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Samadyuti Haldar Hooghly Women's College

Kunal Chakraborty
Indian National Centre for Ocean Information Services

T. K. Kar

Indian Institute Engineering Science and Technology

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Controllability of an eco-epidemiological system with disease transmission delay: A theoretical study

Samadyuti Haldar^{1*}, Kunal Chakraborty² and T. K. Kar³

Department of Mathematics
Hooghly Women's College, Pipulpati
Hooghly-712103, West Bengal, India
samdt_h@yahoo.co.in; samadyuti.h@gmail.com

2Information Services and Ocean Sciences Group
Indian National Centre for Ocean Information Services, Hyderabad

"Ocean Valley", Pragathi Nagar (BO), Nizampet (SO)
Hyderabad-500090, India
kunal.c@incois.gov.in; kcincoishyb@gmail.com

3Department of Mathematics
Indian Institute Engineering Science and Technology

Indian Institute Engineering Science and Technology Shibpur, Howrah-711103, India tkar1117@gmail.com

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Abstract

This paper deals with the qualitative analysis of a disease transmission delay induced prey predator system in which disease spreads among the predator species only. The growth of the predators' susceptible and infected subpopulations is assumed as modified Leslie–Gower type. Sufficient conditions for the persistence, permanence, existence and stability of equilibrium points are obtained. Global asymptotic stability of the system is investigated around the coexisting equilibrium using a geometric approach. The existence of Hopf bifurcation phenomenon is also examined with respect to some important parameters of the system. The criterion for disease a transmission delay the induced Hopf bifurcation phenomenon is obtained and subsequently, we use a normal form method and the center manifold theorem to examine the nature of the Hopf bifurcation. It is clearly observed that competition among predators can drive the system to a stable from an unstable state. Also the infection and competition among predator population enhance the availability of prey for harvesting when their values are high. Finally, some numerical simulations are carried out to illustrate the analytical results.

Keywords: Eco-epidemic; Leslie–Gower; permanence; persistence; Hopf bifurcation;

harvesting

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^{*}Corresponding author

1. Introduction

Two major fields of study, ecology and epidemiology, have some common features in their own right. Eco-epidemiology is a branch in mathematical biology, which considers both the ecological and epidemiological features simultaneously. In the year 1986, Anderson and May merged these two fields and started an investigation of this field. During the last two decades, many researchers developed several prey predator models in the presence of disease and analyzed the dynamics of the system. Many research articles have already appeared, but most of the studies mainly focused on parasite infection in prey population only, For examples see Hethcote et al. (2004), Li et al. (2014), Niu et al. (2011), Sarwardi et al. (2014) and so on. However, there are some predators which are infected by different infectious diseases. Infection in predator can magnify the inter-specific and intra-specific interactions among all the species of a prey-predator system. Therefore, an infectious disease in predator biomass can be a factor to regulate the interactions between prey predator species and their harvesting.

Lotka (1925) shows that the consumption or feeding rate describes the transfer of biomass between trophic levels and, completely describes the dynamic coupling between predator abundance and prey abundance. The mathematical form of different issues like the feeding or consumption rate, infection rate, etc. can influence the distribution of the predator through the space discussed (Van der Meer and Ens (1997)). These also influence the stability of enriched predator-prey systems, correlations between nutrient enrichment and the biomass of lower or higher trophic levels studied by DeAngelis et al. (1975) and Huisman and De Boer (1997), and also influence the length of food chains as discussed by Schmitz (1992). Thus the choice of a predator's instantaneous per capita feeding or consumption rate is an important part in modeling and analysis of a prey-predator system. To describe the functional and numerical response of the prey and predator, it is essential to analyze the manner o the f interaction of the prey and predator species with each other. We find several models in the literature where several functional response are analyzed. Yodzis (1994) investigated with Holling a type-I functional response, Kar and Matsuda (2006) studied with Holling a type-II functional response, the ratio dependent functional response was studied by Arditi and Ginzburg (1989), the Hassel-Varley functional response, Beddington DeAngelis functional response are found in the work of Zhang et al. (2008), while the Crowley-Martin functional response was introduced by Crowley and Martin (1989).

An important aspect in the epidemiological system, which should be kept in mind that, it is often necessary to incorporate time delays into the system in order to reflect the dynamical behaviors of the system depending on the past history of the system. Delay induced epidemiological systems have been studied extensively. The predator prey epidemiological system with disease in predator has been proposed and discussed by Zhang et al. (2008). Stability and Hopf bifurcation of a three species system with Holling type II functional response and feedback delay has been investigated by Meng et al. (2011). Recently, Chakraborty et al. (2013) investigated the global stability and bifurcation of a delay induced prey predator fishery system with stage structure. Sawarbari et al. (2011) developed and analyzed an eco-epidemic model with disease in the predator.

Renewable resource management is very complicated and constructing accurate mathematical models about the effect of harvesting on different biomass is even more complicated. The effect of a constant rate harvesting on the dynamics of predator prey model has been investigated by many authors like Brauer and Saudack (1979), Xiao and Ruan (1999). They investigated very

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rich and interesting dynamical behaviour as stability equilibrium, existence of Hopf bifurcation, limit cycles, Bogdanov-Takens bifurcations. Recently, Kar and Misra (2010) proposed and investigated a resource based stage structured fishery model with selective harvesting of mature species. Through harvesting, we can achieve monetary social benefit and can preserve different species from their extinction. Thus, harvesting is a very effective tool to protect and preserve renewable resources from the socio-economic and bio-economic viewpoints. Thus, to develop a complete eco-epidemic model an introduction of species harvesting is necessary.

In this paper, we have studied the impact of infection of predator and the effect of inter and intra specific competition of the predators on the dynamics of an eco-epidemic model with a constant rate of prey harvesting. We want to investigate how the competition and infection among predators enhance the harvesting of prey. The assumptions towards the construction of the model are described in Section 2. In Section 3, some qualitative analysis of the positivity, persistence and permanence of the solution are established. In Section 4, the existence of equilibrium and local as well as global stability of the system in absence of delay are analyzed. Bifurcation analysis is also included there. The delay-induced dynamics of the system like stability, bifurcation and direction of that stability are analyzed in Section 5. Numerical simulations and result discussions are given in Section 6. Finally in Section 7, a brief conclusion of the biological significance of our findings is provided.

2. Assumptions and model formulation

In order to study the influence of the disease on the species in a homogeneous environment, we consider an eco-epidemiological model system consisting of two species, viz. prey and predator and their population size in time t are x(t) and P(t) respectively.

The ecological set up of the system is based on the following assumptions:

- **A₁:** The growth of prey population in absence of predator is assumed to be logistic with intrinsic growth rate $r \ (>0)$ and carrying capacity $K \ (>0)$.
- **A2:** The disease is only spread among the predator population and it is not genetically inherited. The disease infects some of the predator populations, and they are called infected predator. Predators, which are not infected, are termed as susceptible predator. Let y(t) and z(t) be the size of susceptible and infected predator populations in time t. Thus, in presence of disease or infection, we can write

$$P(t) = y(t) + z(t).$$

A₃: It is assumed that both predator classes consumes prey population. In order to incorporate the fact that the rate of consumption of prey population by susceptible predator population is more than that of the infected predator population, we have consider the Holling type-I functional form of response viz. mxy for the susceptible predator but for the infected predator population it is considered as Holling type-II functional form of response viz. $\alpha xz/(a+x)$ where m and α are respectively the rate of consumption of the susceptible and infected predator to the prey population and a is the half capturing saturation constant.

A4: The growth of the predator is assumed to be of the modified Leslie–Gower type which was also studied by Aziz-Alaoui and Daher Okiye (2003). In this regard, it is to be noted that the Leslie–Gower formulation is based on the assumption that a reduction in the predator population has a reciprocal relationship with per capita availability of its preferred food (prey). Indeed, Leslie introduced a predator prey model where the carrying capacity of the predator environment is proportional to the number of prey. He stresses the fact that there are upper limits to the rates of increase of both prey and predator, which are not recognized in the Lotka–Volterra model. However, In the case of severe scarcity, the predator can switch over to other population, but its growth will be limited by the fact that its most favourite food, the prey, is not available in abundance. The situation can be taken care of by adding a positive constant to the denominator, hence the growth function is considered to be of the modified Leslie–Gower type. Let us also introduce an intra-specific competition among the predators' susceptible and infected subpopulations, with parameters σ and ω , where $\sigma > \omega$ as studied by Sarwardi et al. (2011).

Therefore, the system becomes

$$\frac{dx}{dt} = rx \left(1 - \frac{x}{K} \right) - \frac{\alpha xz}{a+x} - mxy,$$

$$\frac{dy}{dt} = sy - \frac{\sigma y(y+z)}{ux+L},$$

$$\frac{dz}{dt} = \mu z - \frac{\omega z(y+z)}{vx+N},$$

where s and μ ($s \ge \mu$) are respectively the intrinsic growth rate of susceptible and infected predator population, u and v are the prey predators' conversion factor, L and N respectively represent the residual loss in species y and z due to severe scarcity of its favourite x.

A₅: It is usual that a mass action incidence is considered in epidemic models, where βyz is the infection term, β being the infection rate. In this paper, we have replaced the infection term, βyz by $\beta yz/(y+z+M)$, where M is the half saturation type constant. For large populations, an individual's finite and often slow movement prevents it from making contact with a large number of individuals in a unit time. Such a mechanism is better described by $\beta [y/(y+z+M)]z$ than βyz . Pathak et al. (2011) shows that the encounter infection rate makes sense only when the total population is small and steady.

Further, let the parameter τ represent the disease transmission delay, i.e., the time τ is required to become infected after contamination from susceptible predator. Therefore, the term $e^{-\gamma \tau} \beta y(t-\tau)z(t-\tau)/(y(t-\tau)+z(t-\tau)+M)$ denotes the conversion of susceptible predators to infected predators with exponential decay rate γ . The infected prey populations recover and become immune at the rate of ξ . However, in the presence of a disease they are also removed from the system at the rate of η .

A₆: Harvesting has generally a strong impact on the dynamics of the system. The severity of this impact depends on the harvesting strategy implemented which in turn may range from

the rapid depletion to the complete preservation of a population. Problems related to the exploitation of multi-species systems are interesting and difficult both theoretically and practically. It is assumed that only the prey population is harvested and the catch per unit effort (CPUE) is proportional to the stock level as introduced by Clerk (1990). Thus the harvesting function h(t) can be written as h(t) = qEx(t), where q is the catchability coefficient for the prey populations.

Keeping these aspects in view, the dynamics of the system may be governed by the following system of differential equations:

$$\frac{dx}{dt} = rx\left(1 - \frac{x}{K}\right) - \frac{\alpha xz}{a+x} - mxy - qEx,\tag{2.1a}$$

$$\frac{dy}{dt} = sy - \frac{\sigma y(y+z)}{ux+L} - \frac{\beta yz}{y+z+M} + \xi z, \qquad (2.1b)$$

$$\frac{dz}{dt} = \mu z - \frac{\omega z(y+z)}{vx+N} + e^{-\gamma \tau} \frac{\beta y(t-\tau)z(t-\tau)}{y(t-\tau)+z(t-\tau)+M} - \eta z - \xi z.$$
 (2.1c)

The initial conditions for the system (2.1) take the form

$$x(\theta) = \phi_1(\theta), y(\theta) = \phi_2(\theta),$$

$$z(\theta) = \phi_2(\theta), \phi_1(\theta) \ge 0, \ \theta \in [-\tau, 0), \ \phi_1(0) > 0, i = 1, 2, 3,$$
(2.2)

where

$$(\phi_1(\theta), \phi_2(\theta), \phi_3(\theta)) \in C([-\tau, 0], R_+^3),$$

the Banach space of continuous functions mapping in the interval $[-\tau,0]$ into R_+^3 , where

$$R^3_{\perp} = \{(x, y, z) \in \mathbb{R}^3; x \ge 0, y \ge 0, z \ge 0\}.$$

3. Qualitative properties of the system

Discussion and analysis of a system is fundamentally centered on its solution. So it is extremely important to know the nature of the solutions of the system. Here, we discuss the qualitative nature of the solution of the system like positivity, persistence and permanence of the system.

