

Modeling and Stability Analysis of Host-parasite Population Dynamics

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Abstract: In this study, a mathematical model is developed to explore the population dynamics of two host species. Both the hosts depend on the same resources and the availability of such resources is limited in nature. If the host populations increase abnormally the limited natural resources will be used up. Hence, the concept of parasite is brought in to the picture to regulate the host populations. The parasite is a mechanism that reduces the host populations. However, on one hand if the parasite attacks more the hosts may extinct and on the other hand if the parasite do not attack then the host populations may increase and resource may be used up. Hence, the parasite is expected to maintain a balance so that neither the host populations nor the resources extinct. Here, both the hosts are classified in to susceptible and infected and hence the model comprises of four populations: Susceptible Host-1, Infected Host-1, Susceptible Host-2 and Infected Host-2. Thus, the mathematical model comprises of a system of four first order non-linear ordinary differential equations. Mathematical analysis of the model is conducted. Positivity and boundedness of the solution have been verified and thus shown that the model is physically meaningful and biologically acceptable. Equilibrium points of the model are identified and stability analysis is conducted. Simulation study is conducted in order to support the mathematical analysis using software packages Mat lab and DeDiscover.

Keywords: Modeling, Hosts, Parasite-mediated Interactions, Stability, Numerical Simulation

1. Introduction

Parasites can theoretically encourage indirect interactions among ecological communities those live in the same trophic level. These interactions lead to some kind of effects on the competing species. Such interactions are also called as parasite-mediated competition [1-2, 4, 13-15, 19]. However, parasites induce competition-like indirect interactions among species that would not otherwise happen at all [5, 8]. These ideas reflect indirect effects of parasites on community composition leading to coexistence or promoting exclusion.

1.1. Parasite-mediated Competition

Parasites may affect competitive dynamics between native and introduced animals and plants through density and trait effects [3, 6, 13-15].

Models of parasite-mediated competition have provided insight into the role of shared parasites in shaping

communities of competing species. Parasites can regulate the population density of their hosts and, as a result, affect the dynamics of the community, and can cause the extinction of local populations and of whole species [2, 3, 13-15].

In this present study, parasites are predicted to enhance the range of conditions leading to coexistence if, for example, one of the host are more heavily impacted by the parasite. However, some arguments argued that the predominant pressure exerted by parasites on communities might not be the result of catastrophic outbreaks, but rather of less virulent and persistent where the parasites moderate or enhance the competitive capabilities of their hosts [3, 13].

When viewed as part of an ecosystem, the parasitized host is subject to numerous other interactions that convey benefits, no apparent benefits, or harm or not benefits to it [11, 12, 5, 20]. Argued that the direct outlay to the host that reduce its fitness can be either aggravated by indirect consequences (i.e. where parasitism renders predation more likely), or improved by indirect consequences.

1.2. Apparent Competition

In the context of parasitism, apparent competition is predicted to occur when two species that do not otherwise interact both host with the same parasite species [16-18, 22]. As both host species are a resource for the parasite, population density increases in either host lead to reductions in the other, via the, density mediated, negative effects of the parasite. Apparent competition can theoretically lead to the elimination of one host, indirectly coupling the dynamics of host species with different habitat or resource requirements.

In view of these, this study targeted to present these synthesized empirical and theoretical work using mathematical modeling and analysis qualitatively how parasites influences competitive and predatory interactions between species [1, 7, 10, 13, 21, 23]. We highlight the sensitive parameters to assess the impact of parasites on communities by incorporating the theoretical and empirical studies so as to examine how the effects of parasitism. Moreover, parasitism scales up the community-level processes.

In this paper, comparison of the model system would be carried out through the investigation of the interaction made between the populations directly and indirectly to each other and with parasite.

2. Assumption and Model Formulation

Accordingly, Mathematical epidemiology to study the dynamics of diseases spread has become an interesting topic of research study and received much attention from scientists after the pioneering work of [2, 3, 13-15, 20].

In this recent study, by applying a deterministic framework of an ecological model of the infection of parasite, the analysis of the population dynamics of eco-epidemiology would be investigated.

To start with, the mathematical model of a single type of host and its parasite would be defined by considering the following descriptions. These are the changes in the numbers of hosts H and free-living parasite. These changes are due to three minimum processes: host birth, mortality due to parasite infection and natural death of a host. Hence, the classical model of the combination of host and parasite population growth with carrying capacity of host which follows the general deterministic trajectories defined as

$$\frac{dH}{dt} = rH \left(1 - \frac{H}{k}\right) - \delta HP \quad (1)$$

$$\frac{dP}{dt} = \delta mHP - dP; \quad (2)$$

This model is shown with simple diagram in figure 1. This system is similar to a predator-prey system but differing by the quantity m and called in host-parasite system burst size where as in predator-prey system ecological efficiency. Regardless of the potentially widespread effects of parasites on the community, most studies of host-parasite dynamics focus on the effects of a single parasite on a single host,

neglecting the host's interactions with each other and with other species.

The present model considers interaction of two hosts. These hosts do not directly interact with each other but they are mediated by a common enemy or parasite.

In this paper, the recovery of the hosts would not be considered. Moreover, in this study the interactions involving parasites that feed on a host individual usually living on or in it and often causing harm but not immediate death and the carrying capacity is only allowed for the uninfected hosts. i.e., for infected hosts limited resources are available. Further, responses of the hosts are linear rather than saturated. In this assumption, the model for free living parasite is not explicitly modeled.

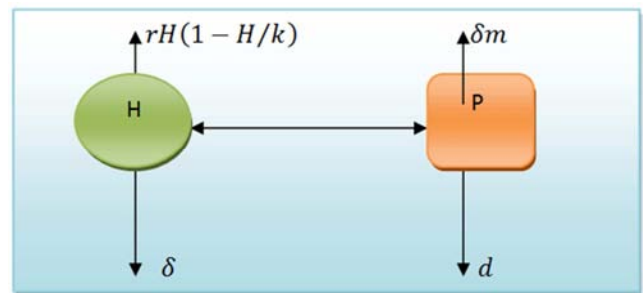


Figure 1. Simple schematic diagram of system (1) and (2).

The interaction between the states of the present model is illustrated in Figure 2 below;

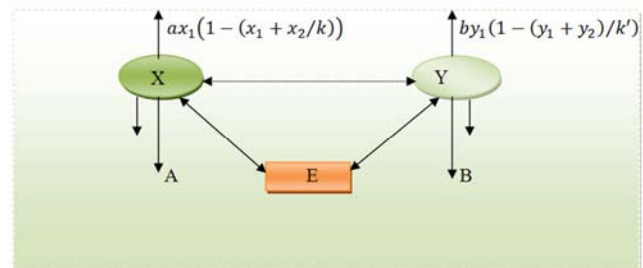


Figure 2. Schematic diagrams of the population dynamics.

