Pelican at Risk in Salton Sea – a Delay-Induced Eco-Epidemiological Model

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ABSTRACT

Elevated salinity, accelerated eutrophication, blooms of Avian botulism and dramatic water quality fluctuation are supposed to be the key factors for massive die-off of Tilapia (prey) and Pelican (predator) in the Salton sea. We modify the model of Chattopadhyay and Bairagi [*Ecological Modelling 136* (2001), pp. 103–112] with an assumption that the growth rate of susceptible fish population is very high and study the dynamics of the system by introducing a delay factor in the predator response function. It is observed that the otherwise stable system exhibit a stable limit cycle solution when the lag factor attains its critical value. It is also observed that there is a high possibility of an epidemic out break in the fish as well as in the Pelican population if the predation process is delayed by a considerable amount of time. Numerical simulations for a hypothetical set of parameter values are presented to illustrate the analytical findings.

Keywords: Avian botulism, susceptible Tilapia, infected Tilapia, Pelican, time delay, Hopf-bifurcation.

1. INTRODUCTION

The Salton Sea has become a dangerous habitat of wild migratory birds. Each year, millions of birds are paralyzed or they die after exposure to a toxin produced by the botulism bacterium. Avian botulism is most likely to occur due to elevated salinity of the water, accelerated eutrophication, algal blooms, reduced dissolved oxygen and dramatic water quality fluctuation. In the Salton Sea, the level of dissolved salt is around 43 ppt (parts per thousand) whereas the normal salinity of the sea water is around 35 ppt. Colorado River while travelling through Imperial and Coachella Valley picks up salt and nutrient and eventually drains into the Salton Sea. Moreover, the Salton Sea has no outlet and hence the salt and nutrient remain in the lake, continue to increase year after year, causing massive algal blooms in the Sea. These algae die almost as quickly as they grow. When they die, oxygen is pulled from the sea water to help the algae decay causing oxygen depletion in the Sea water. This usually happens

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during the late summer when there is little dissolved oxygen in the water, which is a suitable medium for the botulism bacteria to grow and produce toxin [1, 2].

There are four types of sport fish in the Salton Sea, namely, Tilapia (*Oreochromis mossambicus*), Corvina (*Cynoscion xanthulus*), Croaker (*Bairdiella icistius*) and Sargo (*Anisopremus davidsoni*). Out of these four types of fish, Tilapia is the most abundant in the Salton Sea, probably because of its stunning reproduction rate. It is well known that Tilapia is infected by a vivrio class of bacteria, which is very common in salt water fish. Due to this vivrio infection, millions of Tilapia die every year. The Tilapia which are infected by the disease develop some oxygen free portions in their body, and these are a good habitat for botulism. As fish affected with vivrio tend to rot from the inside out while it is alive. Frank Shipley, Director of Northwest Biological Science Center in Seattle, remarked that the Tilapia, while dying of vivrio infections, would also harbouring fatal doses of botulism when they are eaten alive by the Pelican. As the fish struggle in its death it tends to rise to the surface of the sea and it becomes more vulnerable as well as attractive to fish-eating birds, like Pelican [3]. Thus, a unique interaction occurs between the Pelican and sick Tilapia with botulism in their tissues and serves as a source for toxication of birds that feed upon them. Also vivrio is passed from one infected fish to another susceptible fish; the more fish that are in the sea, the more chance that a large number of them will become affected by the disease. This causes terrible bird mortality events at the Salton Sea. It has been observed that over 14,000 water birds, mostly white Pelican, died during the summer of 1996. The similar events also happened in 1992 and 1994 when 15,000 and 20,000 Eared Grebs (water birds) died. This imply that infection spreads from fish to Pelican with catastrophic consequences.

Chattopadhyay and Arino [4] proposed a three species eco-epidemiological model, namely, sound prey (susceptible), infected prey (infective), and their predator. Making an assumption on the growth rate of the susceptible prey population, they converted the three-dimensional model to a two-dimensional one and studied the local stability, extinction and Hopf-bifurcation in a two-dimensional system. By applying a Poincare map, they observed the connection between the reduced and the original system. Chattopadhyay and Bairagi [5] proposed and analysed a three-dimensional eco-epidemiological model, consisting of a susceptible fish population, an infected fish population and their predator; the Pelican population. They studied the local stability, global stability and persistence of the system around the positive interior equilibrium. They observed that if the level of the search rate of the predator is low, the system around the positive interior equilibrium is stable. But the instability sets in with the increase of the search rate level of the predator. Sarkar et al. [6] modified the model of Chattopadhyay and Bairagi [5] by introducing an additive colour noise in the infected Tilapia population and studied the dynamical behaviour of the system. They also concluded that for the persistence of the Tilapia and Pelican in the Salton Sea, reduction of the Tilapia population in considerable amount is required and hence a suitable harvesting strategy should be implemented.