3.1. Positivity

Here, we now discuss the positivity of the solution, which confirms that the solutions of the system (2.1) are all positive. Let us denote

$$\operatorname{int}(R_{+}^{3}) = \{(x, y, z) \in R^{3}; x > 0, y > 0, z > 0\},\$$

the positive octant of R^3 . Then positivity of the solution of the system is confirmed by the following theorem.

Theorem 3.1.

All the solutions of the system of equations (2.1) are positive and belong to positive octant int (R^3_+) .

Proof:

The proof is given in Appendix A.

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3.2. Uniform strong Persistence

Persistence in biological systems is the property which determine whether a population will continue to exist or will be annihilated. It is closely related to the stability of some equilibrium solutions of the dynamical system. Thus the persistence results make the formulation of the model stronger. In this section, we present the conditions for the uniform strong persistence of the system.

Let us consider

$$M_1 = K(r - qE)/r$$
, $M_2 = (s + \xi)(u(M_1 + \varepsilon) + L)/\sigma$, (3.1a)

$$M_3 = \left(\mu + \beta M_2 e^{(\eta + \xi - \gamma)\tau}\right) (vM_1 + N)/\omega, \tag{3.1b}$$

$$N_1 = \frac{K}{r} \left(r - \frac{\alpha M_3}{a} - mM_2 - qE \right) , \qquad (3.1c)$$

$$N_2 = \frac{L}{\sigma} \left(s - \frac{\sigma M_3}{L} - \frac{\beta M_2}{M} \right), \tag{3.1d}$$

$$N_3 = \frac{1}{\omega} \left((\mu - \eta - \xi) N + \omega M_2 \right). \tag{3.1e}$$

Then, we can write the following proposition.

Proposition 3.1.

For the system (2.1) all the biomass has its limit supremum as well as limit infimum as $t \to +\infty$, i.e.,

$$\begin{split} &\limsup_{t\to +\infty} x(t) \leq M_1, \ \limsup_{t\to +\infty} y(t) \leq M_2, \ \limsup_{t\to +\infty} z(t) \leq M_3, \\ &\liminf_{t\to +\infty} x(t) \geq N_1, \ \liminf_{t\to +\infty} \ y(t) \geq N_2 \ \text{and} \ \liminf_{t\to +\infty} \ z(t) \geq N_3. \end{split}$$

The proof is given in Appendix B.

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Using the above proposition we obtain the criterion for uniform persistence. The following theorem gives the result.

Theorem 3.2.

If

(i)
$$r > \frac{\alpha M_3}{a} + mM_2 + qE$$
,
(ii) $s > \frac{\sigma M_3}{L} + \frac{\beta M_2}{M}$ and
(iii) $\mu > (\eta + \xi) + \frac{\omega M_2}{N}$,

then the system (2.1) is uniformly strong persistent, where M_1, M_2, M_3, N_1, N_2 and N_3 are as given above.

Proof:

The proof is given in Appendix C.

3.3. Permanence

Definition:

Any eco-epidemiological system is said to be permanent (Gopalswamy, 1992), if there exists \wp , $\Re \in R$ and $0 < \wp < \Re$ independent of the initial condition, such that for all solution of the system,

$$\wp \leq \min \left\{ \liminf_{t \to +\infty} x(t), \liminf_{t \to +\infty} y(t), \liminf_{t \to +\infty} z(t) \right\},$$

$$\max \left\{ \limsup_{t \to +\infty} x(t), \limsup_{t \to +\infty} y(t), \limsup_{t \to +\infty} z(t) \right\} \leq \Re.$$

According to this definition, it is easy to establish that our system is permanent and we can write the following theorem.

Theorem 3.3.

The system (2.1) is permanent, if it is persistent.

Proof:

The proof is given in Appendix D.

4. Analysis of the model in absence of delay

Here, we analyze the system (2.1) without delay. At first we want to investigate the biologically important points of equilibrium and their stability.

4.1. Equilibria analysis

Our objective in this section is to investigate the equilibrium points and their stability. The system of equation has the following equilibrium:

- i) The trivial equilibrium $E_0(0,0,0)$,
- ii) The predator free axial equilibrium $E_1(K(r-qE)/r,0,0)$, provided r/q > E, i.e., the bio technical productivity (BTP) of prey is greater than the combined harvesting effort.
- iii) The disease free equilibrium $E_2(x_2, y_2, 0)$: Solving the system of equations (2.1) assuming z = 0, we find that the disease free equilibrium $E_2(x_2, y_2, 0)$ is given by

$$x_2 = \frac{(\sigma(r-qE) - sLm)K}{\sigma r + u sm K}$$
 and

$$y_2 = \frac{Kus(r-qE) + sLr}{\sigma r + usmK}$$
, provided $\sigma > \frac{msL}{(r-qE)}$ and $r/q > E$.

iv) The positive interior equilibrium $E^*(x^*, y^*, z^*)$: It is quite difficult to calculate explicitly the interior equilibrium point theoretically due to the complexity of the model. Here we only give the way of finding the interior equilibrium point $E^*(x^*, y^*, z^*)$ as follows:

 x^* and y^* are given by the positive solutions of simultaneous equations

$$\Phi_2(x^*, y^*) = 0$$
 and

$$\Phi_3(x^*, y^*) = 0$$
,

where Φ_2 and Φ_3 are given by

$$\Phi_2(x^*, y^*) = s - \frac{\sigma(y + \Phi_1)}{u x + L} - \frac{\beta \Phi_1}{y + \Phi_1 + M} + \frac{\xi \Phi_1}{y},$$

$$\Phi_{3}(x^{*}, y^{*}) = \mu - \frac{\omega(y^{*} + \Phi_{1})}{vx^{*} + N} + \frac{\beta \Phi_{1}}{y^{*} + \Phi_{1} + M} + \frac{\xi \Phi_{1}}{y^{*}},$$

and

$$\Phi_1(x^*, y^*) = \frac{a + x^*}{\alpha} \left(r(1 - \frac{x^*}{K}) - m y^* - q E \right).$$

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After obtaining x^* and y^* , we can get z^* from the relation $z^* = \Phi_1(x^*, y^*)$.

For biological importance, we are interested about the two equilibrium points viz. the disease free equilibrium and the interior equilibrium.

4.2. Stability analysis

Here we discuss the local stability at different equilibrium point as well as the global stability of the solution of the system. After the theoretical investigation we obtain the following results.

Theorem 4.1.

Trivial equilibrium E_0 is always unstable.

Proof:

The characteristic equation of the given system around equilibrium E_0 is

$$(\lambda - s)(\lambda - (r - qE))(\lambda - (\mu - \eta - \xi)) = 0. \tag{4.1}$$

Since the roots of the characteristic equation (4.1) at E_0 are

$$\lambda_1^{(0)} = s$$
, $\lambda_2^{(0)} = r - q E$, $\lambda_3^{(0)} = \mu - \eta - \xi$,

which are all positive because of the construction of the model r > qE and $\mu > \eta + \xi$ always. Thus, the trivial equilibrium is always unstable. This completes the proof of the theorem.

Theorem 4.2.

The predator free axial equilibrium E_1 is an unstable saddle point with x-axis as its stable manifold and the z axis as its unstable manifold but the y axis as its stable manifold, if $\mu < \eta + \xi$ and unstable manifold, if $\mu > \eta + \xi$.

Proof:

The characteristic equation of the given system at the equilibrium E_1 is

$$(\lambda + (r - qE))(\lambda - (\mu - \eta - \xi))(\lambda - s) = 0. \tag{4.2}$$

Since roots of the characteristic equation (4.2) at E_1 are

$$\lambda_1^{(1)} = -(r - qE), \ \lambda_2^{(1)} = \mu - \eta - \xi, \ \lambda_3^{(1)} = s,$$

the first eigenvalue is negative when

$$\frac{r}{q} > E$$
,

i.e., the BTP of the prey is greater than the harvesting effort and it is quite natural. When E_1 exists, then the above condition is satisfied. The third eigenvalue is positive. The second eigenvalue is negative, if $\mu < \eta + \xi$ and positive, if $\mu > \eta + \xi$. So this axial equilibrium E_1 is unstable saddle point with x-axis as its stable manifold and z axis as its unstable manifold, but y axis as its stable manifold, if $\mu < \eta + \xi$ and unstable manifold, if $\mu > \eta + \xi$. This completes the proof of the theorem.

Theorem 4.3.

Suppose the disease free equilibrium $E_2(x_2, y_2, 0)$ exist. Then,

- (i) if $\overline{\omega} < 0$ and $\overline{\sigma} < 0$, the disease free equilibrium is always stable,
- (ii) if $\overline{\omega} < 0$ and $\overline{\sigma} > 0$, the disease free equilibrium is stable for $\sigma > \overline{\sigma}$ and the system undergoes through a Hopf bifurcation at $\sigma = \overline{\sigma}$,
- (iii) if $\overline{\sigma} < 0$ and $\overline{\omega} > 0$, the disease free equilibrium is stable for $\omega > \overline{\omega}$,
- (iv) if $\overline{\omega} > 0$ and $\overline{\sigma} > 0$, the system is stable for $\sigma > \overline{\sigma}$ and $\omega > \overline{\omega}$. Also the system undergoes through a Hopf bifurcation at $\sigma = \overline{\sigma}$,

where

$$\overline{\omega} = \left(\mu - \eta - \xi + \frac{\beta y_2}{y_2 + M}\right) \left(\frac{v x_2 + N}{y_2}\right) \text{ and}$$

$$\overline{\sigma} = \left(r + s - qE - \frac{2r x_2}{K} - my_2\right) \left(\frac{u x_2 + L}{2y_2}\right), \quad x_2 \text{ and } y_2 \text{ are given above.}$$

Proof:

The characteristic equation of the given system at the equilibrium E_2 is

$$\begin{split} \left(\lambda - \left(\mu - \eta - \xi - \frac{\omega y_2}{v x_2 + N} + \frac{\beta y_2}{y_2 + M}\right)\right) \left(\lambda^2 - \left(r + s - qE - \frac{2 r x_2}{K} - m y_2 - \frac{2 \sigma y_2}{u x_2 + L}\right)\lambda \right. \\ \left. + \left(r - qE - \frac{2 r x_2}{K} - m y_2\right) \left(s - \frac{2 \sigma y_2}{u x_2 + L}\right) + m x_2 \frac{\sigma u y_2^2}{\left(u x_2 + L\right)^2}\right) = 0 \; . \end{split}$$

The eigenvalues are

$$\lambda_1^{(2)} = \mu - \eta - \xi - \frac{\omega y_2}{v x_2 + N} + \frac{\beta y_2}{y_2 + M}, \ \lambda_2^{(2)} \text{ and } \lambda_3^{(2)}$$

that are the roots of the quadratic equation

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$$\lambda^{2} - \left(r + s - qE - \frac{2rx_{2}}{K} - my_{2} - \frac{2\sigma y_{2}}{ux_{2} + L}\right)\lambda + \left(r - qE - \frac{2rx_{2}}{K} - my_{2}\right)\left(s - \frac{2\sigma y_{2}}{ux_{2} + L}\right) + mx_{2}\frac{\sigma u y_{2}^{2}}{\left(u x_{2} + L\right)^{2}} = 0.$$

 E_2 will be locally asymptotically stable, if $\lambda_1^{(2)} < 0$ and $\text{Re}(\lambda_{2,3}^{(2)}) < 0$. Clearly, $\lambda_1^{(2)} < 0$, if

$$\mu - \eta - \xi - \frac{\omega y_2}{v x_2 + N} + \frac{\beta y_2}{y_2 + M} < 0 \text{ or, } \omega > \overline{\omega}$$

and for

$$r+s-qE-\frac{2rx_2}{K}-my_2-\frac{2\sigma y_2}{ux_2+L}<0 \text{ or, } \sigma>\overline{\sigma},$$

the real part of $\lambda_{2,3}^{(2)} < 0$. But, when $\sigma = \overline{\sigma}$, then the real part of $\lambda_{2,3}^{(2)}$ are zero. Moreover, in this case, the transversality condition for Hopf bifurcation

$$\frac{d\lambda_{2,3}^{(2)}(\sigma)}{d\sigma}\bigg|_{\sigma=\bar{\bar{\sigma}}}\neq 0$$

holds. Remember that ω and σ are positive quantity. Thus, (i) when $\overline{\omega}$ and $\overline{\sigma}$ are negative, the conditions $\omega > \overline{\omega}$ and for $\sigma > \overline{\sigma}$ holds automatically. So the system is locally stable always. (ii) If $\overline{\omega} < 0$ and $\overline{\sigma} > 0$, then obviously $\omega > \overline{\omega}$. So the system is locally stable for $\sigma > \overline{\sigma}$ and the system undergoes through a Hopf bifurcation at $\sigma = \overline{\sigma}$. (iii) If $\overline{\sigma} < 0$ and $\overline{\omega} > 0$, the disease free equilibrium is stable for $\omega > \overline{\omega}$ as $\sigma > \overline{\sigma}$ holds normally. (iv) If $\overline{\omega} > 0$ and $\overline{\sigma} > 0$, the system is stable for $\sigma > \overline{\sigma}$ and $\omega > \overline{\omega}$. Also the system undergoes through a Hopf bifurcation at $\sigma = \overline{\sigma}$.

