{r}{k + x} - b_1 \right) xy_1 + (r + mk)y_2 - \frac{rk x}{k + x} - \frac{r y_1 y_2}{k + x} - ny_2
\]

\[
< x \left( 1 - \frac{r k}{k + 1} \right) + \left( \frac{r}{k} - b_1 \right) xy_1 + y_2 (r + mk - n).
\]

So \( \dot{V}_1 < 0 \) if \( r - b_1 k < 0, k + 1 - rk < 0 \) and \( r + km < n \). Also \( \dot{V}_1 = 0 \) when \( (x, y_1, y_2) = (0, k, 0) \). Therefore, \( E_2 \) is globally asymptotically stable (under the stated parametric restrictions) using LaSalle theorem [34].
Theorem 20. The axial equilibrium $E_3(\bar{x}, \bar{y}_1, 0)$ is globally asymptotically stable if

$$\left(\frac{b^2}{k^2} + \frac{r^2}{(k+1)^2} - \frac{2rb_{1}b_{2}}{(k+1)}\right) < \frac{4r}{k+1}$$

along with $b_{2}\bar{x} + m\bar{y}_1 + \frac{r\bar{y}_1}{k} < n$.

Proof. Consider a suitable Lyapunov function as;

$$V_2(x, y_1, y_2) = \left[ x - \bar{x} - \bar{x} \log \left( \frac{x}{\bar{x}} \right) \right] + \left[ y_1 - \bar{y}_1 - \bar{y}_1 \log \left( \frac{y_1}{\bar{y}_1} \right) \right] + y_2. $$

Here, $V_2(x, y_1, y_2)$ is a positive definite function for all $(x, y_1, y_2)$ other than $(\bar{x}, \bar{y}_1, 0)$.

Now time derivative of $V_2$ computed along the solutions of the system (4) is given by;

$$V_2 = \left(1 - \frac{\bar{x}}{x}\right) \frac{dx}{dt} + \left(1 - \frac{\bar{y}_1}{y_1}\right) \frac{dy_1}{dt} + \frac{dy_2}{dt}$$

$$= (x - \bar{x}) \left[ 1 - x - b_{1}y_{1} - b_{2}y_{2} \right] + (y_1 - \bar{y}_1) \left[ r \left( 1 - \frac{y_1 + y_2}{k + x} \right) - my_2 \right] + m y_1 y_2 - ny_2$$

$$= (x - \bar{x}) \left[ 1 - x - b_{1}y_{1} - b_{2}y_{2} \right] + (y_1 - \bar{y}_1) \left[ r \left( 1 - \frac{y_1 + y_2}{k + x} \right) - my_2 \right] + m y_1 y_2 - ny_2$$

$$+ \frac{r(y_1 - \bar{y}_1)}{(k + x)(k + x)} \left[-k(y_1 - \bar{y}_1) + (x\bar{y}_1 - y_1 \bar{x}) \right] - \left( m + \frac{r}{k + x} \right) y_2(y_1 - \bar{y}_1)$$

$$= -(x - \bar{x})^2 + \left[-b_{1} + \frac{r\bar{y}_1}{(k + x)(k + x)} \right] \left( x - \bar{x} \right)(y_1 - \bar{y}_1) - b_{2}y_{2}(x - \bar{x})$$

$$- \frac{r}{(k + x)} (y_1 - \bar{y}_1)^2 + m y_1 y_2 - \frac{r y_1 y_2}{k + x} + \frac{r\bar{y}_1 y_2}{k + x} - ny_2$$

$$< - \left[ (x - \bar{x}) - \frac{1}{2} \left( b_{1} - \frac{r\bar{y}_1}{(k + x)(k + x)} \right) \right] \left( y_1 - \bar{y}_1 \right)^2 + \left( b_{2}\bar{x} + m\bar{y}_1 + \frac{r\bar{y}_1}{k} - n \right) y_2$$

$$+ \left[ \frac{1}{4} \left( b_{1} - \frac{r\bar{y}_1}{(k + x)(k + x)} \right)^2 - \frac{r}{k + x} \right] \left( y_1 - \bar{y}_1 \right)^2$$

$$< - \left[ (x - \bar{x}) - \frac{1}{2} \left( b_{1} - \frac{r\bar{y}_1}{(k + x)(k + x)} \right) \right] \left( y_1 - \bar{y}_1 \right)^2 + \left( b_{2}\bar{x} + m\bar{y}_1 + \frac{r\bar{y}_1}{k} - n \right) y_2$$

$$+ \left[ \frac{1}{4} \left( b_{1}^2 + \frac{r^2\bar{y}_1^2}{k^2(k + x)^2} - \frac{2rb_{1}\bar{y}_1}{k(k + 1)(k + x)} \right) - \frac{r}{k + 1} \right] \left( y_1 - \bar{y}_1 \right)^2.$$ 

So $\dot{V}_2 < 0$ if $\left[ \frac{b^2}{k^2} + \frac{r^2}{(k+1)^2} - \frac{2rb_{1}b_{2}}{(k+1)} \right] < \frac{4r}{k+1}$ and $b_{2}\bar{x} + m\bar{y}_1 + \frac{r\bar{y}_1}{k} < n$. Also $\dot{V}_2 = 0$ when $(x, y_1, y_2) = (\bar{x}, \bar{y}_1, 0)$. Therefore, $E_3$ is globally asymptotically stable (under the stated parametric restrictions) using LaSalle theorem [34].