The present mathematical model is formulated based on the above schematic model, and is given by the following system of differential equation:

$$\frac{dx_1}{dt} = ax_1 \left(1 - \frac{x_1 + x_2}{k}\right) - \mu x_1 x_2 - \alpha x_1 \quad (3)$$

$$\frac{dx_2}{dt} = \mu x_1 x_2 - A x_2; \quad A = \alpha + \delta \quad (4)$$

$$\frac{dy_1}{dt} = by_1 \left(1 - \frac{y_1 + y_2}{k'}\right) - r y_1 y_2 - \theta y_1 \quad (5)$$

$$\frac{dy_2}{dt} = r y_1 y_2 - B y_2; \quad B = \beta + \theta \quad (6)$$

Here, $X(t) = x_1(t) + x_2(t)$ and $Y(t) = y_1(t) + y_2(t)$ denote total population sizes of the first and second hosts respectively. However, the total size of both the hosts in the system is given by $W(t) = X(t) + Y(t)$.

Descriptions of notations, parametric values of the system

are given in the following tables.

Table 1. Notations and descriptions of the model variables.

Variables	Descriptions
$x_1(t)$	Density of first susceptible host population
$x_2(t)$	Density of first infectious host population
$y_1(t)$	Density of second susceptible host population
$y_2(t)$	Density of second infectious host population

Table 2. Notations and descriptions of the parameters used in the model.

Parameters	Descriptions
a	Growth rate of host-1
b	Growth rate of host-2
μ	Infection transmission rate of host-1
r	Infection transmission rate of host-2
α	Natural death rate of host-1
θ	Natural death rate of host-2
β	Mortality rate due to disease of host-2
A	Total death rate of host-1
B	Total death rate of host-2
k	Environmental carrying capacity for host-1
k'	Environmental carrying capacity for host-2

3. Qualitative Analysis of the Model Systems

3.1. Positivity and Boundedness of the System

For the model to be meaningful and well posed, it is necessary that the solutions of (3)–(6) with positive initial data are positive and bounded for all $t > 0$. This fact has been stated and proved in the following:

Proposition 1: All solutions of the model equations (3)–(6) are non-negative and bounded.

Proof:

Positivity of the populations: consider the equation of system (3) - (6).

Up on integrating the model equations (3)–(6) with respect to t the analytical solutions of the model variables are obtained as

$$\begin{aligned}
 x_1(t) &= x_{01} \exp \int_0^t \left\{ a \left(1 - \frac{x_1(s) + x_2(s)}{k} \right) - \mu x_2(s) - \alpha \right\} ds \\
 x_2(t) &= x_{02} \exp \int_0^t \{ \mu x_1(q) - A \} dq \text{ Host - 1} \\
 y_1(t) &= y_{01} \exp \int_0^t \left\{ b \left[1 - \frac{y_1(u) + y_2(u)}{k'} \right] - r y_2(u) - \theta \right\} du \text{ Host - 2} \\
 y_2(t) &= y_{02} \exp \int_0^t \{ r y_1(v) - B \} dv
 \end{aligned}$$

Here, it can be observed that each solution is a product of an initial condition and an exponential function. However, by definition every initial condition and exponential function are non-negative quantities, so are their products.

Therefore, all solutions of the system of equations (3)–(6) are non-negatives for all $t \geq 0$.

Boundedness of the system

In theoretical eco-epidemiology, a system is bounded implies that the system is biologically valid and well behaved. Here in the present study, the biological validity of the model is achieved by showing that the solutions of (3)–(6) are bounded.

In order to show the system is bounded, let the expression for the total population of the system as $W(t) = x_1(t) + x_2(t) + y_1(t) + y_2(t)$ be considered. Also, let $mW = m(x_1 + x_2 + y_1 + y_2)$ and $(dW/dt) = (dx_1/dt) + (dx_2/dt) + (dy_1/dt) + (dy_2/dt)$ where m is any positive constant. Now, using the fore going expressions in the addition of (3)–(6) reduces the result as:

$$\begin{aligned}
 \frac{dW}{dt} + mW &= ax_1 \left(1 - \frac{x_1 + x_2}{k} \right) + by_1 \left(1 - \frac{y_1 + y_2}{k'} \right) - (\alpha x_1 + Ax_2 + \theta y_1 + By_2) + mW \\
 \frac{dW}{dt} + mW &\leq ax_1 \left(1 - \frac{x_1 + x_2}{k} \right) + by_1 \left(1 - \frac{y_1 + y_2}{k'} \right) + mx_1 + my_1, \text{ For } A \text{ and } B > m \\
 &= (a + m)x_1 - a \frac{x_1^2}{k} - a \frac{x_1 x_2}{k} + (b + m)y_1 - b \frac{y_1^2}{k'} - b \frac{y_1 y_2}{k'} \\
 &\leq (a + m)x_1 - a(x_1^2/k) + (b + m)y_1 - b(y_1^2/k') \\
 &= \frac{k}{4a}(a + m)^2 - \frac{a}{k} \left[x_1 - \frac{k}{2a}(a + m) \right]^2 + \frac{k'}{4b}(b + m)^2 \\
 &\quad - \frac{b}{k'} \left[y_1 - \frac{k'}{2b}(b + m) \right]^2 \\
 &\leq (k/4a)(a + m)^2 + (k'/4b)(b + m)^2
 \end{aligned}$$

Now, introducing notation $\omega = (k/4a)(a + m)^2 + (k'/4b)(b + m)^2$ the differential inequality reduces to the form $(dW/dt) + mW \leq \omega$. It is a first order ordinary differential equation with constant coefficients and its analytical solution is given by $W(t) \leq (\omega/m)(1 - e^{-mt}) + W_0 e^{-mt}$. However, in the limit as $t \rightarrow \infty$, the solution takes the form as $W(t) \leq (\omega/m)$ showing that the total population size is bounded. Hence, each population size is also bounded. Thus, the statement holds true.

3.2. Existence of Steady States of the Model

The model equations (3)–(6) possess equilibrium points which are biologically feasible. These are obtained by setting $(dx_1/dt) = (dx_2/dt) = (dy_1/dt) = (dy_2/dt) = 0$ and solving the resultant expressions. Thus, the nine equilibrium points of the model and the corresponding existence conditions are listed as follows:

- (1) Trivial equilibrium $E_0 = (0, 0, 0, 0)$ always exists. At this all the populations are absent.
- (2) Axial equilibrium $E_1 = (x_1^*, 0, 0, 0)$ exists if $a > \alpha$. Here, only susceptible host-1 is present.
- (3) Axial equilibrium $E_2 = (0, 0, y_1^*, 0)$ exists

if $b > \theta$. Here, only susceptible host-2 is present.

- (4) Disease free equilibrium $E_3 = (x_1^*, 0, y_1^*, 0)$ exists. At this equilibrium both susceptible host-1 and host-2 are present.
- (5) Equilibrium $E_4 = (x_1^{**}, x_2^{**}, 0, 0)$ exists if $a[1 - (A/\mu k)] > \alpha$ and $\mu k > A$. Here both susceptible and infected populations of host-1 are present.
- (6) Equilibrium $E_5 = (0, 0, y_1^{**}, y_2^{**})$ exists if $b[1 - (B/rk')] > \theta$ and $rk' > B$. Here both susceptible and infected populations of host-2 only exist.
- (7) Equilibrium $E_6 = (x_1^*, 0, y_1^{**}, y_2^{**})$ exists. This is a disease-free equilibrium of host-1.
- (8) Equilibrium $E_7 = (x_1^{**}, x_2^{**}, y_1^*, 0)$ exists. This is a disease-free equilibrium of host-2.
- (9) Equilibrium $E_8 = (x_1^{**}, x_2^{**}, y_1^{**}, y_2^{**})$ exists. This is a co-existence equilibrium i.e., all types of hosts present here.