This completes the proof of the theorem.

Remarks:

The intra-specific competition coefficients among the predators' susceptible and infected sub-populations plays a role to destabilize the system from stable situation.

We shall now examine the local stability of the system at the point of interior equilibrium $E^*(x^*, y^*, z^*)$. The Jacobian of the system is $(J_{ij}) \in R^{3x^3}$, where

$$J_{11} = -\frac{rx}{K} + \frac{\alpha xz}{(a+x)^{2}}, J_{12} = -mx, \quad J_{13} = -\frac{\alpha x}{(a+x)}, J_{21} = \frac{\sigma u y(y+z)}{(u x+L)^{2}},$$

$$J_{22} = -\frac{\xi z}{y} - \frac{\sigma y}{u x+L} + \frac{\beta yz}{(y+z+M)^{2}}, \quad J_{23} = \xi - \frac{\sigma y}{u x+L} - \frac{\beta y(y+M)}{(y+z+M)^{2}},$$

$$J_{31} = \frac{\omega v z(y+z)}{(v x+N)^{2}}, \quad J_{32} = -\frac{\omega z}{v x+N} + \frac{\beta z(z+M)}{(y+z+M)^{2}}, \quad J_{33} = -\frac{\omega z}{v x+N} - \frac{\beta y z}{(y+z+M)^{2}}.$$

$$(4.3)$$

The characteristic equation at the interior equilibrium point $E^*(x^*, y^*, z^*)$ is

$$\lambda^3 + a_1 \lambda^2 + a_2 \lambda + a_3 = 0 \tag{4.4}$$

where

$$a_2 = a_{21} - a_{22}, \ a_3 = a_{31} - a_{32}$$

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and

If

$$\frac{r}{K} > \frac{\alpha z^*}{(a+x^*)^2},$$

then $a_1 > 0$. Also, if $a_{31} > a_{32}$ then $a_3 > 0$.

According to Routh-Hurwitz criterion, we obtain the following theorem for local stability of the system (2.1) at the interior equilibrium point $E^*(x^*, y^*, z^*)$.

Theorem 4.4.

The model system (2.1) is locally asymptotically stable at the interior equilibrium point

$$E^*(x^*, y^*, z^*)$$
, if $i) \frac{r}{K} > \frac{\alpha z^*}{(a + x^*)^2}$, $ii) a_{31} > a_{32}$ and $iii) a_1 a_2 > a_3$ holds.

4.3. Global stability

In the case of bi-dimensional epidemic systems, Perko (1996) states that the global stability of E^* may be obtained by using the Dulac criterion and Poincar'e–Bendixson theorem. But, for higher dimensional systems, the geometrical approach is a more powerful technique that has been extensively applied to study the global behavior of many epidemic models, as for example see Sun et al. (2007). Following the geometric approach introduced and described by Li and Muldowney (1996), we have obtained the following result which ensures the global stability of the system (2.1) at the interior equilibrium point $E^*(x^*, y^*, z^*)$.

Theorem 4.5.

If the endemic equilibrium of the system (2.1) exists then the sufficient condition for global stability of the system (2.1) at that endemic equilibrium point is $\mu_0 < \mu - \eta - \xi$, where

$$\mu_0 = \max\{\mu_1, \mu_2\}, \ \mu_1 = \mu_{11} + \mu_{12}, \ \mu_2 = \mu_{20} + \mu_{21} + \mu_{22}, \ \mu_{ii}$$

are given in the Appendix D.

Proof:

The proof is given in Appendix D.

4.4. Bifurcation due to competition

When a stable steady state goes through a bifurcation, it will lose its stability or may perish. Even, if the system ends up in another steady state, the transition to that state will often involve the extinction of one or more level of food chain. The entire system may survive in a non-stationary state, but another bifurcation may lead to local extinction of every population. To preserve the system in its natural state, cross bifurcation should be avoided. Thus the estimation of critical values of the bifurcation parameter is a crucial part of the analysis of a system.

Bifurcation may occur for more than one variable. Liu (1994) derived a criterion of Hopf bifurcation without using the eigenvalues of the variational matrix of the interior equilibrium point.

Let $\zeta (= \sigma \ or \ \omega)$ be the bifurcation parameter.

Liu's criterion:

If the characteristic equation at the interior equilibrium point $E^*(x^*, y^*, z^*)$ is given by

$$\lambda^3 + a_1(\zeta)\lambda^2 + a_2(\zeta)\lambda + a_3(\zeta) = 0,$$

where

$$a_1(\zeta), \Delta(\zeta) = a_1(\zeta) a_2(\zeta) - a_3(\zeta), a_3(\zeta)$$

are smooth functions of ζ in an open interval about $\zeta^* \in \Re$ such that

i)
$$a_1(\zeta^*) > 0, a_3(\zeta^*) > 0$$

and

ii)
$$\Delta(\zeta^*) = 0$$
,

but

iii)
$$\left(\frac{d\Delta}{d\zeta}\right)_{\zeta=\zeta^*} \neq 0$$
 then a simple Hopf bifurcation occur at $\zeta = \zeta^*$.

We investigate the bifurcation of the model for $\zeta = \sigma$ (the intra-specific competition factor of sound predator to the whole predator subpopulation) and $\zeta = \omega$ (the intra-specific competition factor of infected predator to the whole predator subpopulation) separately. Due to complexity of the model, we are unable to find theoretically the specific criterion of bifurcation for σ and ω , but numerically we are able to find σ^* and ω^* .

Remarks:

For $\zeta = \sigma$ or ω , since

$$\Delta(\zeta^*) = 0$$
 and $\left(\frac{d\Delta}{d\zeta}\right)_{\zeta=\zeta^*} \neq 0$, $\Delta(\zeta) > 0$, for $\zeta > \zeta^*$,

and according to the Routh-Hurwitz criterion, $E^*(x^*, y^*, z^*)$ is locally asymptotically stable for $\zeta > \zeta^*$ (i.e., $\sigma > \sigma^*$ or $\omega > \omega^*$) and for $0 < \zeta < \zeta^*$ (i.e. $0 < \sigma < \sigma^*$ or $0 < \omega < \omega^*$), it approaches a periodic solution that implies that the system is unstable. So the system where only prey population are harvested, the competition among predators subpopulation for existence of life (specially, for food) bifurcate the system from stable situation to unstable. In other words, to obtain sustainable yield from this system, higher rate of competition is desirable.

5. Dynamics of the delay system

We shall now investigate the dynamics of the delay system (2.1) at the interior point.

5.1. Delay-induced Bifurcation

To study the behavior of the system at the interior equilibrium point $E^*(x^*, y^*, z^*)$ we use the transformation

$$x = x^* + x_1, y = y^* + y_1, z = z^* + z_1.$$

Then, the linear system is given by

$$\frac{dw}{dt} = Mw(t) + Nw(t - \tau),\tag{5.1}$$

where

$$w(t) = [x_{1}, y_{1}, z_{1}]^{T}, \quad M = (m_{ij})_{3x3}, \quad N = (n_{ij})_{3x3},$$

$$m_{11} = -\frac{r x^{*}}{K} + \frac{\alpha x^{*} z^{*}}{(a + x^{*})^{2}}, \quad m_{12} = -mx^{*}, \quad m_{13} = -\frac{\alpha x^{*}}{(a + x^{*})}, \quad m_{21} = \frac{\sigma u y^{*} (y^{*} + z^{*})}{(u x^{*} + L)^{2}},$$

$$m_{22} = -\frac{\xi z^{*}}{y^{*}} - \frac{\sigma y^{*}}{u x^{*} + L} + \frac{\beta y^{*} z^{*}}{(y^{*} + z^{*} + M)^{2}}, \quad m_{23} = \xi - \frac{\sigma y^{*}}{u x^{*} + L} - \frac{\beta y^{*} (y^{*} + M)}{(y^{*} + z^{*} + M)^{2}},$$

$$m_{31} = \frac{\omega v z^{*} (y^{*} + z^{*})}{(v x^{*} + N)^{2}}, \quad m_{32} = -\frac{\omega z^{*}}{v x^{*} + N}, \quad m_{33} = \mu - \frac{\omega (y^{*} + 2z^{*})}{v x^{*} + N} - \eta - \xi,$$

$$m_{32} = \frac{\beta z^{*} (z^{*} + M) e^{-\gamma \tau}}{(y^{*} + z^{*} + M)^{2}}, \quad n_{33} = \frac{\beta y^{*} (y^{*} + M) e^{-\gamma \tau}}{(y^{*} + z^{*} + M)^{2}} \quad \text{and all other } n_{ij} = 0.$$

The characteristic equation of the system (5.1) at $E^*(x^*, y^*, z^*)$ is

$$P(\lambda, \tau) + Q(\lambda, \tau)e^{-\lambda \tau} = 0, \tag{5.2}$$

where

$$P(\lambda,\tau) = \lambda^3 + a_1(\tau)\lambda^2 + a_2(\tau)\lambda + a_3(\tau), \quad Q(\lambda,\tau) = b_1(\tau)\lambda^2 + b_2(\tau)\lambda + b_3(\tau),$$

and

$$\begin{split} a_1(\tau) &= -(m_{11} + m_{22} + m_{33}) \,, \quad a_2(\tau) = m_{11} m_{22} + m_{11} m_{33} + m_{22} m_{33} - m_{12} m_{21} - m_{13} m_{31} \,, \\ a_3(\tau) &= -m_{11} m_{22} m_{33} + m_{12} m_{23} m_{31} + m_{12} m_{21} m_{33} + m_{13} m_{31} m_{22} \,, b_1(\tau) = -n_{33} \,, \\ b_2(\tau) &= (m_{11} + m_{22}) n_{33} - m_{23} n_{32} \,, \, b_3(\tau) = -m_{11} m_{22} n_{33} + m_{11} m_{23} n_{32} + m_{12} m_{21} n_{33} + m_{13} m_{21} n_{32} \,. \end{split}$$

The necessary condition of delay-induced instability of the system (5.1) at $E^*(x^*, y^*, z^*)$ is that the delay induced characteristic equation (5.2) has a purely imaginary solution. One thing to be noticed here is that $P(\lambda, \tau)$ and $Q(\lambda, \tau)$ are delay dependent. The main complication arises when

we proceed to investigate the existence of purely imaginary root, $\lambda = i\omega_1$ of the characteristic equation (5.2) of the system (5.1). Here we follow the approach developed by Beretta and Kuang (1998).