Theorem 21. The equilibrium point $E_4(0, \bar{y}_1, \bar{y}_2)$ exists and is globally asymptotically stable if

$$\frac{b^2}{2} \left[ \frac{r}{4(k+1)} \left( b_{1} - \frac{r(r + n)}{k(km + r)} \right) - \frac{rb_{2}}{2k} \right] + \frac{r}{k + 1} \left[ \frac{b_{2}}{4} \left( b_{1} - \frac{r(r + n)}{k(km + r)} \right) - \frac{r}{2k} \right] > 0, \quad (9)$$

$$\quad \frac{kb_{1}(km + r)}{r} > r(r + n) \quad (10)$$

and

$$\frac{r}{k + 1} \left[ \frac{b_{1}^2 + \frac{r^2(r + n)^2}{k^2(km + r)^2} - \frac{2rb_{1}(r + n)}{(k + 1)(km + r)}}{k(k + 1)(km + r)} \right] > 0. \quad (11)$$

Proof. Consider a Lyapunov function as:

$$V_3(x, y_1, y_2) = x + \left[ y_1 - \bar{y}_1 - \bar{y}_1 \log \left( \frac{y_1}{\bar{y}_1} \right) \right] + \left[ y_2 - \bar{y}_2 - \bar{y}_2 \log \left( \frac{y_2}{\bar{y}_2} \right) \right].$$
Here, $V_3(x, y_1, y_2)$ is a positive definite function for all $(x, y_1, y_2)$ other than $(0, y_1, y_2)$.

Now time derivative of $V_3$ computed along the solutions of system (4) is given by:

$$
\dot{V}_3 = \frac{dx}{dt} + \left(1 - \frac{\dot{y}_1}{y_1}\right) \frac{dy_1}{dt} + \left(1 - \frac{\dot{y}_2}{y_2}\right) \frac{dy_2}{dt}
$$

$$
= [x(1 - x) - b_1xy_1 - b_2xy_2] + (y_1 - \frac{\dot{y}_1}{y_1}) \left\{ r \left(1 - \frac{y_1 + y_2}{k + x}\right) - my_2\right\} + (y_2 - \frac{\dot{y}_2}{y_2}) (my_1 - n)
$$

$$
= x[-x - b_1(y_1 - \frac{\dot{y}_1}{y_1}) - b_2(y_2 - \frac{\dot{y}_2}{y_2})] - \frac{r}{k + x}(y_1 - \frac{\dot{y}_1}{y_1})^2 - \frac{r}{k + x}(y_1 - \frac{\dot{y}_1}{y_1})(y_2 - \frac{\dot{y}_2}{y_2})
$$

$$
+ \frac{r(\frac{\dot{y}_1}{y_1} + \frac{\dot{y}_2}{y_2})}{k(k + x)}x(y_1 - \frac{\dot{y}_1}{y_1}) + [1 - b_1\frac{\dot{y}_1}{y_1} - b_2\frac{\dot{y}_2}{y_2}]x.
$$

Adding these contributions, for $U = (x, (y_1 - \frac{\dot{y}_1}{y_1}), (y_2 - \frac{\dot{y}_2}{y_2}))^T$, we find

$$
\frac{dV}{dt} = Ax^2 + B(y_1 - \frac{\dot{y}_1}{y_1})^2 + C(y_2 - \frac{\dot{y}_2}{y_2})^2 + 2Hx(y_1 - \frac{\dot{y}_1}{y_1}) + 2F(y_1 - \frac{\dot{y}_1}{y_1})(y_2 - \frac{\dot{y}_2}{y_2})
$$

$$
+ 2Gx(y_2 - \frac{\dot{y}_2}{y_2}) = -U^T MU + [1 - b_1\frac{\dot{y}_1}{y_1} - b_2\frac{\dot{y}_2}{y_2}]x.
$$

Here $M$ is the symmetric quadratic form given by

$$
M = \begin{pmatrix}
A & H & G \\
H & B & F \\
G & F & C
\end{pmatrix}
$$

with entries that are functions only of the variable $x$.

$A = 1$, $B = \frac{r}{k + x}$, $C = 0$, $H = \frac{1}{2} \left\{ b_1 \frac{r(\frac{\dot{y}_1}{y_1} + \frac{\dot{y}_2}{y_2})}{k(k + x)} \right\}$, $F = \frac{r}{2(k + x)}$, $G = \frac{b_2}{2}$.