Here in the equilibrium points some notations are used in the coordinates those represent the following expressions:

$$x_1^* = [k(a - \alpha)/a], x_1^{**} = (A/\mu), x_2^{**} = [k/(\mu k + a)][a - \alpha - (aA/\mu k)],$$

$$y_1^* = [k'(b - \theta)/b], y_1^{**} = B/r, y_2^{**} = [k'/(b + rk')][b - \theta - (bB/rk')].$$

3.3. Variational Matrix of the Model Equations

Set the model equations (3)–(6) as

$$dx_1/dt = f(x_1, x_2, y_1, y_2),$$

$$M = \begin{bmatrix} a \left(1 - \frac{2x_1 + x_2}{k} \right) - \mu x_2 - \alpha & -\frac{ax_1}{k} - \mu x_1 & 0 & 0 \\ \mu x_2 & \mu x_1 - A & 0 & 0 \\ 0 & 0 & b \left(1 - \frac{2y_1 + y_2}{k'} \right) - ry_2 - \theta & -\frac{by_1}{k'} - ry_1 \\ 0 & 0 & ry_2 & ry_1 - B \end{bmatrix}$$

3.4. Stability Analysis of Model System

3.4.1. Local Stability of the System

Recall that,

- (i) An equilibrium point is said to stable if the variational matrix at that equilibrium has all negative eigenvalues,
- (ii) An equilibrium point is said to unstable if the variational matrix at that equilibrium has at least one positive eigenvalues,

This fact is used in verifying whether a given equilibrium is stable or unstable. Here, Local stability analysis of the system at the equilibrium points is conducted and the results are presented in form of theorems and proofs in what follows:

Theorem 1 The trivial equilibrium point $E_0 = (0, 0, 0, 0)$ is unstable.

Proof:

The variational matrix M at the equilibrium point E_0 is denoted by M_0 and is given by

$$dx_2/dt = g(x_1, x_2, y_1, y_2),$$

$$dy_1/dt = h(x_1, x_2, y_1, y_2),$$

$$dy_2/dt = k(x_1, x_2, y_1, y_2)$$

The variational matrix is defined as

$$M = \begin{pmatrix} \frac{\partial f}{\partial x_1} & \frac{\partial f}{\partial x_2} & \frac{\partial f}{\partial y_1} & \frac{\partial f}{\partial y_2} \\ \frac{\partial g}{\partial x_1} & \frac{\partial g}{\partial x_2} & \frac{\partial g}{\partial y_1} & \frac{\partial g}{\partial y_2} \\ \frac{\partial h}{\partial x_1} & \frac{\partial h}{\partial x_2} & \frac{\partial h}{\partial y_1} & \frac{\partial h}{\partial y_2} \\ \frac{\partial k}{\partial x_1} & \frac{\partial k}{\partial x_2} & \frac{\partial k}{\partial y_1} & \frac{\partial k}{\partial y_2} \end{pmatrix}$$

Now, the elements of the matrix M are computed as:

$$\partial f / \partial x_1 = a \{ 1 - [(2x_1 + x_2)/k] \} - \mu x_2 - \alpha, \partial f / \partial x_2 = -[(ax_1/k) + \mu x_1],$$

$$\partial f / \partial y_1 = \partial f / \partial y_2 = 0, \partial g / \partial x_1 = \mu x_2, \partial g / \partial x_2 = \mu x_1 - A,$$

$$\partial g / \partial y_1 = \partial g / \partial y_2 = 0, \partial h / \partial x_1 = \partial h / \partial x_2 = 0, \partial h / \partial y_1 = b \{ 1 - [(2y_1 + y_2)/k'] \} - ry_2 - \theta$$

$$\partial h / \partial y_2 = -[(by_1/k') + ry_1], \partial k / \partial x_1 = \partial k / \partial x_2 = 0, \partial k / \partial y_1 = ry_2, \partial k / \partial y_2 = ry_1 - B.$$

Thus, the variational matrix takes the form as

$$M_0 = \begin{bmatrix} a - \alpha & 0 & 0 & 0 \\ 0 & -A & 0 & 0 \\ 0 & 0 & b - \theta & 0 \\ 0 & 0 & 0 & -B \end{bmatrix}$$

Its characteristic equation defined by $\det(M_0 - \lambda I) = 0$ takes the form as $(a - \alpha - \lambda)(-A - \lambda)(b - \theta - \lambda)(-B - \lambda) = 0$. On solving which eigenvalues of M_0 are obtained as $\lambda_1 = a - \alpha, \lambda_2 = -A, \lambda_3 = b - \theta, \lambda_4 = -B$. Here, λ_1 and λ_3 are positive eigenvalues since the parameters satisfy the relations $a > \alpha$ and $b > \theta$. That is, here at least one eigenvalues is positive. Hence, the equilibrium point E_0 is unstable.

Theorem 2 The disease-free equilibrium point $E_1 = (x_1^*, 0, y_1^*, 0)$ is stable if the following conditions on the parameters hold true: $[(A/\mu k) + (\alpha/a)] > 1$ and $[(B/rk') + (\theta/b)] > 1$.

Proof:

The variational matrix M at the disease-free equilibrium point E_1 is denoted by M_1 and given by:

$$M_1 = \begin{bmatrix} \alpha - a & 0 & 0 & 0 \\ 0 & \frac{\mu k(a - \alpha)}{a} - A & 0 & 0 \\ 0 & 0 & \theta - b & 0 \\ 0 & 0 & 0 & -B \end{bmatrix}$$

Its characteristic equation is given by $\det(M_1 - \lambda I) = 0$. On solving which the eigenvalues of M_1 are obtained as $\lambda_1 = \alpha - a, \lambda_2 = \{[\mu k(a - \alpha)/a] - A\}, \lambda_3 = (\theta - b), \lambda_4 = [rk'(b - \theta)/b] - B$. Clearly, λ_1 and λ_3 are

$$M_2 = \begin{bmatrix} a - \alpha & 0 & 0 & 0 \\ 0 & -A & 0 & 0 \\ 0 & 0 & (b - \theta - bB/rk')(rk'/b + rk') - bB/rk' & -B/r(b + rk'/b) \\ 0 & 0 & (rk'/b + rk')(b - \theta - bB/rk') & 0 \end{bmatrix}$$

Its characteristic equation is given by $\det(M_2 - \lambda I) = 0$. By solving the eigenvalues of M_2 becomes $\lambda_1 = \alpha - a, \lambda_2 = -A, \lambda_3 = (1/2)[-Q + \sqrt{Q^2 - 4K}], \lambda_4 = (1/2)[-Q - \sqrt{Q^2 - 4K}]$ where $Q = [b - \theta - (bB/rk')][rk'/(b + rk')] - (bB/rk'), K = (B/b)[\theta - b + (bB/rk')]$. It can be observed that both eigenvalues λ_1 and λ_2 are negative quantities. Also, the values λ_3 and λ_4 are also negatives if both the conditions $Q^2 - 4K > 0$ and $K > 0$ hold true simultaneously.