Let τ_{\max} be the maximum value of τ for which $E^*(x^*, y^*, z^*)$ exists. For $\tau \in [0, \tau_{\max}]$, we assume the following:

(i)
$$P(0,\tau) + Q(0,\tau) = a_3(\tau) + b_3(\tau) \neq 0$$
,

(ii)
$$P(i\omega_1, \tau) + Q(i\omega_1, \tau) = a_3(\tau) + b_3(\tau) - \omega_1^2 \{a_1(\tau) + b_1(\tau)\} - i\{\omega_1^3 - \omega_1 a_2(\tau) - \omega_1 b_2(\tau)\} \neq 0$$
.

We further noticed that

(iii)
$$\frac{\lim_{|\lambda| \to \infty} \left| \frac{Q(\lambda, \tau)}{P(\lambda, \tau)} \right| = \lim_{|\lambda| \to \infty} \left\{ \frac{b_1(\tau)\lambda^2 + b_2(\tau)\lambda + b_3(\tau)}{\lambda^3 + a_1(\tau)\lambda^2 + a_2(\tau)\lambda + a_3(\tau)} \right\} = 0 < 1.$$

Now, $F(\omega_1, \tau) = |P(i\omega_1, \tau)|^2 - |Q(i\omega_1, \tau)|^2$ is a polynomial of degree 6. Therefore,

(iv) $F(\omega_1, \tau)$ has infinite number of zeroes.

By the implicit function theorem, we have

(v) every positive simple root $\omega_1(\tau)$ of $F(\omega_1, \tau) = 0$ is continuous and differentiable in τ whenever it exists.

Let $\lambda(\tau) = \mu_1(\tau) + i\omega_1(\tau)$ be an eigen value of the system. It is assumed that the change of stability will occur for $\text{Re}(\lambda) = 0$. Therefore, we substitute $\lambda = i\omega_1$ in (5.2) and separating the real and imaginary parts, we get

$$-a_{1}\omega_{1}^{2} + a_{3} = (b_{1}\omega_{1}^{2} - b_{3})\cos(\omega_{1}\tau) - b_{2}\omega\sin(\omega_{1}\tau),$$

$$\omega_{1}^{3} - a_{2}\omega_{1} = b_{2}\omega_{1}\cos(\omega_{1}\tau) + (b_{1}\omega_{1}^{2} - b_{3})\sin(\omega_{1}\tau),$$

which gives

$$\sin(\omega_1 \tau) = \frac{b_2 \omega_1 (a_1 \omega_1^2 - a_3) + (b_1 \omega_1^2 - b_3)(\omega_1^3 - a_2 \omega_1)}{b_2^2 \omega_1^2 + (b_3 - b_1 \omega_1^2)^2}$$
(5.3)

and

$$\cos(\omega_1 \tau) = \frac{b_2 \omega_1(\omega_1^3 - a_2 \omega_1) - (a_3 - a_1 \omega_1^2)(b_3 - b_1 \omega_1^2)}{b_2^2 \omega_1^2 + (b_3 - b_1 \omega_1^2)^2},$$
(5.4)

where we notice that

$$b_2^2 \omega_1^2 + (b_3 - b_1 \omega_1^2)^2 = |Q(i\omega_1, \tau)|^2 \neq 0$$

(because $Q(i\omega_1, \tau) = 0$ would imply $P(i\omega_1, \tau) = 0$, a contradiction to (ii) above).

Squaring and adding (5.3) and (5.4), we get

$$F(\omega_1, \tau) = \omega_1^6 + d_1 \omega_1^4 + d_2 \omega_1^2 + d_3 = 0, \tag{5.5}$$

where

$$d_1 = a_1^2 - 2a_2 - b_1^2$$
, $d_2 = a_2^2 - 2a_1a_3 + 2b_1b_3 - b_2^2$, $d_3 = a_3^2 - b_3^3$.

Thus, ω_1 in terms of τ can be obtained from equation (5.5). For each τ , equation (5.5) has at most a finite number of roots, and ensures that only a finite number of 'gates' for the roots to cross the imaginary axis. Let us define

$$J = \{\tau : \tau > 0, \text{ where } \omega_1(\tau) \text{ is a positive root of } (5.5) \}.$$

Thus, if $\tau \in J^c$ then there is no positive solution of (5.5), and thus no stability switches occur. Now, for any $\tau \in J$, we can define $\theta(\tau) \in (0,2\pi)$ as the solution of (5.2). Therefore,

$$\sin(\theta(\tau)) = \frac{b_2 \omega_1 (a_1 \omega_1^2 - a_3) + (b_1 \omega_1^2 - b_3)(\omega_1^3 - a_2 \omega_1)}{b_2^2 \omega_1^2 + (b_3 - b_1 \omega_1^2)^2} = \frac{\Omega}{|Q(i\omega_1, \tau)|},$$
(5.6)

$$\cos(\theta(\tau)) = \frac{b_2 \omega_1(\omega_1^3 - a_2 \omega_1) - (a_3 - a_1 \omega_1^2)(b_3 - b_1 \omega_1^2)}{b_2^2 \omega_1^2 + (b_3 - b_1 \omega_1^2)^2} = \frac{\Psi}{|Q(i\omega_1, \tau)|},$$
(5.7)

where Ω and Ψ are continuous and differentiable functions of τ such that

$$\Omega^2 + \Psi^2 = |P(i\omega_1, \tau)|^4$$
 and $|Q(i\omega_1, \tau)|^2 = |P(i\omega_1, \tau)|^2$.

Putting $\omega_1 = \omega_1(\tau)$ in (5.6) and (5.7), $\theta(\tau) \in (0,2\pi)$ can be determined as follows:

$$\begin{cases} \arctan \frac{\Omega}{\Psi}, & \text{if } \sin(\theta(\tau)) > 0, \cos(\theta(\tau)) > 0, \\ \frac{\pi}{2}, & \text{if } \sin(\theta(\tau)) = 1, \cos(\theta(\tau)) = 0, \\ \pi + \arctan \frac{-\Omega}{\Psi}, & \text{if } \cos(\theta(\tau)) < 0, \\ \frac{3\pi}{2}, & \text{if } \sin(\theta(\tau)) = -1, \cos(\theta(\tau)) = 0, \\ 2\pi + \arctan \frac{\Omega}{\Psi}, & \text{if } \sin(\theta(\tau)) < 0, \cos(\theta(\tau)) > 0. \end{cases}$$
 (5.8)

We noticed that for $\tau \in J$, $\theta(\tau)$ defined as above, is continuous at τ and, if $\theta(\tau) \in (0,2\pi)$, $\tau \in J$, then $\theta(\tau)$ is also differentiable at τ .

Now from equations (5.3), (5.4) and (5.8), we have $\omega_1(\tau)\tau = \theta(\tau) + 2n\pi$, $\tau \in J$, $n \in \mathbb{N}_0$.

Let us define the maps $\tau_n: J \to \mathbb{R}_{+0}$ given by

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$$\tau_n(\tau) = \frac{1}{\omega_1(\tau)}(\theta(\tau) + 2n\pi), \quad n \in \mathbb{N}_0, \ \tau \in J, \ \omega_1(\tau)$$

is a positive simple root of

$$F(\omega_1, \tau) = 0$$
.

We now introduce the function

$$S_n: J \to \mathbb{R} \text{ by } S_n(\tau) = \tau - \tau_n(\tau), \ \tau \in J, \ n \in \mathbb{N}_0,$$
 (5.9)

which are continuous and differentiable at τ . We notice that, the values of $\tau \in J$ at which stability switches may occur, are the solutions of $S_n(\tau) = 0$ for some $n \in \mathbb{N}_0$, provided the corresponding transversality condition is satisfied. To find out the transversality condition, we differentiate the characteristic equation (5.2) with respect to τ , which gives us

$$\left(\frac{d\lambda}{d\tau}\right)^{-1}\bigg|_{\lambda=i\omega} = \frac{G+iH}{K+iL},\tag{5.10}$$

where the values of G, H, K, L are given in the Appendix F. Therefore,

$$\left\{ \operatorname{Re} \left(\frac{d\lambda}{d\tau} \right)^{-1} \right|_{\lambda = i\omega} \right\} = \frac{GK + HL}{K^2 + L^2}.$$

Now we assume

$$\eta(\tau) = \operatorname{sign}\left\{\operatorname{Re}\left(\frac{d\lambda}{d\tau}\right)^{-1}\Big|_{\lambda=i\omega}\right\}.$$

Then, we have the following theorem:

Theorem 5.1.

Let $\omega_1(\tau)$ be the positive root of equation (5.5) for $\tau \in J$ and for some $\tau^* \in J$ and $n \in \mathbb{N}_0$, $S_n(\tau^*) = 0$. Then a pair of simple conjugate pure imaginary roots $\lambda_+(\tau^*) = i\omega_1(\tau^*)$,

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 $\lambda_{-}(\tau^{*}) = -i\omega_{1}(\tau^{*})$ of (5.2) exists at $\tau = \tau^{*}$, which crosses the imaginary axis from left to right, if $\eta(\tau^{*}) > 0$ and crosses the imaginary axis from right to left, if $\eta(\tau^{*}) < 0$.

Now subsequently, using the stability criterion of the interior equilibrium, $E^*(x^*, y^*, z^*)$ of system (2.1) in the absence of delay (Theorem 4.4) and the Hopf bifurcation theorem, we can have the following theorem for the existence of Hopf bifurcation near $E^*(x^*, y^*, z^*)$.

Theorem 5.2.

Let $a_1 > 0$, $a_3 > 0$ and $\Delta(=a_1a_2 - a_3) > 0$. Again, let $\omega_1(\tau)$ be a positive root of (5.5) defined for $\tau \in J$ and at some $\tau^* \in J$, $S_n(\tau^*) = 0$ for some $n \in \mathbb{N}_0$. Then the system (2.1) exhibits a Hopf bifurcation near E^* , provided that $\eta(\tau^*) \neq 0$. As τ increases from zero upward the first such Hopf bifurcation will be from stable to unstable, the second from unstable to stable, and the third from stable to unstable and so on.

5.2. Direction of the Hopf-bifurcation

In this section we shall study the direction of the Hopf-bifurcation and the stability of the bifurcating periodic solutions. In the previous section we obtain the conditions under which a family of periodic solutions bifurcates from the positive equilibrium E^* at the critical value τ^* . Assume that the system (2.1) under goes a Hopf bifurcation at the positive equilibrium E^* for $\tau = \tau^*$, i.e., the Theorem 5.1 hold. Following the ideas of Hasserd et al. (1981), here we derive the explicit formula for determining the properties of Hopf Bifurcation at τ^* by using the normal form and centre manifold theory.

Let

$$x_1 = x - x^*, \ x_2 = y - y^*, \ x_3 = z - z^*, \ \overline{x}_i(t) = x_i(\pi), \ i = 1,2,3$$

(rescale the time by $t \to (t/\tau)$ to normalize the delay) and $\tau = \tau^* + \mu$ (here μ is the bifurcation parameter). Leaving the bars for the simplifications of notations, the system (2.1) becomes a functional differential equation in $C = C([-1,0], R^3)$ as

$$\frac{dX}{dt} = L_{\mu}(X_t) + f(\mu, X_t), \qquad (5.11)$$

where

$$X_t = (x_1(t), x_2(t), x_3(t))^T \in R^3 \text{ and } L_\mu : C \to R^3, \quad f : R \times C \to R^3$$

are, respectively, the linear and nonlinear term of the Tailor series expansion of the right hand side of the system (2.1).

By the Riesz representation theorem described by Hale and Verduyn (1993), \exists a function $\kappa(\theta,\mu)$ of bounded variation for $\theta \in [-1,0]$ such that $L_{\mu}(\phi) = \int_{-1}^{0} d\kappa(\theta,\mu)\phi(\theta)$, for $\phi \in C$.