Local stability condition of $E_4$ gives that $1 - b_1\frac{\dot{y}_1}{y_1} - b_2\frac{\dot{y}_2}{y_2} < 0$ and hence the last term of the formula (12) is also negative. Thus, if the matrix $M$ is positive definite, then $\frac{dV}{dt} < 0$. We need all of the principal minors of $M$, namely $P_1 \equiv A$, $P_2 \equiv AB - H^2$, $P_3 \equiv ABC + 2FGH - AF^2 - BG^2 - CH^2$, to be positive, i.e., $P_1 = 1 > 0$,

$$
P_2 = \frac{r}{k + x} - \frac{1}{4} \left\{ b_1 \frac{r(\frac{\dot{y}_1}{y_1} + \frac{\dot{y}_2}{y_2})}{k(k + x)} \right\} > 0,
$$

$$
P_3 = C(AB - H^2) + G(FH - BG) + F(GH - AF) > 0.
$$

For $P_2$, using condition (11) we have

$$
P_2 = \frac{r}{k + x} - \frac{1}{4} \left\{ b_1 \frac{r(\frac{\dot{y}_1}{y_1} + \frac{\dot{y}_2}{y_2})}{k(k + x)} \right\}
$$

$$
= \frac{r}{k + x} - \frac{1}{4} \left\{ b_1 \frac{r - m\frac{\dot{y}_2}{y_2}}{k + x} \right\}
$$

$$
> \frac{r}{k + 1} - \frac{1}{4} \left\{ b_1^2 + \frac{r^2(r + n)^2}{k^2(km + r)^2} \right\} > 0.
$$

Now $P_3 = C(AB - H^2) + G(FH - BG) + F(GH - AF) = G(FH - BG) + F(GH - AF)$. 
For $P_3$, using conditions (9) and (10) we have

$$FH - BG = \frac{r}{4(k + x)} \left[ b_1 - \frac{r(y_1 + y_2)}{k(k + x)} \right] - \frac{rb_2}{2(k + x)}$$

$$> \frac{r}{4(k + 1)} \left[ b_1 - \frac{r(y_1 + y_2)}{k^2} \right] - \frac{rb_2}{2k}$$

$$= \frac{r}{4(k + 1)} \left[ b_1 - \frac{r(r + n)}{k(km + r)} \right] - \frac{rb_2}{2k}$$

and $GH - AF = \frac{b_2}{4} \left[ b_1 - \frac{r(y_1 + y_2)}{k(k + x)} \right] - \frac{r}{2(k + x)}$

$$> \frac{b_2}{4} \left[ b_1 - \frac{r(y_1 + y_2)}{k^2} \right] - \frac{r}{2k}$$

$$= \frac{b_2}{4} \left[ b_1 - \frac{r(r + n)}{k(km + r)} \right] - \frac{r}{2k}$$

So, $P_3 > \frac{b_2}{2} \left[ \frac{r}{4(k + 1)} \left( b_1 - \frac{r(r + n)}{k(km + r)} \right) - \frac{rb_2}{2k} \right] + \frac{r}{k + 1} \left[ \frac{b_2}{4} \left( b_1 - \frac{r(r + n)}{k(km + r)} \right) - \frac{r}{2k} \right] > 0.$

Hence, the symmetric matrix $M$ is positive definite, implying $\frac{dV}{dt} < 0$ along the trajectories. Also $\dot{V}_3 = 0$ when $(x, y_1, y_2) = (0, \bar{y}_1, \bar{y}_2)$. Therefore, $E_4$ is globally asymptotically stable (under the stated parametric restrictions) using LaSalle theorem [34].

Now, we shall check the global stability of the interior equilibrium point $E^*$.  

**Theorem 22.** The interior equilibrium $E^*$ is globally asymptotically stable if

$$b_1 - \frac{r(y_1^* + y_2^*)}{k(k + x^*)} > \max \left\{ \frac{2r}{kb_2}, \frac{2(k + 1)b_2}{k} \right\}, \quad (13)$$

$$kb_1(k + x^*) < r(y_1^* + y_2^*) \quad (14)$$

and

$$\left[ b_1^2 + \frac{r^2(y_1^* + y_2^*)^2}{k^2(k + x^*)^2} - \frac{2rb_1(y_1^* + y_2^*)}{k(k + x^*)} \right] < \frac{4r}{k + 1}. \quad (15)$$

**Proof.** Consider a appropriate lyapunov function:

$$V(x, y, z) = \left[ x - x^* - x^* \log \left( \frac{x}{x^*} \right) \right] + \left[ y_1 - y_1^* - y_1^* \log \left( \frac{y_1}{y_1^*} \right) \right] + \left[ y_2 - y_2^* - y_2^* \log \left( \frac{y_2}{y_2^*} \right) \right],$$