Thus, equilibrium point E_2 is stable under the considered condition.

Theorem 4: The equilibrium point $E_3 = (x_1^*, x_2^*, 0, 0)$ is stable if $b < \theta$ and $H > 1$ otherwise unstable.

Proof:

Consider

$$M_3 = \begin{bmatrix} (-aA/\mu k) & -A(a + k\mu)/\mu & 0 & 0 \\ (\mu k/\mu k + a)(a - \alpha - aA/\mu k) & 0 & 0 & 0 \\ 0 & 0 & b - \theta & 0 \\ 0 & 0 & 0 & -B \end{bmatrix}$$

But applying the elementary linear algebra on interchanging column operation this M_3 is equivalent to;

$$M'_3 = \begin{bmatrix} -A(a + k\mu)/\mu & (-aA/\mu k) & 0 & 0 \\ 0 & (\mu k/\mu k + a)(a - \alpha - aA/\mu k) & 0 & 0 \\ 0 & 0 & b - \theta & 0 \\ 0 & 0 & 0 & -B \end{bmatrix}$$

Now, the variational matrix at equilibrium E_3 is equivalently denoted by M_3' and its characteristic equation is given by $\det(M_3' - \lambda I) = 0$. This gives the eigenvalues

$\lambda_1 = -A(a + k\mu)/\mu, \lambda_2 = H, \lambda_3 = b - \theta, \lambda_4 = -B$. Where, $H = (\mu k/\mu k + a)(a - \alpha - aA/\mu k)$. Here, unconditionally $\lambda_1 < 0$ and $\lambda_4 < 0$. However, λ_2 and λ_3 would be negative if the condition $b < \theta$ and $H > 1$.

Hence, the equilibrium E_3 is stable under the stated condition.

Theorem 5: The equilibrium point $(0, 0, y_1^*, 0)$ is stable if $[rk'(b - \theta)/b] < B$.

Proof:

The variational matrix M at this equilibrium is denoted by M_6 and given by

negative values. But, λ_2 and λ_4 are negative quantities if the conditions $[(A/\mu k) + (\alpha/a)] > 1$ and $[(B/rk') + (\theta/b)] > 1$ are satisfied. Hence, the equilibrium point E_1 is stable under the considered assumption.

Theorem 3: The equilibrium point $E_2 = (0, 0, y_1^*, y_2^*)$ is stable if $Q^2 - 4K > 0$.

Proof:

The variational matrix M at equilibrium E_2 is denoted by M_1 and given by

$$M_6 = \begin{bmatrix} a - \alpha & 0 & 0 & 0 \\ 0 & -A & 0 & 0 \\ 0 & 0 & \theta - b & -\frac{(b + rk')(b - \theta)}{b} \\ 0 & 0 & 0 & rk'(b - \theta)/b - B \end{bmatrix}$$

The characteristic equation $\det(M_6 - \lambda I) = 0$ of the variation matrix M_6 at the equilibrium E_6 takes the form as $(\alpha - a - \lambda)(-A - \lambda)(\theta - b - \lambda)r[k'(b - \theta)/b] - B - \lambda = 0$ giving the eigenvalues $\lambda_1 = \alpha - a, \lambda_2 = -A, \lambda_3 = \theta - b, \lambda_4 = r[k'(b - \theta)/b] - B$. Here, $\lambda_2 < 0$ unconditionally. However, the remaining eigenvalues are conditionally negatives with the conditions

- (i) $\lambda_1 < 0$ if $\alpha < a$
- (ii) $\lambda_3 < 0$ if $\theta < b$
- (iii) $\lambda_4 < 0$ if $rk'(b - \theta)/b < B$.

Here, under the considered condition the equilibrium point E_6 is stable.

Theorem 6: The equilibrium point $E_7(x_1^*, 0, 0, 0)$ is stable if $[\mu k(a - \alpha)/a] < A$.

Proof:

$$M_7 = \begin{bmatrix} \alpha - a & -(a - \alpha)(a + \mu k)/a & 0 & 0 \\ 0 & (\mu k(a - \alpha)/a) - A & 0 & 0 \\ 0 & 0 & b - \theta & 0 \\ 0 & 0 & 0 & -B \end{bmatrix}$$

The characteristic equation $\det(M_7 - \lambda I) = 0$ of the variation matrix M_7 at the equilibrium E_7 takes the form as $(\alpha - a - \lambda)\{[\mu k(a - \alpha)/a] - A - \lambda\}(b - \theta - \lambda)(-B - \lambda) = 0$ giving the eigenvalues $\lambda_1 = \alpha - a, \lambda_2 = [\mu k(a - \alpha)/a] - A, \lambda_3 = b - \theta, \lambda_4 = -B$. Here, it can be observed that λ_4 is unconditionally negative while the other eigenvalues are negatives conditionally with the following conditions:

- (i) $\lambda_1 < 0$ if $\alpha < a$
- (ii) $\lambda_2 < 0$ if $\mu k(a - \alpha)/a < A$
- (iii) $\lambda_3 < 0$ if $b < \theta$. Based on the set condition, the equilibrium point is stable.

3.4.2. Global Stability of the System

To determine the global stability of the system Lyapunov function method is followed in this study.

Theorem 7: The interior equilibrium point

$(x_1^*, x_2^*, y_1^*, y_2^*)$ is globally stable.

Proof:

Construct the Lyapunov function as

$$\begin{aligned} v(x_1, x_2, y_1, y_2) &= x_1 - x_1^* - x_1^* \ln(x_1/x_1^*) \\ &+ l[x_2 - x_2^* - x_2^* \ln(x_2/x_2^*)] \\ &+ m[y_1 - y_1^* - y_1^* \ln(y_1/y_1^*)] \\ &+ s[y_2 - y_2^* - y_2^* \ln(y_2/y_2^*)]. \end{aligned}$$

Where m, l, s are chosen constants.