Delay models are much more realistic, as in reality time delay occurs in almost every situation. Time delay (discrete or distributed) factor have been incorporated into different biological situations by several authors [7–10]. Generally, delay differential equations exhibit much more complicated dynamics than ordinary differential equations, since the stability of the steady state depends on the delay factor and delay-induced oscillations could occur via instability. It is already mentioned that the massive death of the Pelican population occurs due to predation of infected fish. But the predation process is not instantaneous as there is a time delay between the events of getting the infection and coming closer to the surface of the sea by the infected fish. Hence, to make the model biologicaly more plausible, this time delay factor should be included in the predator response function.

In this work, we first simplify the eco-epidemiological model proposed by Chattopadhyay and Bairagi [5] by making an assumption on the growth rate of susceptible fish as done by Chattopadhyay and Arino [1] and studied the existence and stability around the positive interior equilibrium of the system. Then we introduce a discrete time delay (τ) into the predator response function. Conditions for which the delay induced system enters into Hopf-bifurcation are studied. Numerical solutions for a hypothetical set of parameter values are presented to justify the analytical findings.

The organization of the paper is as follows: Section 2 deals with the basic model and some basic results. In Section 3, we present the delay-induced eco-epidemiological model. The stability analysis of the system for $\tau > 0$ is studied in Section 4. Finally a discussion is presented in Section 5.

2. THE MATHEMATICAL MODEL

2.1. The Basic Ecological Assumptions

We have two populations:

1. The fish, Tilapia, whose population is denoted by $N([N] =$ number of Tilapia per unit designated area).
2.. The bird, whose population is denoted by $P([P] =$ number of birds per unit designated area).

The following assumptions are made for formulating the basic differential equations.

(A1): In the absence of a bacterial infection, the fish population grows according to a logistic fashion with carrying capacity $K(K \in R_+)$, with an intrinsic birth rate constant $r(r \in R_+)$ such that

$$\frac{dN}{dt} = rN\left(1 - \frac{N}{K}\right). \tag{1}$$

(A2): In the presence of a bacterial infection, we assume that the total fish population N is divided into two classes, namely, the susceptible fish population, denoted by S, and the infected fish population, denoted by I. Therefore, at any time t the total number of fish population is

$$N(t) = S(t) + I(t). \tag{2}$$

(A3): We assume that only the susceptible fish population, S, is capable to reproduce itself to logistic law [Equation (1)] and the infective fish population, I, dies before having the capability of reproducing. However, the infective fish, I, still contributes with S to the population growth towards the carrying capacity.

(A4): The mode of disease transmission follows the simple law of mass action. Therefore, the evolution equation for the susceptible fish population, S, according to Equation (1) and assumptions (A3) and (A4), can be written as

$$\frac{dS}{dt} = rS\left(1 - \frac{S+I}{K}\right) - \lambda SI, \tag{3}$$

where $\lambda(\lambda \in R_+)$ is the rate of transmission (or force of infection).

(A5): The disease spreads among the prey population only and is not genetically inherited. The infected population does not recover or become immune. The predator (bird) population preys mostly on infected fish population. Also the predator eats a small fraction $\eta > 0$, of the susceptible fish population. The death rate of infected prey (not due to predation) is denoted by $\mu(\mu \in R_+)$. The natural death rate of predator population is denoted by $e(e \in R_+)$ and the rate of death due to predation of infected prey is denoted by $\psi(\psi \in R_+)$.

2.2. The Basic Mathematical Model

From the above assumptions we can now make the following differential equations:

$$\begin{aligned}
\frac{dS}{dt} &= rS\left(1 - \frac{S+I}{K}\right) - \lambda IS - \frac{\eta m_1 SP}{a_1 + S} \\
\frac{dI}{dt} &= \lambda IS - \frac{mIP}{a+I} - \mu I \\
\frac{dP}{dt} &= \frac{\theta IP}{a+I} + \frac{\eta\theta SP}{a_1 + S} - dP
\end{aligned} \tag{4}$$

as our model. Here $d = e + \psi$ is the total death rate of predator population and m_1, m are the search rates, $\theta(\leq m)$ is the conversion factor and a_1, a are the half saturation coefficients. To account for the stability of the marine ecosystem of multi-species fisheries, Holling type II or Holling type III is more appropriate than that of a crude Lotka-Volterra predational form [11].