Here, $V(x, y_1, y_2)$ is a positive definite function for all $(x, y_1, y_2)$ except $(x^*, y_1^*, y_2^*)$. The time derivative of $V$ computed along the solutions of the system (4) is given by;
\[ \dot{V} = \left(1 - \frac{x^*}{x}\right) \frac{dx}{dt} + \left(1 - \frac{y_1^*}{y_1}\right) \frac{dy_1}{dt} + \left(1 - \frac{y_2^*}{y_2}\right) \frac{dy_2}{dt} \]

\[ = (x - x^*) \left[1 - x - b_1(y_1 - b_2y_2) + (y_1 - y_1^*) \right] \left[ r \left(1 - \frac{y_1 + y_2}{k + x}\right) - my_2 \right] + (y_2 - y_2^*) \left[ m(y_1 - n) \right] \]

\[ = -(x - x^*)^2 - b_1(x - x^*)(y_1 - y_1^*) - b_2(x - x^*)(y_2 - y_2^*) \]

\[ + \frac{r(y_1 - y_1^*)}{(k + x)(k + x^*)}(-(k + x^*)(y_1 - y_1^*) - (k + x^*)(y_2 - y_2^*) + (x - x^*)(y_1^* + y_2^*)) \]

\[ = -(x - x^*)^2 - \frac{r}{k + x}(y_1 - y_1^*)^2 - b_2(x - x^*)(y_2 - y_2^*) \]

\[ - \left[b_1 - \frac{r(y_1^* + y_2^*)}{(k + x)(k + x^*)}\right](x - x^*)(y_1 - y_1^*) - \frac{r}{k + x}(y_1 - y_1^*)(y_2 - y_2^*). \]

Adding these contributions, for \( U = ((x - x^*), (y_1 - y_1^*), (y_2 - y_2^*))^T \), we find

\[ \frac{dV}{dt} = A(x - x^*)^2 + B(y_1 - y_1^*)^2 + C(y_2 - y_2^*)^2 + 2H(x - x^*)(y_1 - y_1^*) + 2F(y_1 - y_1^*)(y_2 - y_2^*) \]

\[ + 2G(x - x^*)(y_2 - y_2^*) = -UTMU. \]  

(16)

Here \( M \) is the symmetric quadratic form given by

\[ M = \begin{pmatrix} A & H & G \\ H & B & F \\ G & F & C \end{pmatrix} \]

with entries that are functions only of the variable \( x \).

\( A = 1, \ B = \frac{r}{k + x}, \ C = 0, \ H = \frac{1}{2} \left\{ b_1 - \frac{r(y_1^* + y_2^*)}{(k + x)(k + x^*)}\right\} \), \( F = \frac{r}{2(k + x)}, \ G = \frac{b_2}{2} \).

Thus, if the matrix \( M \) is positive definite, then \( \frac{dV}{dt} < 0 \). We need all of the principal minors of \( M \), namely \( P_1 \equiv A, \ P_2 \equiv AB - H^2, \ P_3 \equiv ABC + 2FGH - AF^2 - BG^2 - CH^2 \), to be positive, i.e., \( P_1 = 1 > 0 \),

\[ P_2 = \frac{r}{k + x} - \frac{1}{4} \left\{ b_1 - \frac{r(y_1^* + y_2^*)}{(k + x)(k + x^*)}\right\}^2 > 0, \ P_3 = C(AB - H^2) + G(FH - BG) + F(GH - AF) > 0. \]

For \( P_2 \), using condition (15) we have

\[ P_2 = \frac{r}{k + x} - \frac{1}{4} \left\{ b_1 - \frac{r(y_1^* + y_2^*)}{(k + x)(k + x^*)}\right\}^2 \]

\[ > \frac{r}{k + 1} - \frac{1}{4} \left\{ b_1^2 + \frac{r^2(y_1^* + y_2^*)^2}{k^2(k + x^*)^2} - \frac{2r^2b_1(y_1^* + y_2^*)}{(k + 1)(k + x^*)}\right\} > 0. \]

Now \( P_3 = C(AB - H^2) + G(FH - BG) + F(GH - AF) = G(FH - BG) + F(GH - AF). \)

As \( F, G > 0 \), then \( FH - BG, GH - AF > 0 \) implies \( P_3 > 0 \). For \( P_3 \), using conditions (13)
and (14) we have
\[
FH - BG = \frac{r}{4(k + x)} \left[ b_1 - \frac{r(y_1^* + y_2^*)}{(k + x)(k + x^*)} \right] - \frac{rb_2}{2(k + x)} \]
\[
> \frac{r}{4(k + 1)} \left[ b_1 - \frac{r(y_1^* + y_2^*)}{k(k + x^*)} \right] - \frac{rb_2}{2k} > 0
\]
and
\[
GH - AF = \frac{b_2}{4} \left[ b_1 - \frac{r(y_1^* + y_2^*)}{(k + x)(k + x^*)} \right] - \frac{r}{2k} > 0.
\]
Combining these results it follows that \( P_3 > 0 \). Hence, the symmetric matrix \( M \) is positive definite, implying \( \frac{dV}{dt} < 0 \) along the trajectories. Thus, \( V \) is a Lyapunov function and global stability for \( E^* \) follows.