Now, the differentiation of v with respect to t and after some algebraic manipulation reduces to the form as follow;

$$\begin{aligned} \frac{dv}{dt} &= \left(\frac{x_1 - x_1^*}{x_1}\right) \left(\frac{dx_1}{dt}\right) + l \left(\frac{x_2 - x_2^*}{x_2}\right) \left(\frac{dx_2}{dt}\right) \\ &+ m \left(\frac{y_1 - y_1^*}{y_1}\right) \left(\frac{dy_1}{dt}\right) + s \left(\frac{y_2 - y_2^*}{y_2}\right) \left(\frac{dy_2}{dt}\right) \\ &= \left(\frac{x_1 - x_1^*}{x_1}\right) a x_1 \left(1 - \frac{x_1 + x_2}{k}\right) - \mu(x_1 x_2) - \alpha x_1 \\ &\quad + l \left(\frac{x_2 - x_2^*}{x_2}\right) \mu(x_1 x_2) - A x_2 \\ &\quad + m \left(\frac{y_1 - y_1^*}{y_1}\right) b y_1 \left(1 - \frac{y_1 + y_2}{k'}\right) - r(y_1 y_2) - \theta y_1 \end{aligned}$$

$$\begin{aligned} &+ s \left(\frac{y_2 - y_2^*}{y_2}\right) r(y_1 y_2) - B y_2 \\ &= (x_1 - x_1^*) \left[a \left(1 - \frac{x_1 + x_2}{k}\right) - \mu x_2 - \alpha \right] \\ &\quad + l(x_2 - x_2^*) [\mu x_1 - A] \\ &+ m(y_1 - y_1^*) \left[b \left(1 - \frac{y_1 + y_2}{k'}\right) - r y_2 - \theta \right] \\ &\quad + s(y_2 - y_2^*) r[(y_1) - B] \\ &= -(a/k)(x_1 - x_1^*)^2 - \mu(x_2 - x_2^*)(x_1 - x_1^*) + \\ &\quad l\mu(x_2 - x_2^*)(x_1 - x_1^*) \\ &- (bm/k')(y_1 - y_1^*)^2 - mr(y_1 - y_1^*)(y_2 - y_2^*) \\ &\quad + sr(y_2 - y_2^*)(y_1 - y_1^*) \end{aligned}$$

For simplicity, choosing $l = 1$ and $m = s$ the foregoing time derivative reduces to $(dv/dt) = -(a/k)(x_1 - x_1^*)^2 - (bm/k')(y_1 - y_1^*)^2$. Now, it can be observed that $(dv/dt) < 0$

Therefore, the interior equilibrium point is globally stable.

3.5. Numerical Simulations

In this section, numerical simulations of the dynamics of the model equation around some of the steady state for certain range of parametric values are used to support the analytical results.

The parameters and its values used in this study are mentioned in the following table as follow;

Table 3. Parameters and their values for figures 3-9.

Figures	Parameters and their values									
	a	b	μ	r	α	θ	δ	β	k	k'
3	1.1940	0.2550	0.3900	3.3920	2.4150	0.6000	2.1600	1.0980	0.6250	2.3920
4	3.1260	3.9550	0.1600	6.2000	3.1050	0.4800	15.2200	1.6470	0.5450	6.9200
5	3.0660	2.6200	6.0300	4.1440	8.0700	0.4800	11.9400	2.1960	2.6350	7.6400
6	5.5380	0.8700	8.4600	1.1040	1.7250	5.3820	1.0400	6.0210	3.5100	1.6240
7	5.5380	1.1250	8.4600	5.0960	1.7250	5.2680	4.4000	7.9020	3.5100	6.0160
8	5.4960	1.4950	0.2300	1.8240	0.5850	0.2520	2.4200	3.9060	1.2500	2.8320
9	5.4960	2.5400	0.2300	3.1600	0.5850	0.2700	2.4200	8.0730	1.2500	5.0640

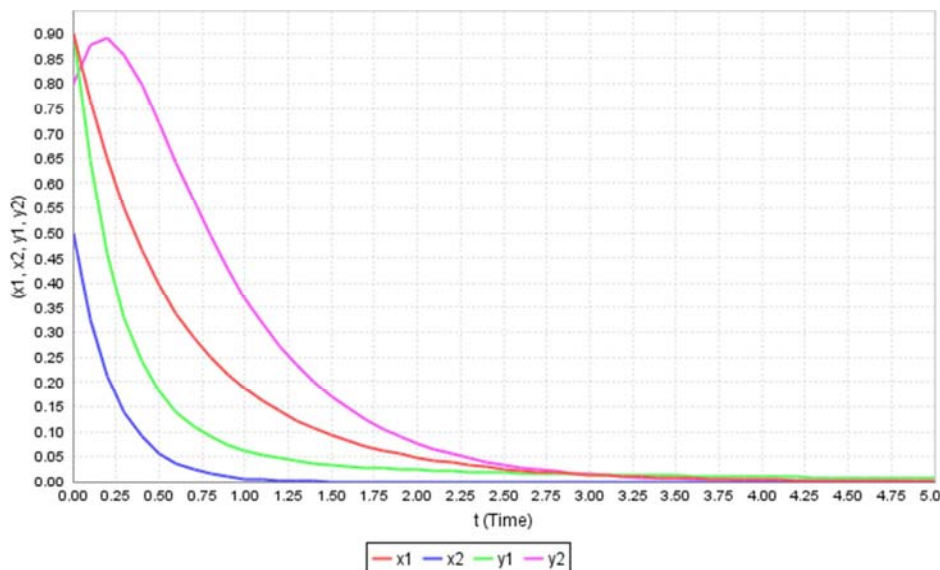


Figure 3. All populations vanish together over time.

This figure shows that when growth rate of both populations is less than the natural death rate and attacked by disease all the populations become completely extinct.

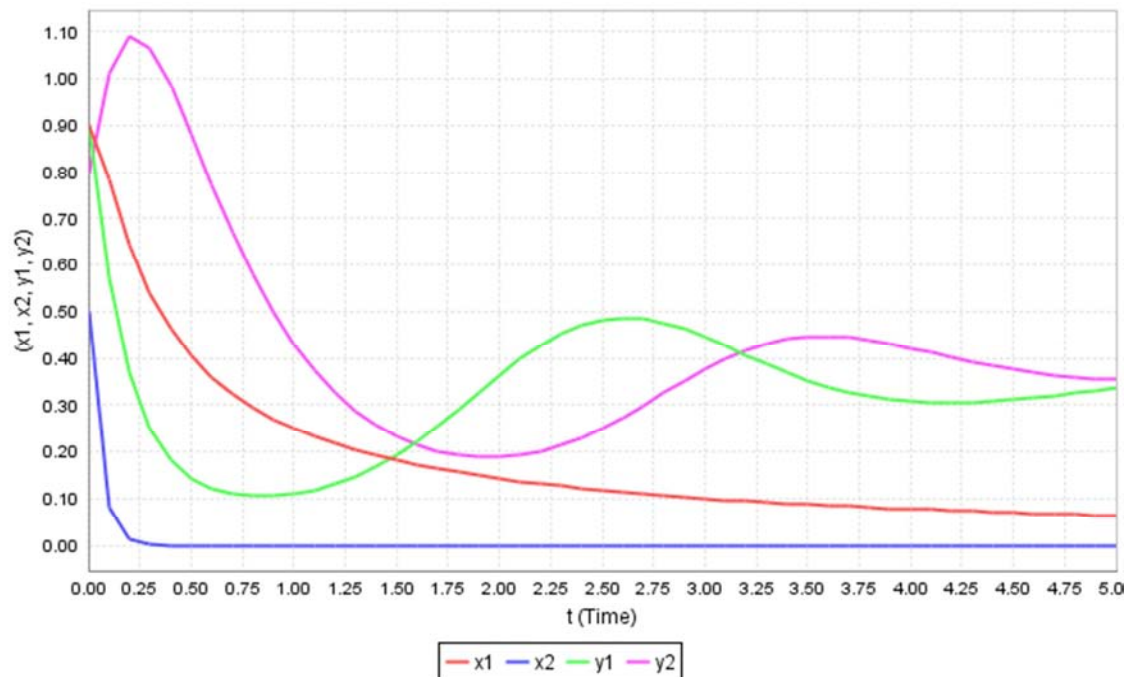


Figure 4. The populations of the second host exist with disease.