Since the prey population is infected by a (lethal) disease, infected preys are weakened and become easier to predate, while susceptible preys easily escape and predation becomes difficult. Considering this fact, we assume in the subsequent part of this paper that $\eta = 0$, that is to say that the predator eats only infected prey, thus Eqaution (4) becomes

$$\begin{aligned} \frac{dS}{dt} &= rS\left(1 - \frac{S+I}{K}\right) - \lambda IS \\ \frac{dI}{dt} &= \lambda IS - \frac{mIP}{a+I} - \mu I \\ \frac{dP}{dt} &= \frac{\theta IP}{a+I} + -dP \end{aligned} \tag{5}$$

Equation (5) has to be analyzed with the following initial conditions:

$$S(0) > 0, I(0) > 0, P(0) > 0.$$

2.3. Some Basic Results

We observe that the right hand-side of Equation (5) is a smooth function of the variables (S, I, P) and the parameters, as long as these quantities are non-negative, so local existence and uniqueness properties hold in the positive quadrant.

From the third equation of Equation (5), it follows that $P = 0$ is an invariant sub set, that is, $P \equiv 0$ if and only if $P(t) = 0$ for some t. Thus $P(t) > 0$ for all t if $P(0) > 0$. The same argument follows for the second equation of Equation (5).

So, either $I = 0$ in which case the first equation of Equation (5) reduces to a pure logistic law verified by S, and P is going to zero asymptotically; or, $I(t) > 0$ for all t. Summing up the first two equations of Equaiton (5), one obtains

$$\frac{d(S+I)}{dt} = rS\left(1 - \frac{S+I}{K}\right) - \frac{mIP}{a+I} - \mu I$$

from which one can see that

$$(S+I)(t_0) \leq K, \Longrightarrow (S+I)(t) \leq K, \; for \; t \geq t_0$$

and $(S+I)(t)$ is asymptotically $\leq K$.

We should notice that positivity of S is not guaranteed and, in fact, if we assume that $S(0) = 0$ and $I(0) > K$ then we have $S(t) < 0$ for $t > 0$ small. This inadequacy is of course entailed by the assumed dependence of the logistic part of the equation upon $S + I$. There is no problem, however, if $S + I \leq K$. One can also correct the problem by putting $(1 - \frac{S+I}{K})^+$ instead of $(1 - \frac{S+I}{K})$. This means that the logistic part of the equation is just counting births and there is no birth if the total population exceeds the carrying capacity.

Throughout the paper, we will assume that $S(0)+I(0)\leq K$. We can also relax the above assumption and allow $S(0)+I(0)$ to exceed K, that is,

$$S(0)+I(0)\leq K+\frac{\mu}{\lambda},\quad I(0)\leq K$$

With these latter conditions on the initial values, we have

$$S(t)+I(t)\leq K+\frac{\mu}{\lambda},\quad I(t)\leq K$$

for all $t\geq 0$.

In this case, we first show that $I(t)\leq K$ for all $t\geq 0$. In fact, if these were not true, and for some $t_0\geq 0$, we have $I(t_0)=K$, we will have at the same time $S(t_0)\leq \frac{\mu}{\lambda}$, therefore, $\frac{dI}{dt(t_0)}\leq 0$.

By Standard argument on invarint subsets, we conclude that: $I(t)$ cannot exceed K. Now this in turn implies that $S(t)\geq 0$ for all $t\geq 0$. So we can allow $S+I$ to exceed K provided that it does not exceed $K+\frac{\mu}{\lambda}$ and $I\leq K$.

Thus we have $0\leq S,I\leq K$. It remains to show that P is ultimately bounded too. Adding together the second equation and $\frac{m}{\theta}$ times the third equation of Equation (5); we obtain

$$\frac{d\left(I+\frac{mP}{\theta}\right)}{dt}=(\lambda S-\mu)I-\frac{dm}{\theta}P\leq \lambda K^2-\min(\mu,d)\left(I+\frac{m}{\theta}P\right),$$

which implies that

$$I(t)+\frac{m}{\theta}P(t)\leq \max\left(\frac{\lambda K^2}{\min(\mu,d)},I(0)+\frac{m}{\theta}P(0)\right).$$

This implies that

$$I(t)+\frac{m}{\theta}P(t)\leq \frac{\lambda K^2}{\min(\mu,d)}\ \textit{for all}\ t\geq 0,$$

if it is true for $t=0$, and

$$\lim_{t\to+\infty}\sup\ \left(I(t)+\frac{m}{\theta}P(t)\right)\leq \frac{\lambda K^2}{\min(\mu,d)}$$

for solutions defined up to $+\infty$, as long as the solution is defined on positive axis. We summarize the above results in in the next proposition.

Proposition 1: Every solution initiating in the positive octant and such that $S(0)+I(0)\leq K$ satisfies the same properties for all $t\geq 0$ as long as it exists. Moreover, the following inequality holds:

$$I(t)+\frac{m}{\theta}P(t)\leq \max\left(\frac{\lambda K^2}{\min(\mu,d)},I(0)+\frac{m}{\theta}P(0)\right)$$

As a consequence, every solution with initial value in R^3 verifying, in addition to the condition $(S+I)(0) \leq K$ can be extended upto $+\infty$.