Let us define

$$A(\mu)\phi = \begin{cases} \frac{d\phi(\theta)}{d\theta}, & \theta \in [-1,0) \\ \int_{-1}^{0} d\kappa(\mu, s)\phi(s), & \theta = 0 \end{cases} \text{ and } R(\mu)\phi = \begin{cases} 0, & \theta \in [-1,0) \\ f(\mu, \phi), & \theta = 0, \end{cases}$$

for $\phi \in C([-1,0], \mathbb{R}^3)$. Then, the system (5.11) is equivalent to

$$\frac{dX}{dt} = A(\mu)X_t + R(\mu)X_t$$

where

$$X_{t}(\theta) = X(t+\theta), \text{ for } \theta \in [-1,0].$$

If q is the eigen vectors and q^* is the adjoint eigen vector of A(0) corresponding to the eigenvalue $+i\omega_1^*\tau^*$ of A(0), then due to Kuznetsov (1997), we can write the following expression:

$$g_{rs} = \frac{\partial^{r+s}}{\partial z^r \partial \overline{z}^s} \langle \overline{q}^*, F(zq + \overline{z}\overline{q}) \rangle \bigg|_{z=0}, \quad r+s \ge 2, \quad r, s = 0,1,2,...,$$

where $\langle \circ, \circ \rangle$ is the standard bilinear scalar product in R^n and z and \bar{z} are local co-ordinates for the center manifold C_0 in the direction of q and \bar{q}^* . We have calculated thoroughly and obtained the expressions of g_{rs} , which are not given here. One can follow Chakraborty et al. (2013) for obtaining those terms and techniques. According to that, we can easily compute the following values:

$$c_{1}(0) = \frac{i}{2\omega_{1}^{*}\tau^{*}} \left(g_{20}g_{11} - 2|g_{11}|^{2} - \frac{|g_{02}|^{2}}{3} \right) + \frac{g_{21}}{2},$$

$$\delta_{2} = -\frac{\operatorname{Re}\{c_{1}(0)\}}{\operatorname{Re}\{\lambda'(\tau^{*})\}},$$

$$\rho_{2} = 2\operatorname{Re}\{c_{1}(0)\},$$

$$T_{2} = \frac{\operatorname{Im}\{c_{1}(0)\} + \delta_{2}\operatorname{Im}\{\lambda'(\tau^{*})\}}{\omega_{1}^{*}\tau^{*}}, \quad k = 0, \pm 1, \pm 2, \dots$$

$$(5.12)$$

Now, we state the following theorem:

Theorem 5.3.

(i) The sign of δ_2 determined the direction of Hopf bifurcation, i.e., if $\delta_2 > 0(\delta_2 < 0)$ then the Hopf bifurcation is super critical (sub critical) and the bifurcating periodic solution exist for $\tau > \tau^* \left(\tau < \tau^*\right)$.

- (ii) The sign of ρ_2 determines the stability of the bifurcating periodic solution i.e. the bifurcating periodic solution is stable (unstable), if $\rho_2 < 0(\rho_2 > 0)$.
- (iii) The sign of T_2 determines the period of the bifurcating periodic solution i.e. the period increase or decrease according as $T_2 > 0$ or $T_2 < 0$.

6. Numerical simulations and result discussions

It is quite difficult to have numerical value of the parameters of the system based on real world observations. As our present work is not a case study no real world data is available. Hence for the purpose of simulation we have used a simulated set of parameters in appropriate units. On the other hand, it is necessary to have some idea regarding the sensitivity of the parameters in connection to the observed real system. Therefore, the major results described by the simulations presented should be considered from a qualitative, rather than a quantitative point of view. Even in absence of real world data, our simulation covering the breath of the biological feasible parameter space makes some sense from all the scenarios tested. For the purpose of simulations, we consider the following set of simulated parameters:

$$P_1 = \{r = 6.8, K = 70, m = 0.3, N = 0.71, L = 1.8, M = 1.4, a = 0.5, q = 0.1, E = 0.9, v = 4.5, s = 0.2, \mu = 3.15, u = 2.6, \xi = 1.6, \eta = 0.7, \alpha = 0.25, \beta = 0.45, \sigma = 0.316, \gamma = 0.12, \omega = 0.23\}.$$

MATLAB 7.10 and Mathematica 9.0 are the main software used for the purpose of simulation experiments.

6.1. Stability and bifurcation due to competition

For the above set of parameters, the infection free planer boundary equilibrium is $E_2(0.655, 2.215, 0)$, the stability of which is confirmed by the phase plane diagram in Fig.1. So without infection prey and predator drive the system in equilibrium. The interior coexistence equilibrium with respect to this parameter set is $E^*(1.36733,14.3872,16.8882)$ which is asymptotically stable. The stability confirms by the time plot and the phase plot for $\sigma=0.316$ in Figure 2.a where there is no limit cycle. Next we try to establish bifurcation of the system (2.1) in absence of delay for the parameter σ and ω .

At first, we consider the parameter σ . We decrease the value of σ keeping other parameters same and observed that the system becomes unstable for $\sigma = 0.315$. Figure 2.b shows the oscillatory solution with a limit cycle in phase plot. Here the critical value of σ is $\sigma^* = 0.3158$. Since the system becomes unstable as it passes through σ^* , there arises a Hopf bifurcation at σ^* . Hence, the parameter σ has a property of stabilization when $\sigma > \sigma^*$ as shown in Table 1. The Table 1 depicts the changes of nature of the point of interior equilibrium from unstable to stable as we increase σ through σ^* . We see that x^* and z^* i.e. equilibrium prey and infected predator are increasing but y^* i.e., susceptible predators are decreasing.

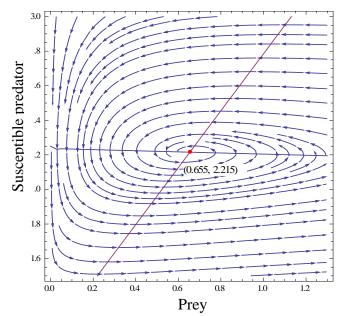
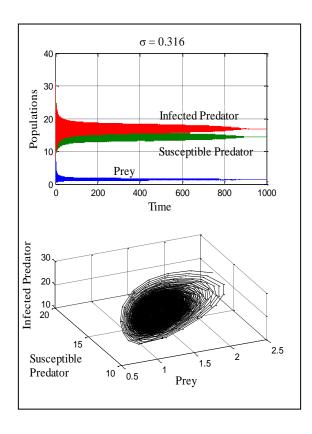


Figure 1. Phase plot of infection free equilibrium



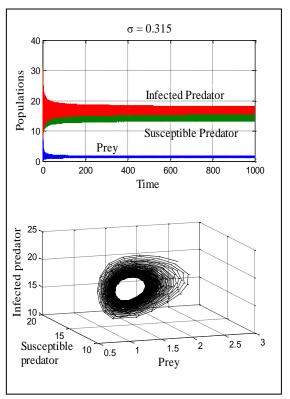
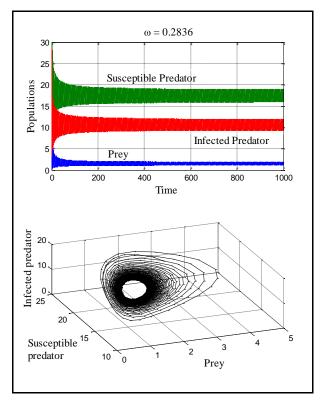


Figure 2.a. Time and phase plot of populations without delay with parameter set P_1 for $\sigma = 0.316 > \sigma^*$

Figure 2.b. Time and phase plot of populations without delay with parameter set P_1 for $\sigma = 0.315 < \sigma^*$

Table 1. Point of equilibrium with different sigma around critical value $\sigma^* = 0.3158$

Observations	Value of	Interior equilibrium E^*			Nature of the
No.	σ	x*	<i>y</i> *	z^*	system
1	0.310	1.35521	14.4569	16.6322	Unstable
2	0.315	1.36531	14.3987	16.8456	Unstable
3	0.316	1.36733	14.3872	16.8882	Stable
4	0.320	1.37539	14.3416	17.0579	Stable



 $\omega = 0.2842$ 40 Populations Susceptible Predator 20 10 Infected Predator 200 400 600 800 1000 Time Infected predator 25 20 Susceptible 10 predator 0 Prey

Figure 3.a. Time and phase plot of populations without delay with parameter set P_1 for $\omega = 0.2836 < \omega^*$

Figure 3.b. Time and phase plot of populations without delay with Parameter set P_1 for $\omega = 0.2842 > \omega^*$

Now, we want to investigate the stabilizing effect of ω . For that we consider $\sigma = 0.2$ and $\omega = 0.2836$ with the same value of other parameters in the parameter set P_1 . The time plot in Figure 3.a reveals that the system is unstable and there is a limit cycle in the corresponding phase plot. But, if we gradually increase the value of ω , then the similar figure in Figure 3.b for $\omega = 0.2842$ shows that the system becomes asymptotically stable which confirms by the absence of limit cycle in the corresponding phase plot. Thus there arises a Hopf bifurcation when $\omega = \omega^*$, where $\omega^* = 0.2841$ is the critical value of ω . Therefore, the parameter ω has a property of stabilization when it is increasing through it's critical value ω^* . Table 2 also shows the nature of the system with the change of ω through ω^* . It is a natural phenomenon in a prey predator model that several species when coexists, must compete with each other for their food and shelter i.e. for their existence. This competition reduces them in general. Here this competition plays among predator subpopulations only.

Observations	Value of	Interior equilibrium E^*			Nature of the
No.	ω	<i>x</i> *	<i>y</i> *	z^*	system
1	0.2836	1.41807	17.3043	10.5952	Unstable
2	0.2838	1.41910	17.3080	10.5915	Unstable
3	0.2840	1.42014	17.3117	10.5878	Unstable
4	0.2842	1.42117	17.3155	10.5841	Stable
5	0.2844	1.42220	17.3192	10.5804	Stable

Table 2. Point of equilibrium with different sigma around critical value $\omega^* = 0.2841$

In our model, we consider two separate competition rate. One is among the susceptible with the whole predator biomass and the other, is among the infected with whole predator biomass. We have separately investigated their influence on every species. But species are interacting among themselves under the simultaneous influence of both the parameters σ and ω . Here we have depicted three different figures which reveal the quantitative change of three population with respect to σ and ω .

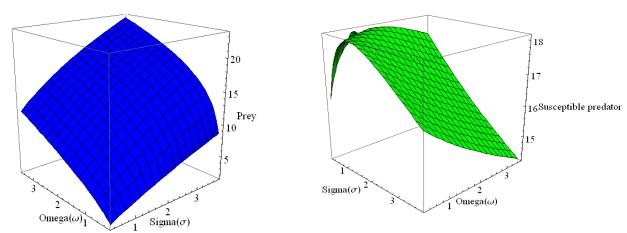


Figure 4.a. Variation of x(t) w.r.t. σ and ω **Figure 4.b.** Variation of y(t) w.r.t. σ and ω

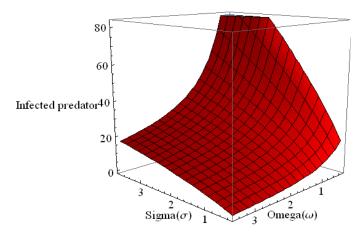


Figure 4.c. Variation of z(t) w.r.t. σ and ω

Figure 4.a clearly shows that prey population x(t) is increasing with the increase of both the parameters σ and ω . Figure 4.b reveals that at low σ and ω susceptible predator y(t) increases, but at their high value, y(t) decreases finally. In figure 4.c we see that for increase of σ , z(t) increase rapidly at low value of ω , but at high value of ω , z(t) increases moderately. However, z(t) increases for increase of σ . On the other hand for increase of ω , z(t) decreases slowly at low σ , but it decreases rapidly at high σ . In any ways, z(t) decreases for increase of ω . The above phenomena are summaries in the following Table 3 and Table 4.