10. NUMERICAL SIMULATION

Now we shall analyze the proposed model with some numerical figures, focusing on the effect of disease transmission rate, \( m \) and consumption rate of healthy pest, \( b_1 \) of the system. The values of the following parameters are stated in Table 1: attack rate of infected pest on plants (\( b_2 \)), intrinsic growth rate of healthy plants (\( r \)), intake rate (for pest) of secondary food source (\( k \)) and mortality rate of infected plants (\( n \)). These values imply that for \( b_1 = 0.5 \), \( 1 - kb_1 > 0 \) and so \( E_3 \) exists when the bio-control pathogen (infected pest) is not present in the system. The trajectories of system (4) starting from (a) (0.5,2,0.3), (b) (0.1,0.8,0.1) and (c) (0.4,1.6,0.2) approach the equilibrium \( E_3 = (0.25, 1.5, 0) \) (see Figure 1). That means the healthy pest can coexist with wild plant species and, in long run, population biomass of both species are approaching stationary values.

| Parameters | Values |
|------------|--------|
| \( b_2 \)   | 0.07   |
| \( r \)     | 0.125  |
| \( k \)     | 1.25   |
| \( n \)     | 0.125  |

For \( b_1 = 1 \) (implies \( b_1k > 1 \)) and \( m = 0.01 \), we have a stable situation where infected pests are going to extinct in absence of wild plants. For \( m < 0.1 \), the trajectories starting in the neighborhood of \( E_2 = (0, 1.25, 0) \) converge to \( E_2 \) (see Figure 5). Introducing disease (insecticide) into the herbivore pest species and choosing the attack rate of the healthy pest on the plant \( b_1 = 0.5 \), we calculate the equilibria as \( m \) increases: (i) equilibrium \( E_3 = (0.25, 1.5, 0) \) is stable if \( m < 0.0836 \) (ii) when \( m \) exceeds 0.0836, \( E^* \) exists and is stable. For \( m = 0.1 \), the trajectories converge to \( E^* = (0.36, 1.25, 0.16) \) (see Figure 2). This means that with comparatively large transmission rates above 0.084 the disease can invade and we get an endemic situation. So, it can be stated that biological control has a high stabilizing effect.

It is evident that in natural ecosystems the asymptotic behaviour of system trajectories are rarely seen and their dynamics are essentially transient [35, 36, 37]. Transient dynamics mainly occurs when the population dynamics mimic the asymptotic behaviour of one kind for a very long time and suddenly convergence to a different attractor [38]. In this system, if we start to increase the value of transmission rate coefficient (\( m \)), a transient dynamics can be observed:
Fig. 1. Stable behaviour of $E_3$. 

Fig. 2. Stable behaviour of $E^*$ for $m = 0.1$. 

Fig. 3. Stable behaviour of $E^* = (0.86, 0.25, 0.20)$ for $m = 0.5$. 

Mathematical Biology and Bioinformatics. 2020. V. 15, № 2. doi: 10.17537/2020.15.268
Fig. 4. Stable behaviour of the system around \( E^* \) for higher disease transmission rate.

The trajectories start to oscillate first but after some time it converges to the steady-state (see Figure 3). Further figure 4 depict that increased transmission rate \( (m = 20) \) can result in transient oscillations of the populations where the final attractor is the stationary (steady) state. Depending on the parameter value \( (m) \), the population size can exhibit oscillations around the unstable equilibrium \( (E_1) \) for a long time until it eventually settles to the interior steady state of the system. Here for higher transmission rate, the system exhibits substantially different behaviour: the population size shows oscillations around the unstable state \( E_1 \) over a long time period but then ultimately tends to the interior stationary state \( E^* \). The dynamics implies of occurrence of a transient limit cycle as the trajectory initially approaches the cycle, remains on it over a long time and finally leaves the cycle and ultimately approaches to interior steady state. The duration of such oscillations depends on the transmission rate coefficient.

Moreover, the values \( b_1 = 1 \) and \( m = 0.01 \) along with other parametric values stated in Table 1 imply that \( mk < n \) and so the wild plant species going to extinct in absence of biological control of the insect pest. It is already mentioned that the equilibrium \( E_2 = (0, 1.25, 0) \) is stable.
if \( m < 0.1 \) and trajectories of the system approach the equilibrium while starting from (a) \((0.5,3,0.3)\), (b) \((0.2,2,0.15)\) and (c) \((0.4,1,0.2)\) (see Figure 5). That means the herbivore pest can survive with the help of other food sources in absence of wild plant species. Moreover, in absence of wild plants, the pest species approaches to the steady state where both healthy and pathogen infected pets live if \( km > n \) holds. The threshold parameter \( R_0 = \frac{m(r+mk)}{rb_2(mk-n)+nb_1(r+mk)} \) determines the development of wild plant species. In absence of wild plants, the predator population approaches to \( \bar{y}_1 = \frac{n}{m} \) and \( \bar{y}_2 = \frac{r(\mk-n)}{m(r+mk)} \), provided \( R_0 < 1 \). If \( R_0 > 1 \), then \( 1 - b_1\bar{y}_1 - b_2\bar{y}_2 > 0 \) implying the growth rate of prey (plant) species is an increasing function at \( E_4 = (0, \bar{y}_1, \bar{y}_2) \). So, the wild plant can successfully invade the pest equilibrium as a result of coexistence state. If \( m \) increases above 0.1, the disease can invade and approach the endemic level with the presence of wild plants in the system. The pest species approaches to \( E_4 = (0, 1.09, 0.08) \) while \( m = 0.115 \) and \( b_1 = 1 \) (see Figure 6). Keeping the value of \( m \) as 0.1, if \( b_1 \) decreases, \( E^* \) exists. Figure 2 depicts that for \( b_1 = 0.5 \), the system tends to the interior equilibrium point \( E^* \). Thus the disease can affect the healthy pest by reducing its biomass to such a level that the predation pressure on the wild plant species is small and it can coexist with pest.