In Equation (5), the number r represents the growth rate of the fish population. The greater r, the faster the population reaches its carrying capacity. For $r = \infty$, one can consider that $S + I = K$ and Equation (5) reduces to the following two-dimensional system:

$$\frac{dI}{dt} = \lambda I(K - I) - \frac{mIP}{a+I} - \mu I$$
$$\frac{dP}{dt} = \frac{\theta IP}{a+I} - dP \tag{6}$$

The subsequent part of this paper is devoted to the study of Equation (6). The model Equation (6) have the following equilibria on all the coordinate planes, viz, $E_0(0,0)$, $E_1(\frac{\lambda K-\mu}{\lambda}, 0)$, and $E^*(I^*, P^*)$ where $I^* = \frac{da}{\theta - d}$, $P^* = 1/m(a+I^*)\ (\lambda K - \mu - \lambda I^*)$.

Now first equation of Equation (6) can be written as

$$\frac{dI}{dt} = [(\lambda K - \mu) - \lambda I]I - \frac{mIP}{a+I}$$

If $\lambda < \frac{\mu}{K}$ it follows that $I(t) \to 0$ as $t \to \infty$ and so does P. Now one can state the following theorem:

Theorem 1: If $\lambda < \frac{\mu}{K}$, then the trivial equilibrium E_0 is globally asymptotically stable. Hence one may now assume that

$(H_1)\lambda K - \mu > 0$

setting $r_1 = \lambda K - \mu > 0$, $K_1 = K - \frac{\mu}{\lambda}, \alpha = \frac{m}{a}(> 0) \& \beta = \frac{1}{a}(> 0)$, we get,

$$\frac{dI}{dt} = r_1\left(1 - \frac{I}{K_1}\right)I - \frac{\alpha IP}{1+\beta I} \tag{6a}$$
$$\frac{dP}{dt} = \left(\frac{\theta \beta I}{1+\beta I} - d\right)P \tag{6b}$$

From Equation (6a) one can deduce that $\lim_{t\to+\infty} \sup I(t) \leq K_1$. The functional response to predation is being increasing with respect to the prey state variable. When $\frac{\theta\beta K_1}{1+\beta K_1} < d$, that is, $\lambda < \frac{\mu(\theta-d)}{K\theta - Kd - ad}$ (in original parameters) it follows that $P(t) \to 0$ as $t \to \infty$ and $I(t) \to K_1$. Hence one can state the following theorem:

Theorem 2: If (H_1) holds and $\lambda < \frac{\mu(\theta-d)}{K\theta-Kd-ad}$, then E_1 is globally asymptotically stable.

Again we have, $K_1 < \frac{d}{\beta(\theta-d)}$; setting $K_1{}^* = \frac{d}{\beta(\theta-d)}$, one may now assume that $(H_2)\ \theta - d > 0$. It is known [12–14] that if (H_1) and (H_2) hold then one can state the following theorem:

Theorem 3: When $K_1{}^* < K_1 < 2K_1{}^* + \frac{1}{\beta}$ i.e. when $\frac{\mu(\theta-d)}{K\theta-Kd-ad} < \lambda < \frac{\mu(\theta-d)}{K\theta-Kd-ad-a\theta}$ then the predator population controls the prey population at density $I^* = K^*$ and settle at density $P^* = \frac{r_1\theta(K_1-K_1{}^*)}{K_1\alpha(\theta-d)}$. And when $2K_1{}^* + \frac{1}{\beta} < K_1$ that is, when $\lambda > \frac{\mu(\theta-d)}{K\theta-Kd-ad-a\theta}$ the

equilibrium (I^*, P^*) loses its stability and a unique and globally stable periodic solution exists and a Hopf-bifurcation occur at $K_1 = 2K_1^* + \frac{1}{\beta}$ that is, when $\lambda = \frac{\mu(\theta-d)}{K\theta-Kd-ad-a\theta}$

3. The Delay Model

In this section we modify the Equation (6) by incorporating a discrete time delay (τ) into the predator response function. The modified model is given as follows:

$$\begin{aligned}\frac{dI}{dt} &= \lambda I(K-I) - \frac{mI(t-\tau)P}{a+I(t-\tau)} - \mu I,\\ \frac{dP}{dt} &= \frac{\theta I(t-\tau)P}{a+I(t-\tau)} - dP\end{aligned} \tag{7}$$

Equation (7) has to be analyzed with the following initial conditions:

$$I(0) > 0, P(0) > 0.$$

We observe that the right- hand side of Equation (7) is a smooth function of the variables (I, P) and the parameters, as long as these quantities are non-negative, so local existence and uniqueness properties hold in the positive quadrant.