Table 3. Tendency of biomasses with increase of σ

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As ω increase	At low σ	At high σ	
x (t) (Figure 4.a)	Increases		
y(t) (Figure 4.b)	Initially increases and then decreases	Decreases	
z (t) (Figure 4.c)	Decreases with low rate	Decreases with high rate	

Table 4. Tendency of biomasses with increase of ω

As σ increase	At low ω	At high ω	
x (t) (Figure 4.a)	Increases		
y(t) (Figure 4.b)	Initially increases and then decreases	decreases	
z (t) (Figure 4.c)	Increase with high rate	Increase moderately	

One thing to remember is that, σ should be greater than ω , otherwise the system may be unstable due to extinction of one or more species (Sarwardi et al., 2011). We observed in our previous discussion that under the considered parameter set P_1 , the system becomes stable with respect to the two competition rate σ and ω at their critical values $\sigma^* = 0.3158$ and $\omega^* = 0.2841$ where $\sigma^* > \omega^*$ also.

The obtained results can be described ecologically. Note that ω and σ must reduce infected predator as well as susceptible predator and that also reduce the consumption of prey. As a result prey population increases. When both σ and ω are small, competition among predator is very low. Naturally, susceptible predator consumes more prey and increase themselves. But when σ increases, susceptible predator will reduce more than infected class. Not only that when ω is low, reduction of infected predator is low than that at high value of ω . So the existing infected class will get more food than before and increase them. Simultaneously, infection among susceptible predator increases the infected class also. These combined effects finally cause an increase in z(t). But with the increase of ω , competition of infected class with the whole predator biomass becomes high which in turn reduce the increment rate of z(t) and z(t) increases moderately at high ω . Thus to increase prey abundance for the interest of harvesting in this ecoepidemic system, higher value of ω is desirable with the higher value of σ .

6.2.2. Effect of varying infection rate

The rate of infection (β) has a great importance in this eco-epidemic system. In Figure 5, we see that infected predator populations are increasing (Figure 5.a) noticeably but susceptible predators are decreasing (Figure 5.b) with increasing infection rate. This is obvious because increase of

infection in susceptible predator reduce them and increase the infected class. Since infected predator consumes less number of prey than susceptible predator due to their physical weakness as described in (A_3) of section 2, total predation by the predators (both susceptible and infected) reduces. This is shown in Figure 5.c. and it's inset figure. On the other hand, decrease of predation increase the prey population which is shown in Figure 5.d. (clearly shown at the inset figure). This is desired and achieved. Therefore, infection rate among predators should increase the harvesting of prey, and more infection among predator is desirable for obtaining MSY(maximum sustainable yield) in this prey harvested system. So infection rate can be a regulatory parameter in our system.

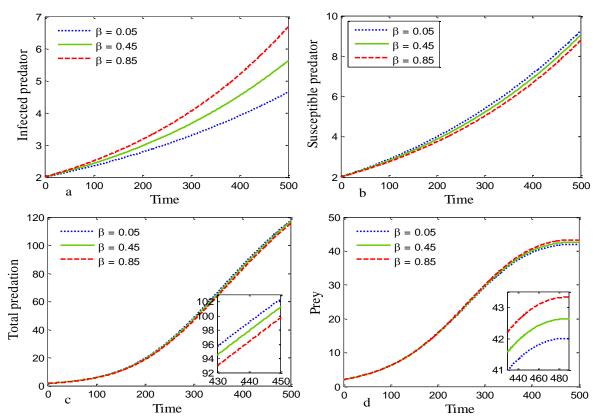


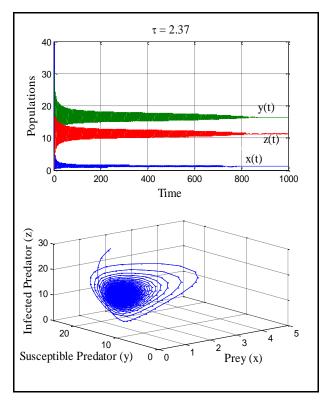
Figure 5. Variation of infected predator (a), susceptible predator (b), total predation and prey(a) for different values of β .

6.3. Delay-induced bifurcation: switching stability

In this section, we present some numerical results of the delayed system with graphical illustrations to establish the theoretical results derived earlier. For this purpose, we shall use the parameter set mentioned above. We have numerically studied delay induced stability of the system. It is to be noted that the critical value of delay is $\tau_1^* = 2.378$ which satisfies the conditions, $\rho_2 < 0$, $\delta_2 > 0$, $T_2 > 0$. Therefore, according to Theorem 5.2, we can state that the system is locally asymptotically stable around $E^*(x^*, y^*, z^*)$ when $\tau < \tau_1^*$ and unstable when $\tau > \tau_1^*$. The time series solution curves and phase space trajectories of the system (2.1) for $\tau = 2.37 (< \tau_1^*)$ in Fig-

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ure 6.a shows that system is stable. But for $\tau = 2.38 > \tau_1^*$, the system is unstable which is confirmed by the Figure 6.b with limit cycle in the corresponding phase plot.



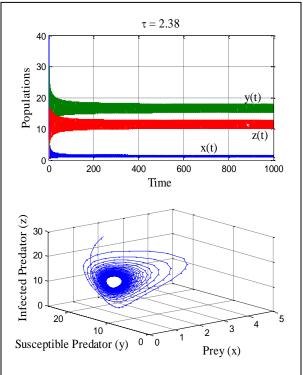


Figure 6.a. Time and phase plot of populations for parameter set P_1 with time delay $\tau = 2.37$

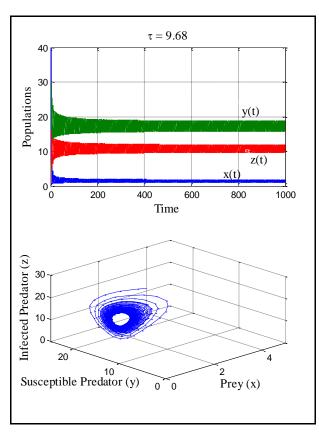
Figure 6.b. Time and phase plot of populations for parameter set P_1 with time delay $\tau = 2.38$

Moreover, Theorem 5.2 states that, if the necessary conditions, for the existence of Hopf bifurcation, are satisfied and τ increases from zero upward then first such Hopf bifurcation would be stable to unstable, the second from unstable back to stable, the third from stable to unstable and so on. In order to validate the theorem, if we gradually increase the value of τ , we obtain another critical value $\tau_2^* = 9.75$, and for $\tau = 9.68 < \tau_2^*$ the system remains unstable around $E^*(x^*, y^*, z^*)$. This is shown in Figure 8.a, where we see a limit cycle around E^* . Further we increase τ and for $\tau = 9.76 > \tau_2^*$, it is clearly observed from Figure 8.b that the positive equilibrium $E^*(x^*, y^*, z^*)$ become stable and the periodic orbit near $E^*(x^*, y^*, z^*)$ disappears.

7. Conclusion

In this paper, we have analyzed the dynamical behavior of a prey-predator type system with disease in the predator population. The growth of the predators' susceptible and infected subpopulations is assumed as the modified the Leslie–Gower type. In addition to the inter-specific competition among the predators' subpopulations, we have incorporated the intra-specific competition among the predators' susceptible and infected subpopulations. Existence and stability criterion of different equilibrium points and the criterion for uniform strong persistence of the system are derived. In this regard, it is observed that a structured population is permanent, if it is uniformly (strongly) persistent. We have derived the existence and stability criterion of the different equi-

librium points. The equilibrium system without predator exists only when the BTP of the prey is greater than the harvesting effort, but this equilibrium is unstable. Disease free equilibrium exists under certain conditions. The stability criteria of disease free equilibrium points are discussed. The system is also locally, as well as, globally stable at the interior equilibrium point under certain conditions.



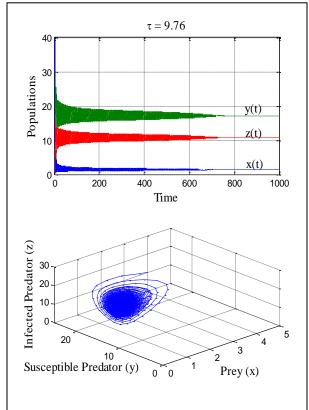


Figure 7.a. Time and phase plot of populations for parameter set P_1 except $\sigma = 0.2$, $\omega = 0.23$ with time delay $\tau = 9.68 < \tau^*$.

Figure 7.b. Time and phase plot of populations for parameter set P_1 except $\sigma = 0.2$, $\omega = 0.23$ with time delay $\tau = 9.76 > \tau^*$.

It is well accepted that the competition among the predators is influencing the interaction of the species of a prey-predator system. It is evident from the obtained results that the (inter specific and intra specific) competition among predator species can cause an unstable system to become stable equilibrium and even a simple Hopf bifurcation can occur when it passes through its critical value. Not only that, the high competition among predators increase prey biomass and decrease the predator biomass. As a result, total harvest increases by the increase of prey abundance. Hence to obtain maximum sustainable yield (MSY) for long time prey harvesting, competition among predators can enhance the harvesting.

Again, the consideration of a disease in predator population makes the system extremely complex. It is reasonable that the increase of disease transmission rate in predator population reduce them. But here increase of this infection rate increase the prey population with the reduction of predator which in turn increase the maximum sustainable yield (MSY). Thus to control an ex-

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ploited prey predator eco-epidemic system leading to increase harvesting, diseases transmission rate in predator is a controlling parameter and it is desirable. However, the consequent results are important towards the conservation of the species and the sustainability of the system.

The dynamic behavior of the system with diseases transmission delay is examined in the second part of the paper. In order to prove that delay differential equations exhibit much more complicated dynamics compared to ordinary differential equations, we have rigorously studied the effects of delay on the dynamics of a prey-predator system. It is clearly observed from the obtained results that the time delay can cause a stable equilibrium to become unstable, and even a simple Hopf bifurcation may occur when the time delay passes through its critical value. Subsequently, the system may exhibit cycle behavior and due to this cyclic nature some population exhibit periodic fluctuation in abundance. It is also clear that, when the time delay is small, both the prey and predator populations reach periodic oscillations around the equilibrium in finite time and then converge to their equilibrium values. As the diseases transmission delay increases, the oscillations also increases.

But, when the diseases transmission delay is too long, the positive steady state disappears. However, if we continuously increase the value of diseases transmission delay then the stability of the system may be resumed due to the conversion efficiency of predator (susceptible and infected) into new predators. Therefore, we can conclude that diseases transmission delay of certain dimensions can induce instability oscillation via Hopf bifurcation due to some impulsive phenomenon, consequently, switching stability behavior may be incurred to the system. The conditions to determine the direction of Hopf bifurcation are obtained. However, the model and its dynamical behavior are studied mainly on the deterministic framework. In this regard, we can say that it will be more realistic to consider the model in a stochastic environment due to ecological fluctuations. This needs further future work in this context.

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APPENDICES

Appendix A: Positivity (Proof of Theorem 3.1)

From the first equation of the system (2.1), we get

$$\frac{dx}{dt} = x(t) \left(r \left(1 - \frac{x(t)}{K} \right) - \frac{\alpha z(t)}{a + x(t)} - my(t) - qE \right),$$

or,

$$\frac{dx}{x} = \left(r\left(1 - \frac{x(t)}{K}\right) - \frac{\alpha z(t)}{a + x(t)} - my(t) - qE\right)dt.$$

Taking integration over [0,t], we get $x(t) = x(0) \exp\left(\int_{0}^{t} \aleph_1 dp\right) > 0$ as x(0) > 0,

where

$$\aleph_1 = r \left(1 - \frac{x(p)}{K} \right) - \frac{\alpha z(p)}{a + x(p)} - my(p) - qE.$$

Similarly, from second and third equations of the system (2.1), with y(0) > 0, and z(0) > 0, we can write

$$y(t) = y(0) \exp\left(\int_{0}^{t} \aleph_{2} dp\right) > 0 \text{ and } z(t) = z(0) \exp\left(\int_{0}^{t} \aleph_{3} dp\right) > 0,$$

where

$$\aleph_2 = s - \frac{\sigma(y(p) + z(p))}{ux(p) + L} - \frac{\beta z(p)}{y(p) + z(p) + M} + \frac{\xi z(p)}{y(p)},$$

$$\aleph_3 = \mu - \frac{\omega(y(p) + z(p))}{vx(p) + N} + e^{-\gamma \tau} \frac{\beta y(p - \tau)z(p - \tau)}{(y(p - \tau) + z(p - \tau) + M)z(p)} - \eta - \xi.$$

Thus, all the solutions of the system of equation (2.1) are positive. Hence the desired result is obtained.