From the coordinates of \( E_3 \) and \( E^* \), it is observed that susceptible pest’s biomass decreases in the presence of insecticide. Also, it is noted that the biomass of wild plant increases compares to \( E_3 \). So, the disease into pest reduces the predation pressure on wild plant resulting in a larger plant population. It indicates that biological control is beneficial to plant species. From Figure 7, it is observed that the healthy pest population of the system (4) is decreased with the increasing transmission rate. The healthy pest as well as total pest biomass of \( E^* \) is smaller than that of \( E_3 \). Hence, the damages done by herbivore pest on the crop are reduced as densities of susceptible pests decrease. It implies that biological control is also beneficial to crop species.

In the absence of any pathogen infection, if the healthy prey starts to consume the wild plants with a higher rate, then gradually plant biomass starts to decrease. This situation can occur if the secondary food source is scarce or if the biomass of pest become much higher. Continuation of this situation leads to the extinction of plants gradually. In fact, after crossing a threshold value of \( b_1 \) we get a system without any wild plants and the pest have to live with the secondary food source only. So, at \( b_1 = 0.8 \), a transcritical bifurcation occurs around \( E_2 \) and the equilibrium point becomes stable when \( b_1 \) exceeds 0.8 (see Figure 8).

Moreover, in absence of any plant species, if insecticide pathogen is transmitted in the system
Fig. 7. Steady states of the model (4) for different transmission rate.

Fig. 8. Transcritical Bifurcation around $E_2$ taking $b_1$ as bifurcation parameter.
with a lower quantity, then a lesser number of pests get infected. Hence as time goes we get a system where only healthy pest exists as a stable equilibrium and infected pests washout from the system when the transmission rate crosses a threshold value. As a result, a transcritical bifurcation occurs at \( m = 0.1 \) around \( E_2 \) and the equilibrium point becomes stable when \( m \) is less than 0.1 (see Figure 9).

In a system where all the species coexist, if the transmission rate starts to decrease due to some unavoidable issues, then the biomass of infected pest starts to decrease. Continuation of this situation leads to the extinction of pathogen-infected pests. It is observed that, when \( m \) goes below 0.083, infected species wash out and we get a system consists of wild plants and healthy prey as a stable equilibrium. So, a transcritical bifurcation occurs at \( m = 0.083 \) around \( E_3 \) and the equilibrium point becomes stable when \( m \) is less than 0.083 (see Figure 10).

Further, if alternative prey is scarce, the pest prefers to have plants as their food source with a higher rate resulting in an increased consumption rate of the healthy predator (pest). Now in this situation, the biomass of plants starts to decrease and ultimately we shall reach to a system consists of pest species (both healthy and infected) only. It is observed that, when \( b_1 \) exceeds 0.88, plant species washout from the system and hence it can be concluded that a transcritical

**Fig. 9.** Transcritical Bifurcation around \( E_2 \) taking \( m \) as bifurcation parameter.

**Fig. 10.** Transcritical Bifurcation around \( E_3 \) taking \( m \) as bifurcation parameter.
bifurcation occurs at $b_1 = 0.88$ around $E_4$ and the equilibrium point becomes stable when $b_1$ is greater than 0.88 (see Figure 11).

We have chosen different set of parameters to show the global stability of the equilibrium points $E_2$, $E_3$ and $E_4$ (see Figure 12). It can be observed from figure (12.a) that $E_2$ is globally asymptotic stable (GAS) for $r = 2$, $b_1 = 1$, $b_2 = 0.07$, $n = 2.15$, $m = 0.01$ and $k = 2.25$. In fact, when $n$ exceeds 2.04, $E_2$ becomes GAS and it remains globally stable for increasing value of $n$. Similarly, figure (12.b) depicts that $E_3$ is globally asymptotic stable for $r = 0.125$, $b_1 = 0.5$, $b_2 = 0.07$, $n = 0.5$, $m = 0.01$ and $k = 1.25$. In fact, when $n$ exceeds 0.19, $E_3$ becomes GAS and it remains globally stable for increasing value of $n$. Moreover, figure (12.c) shows that $E_4$ is globally asymptotic stable for $r = 20$, $b_1 = 0.5$, $b_2 = 0.35$, $n = 0.5$, $m = 0.2$ and $k = 15.25$. In fact, when $n$ lies in $(0.3, 0.44)$, $E_4$ remains GAS.