4. LOCAL STABILITY ANALYSIS FOR $\tau > 0$

In this section, we shall study and analyze the local stability of the equilibria of Equation (7) for $\tau > 0$. Let $x(t) = I(t) - I^*$, $y(t) = P(t) - P^*$ are the perturbed variables. After removing the nonlinear terms, we obtain the linear variational system, by using equilibrium conditions, as

$$\begin{aligned}\frac{dx}{dt} &= \left(-\lambda I^* + \frac{mP^*}{a+I^*}\right)x(t) - \frac{mI^*}{a+I^*}y(t) - \frac{maP^*}{(a+I^*)^2}x(t-\tau)\\ \frac{dy}{dt} &= \frac{\theta aP^*}{(a+I^*)^2}x(t-\tau).\end{aligned} \tag{8}$$

The associated characteristic equation $\Delta(\xi, \tau) = 0$ with eigen value ξ can be written as

$$\xi^2 + A_1\xi + A_2 + (B_1\xi + B_2)e^{-\xi\tau} = 0 \tag{9}$$

where

$$\begin{aligned}A_1 &= -\lambda K + 2\lambda I^* + \mu + d - \frac{\theta I^*}{a+I^*},\\ A_2 &= \left(\frac{\theta I^*}{a+I^*} - d\right)(\lambda K - 2\lambda I^* - \mu),\\ B_1 &= \frac{maP^*}{(a+I^*)^2},\end{aligned}$$

$$B_2 = \left(\frac{\theta I^*}{a+I^*} - d\right)\frac{maP^*}{(a+I^*)^2} + \frac{m\theta aI^*P^*}{(a+I^*)^3}. \tag{10}$$

For the zero equilibrium, $E_0(0,0)$, $A_1 = -\lambda K + \mu + d$, $A_2 = -d(\lambda K - \mu)$, $B_1 = 0$, $B_2 = 0$. Therefore, the characteristic Equation (9) becomes

$$\xi^2 + (\mu + d - \lambda K)\xi - d(\lambda K - \mu) = 0.$$

Hence $\xi_1 = \lambda K - \mu$ and $\xi_2 = -d(< 0)$ and then E_0 is stable if $\lambda < \frac{\mu}{K}$ and unstable if $\lambda > \frac{\mu}{K}$. It is to be noted that if E_0 is stable then E_1 and E^* do not exist.

For the axial equilibrium $E_1(\frac{\lambda K-\mu}{\lambda}, 0)$, $A_1 = (\lambda K - \mu) + d - \frac{\theta(\lambda K-\mu)}{\lambda a+\lambda K-\mu}$, $A_2 = (\lambda K - \mu)[d - \frac{\theta(\lambda K-\mu)}{\lambda a+\lambda K-\mu}]$, $B_1 = 0$, $B_2 = 0$. Hence the characteristic Equation (9) becomes

$$\xi^2 - \left[(\lambda K - \mu) + d - \frac{\theta(\lambda K - \mu)}{\lambda a + \lambda K - \mu}\right]\xi + (\lambda K - \mu)\left[d - \frac{\theta(\lambda K - \mu)}{\lambda a + \lambda K - \mu}\right] = 0.$$

Therefore $\xi_1 = \mu - \lambda K\,(< 0)$ and $\xi_2 = \frac{\theta(\lambda K-\mu)-d(\lambda a+\lambda K-\mu)}{\lambda a+(\lambda K-\mu)}$.

Thus E_1 is locally stable if $\lambda < \frac{\mu(\theta-d)}{K\theta-Kd-ad}$ and $\theta > d + \frac{ad}{K}$. It is interesting to see that in this case also if E^* exists then E_1 is unstable saddle.

Remark 1: It is to be noted that the delay factor in the Equation (7) has no effect on the existence and stability properties of the zero and axial equilibria.

We now investigate the local asymptotic stability of the positive interior equilibrium E^*. The associated characteristic equation is given by

$$\Delta(\xi, \tau) = \xi^2 + A\xi + (B_1\xi + B_2)e^{-\xi\tau} = 0. \tag{11}$$

Where

$$\begin{aligned} A &= \lambda I^* - \frac{mP^*}{a+I^*} \\ B_1 &= \frac{maP^*}{(a+I^*)^2} \\ B_2 &= \frac{m\theta aP^*I^*}{(a+I^*)^3}. \end{aligned} \tag{12}$$

Equation (11) can be written as

$$\Delta(\xi, \tau) = P_1(\xi) + Q_1(\xi)\,e^{-\xi\tau} = 0 \tag{13}$$

where

$$\begin{aligned} P_1(\xi) &= \xi^2 + A\xi \\ Q_1(\xi) &= B_1\xi + B_2. \end{aligned} \tag{14}$$

Lemma 1: There exists a unique pair of ω_0, τ_0 with ω_0, $\tau_0 \geq 0$, $\omega_0\tau_0 < 2\pi$ such that $\Delta(i\omega_0, \tau_0) = 0$ if the condition $\lambda < \gamma$ holds (a sufficient condition).