Appendix B (Proof of Proposition 3.1)

For this we shall first state a result according to Chen (2005) as follows:

Lemma 1.

If a > 0, b > 0 and $\dot{S}(t) \ge (\le) S(t) (b - aS^{\alpha}(t))$, where α is a positive constant, when $t \ge 0$ and

$$S(0) > 0$$
, we have $S(t) \ge \left(\le \left(\frac{b}{a} \right)^{\frac{1}{\alpha}} \left[1 + \left(\frac{bS^{-\alpha}(0)}{a} - 1 \right) e^{-b\alpha t} \right]^{-\frac{1}{\alpha}}$.

Now, from the first equation of the system (2.1) we have for all t > 0,

$$x(t) < x(t)\left(r - qE - \frac{rx(t)}{K}\right).$$

According to the Lemma 1, we have

$$\limsup_{t \to +\infty} x(t) \le K(r - qE)/r = M_1 \text{ (say)}.$$
(B.1)

Now, we can say that, for any $\varepsilon > 0$ there exists $T_1 > 0$ such that,

$$x(t) < M_1 + \varepsilon, \quad t > T_1.$$

From the second equation of the system (2.1) we have for all $t \in [0, H[$,

$$\dot{y}(t) < y(t) \left(s + \xi - \frac{\sigma y(t)}{ux(t) + L} \right)$$

$$< y(t) \left(s + \xi - (\frac{\sigma}{u(M_1 + \varepsilon) + L}) y(t) \right).$$

Using the lemma 1, we can write

$$\limsup_{t \to +\infty} y(t) \le (s+\xi)(u(M_1+\varepsilon)+L)/\sigma = M_2 \text{ (say)}. \tag{B.2}$$

Then we can say that, for any $\varepsilon > 0$ there exists $T_2 > T_1 > 0$ such that,

$$y(t) < M_2 + \varepsilon, \quad t > T_2$$
.

From the third equation of the system (2.1) we have for all $t \in [0, H[, \dot{z}(t) \ge -z(t)(\eta + \xi)]$.

Thus, for $t > \tau$, we have

$$z(t) \ge z(t-\tau)e^{-(\eta+\xi)\tau} \Rightarrow z(t-\tau) \le z(t)e^{(\eta+\xi)\tau} \text{ for } t > \tau.$$

Therefore, we can write

$$\begin{split} \dot{z}(t) &\leq z(t) \Bigg[(\mu - \eta - \xi) - \frac{\omega(y+z)}{vx+N} + e^{-\gamma\tau} \frac{\beta y(t-\tau)e^{(\eta+\xi)\tau}}{y(t-\tau)+z(t-\tau)+M} \Bigg] \\ &\leq z(t) \Bigg[(\mu - \eta - \xi) - \frac{\omega(y+z)}{vx+N} + e^{-\gamma\tau} \beta y(t-\tau)e^{(\eta+\xi)\tau} \Bigg] \\ &\leq z(t) \Bigg[(\mu - \eta - \xi) - \frac{\omega z(t)}{vM_1+N} + \beta M_2 e^{(\eta+\xi-\gamma)\tau} \Bigg], \quad \text{since } y(t-\tau) \leq M_2 \\ &\leq z(t) \Bigg[(\mu + \beta M_2 e^{(\eta+\xi-\gamma)\tau} - \eta - \xi) - \frac{\omega}{vM_1+N} z(t) \Bigg]. \end{split}$$

Then, from the Lemma 1, we can write

$$\limsup_{t \to +\infty} z(t) \le \left(\mu + \beta M_2 e^{(\eta + \xi - \gamma)\tau} - \eta - \xi\right) \left(vM_1 + N\right) / \omega = M_3 \text{ (say)}. \tag{B.3}$$

Then, we can say that, for any $\varepsilon > 0$ there exists $T_3 > T_2 > 0$ such that,

$$z(t) < M_3 + \varepsilon, \quad t > T_3$$
.

Further, from the first equation of the system (2.1) we have,

$$\begin{split} \dot{x}(t) &= x(t) \Bigg(r \bigg(1 - \frac{x(t)}{K} \bigg) - \frac{\alpha z(t)}{a + x(t)} - m y(t) - q E \Bigg) \\ &\geq x(t) \Bigg(r \bigg(1 - \frac{x(t)}{K} \bigg) - \frac{\alpha M_3}{a} - m M_2 - q E \Bigg) \\ &\geq x(t) \Bigg(\Bigg(r - \frac{\alpha M_3}{a} - m M_2 - q E \Bigg) - \frac{r x(t)}{K} \Bigg). \end{split}$$

By the above lemma 1, we can conclude that,

$$\lim_{t \to +\infty} \inf x(t) \ge \frac{K}{r} \left(r - \frac{\alpha M_3}{a} - mM_2 - qE \right) = N_1 \text{ (say)}. \tag{B.4}$$

Then, we can say that, for any $\varepsilon > 0$ there exists $T_4 > T_3 > 0$ such that,

$$x(t) > N_1 + \varepsilon, \quad t > T_4.$$

From the second equation of the system (2.1) we have,

$$\dot{y}(t) = y(t) \left(s - \frac{\sigma(y(t) + z(t))}{ux(t) + L} - \frac{\beta z(t)}{y(t) + z(t) + M} + \xi \frac{z(t)}{y(t)} \right)$$

$$\geq y(t) \left(\left(s - \frac{\sigma M_3}{L} - \frac{\beta M_3}{M} \right) - \frac{\sigma y(t)}{L} \right).$$

By the above lemma 1, we can write that,

$$\liminf_{t \to +\infty} y(t) \ge \frac{L}{\sigma} \left(s - \frac{\sigma M_3}{L} - \frac{\beta M_2}{M} \right) = N_2 \text{ (say)}.$$
(B.5)

Then, we can say that, for any $\varepsilon > 0$ there exists $T_5 > T_4 > 0$ such that,

$$y(t) > N_2 + \varepsilon$$
, $t > T_5$.

Finally from the third equation of the system (2.1) we have,

$$\dot{z}(t) = z(t) \left(\mu - \frac{\omega(y(t) + z(t))}{vx(t) + N} + e^{-\gamma \tau} \frac{\beta y(t - \tau)z(t - \tau)}{z(t)(y(t - \tau) + z(t - \tau) + M)} - \eta - \xi \right)
\geq z(t) \left(\mu - \eta - \xi - \frac{\omega y(t) + \omega z(t)}{N} \right)
\geq z(t) \left(\mu - \eta - \xi - \frac{\omega M_2}{N} - \frac{\omega z(t)}{N} \right).$$

By the Lemma 1, we can write

$$\lim_{t \to +\infty} \inf z(t) \ge \frac{1}{\omega} \left((\mu - \eta - \xi) N + \omega M_2 \right) = N_3 \text{ (say)}. \tag{B.6}$$

This completes the proof of the theorem.

Appendix C: Persistence (Proof of Theorem 3.2)

From the Proposition 3.1, it is observed that the system (2.1) has limit supremum and limit infimum as $t \to \infty$. For the persistence all constants M_1, M_2, M_3, N_1, N_2 and N_3 defined by the equations (B.1)-(B.6) (in Appendix B) respectively must be greater than zero, i.e. $M_i > 0, N_i > 0$ for i=1,2,3.

Now $M_1 > 0 \implies E < r/q$, the BTP of the prey species. It is easy to state that for the existence of positive boundary equilibrium (x(t),0,0), E < r/q always true and we can take

$$r/q-E>0$$
.

Again $M_2 > 0, M_3 > 0$, without confusion by their expression.

Also

$$N_1 > 0 \implies r > \frac{\alpha M_3}{a} + mM_2 + qE$$

and

$$N_2 > 0 \implies s > \frac{\sigma M_3}{L} + \frac{\beta M_2}{M}$$
.

Further, $N_3 > 0 \implies \mu > (\eta + \xi) + \omega M_2 / N$. Hence, the required results are obtained.

Appendix D: Permanence (Proof of Theorem 3.3)

When the system (2.1) is persistent, all constants M_1, M_2, M_3, N_1, N_2 and N_3 are greater than zero. So,

$$\min\{N_1, N_2, N_3\} \ge 0 \text{ and } \max\{M_1, M_2, M_3\} \ge 0.$$

Also it is obvious that, $\liminf < \limsup \sup$. So, $\min(\liminf) < \max(\limsup \sup)$. Thus, there exists i, g defined by $i \le \min\{N_1, N_2, N_3\}$ and $\max\{M_1, M_2, M_3\} \le g$ then 0 < i < g. Hence, by the definition, the system is permanent.

Appendix-E: Global stability (Proof of Theorem 4.5.)

Here we will use geometric approach described by Li and Muldowney (1996) to show an n-dimensional autonomous dynamical system

$$\frac{dX}{dt} = f(X) , \qquad (E.1)$$

where $f: D \to \mathbb{R}^n$. $D \subset \mathbb{R}^n$ an open and simply connected set and $f \in C^1(D)$ is globally stable under certain parametric conditions. For details see Buonomo et al., 2008, Chakraborty et al., 2013.

A steady-state \overline{X} of (E.1) is said to be globally stable in D, if it is locally stable and all trajectories in D converge to \overline{X} . Let $X(t,X_0)$ denote the solution of (E.1) satisfying $X(0,X_0)=X_0$ and Df the Jacobian matrix of f at X. Assume that (E.1) satisfies the following two conditions:

(H1) System (E.1) has a unique steady-state \overline{X} in D.

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(H2) System (E.1) has a compact absorbing set $\Omega \subset D$.