11. DISCUSSION

Tree and crops are often affected by several insects and mites. Herbivorous insects, per year, destroy almost one-fifth of the world’s total crop production. Some of them affect the production of crops whereas some others causing tree or crop deaths. Brown blight (Peronophythora litchii) is a disease which infects leaves and fruits. On the other hand, there are some diseases like Anthracnose (Colletotrichum gloeosporioides) etc. which attack trees in different countries like China, India and Australia. Parasitic algae and nematodes affect some orchards. There are other species such as different leaf-eating and flower-eating caterpillars and beetles, bark borers, fruit-sucking bugs, leaf mites, fruit-piercing moths etc. Stem borers are one kind of caterpillars which causes almost 10–48 % yield loss per year. Besides, there are more pests such as bollworm, root borer, weevil, aphids etc. which reduce grain production by about 5–30 % per year. In this paper, we have incorporated a plant-herbivore system where biologically-based technologies have been applied to the herbivore population to control the pest. The proposed model is biologically suitable. For example, it is quite obvious that due to the frequent or periodic use of pesticides and insecticides pest population becomes infected. Also, the growth of the pest population depends on plant abundance and so, carrying capacity of plant population is taken as a function which is dependent on plant population. The crop species has no dynamics of its own as it is always controlled by the farmer. The growth of the plants depends on the frequency of plantation and its demand. The biomass of the wild plants is decreased by both healthy and infected pest but it is considered that the digestion power of infected ones is too less to contribute
Fig. 12. (12.a) Global stability of $E_2$ for $n = 2.15$. (12.b) Global stability of $E_3$ for $n = 0.5$. (12.c) Global stability of $E_4$ for $b_2 = 0.35$. 
to their growth. So, the consumption of plant can not contribute to the biomass conversion of infected pest.

We have analyzed the local as well as global properties of the equilibrium points of this model. The proposed model involves the dynamics of insect-pathogen interaction along with the dynamics of a secondary host species (wild plant). From the results of system (4) it can be concluded that when a secondary wild host present, the system exhibits a stable co-existent state for any transmission rate higher than 0.086, i.e., wild plants have a stabilizing effect.

The effect of bio-control on the community has also been obtained from the results of system (4). Numerical simulations indicate that bio-control has a highly stabilizing effect as the interior equilibrium, if exists, remains stable for any transmission value. Moreover, biological control can bring the wild plant species back to the system (by reducing the pest population) even after it has gone to extinction in its absence. Thirdly, it follows from figure 7 that with increasing transmission rate wild plant biomass increases and susceptible pest biomass decreases. This happens because if the herbivore pest is affected with a disease, then the predation rate on wild plant species becomes lower resulting increment in wild plant biomass. Also, lower biomass of susceptible pest leads to lesser damage to the crop. Hence biological control strategy is beneficial for wild plant species as well as crop. The work presented in this paper is an approach to analyze the dynamics of the bio-control program where the pest consumes wild plant species too. Analytical results show that the presence of wild host species has a significant impact on the dynamics of a bio-control program.

The proposed model reveals rich dynamics but some other factors can be included further to make the model more realistic to the environment. We can consider the predatory functions for the pest population (both healthy and infected) as such functions which depend on the plant biomass also. Moreover, the case should be taken into consideration where the plant population is infected with some disease. As, the consumption procedure of pest population is not instantaneous, so, ‘gestation delay’ can be introduced in the system to analyse the effect of the delay parameter. Besides, environmental fluctuations can also be incorporated with the help of white noise and the spatio-temporal model can also be taken with diffusion terms to study the dynamics. So, in future, some models can be formulated with all these assumptions to observe different dynamics and to analyse the impact of all such phenomenon in the model.

The first author (Sangeeta Saha) is thankful to the University Grants Commission, India for providing SRF.