This figure shows that the growth rate of host-2 is greater than its natural death rate and the first host more affected and hence declines more.

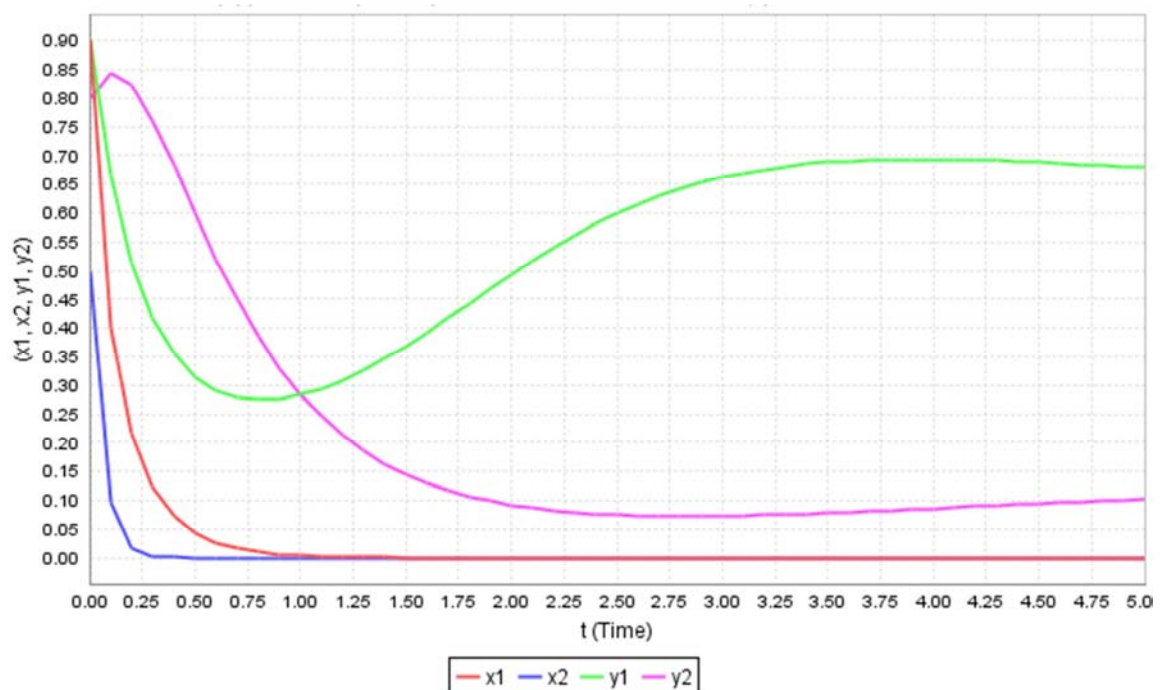


Figure 5. The population of the second host dominant over the other.

This figure shows that the infected host declines more due to the infection rate and natural death rate of host-1 is increased keeping constant death rate of host-2.

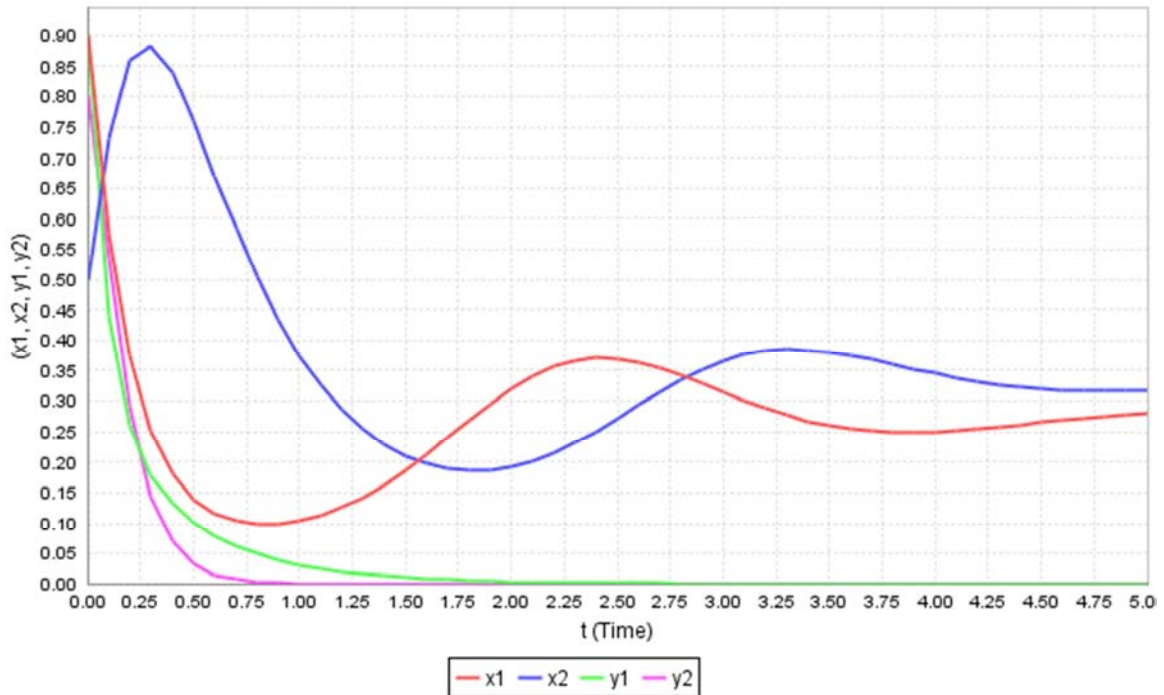


Figure 6. The host population around the equilibrium point E_3 .

One can observe from this figure that host-2 is more affected by disease and die due to natural death.

But once the populations of the infected starts to decline, the first host become slightly increasing and regulate each other by forcing the second host to extinct without recovery.