Proof: We note that $\Delta(0,\tau) \neq 0$.
Now consider $|P_1(i\omega)|^2 - |Q_1(i\omega)|^2$ for $\omega \in R$, we have

$$|P_1(i\omega)|^2 - |Q_1(i\omega)|^2 = \omega^4 + k_1\omega^2 + k_2 \tag{15}$$

where

$$k_1 = A^2 - {B_1}^2 \tag{16}$$

and

$$k_2 = -{B_2}^2 (< 0) \tag{17}$$

where A, B_1 and B_2 are defined in Equation (12). Now $k_1 > 0$ if $\lambda < \gamma$ (for details see Appendix), where

$$\gamma = \frac{\mu(\theta - d)(2\theta - d)}{(K\theta - Kd - ad)(2\theta - d) - a\theta d}. \tag{18}$$

Let

$$|P_1(i\omega)|^2 - |Q_1(i\omega)|^2 = \nu^2 + k_1\nu + k_2 = 0 \tag{19}$$

has a unique positive root

$$\nu_0 = \frac{1}{2}(-k_1 + \sqrt{{k_1}^2 - 4k_2}) > 0. \tag{20}$$

Consequently $|P_1(i\omega)|^2 - |Q_1(i\omega)|^2 = 0, \omega \in R$ if $\omega = \pm\omega_0, \omega_0 = \sqrt{\nu_0} > 0$. Therefore, $|P_1(i\omega)|^2 - |Q_1(i\omega)|^2 = 0$ implies that there is a unique $\tau_0 \geq 0$ such that $\omega_0\tau_0 < 2\pi$ and

$$\Delta(i\omega_0, \tau_0) = P_1(i\omega_0) + Q_1(i\omega_0)e^{-i\omega_0\tau_0} = 0. \tag{21}$$

Moreover, it is to be noted that the critical value of τ_0 can be calculated from ω_0 after computing ν_0 from Equation (20).

From Equation (18) and Lemma 1 we see that

$$\Delta(i\omega, \tau) = 0, \omega \in R, \tau \geq 0$$

if $\omega = \pm\omega_0, \tau = \tau_n = \tau_0 + \frac{2\pi n}{\omega_0}, n = 0, 1, 2, \ldots\ldots$ where $\omega_0 > 0$ and $\tau_0 \geq 0$ as is defined in Lemma 1.

Lemma 2:

$$Re\left[i\omega_0 \frac{\partial\overline{\Delta}(i\omega_0, \tau_n)}{\partial\xi} Q_1(i\omega_0)e^{-i\omega_0\tau_n}\right] > 0, n = 1, 2, \ldots\ldots \tag{22}$$

Proof: Now,

$$i\omega_0 \frac{\partial \overline{\Delta}(i\omega_0, \tau_n)}{\partial \xi} Q_1(i\omega_0) e^{-i\omega_0\tau_n} = i\omega_0(-2i\omega_0 + A + B_1 e^{i\omega_0\tau_n} - \tau_n \overline{Q_1}(i\omega_0) e^{i\omega_0\tau_n}) Q_1(i\omega_0) e^{-i\omega_0\tau_n}$$
$$= -2(\omega_0)^2 P_1(i\omega_0) - i\omega_0 A P_1(i\omega_0) + i\omega_0 B_1 Q_1(i\omega_0) - i\omega_0\tau_n |Q_1|^2$$
$$= (2{\omega_0}^4 + A^2\omega_0^2 - \omega_0^2 {B_1}^2 + i({\omega_0}^3 A + \omega_0 B_1 B_2 - 2{\omega_0}^2 A - \omega_0\tau_n |Q_1|^2).$$

Therefore,

$$Re\left[i\omega_0 \frac{\partial \overline{\Delta}(i\omega_0, \tau_n)}{\partial \xi} Q_1(i\omega_0) e^{-i\omega_0\tau_n} \right]$$
$$= {\omega_0}^2 (2{\omega_0}^2 + A^2 - B_1^2)$$
$$= \nu_0 \sqrt{{k_1}^2 - 4k_2} > 0$$

Hence the lemma:

Lemma 3: If $\lambda < \gamma$ and $\omega_0, \tau_n,\ n = 0, 1, 2, \ldots$ be defined as Lemma 1, then for each τ_n there exists a neighbourhood $I_n \subset R$ of τ_n and a continuously differentiable function $\lambda_n : I_n \to C$ such that

(i) $\xi_n(\tau_n) = i\omega_0$.
(ii) $\Delta(\xi_n(\tau), \tau) = 0, \tau \in I_n$.
(iii) $Re\left[\frac{\partial \xi_n(\tau)}{\partial \tau}|_{\tau=\tau_n}\right] > 0$.