Let $X \to M(X)$ be an $\binom{n}{2} \times \binom{n}{2}$ matrix-valued function that is C^1 on D. Assume that $M^{-1}(X)$ exists and is continuous for $X \in D$. For a Lozinskii measure Γ_1 , a quantity \overline{q}_2 is defined as

$$\overline{q}_2 = \lim_{t \to \infty} \sup \sup_t \frac{1}{t} \int_0^t \Gamma_1(B) ds$$
 where $B = M_f M^{-1} + M(Df) M^{-1}$ and $M_f = \frac{dM}{dX}$,

where
$$f(X) = \begin{bmatrix} rx\left(1 - \frac{x}{K}\right) - \frac{\alpha xz}{a + x} - mxy - qEx \\ sy - \frac{\sigma y(y + z)}{ux + L} - \frac{\beta yz}{y + z + M} + \xi z \\ \mu z - \frac{\omega z(y + z)}{vx + N} + \frac{\beta yz}{y + z + M} - \eta z - \xi z \end{bmatrix} \text{ and } X = \begin{bmatrix} x \\ y \\ z \end{bmatrix}.$$

The variational matrix of the system can be written a $V = \frac{\partial f}{\partial X} = (J_{ij})$. If $V^{[2]}$ be the second additive compound matrix of V then due to Buonomo et al. (2008) we can write

$$V^{[2]} = \begin{pmatrix} J_{11} + J_{22} & J_{21} & -J_{13} \\ J_{32} & J_{11} + J_{33} & J_{12} \\ -J_{31} & J_{21} & J_{22} + J_{33} \end{pmatrix}.$$

We consider M(X) in a way that $M = \text{diag}\{\frac{x}{z}, \frac{x}{z}, \frac{x}{z}\}$ in $C^1(D)$. Then, we have

$$M^{-1} = \operatorname{diag}\left\{\frac{z}{x}, \frac{z}{x}, \frac{z}{x}\right\},\,$$

$$M_f = \frac{dM}{dX} = \operatorname{diag} \left\{ \dot{x}/z - (x/z^2)\dot{z}, \dot{x}/z - (x/z^2)\dot{z}, \dot{x}/z - (x/z^2)\dot{z} \right\} \text{ and } MV^{[2]}M^{-1} = V^{[2]}$$

So calculating we get $B = M_f M^{-1} + MV^{[2]} M^{-1} = \begin{bmatrix} B_{11} & B_{12} \\ B_{21} & B_{22} \end{bmatrix}$, where

$$B_{11} = \frac{\dot{x}}{x} - \frac{\dot{z}}{z} - \frac{rx}{K} + \frac{\alpha xz}{(a+x)^2} - \frac{\xi z}{y} - \frac{\sigma y}{ux+L} + \frac{\beta yz}{(y+z+M)^2},$$

$$B_{12} = \left[\xi - \frac{\sigma y}{ux+L} - \frac{\beta y(y+M)}{(y+z+M)^2}, \quad \frac{\alpha x}{(a+x)} \right],$$

$$B_{21} = \left[-\frac{\omega z}{vx+N} + \frac{\beta z(z+M)}{(y+z+M)^2}, \quad -\frac{\omega vz(y+z)}{(vx+N)^2} \right]^T,$$

$$B_{22} = \begin{bmatrix} \frac{\dot{x}}{x} - \frac{\dot{z}}{z} - \frac{rx}{K} + \frac{\alpha xz}{(a+x)^2} - \frac{\omega z}{vx+N} - \frac{\beta yz}{(y+z+M)^2}, & -mx \\ \frac{\sigma u y(y+z)}{(ux+L)^2}, & \frac{\dot{x}}{x} - \frac{\dot{z}}{z} - \frac{\xi z}{y} - \frac{\sigma y}{ux+L} - \frac{\omega z}{vx+N} \end{bmatrix}.$$

Let us now define the following vector norm in \mathbb{R}^3 as:

$$|(u, v, w)| = \max\{|u|, |v| + |w|\},\$$

where (u, v, w) is the vector in \mathbb{R}^3 and denote by M, the Γ_1 is the Lozinskii measure with respect to this norm. Therefore,

$$\Gamma_1(B) \le \sup\{l_1, l_2\},\tag{E.2}$$

where $l_i = \Gamma_1(B_{ii}) + \left| B_{ij} \right|$ for i = 1, 2 and $i \neq j$, $\left| B_{12} \right|$ and $\left| B_{21} \right|$ are matrix norms with respect to the vector norm. Therefore, we can easily obtain the following terms:

$$\Gamma_{1}(B_{11}) = \frac{\dot{x}}{x} - \frac{\dot{z}}{z} + \frac{\alpha xz}{(a+x)^{2}} + \frac{\beta yz}{(y+z+M)^{2}} - \frac{rx}{K} - \frac{\xi z}{y} - \frac{\sigma y}{ux+L},$$
(E.3)

$$\left|B_{12}\right| = \max\left\{\left|\frac{\sigma y}{u x + L} + \frac{\beta y(y + M)}{\left(y + z + M\right)^{2}} - \xi\right|, \quad \frac{\alpha x}{\left(a + x\right)}\right\},\tag{E.4}$$

$$\left|B_{21}\right| = \max\left\{\left|\frac{\beta z(z+M)}{\left(y+z+M\right)^{2}} - \frac{\omega z}{vx+N}\right|, \quad \left|-\frac{\omega v z(y+z)}{\left(vx+N\right)^{2}}\right|\right\},\tag{E.5}$$

$$\Gamma_1(B_{22}) = \frac{\dot{x}}{x} - \frac{\dot{z}}{z} - \frac{\omega z}{v x + N}$$

$$+ \max \left\{ \frac{\alpha xz}{(a+x)^{2}} - \frac{rx}{K} - \frac{\beta yz}{(y+z+M)^{2}} + \frac{\sigma u y(y+z)}{(ux+L)^{2}}, -\frac{\xi z}{y} - \frac{\sigma y}{ux+L} + \left| -mx \right| \right\}$$
 (5.6)

Now from the third equation of the system (2.1) we can write

$$\frac{\dot{z}}{z} = \mu - \eta - \xi + \frac{\beta y}{y + z + M} - \frac{\omega(y + z)}{v x + N}.$$
(E.7)

Let

 $\mu_l < \min\{\liminf x(t), \liminf y(t), \liminf z(t)\}$

and

 $\max\{\limsup x(t), \limsup y(t), \limsup z(t)\} < \mu_m$.

Obviously, $\mu_i < \mu_m$. Using this we have

$$l_1 \le \frac{\dot{x}}{x} - (\mu - \eta - \xi) + \mu_1,$$
 (E.8)

where

$$\mu_1 = \mu_{11} + \mu_{12}$$

$$\mu_{11} = \frac{2\omega \,\mu_m}{v \,\mu_l + N} + \frac{\alpha \,\mu_m^2}{(a + \mu_l)^2} + \frac{\beta \,\mu_m^2}{(2 \,\mu_l + M)^2} - \frac{\beta \,y}{2 \,\mu_m + M} - \frac{r \,\mu_l}{K} - \frac{\xi \,\mu_l}{\mu_m} - \frac{\sigma \,\mu_l}{u \,\mu_m + L}, \tag{E.9a}$$

and

$$\mu_{12} = \max \left\{ \frac{\sigma \mu_{m}}{u \mu_{l} + L} + \frac{\beta \mu_{m} (\mu_{m} + M)}{(2 \mu_{l} + M)^{2}} - \xi \right|, \frac{\alpha \mu_{m}}{(a + \mu_{l})} \right\}.$$
 (E.9b)

Similarly, $l_2 \le \frac{\dot{x}}{x} - (\mu - \eta - \xi) + \mu_2$, where

$$\mu_2 = \mu_{20} + \mu_{21} + \mu_{22},$$
 (E.10)

$$\mu_{20} = \frac{\omega \mu_{m}}{v \mu_{l} + N} - \frac{\beta \mu_{l}}{2\mu_{m} + M},$$

$$\mu_{21} = \max \left\{ \frac{\alpha \mu_{m}^{2}}{(a + \mu_{l})^{2}} + \frac{\sigma u 2\mu_{m}^{2}}{(u \mu_{l} + L)^{2}} - \frac{r \mu_{l}}{K} - \frac{\beta \mu_{l}^{2}}{(2\mu_{m} + M)^{2}}, -\left(\frac{\sigma \mu_{l}}{u \mu_{m} + L} + \frac{\xi \mu_{l}}{\mu_{m}} + m \mu_{l}\right) \right\},$$

$$\mu_{22} = \max \left\{ \frac{\omega 2\mu_{m}^{3}}{(v \mu_{l} + N)^{2}}, \frac{\beta \mu_{m} (\mu_{m} + M)}{(2\mu_{l} + M)^{2}} - \frac{\omega \mu_{l}}{v \mu_{m} + N} \right\}.$$
(E.11)

Hence, from (E.2)
$$\Gamma_1(B) \le \frac{\dot{x}}{x} - (\mu - \eta - \xi - \mu_0)$$
 where $\mu_0 = \max\{\mu_1, \mu_2\}$ (E.12)

$$\Gamma_1(B) \le \frac{\dot{x}}{x} - \pi$$
, where $\pi = \mu - \eta - \xi - \mu_0 > 0$ when $\mu_0 < \mu - \eta - \xi$.

Along each solution $X(t, X_0)$ to (D.2) such that $X_0 \in \Omega$, we have

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$$\frac{1}{t} \int_{0}^{t} \Gamma_1(B) ds \leq \frac{1}{t} \log \frac{x(t)}{x(0)} - \pi.$$

So

$$\lim_{t\to\infty}\sup\sup_{t}\sup_{t}\int_{0}^{t}\Gamma_{1}(B)ds\leq\lim_{t\to\infty}\sup_{X_{0}\in\Omega}\left\{\frac{1}{t}\log\frac{x(t)}{x(0)}-\pi\right\}<-\pi.$$

Therefore, $\overline{q}_2 < 0$ provided $\mu_0 < \mu - \eta - \xi$. Hence, we can conclude that the system is globally stable and the Theorem 4.5 thus verified.

Appendix F

$$G = (a_{2} - 3\omega_{1}^{2})\{b_{2}^{2}\omega_{1}^{2} + (b_{3} - b_{1}\omega_{1}^{2})^{2}\} + (b_{2} + \varpi_{1}\omega_{1}^{2} - b_{3}\tau)\{b_{2}\omega_{1}(\omega_{1}^{3} - a_{2}\omega_{1}) - (a_{3} - a_{1}\omega_{1}^{2})\}$$

$$(b_{3} - b_{1}\omega_{1}^{2})\} + (2b_{1}\omega_{1} - \varpi_{2}\omega_{1})\{b_{2}\omega_{1}(a_{1}\omega_{1}^{2} - a_{3}) + (b_{1}\omega_{1}^{2} - b_{3})(\omega_{1}^{3} - a_{2}\omega_{1})\}$$

$$H = 2a_{1}\omega_{1}\{b_{2}^{2}\omega_{1}^{2} + (b_{3} - b_{1}\omega_{1}^{2})^{2}\} + (2b_{1}\omega_{1} - \varpi_{2}\omega_{1})\{b_{2}\omega_{1}(\omega_{1}^{3} - a_{2}\omega_{1}) - (a_{3} - a_{1}\omega_{1}^{2})\}$$

$$(b_{3} - b_{1}\omega_{1}^{2})\} + (\varpi_{3}\omega_{1} - b_{2} - \varpi_{1}\omega_{1}^{2})\{b_{2}\omega_{1}(a_{1}\omega_{1}^{2} - a_{3}) + (b_{1}\omega_{1}^{2} - b_{3})(\omega_{1}^{3} - a_{2}\omega_{1})\}$$

$$K = (a'_{1}\omega_{1}^{2} - a'_{3})\{b'_{2}\omega_{1}^{2} + (b_{3} - b_{1}\omega_{1}^{2})^{2}\} + (b'_{1}\omega_{1}^{2} - b'_{3} - b_{2}\omega_{1}^{2})\{b_{2}\omega_{1}(\omega_{1}^{3} - a_{2}\omega_{1}) - (a_{3} - a_{1}\omega_{1}^{2})\}$$

$$(b_{3} - b_{1}\omega_{1}^{2})\} + (b_{3}\omega_{1}^{2} - b'_{2}\omega_{1} - b_{1}\omega_{1}^{3})\{b_{2}\omega_{1}(a_{1}\omega_{1}^{2} - a_{3}) + (b_{1}\omega_{1}^{2} - b_{3})(\omega_{1}^{3} - a_{2}\omega_{1})\}$$

$$L = -a'_{2}\omega_{1}\{b'_{2}\omega_{1}^{2} + (b_{3} - b_{1}\omega_{1}^{2})^{2}\} + (b_{3}\omega_{1} - b'_{2}\omega_{1} - b_{1}\omega_{1}^{3})\{b_{2}\omega_{1}(a_{1}\omega_{1}^{2} - a_{3}) + (b_{1}\omega_{1}^{2} - b_{3})(\omega_{1}^{3} - a_{2}\omega_{1})\}$$

$$(b_{3} - b_{1}\omega_{1}^{2})\} + (b'_{3}\omega_{1}^{2} + b_{2}\omega_{1}^{2} - b'_{1}\omega_{1}^{2})\{b_{2}\omega_{1}(a_{1}\omega_{1}^{2} - a_{3}) + (b_{1}\omega_{1}^{2} - b_{3})(\omega_{1}^{3} - a_{2}\omega_{1})\}$$

(dash denotes the derivative with respect to τ)