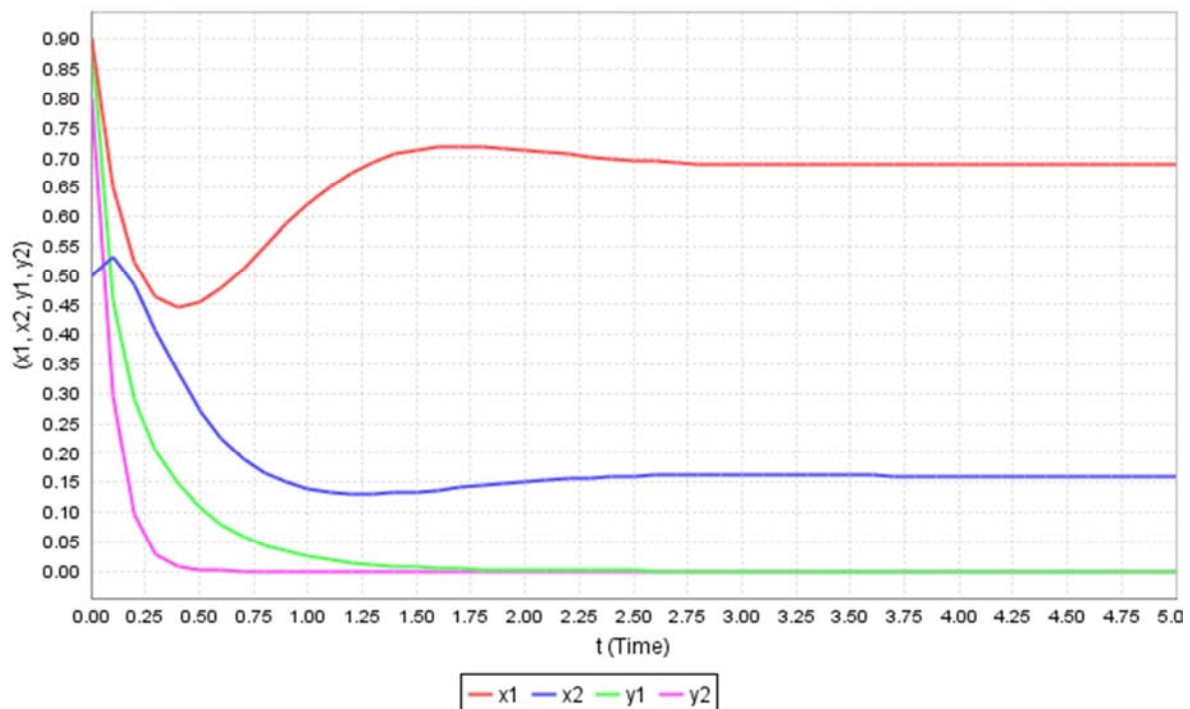


Figure 7. Host-1 population dominant over host-2 population.

This figure shows that host-2 is more infected and hence the mortality rate due to disease increases while the first host keeps increasing up to certain time.

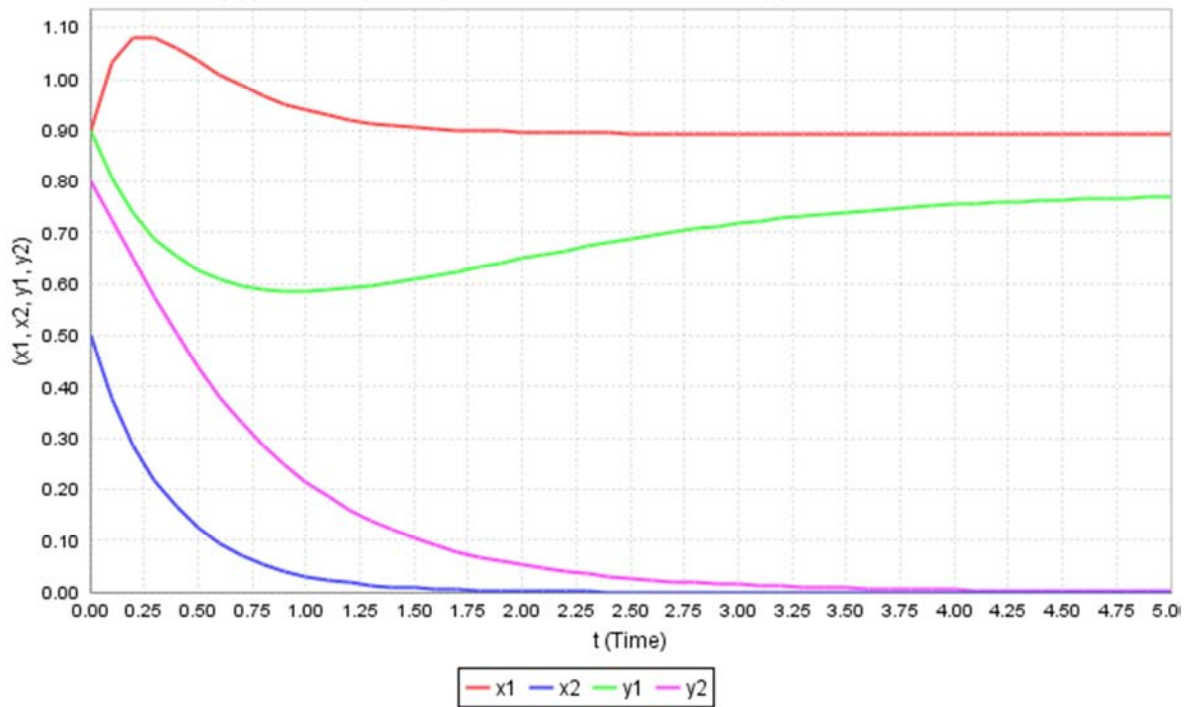


Figure 8. Population dynamics around the equilibrium point E_1 .

This figure shows that both host populations would live together without harming each other when the infected hosts are decline and extinct without recovery.