Proof: It is clear from Lemma 1 that $\frac{\partial \Delta(i\omega_0, \tau_n)}{\partial \xi} \neq 0$, (for n = 0, 1, 2,...). From the implicit function theorem there exist a neighbourhood I_n and a continuously differentiable function ξ_n which satisfies the condition (i) and (ii) of Lemma 3.
Now differentiating condition (ii), we have

$$\frac{\partial \Delta(i\omega_0, \tau_n)}{\partial \xi} \frac{\partial \xi_n}{\partial \tau} - i\omega_0 Q_1(i\omega_0) e^{-i\omega_0\tau_n} = 0.$$

Therefore it follows from Lemma 1 and Lemma 2 that

$$Re\left(\frac{\partial \xi_n(\tau_n)}{\partial \tau}\right) = Re\left[\frac{1}{\left(\frac{\partial \Delta(i\omega_0, \tau_n)}{\partial \xi}\right)^2} \left\{ i\omega_0 \frac{\partial \overline{\Delta}(i\omega_0, \tau_n)}{\partial \xi} Q_1(i\omega_0) e^{-i\omega_0\tau_n} \right\} \right]$$
$$= \frac{1}{\left(\frac{\partial \Delta(i\omega_0, \tau_n)}{\partial \xi}\right)^2} Re\left[\left\{ i\omega_0 \frac{\partial \overline{\Delta}(i\omega_0, \tau_n)}{\partial \xi} Q_1(i\omega_0) e^{-i\omega_0\tau_n} \right\} \right] > 0.$$

Next we state the following theorem due to Cooke and Van den Driessche [15] as modified by Boese [9].

Theorem 4: If $P_1(\xi)$ and $Q_1(\xi)$ are analytic functions in $Re\xi > 0$ and satisfy the following conditions:

(i) $P_1(\xi)$ and $Q_1(\xi)$ have no common imaginary root;
(ii) $\overline{P_1}(-iy) = P_1(iy), \overline{Q_1}(-iy) = Q_1(iy)$ for real y;
(iii) $P_1(0) + Q_1(0) \neq 0$;
(iv) $lim\ sup\{Q_1(\xi)/P_1(\xi)| : |\xi| \to \infty, Re\xi \geq 0\} < 1$;

$F(y) \equiv |P_1(iy)|^2 - |Q_1(iy)|^2$ for real y has atmost a finite number of real zeros.

Then the following statements are true:

(a) If $F(y) = 0$ has no positive roots, then no stability switch may occur.
(b) If $F(y) = 0$ has at least one positive root and each of them is simple, then as τ increases, a finite number of stability switches may occur, and eventually the considered equation becomes unstable.

By applying Lemma 1, Lemma 3 and the above theorem, we finally conclude:

Theorem 5: For $\lambda < \gamma$, the equilibrium (I^*, P^*) is locally asymptotically stable if $0 \leq \tau < \tau_0$ and unstable if $\tau > \tau_0$ where τ_0 is defined in Lemma 1.

Remark 2: As τ passes through the value τ_0, the equilibrium (I^*, P^*) losses its stability and Hopf bifurcation occurs with emergence of a small amplitude periodic oscillations.

Remark 3: The interior equilibrium E^* is locally asymptotically stable in the range $0 \leq \tau < \tau_0$ for $\lambda < \gamma$. It is interesting to note that the value of λ in delay induced system is smaller than that of the upper value of λ in non-delayed system, which shows that delay has destabilizing effect on the system around the interior equilibrium.

5. DISCUSSION

In this paper, we have modified the model of Chattopadhyay and Bairagi [5] with an assumption that the growth rate of susceptible fish is very high. Since a time delay factor is always present in between the infection and predation process, we have incorporated this into the predator response function of the modified model and analyse this delay-induced eco-epidemiological model.