REFERENCES

1. Van Lenteren J.C., Woets J. Biological and integrated pest control in greenhouses. Ann. Rev. Entomol. 1988. V. 33. P. 239–250.
2. Van Lenteren J.C. Integrated pest management in protected crops. In: Integrated pest management. Ed. Dent D. London: Chapman & Hall. 1995. P. 311–343.
3. Burges H.D. Microbial Control of Pests and Plant Diseases 1970-1980 London, New York: Academic Press, 1981.
4. Glare J.R., Jackson T.A. Use of Pathogens in Scarab Pest Management. Andover, Hampshire, UK: Intercept Ltd., 1992.
5. Hochberg M.E. The potential role of pathogens in biological control. Nature. 1989. V. 337. P. 262–265.
6. Anderson R.M., May R.M. Infectious disease and population cycles of forest insect. Science. 1980. V. 210. P. 658–661.
7. Anderson R.M., May R.M. The population dynamics of microparasities and their invertebrate hosts. Philos. Trans. R. Soc. Lond. Ser. B. 1980. V. 291. P. 451–524.
8. Briggs C.J., Godfray H.C.J. The dynamics of insect-pathogen interactions in stage-structured populations. *Am. Nat.* 1995. V. 145. P. 855–887.
9. Brown G.C. Stability in an insect-pathogen mode incorporating age-dependent immunity and seasonal host reproduction. *Bull. Math. Biol.* 1984. V. 46. P. 139–153.
10. Browers R.G., Begon M., Hodgkinson D.E. Host-pathogen population cycles in forest insect? Lwssons from simple models reconsidered. *Oikos.* 1993. V. 67. P. 529–538.
11. Myers J.H. Can a general hypothesis explain population cycles of forest Lepidoptera? *Adv. Ecol. Res.* 1988. V. 18. P. 179–242.
12. Begon M., Bowers R.G., Kadianakis N., Hodgkinson D.E. Disease and community structure: the importance of host self-regulation in a host-pathogen model. *Am. Nat.* 1992. V. 139. P. 1131–1150.
13. Moerbeek, M., Van Den Bosch, F. Insect-pathogen dynamics: stage-specific susceptibility and insect density dependence. *Math. Biosci.* 1997. V. 141. P. 115–148.
14. Holt R.D., Pickering J. Infectious disease and species coexistence: a model in Lotka-Volterra form. *Am. Nat.* 1985. V. 126. P. 196–211.
15. Dwyer G. Density dependence and spatial structure in the dynamics of insect pathogens. *Am. Nat.* 1994. V. 143. P. 533–562.
16. Browers R.G., Begon M. A host-pathogen model with free living infective stages, applicable to microbial pest control. *J. Theor. Biol.* 1991. V. 148. P. 305–329.
17. Begon M., Bowers R.G. Host-pathogen models and microbial pest control: the effect of host self-regulation. *J. Theor. Biol.* 1995. V. 169. P. 275–287.
18. Sharma S., Samanta G.P. A ratio-dependent predator-prey model with Allee effect and disease in prey. *Journal of Applied Mathematics and Computing.* 2014. V. 47. No. 1–2. P. 345–364.
19. Hadeler K.P., Freedman H.I. Predator-prey population with parasitic infection. *J. Math. Biol.* 1989. V. 27. P. 609–631.
20. Freedman H.I. A model of predator-prey dynamics as modified by the action of parasite. *Math. Biosci.* 1990. V. 99. P. 143–155.
21. Beltrami E., Carroll T.D. Modelling the role of viral disease in recurrent phytoplankton blooms. *J. Math. Biol.* 1994. V. 32. P. 857–863.
22. Venturino E. The effects of disease on competing species. *Math. Biosci.* 2001. V. 174. P. 111–131.
23. Venturino E. The influence of disease on Lotka-Volterra systems. *Rockymount. J. Math.* 1994. V. 24. P. 389–402.
24. Xiao Y., Chen L. Modelling and analysis of a predator-prey model with disease in the prey. *Math. Biosci.* 2001. V. 171. P. 59–82.
25. Xiao, Y., Chen, L. Analysis of a three species eco-epidemiological model. *J. Math. Anal. Appl.* 2001. V. 171. P. 59–82.
26. Mondal A., Pal A.K., Samanta G.P. On the dynamics of evolutionary Leslie-Gower predator-prey eco-epidemiological model with disease in predator. *Ecological Genetics and Genomics.* 2019. V. 10. 100034.
27. Hale J.K. *Theory of functional Differential Equations.* Springer-Verlag, Heidelberg, 1977.
28. Saha S., Samanta G.P. Local dynamics of a predator–prey community in a moderate period of time. *Energy, Ecology and Environment.* 2020. V. 5. No. 1. P. 47–60.
29. Freedman H.I., Ruan S. Uniform persistence in functional differential equations. *J Differ Equ.* 1995. V. 115 P. 173–192.
30. Perko L. *Differential equations and dynamical systems.* Berlin: Springer, 2013.
31. Murray J.D. *Mathematical biology.* New York: Springer-Verlag, 1993.
32. Saha S., Samanta G.P. Analysis of a predator–prey model with herd behaviour and disease
33. Saha S., Maiti A., Samanta G.P. A Michaelis–Menten Predator–Prey Model with Strong Allee Effect and Disease in Prey Incorporating Prey Refuge. *International Journal of Biomathematics*. 2019. V. 12. No. 1. 1950007.

34. LaSalle J. The stability of dynamical systems. *Regional conference series in applied mathematics*. SIAM, Philadelphia, 1976.

35. Cushing J.M., Dennis B., Desharnais R.A., Costantino R.F. Moving toward an unstable equilibrium: saddle nodes in population systems. *J. Anim. Ecol.* 1998. V. 67 P. 298–306.

36. Hastings A. Transient dynamics and persistence of ecological systems. *Ecol. Lett.* 2001. V. 4. P. 215–220.

37. Hastings A. Transients: the key to long-term ecological understanding? *Trends in Ecology and Evolution*. 2004. V. 19. P. 39–45.

38. Lai Y.C., Winslow R.L. Geometric-properties of the chaotic saddle responsible for supertransients in spatiotemporal chaotic systems. *Phys. Rev. Lett.* 1995. V. 74. P. 5208–5211.

Accepted 29.03.2020.
Revised 30.10.2020.
Published 18.11.2020.