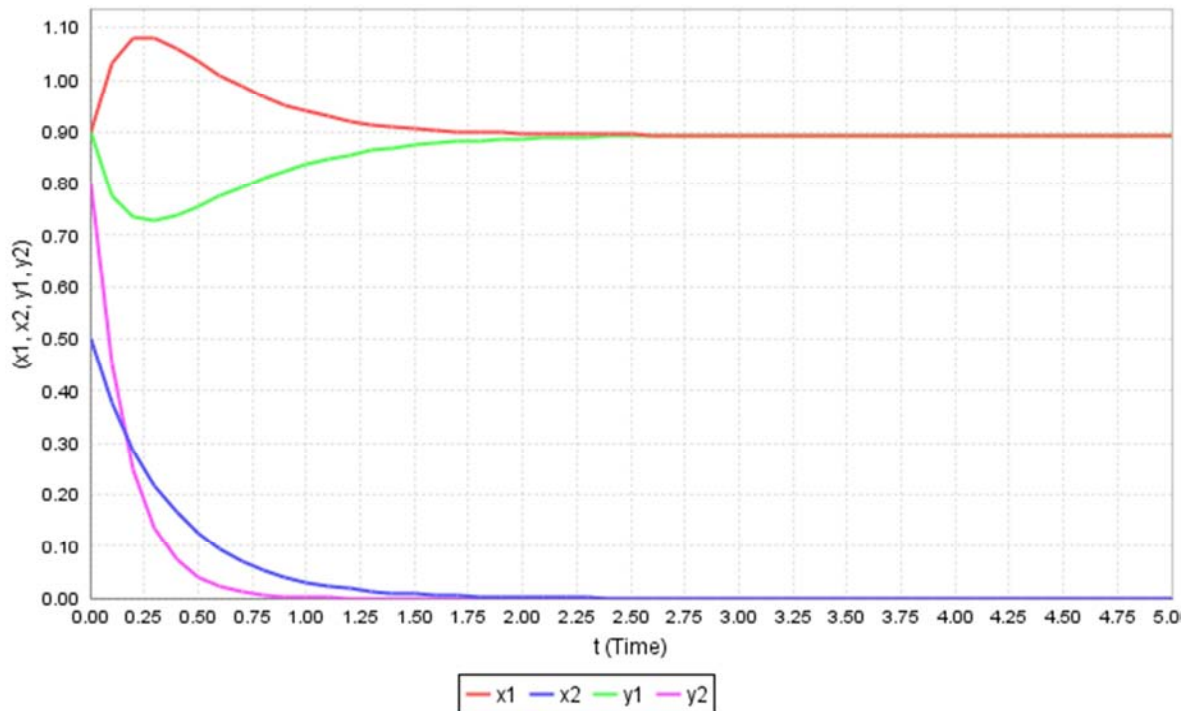


Figure 9. Stabilization of both populations.

This figure shows that when infection transmission rate of host-1 and mortality rate due to disease of host-2 increased keeping the other parameters constant the populations become stabilized.

4. Conclusion

In the present study the population dynamics of four dimensional systems has been taken up for investigation. A

mathematical model for this four dimensional system is developed by studying carefully and incorporating the futures of each system together. The positivity and boundedness of the model variables are verified and hence shown that the developed four-dimensional system is biologically well behaved.

In this paper, species have strong indirect effects on others, and predicting these effects is a central challenge in ecology. Host species sharing an enemy can be linked by apparent competition, that is interaction between two hosts and parasite is carried out for investigation. In the case where infection was permitted, parasite coexists in the population, but only under particular conditions, namely when rates of infection varies both hosts.

The results from the model system demonstrate that the important role that parasite may play in the establishment of community structures within host populations' dynamics. This was indicated from the stability of interior equilibrium point in which the populations are coexisting.

We need to point out here that, although the model considered in this study is two host populations via shared enemy model. Even though there is no direct interactions specifically associated with the hosts populations, the effects of each host was investigated by altering parameters involved in the system. It has been shown that, this process is strong enough to be a community-wide structuring mechanism that could be used to predict future states of diversification. To show this, numerical study of the model was carried out to support the finding. Moreover, the model system would be fully screened when there is a competition between the hosts that would be mediated by parasite interaction providing shared resources. This would be our next further investigation.

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References

- [1] AlyssaLois M. (2016). Non-native parasite enhances susceptibility of host to native predators, Springer-Verlag, Berlin, Heidelberg.
- [2] Anderson R. M., May R. M. (1981). The population dynamics of micro-parasites and their invertebrate hosts. *Phil. Trans. R. Soc. Lond.* 291, 451–524.
- [3] Anderson, R. and May R. *Infectious Diseases of Humans, Dynamics and Control*. Oxford University Press, Oxford (1991).
- [4] Benjamin J. Z., Buckling A. (2012). The mode of host-parasite interaction shapes co-evolutionary dynamics and the fate of host cooperation. *Proc. R. Soc. Lond., B* 279, 3742–48.
- [5] Bowers R. G., Boots M., Begon M. (1994). Life-history trade-offs and the evolution of pathogen resistance: competition between host strain. *Proc. R. Soc. Lond., B* 257, 247–53.
- [6] Chakra M. A., Hilbe C. and Treutlen A. (2014). Plastic behaviors in hosts promote the emergence of retaliatory parasites, *Sci. Rep.*, 4, 4251.
- [7] D. Adak and N. Bairagi. (2014). Dynamical behavior of a predator-prey-parasite model with nonlinear incidence rate, *J. Biol. Syst.*, 1 (1).
- [8] Diekmann O., Heesterbeek J. A. P. (2000). *Mathematical Epidemiology of Infectious Diseases: Model Building, Analysis and Interpretation*. Wiley, New York, p. 365.
- [9] Dobson A. P. (2004). Population dynamics of pathogens with multiple host species. *Am. Nat.*, 164, S64–S78.
- [10] Fanghong Zhang and Cuncheng Jin. (2017). Analysis of an eco-epidemiological model with Disease in the prey and predator. 6 (1), 22–28.
- [11] GeremewKenassa Edessa, BokaKumsa, Purnachandra Rao Koya. (2018) Modeling and Simulation Study of the Population Dynamics of Commensal-Host-Parasite System. *American Journal of Applied Mathematics*. Vol. 6, No. 3, pp. 97–108.
- [12] GeremewKenassa Edessa, BokaKumsa, Purnachandra Rao Koya (2018). Dynamical behavior of Susceptible prey–Infected prey–Predator Populations. *IOSR Journal of Mathematics (IOSR-JM)* 14.4 PP: 31–41.
- [13] Hatcher P. E., Moore J., Taylor J. E., Tinney G. W. and Paul N. D. (2004). Phytohormones and plant–herbivore–pathogen interactions: integrating the molecular with the ecological. *Ecology*, 85, 59–69.
- [14] Hatcher P. E. and Paul N. D. (2000) Beetle grazing reduce natural infection of *Rumex obtusifolius* by fungal pathogens, *New Phytol*, 146, 325–33.
- [15] Hatcher P. E., Paul N. D., Ayres P. G. & Whittaker J. B. (1994). The effect of an insect herbivore and a rust fungus individually and combined in sequence on the growth of two *Rumex* species. *New Phytol*, 128, 71–78.
- [16] Holt R. D. & Dobson A. P. (2006). Extending the principles of community ecology to address the epidemiology of host pathogen systems. In: *Disease Ecology: Community Structure and Pathogen Dynamics*. Oxford University Press, Oxford, pp. 6–27.
- [17] Holt R. D. & Hochberg M. E. (1998). The coexistence of competing parasites. Part II–Hyper parasitism and food chain dynamics. *J. Theor. Biol.*, 193, 485–95.
- [18] Holt R. D. (1977) Predation, apparent competition, and the structure of prey communities. *Theoretical Population Biology*, 12: 197–229.
- [19] Rainey P. B. (2002). Antagonistic co-evolution between a bacterium and a bacteriophage. *Proc. R. Soc. Lond. B* 269, 931–36.
- [20] Sand land G. J., Minchella D. J. (2004). Life-history plasticity in hosts exposed to differing resources and parasitism. *Can. J. Zool.*, 82, 1672–77.
- [21] Sen P., Das k. (2017). Simultaneous Effects of Prey Defence and Predator Infection on a Predator Prey System. *Ann. Bio. Sci.*, 5 (1): 37–46.

- [22] Siekmann. I., (2013). On competition in ecology, epidemiology and eco - epidemiology. Ecol. Complex, <http://dx.doi.org/10.1016/j.ecocom.2013.01.003>.
- [23] Sule H., MuhamadR., and Omar D. (2014). Parasitism rate host stage preference and functional response of *Tamarixiaradiata* on *Diaphorinacitri*. Int. J. Agric. Biol. 16, 783–88.