We have observed that the time delay factor has no effect on the existence and stability behaviour of the system around the trivial and axial equilibria. However, incorporation of delay factor in the predator response function drives the otherwise stable system into Hopf bifurcation, and small amplitude periodic solutions occur around the non-zero equilibrium point, when the delay factor τ reaches its critical

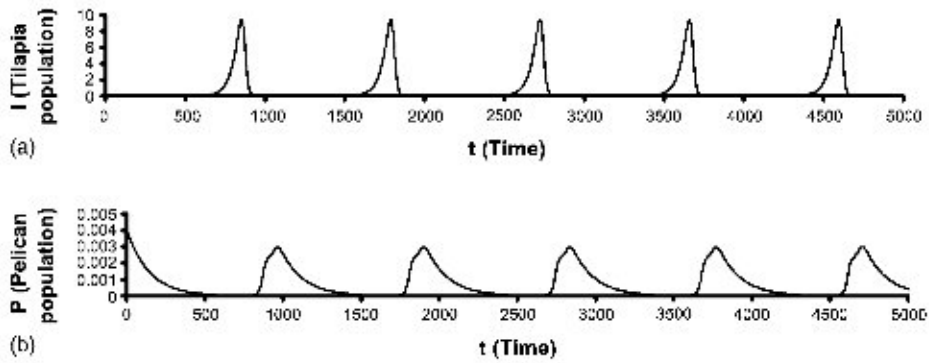

Fig. 1. (a) Stable periodic solutions of Tilapia population for $\tau = 88$ and $\lambda = 0.006$, for other parameters see main text. (b) Stable periodic solutions of Pelican population for $\tau = 88$ and $\lambda = 0.006$. For other parameters, see main text.

value $\tau = \tau_0$. To illustrate our analytical findings, numerical studies have been performed, based on the following hypothetical set of parameter values: $K = 400$, $m = 14.5/day, a = 0.004$ *tonnes*, $\theta = 0.8/day$, $\mu = 0.0019/day, d = 0.7/$ *day*. It has been observed that when $\lambda = 0.006$ and $\tau < 88(h)$ the system becomes stable (with decaying oscillation) and for $\tau > 88$ the system becomes unstable (with growing oscillation). Further for $\tau = 88$ we observe that the system exhibits stable limit cycle with the emergence of small amplitude periodic solutions [see Fig. 1(a) and 1(b)]. The growing oscillations (unstable situation) indicate both the population will go to extinction in the long run. These findings may be related to the comments of Horvitz on the problem of the Salton Sea [2]. He remarked that thousands of birds depend upon the fish for food and 380 species of birds have been counted in the Sea, which is nearly half of the total species known to exist in the United States. As the salinity of the sea is gradually increasing, there will be massive algal blooms causing oxygen depletion in the Sea water due to decomposition of algae. Consequently, the Salton Sea is becoming a suitable medium for the botulism bacteria to grow and produce toxin and in future, there will be no more fish in the Sea and naturally many of the birds that use this Sea will no longer be able to survive there. Thus our observations for the extinction of the species (due to growing oscillation) are to some extent similar to that of Horvitz (due to enhancement of salinity, eutrophication, etc.). Again if $\tau = 0$ and the value of λ is increased from 0.006 to 0.06, keeping other parameters fixed, the system becomes locally asymptotically stable around the interior equilibrium [see Fig. 2(a) and 2(b)] but in the presence of delay for this value of λ we observe that the system becomes unstable for sufficient lower value of τ. Thus, both the analytical and numerical studies confirm that the delay in predator response function has a destabilizing effect on the system. In other words, the non-delayed system can tolerate more infection than that of the delay-induced system. These findings are realistic from

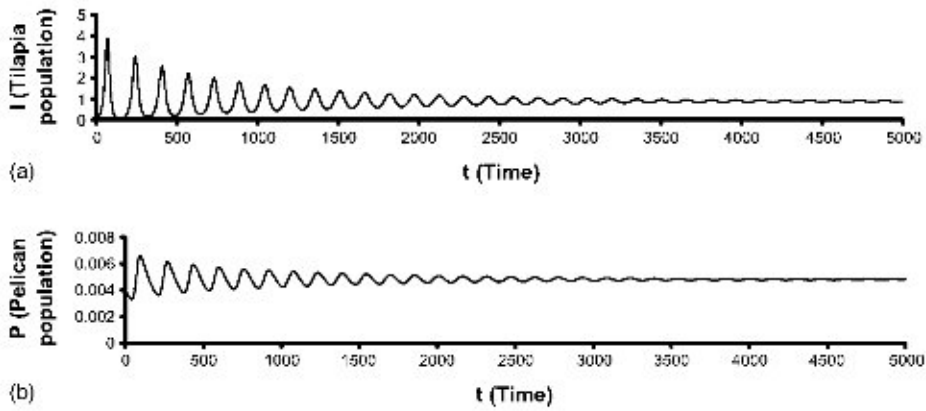

Fig. 2. (a) Asymptotically stable solutions of Tilapia population for $\tau = 0$ and $\lambda = 0.06$, for other parameters see main text. (b) Asymptotically stable solutions of Pelican population for $\tau = 0$ and $\lambda = 0.06$, for other parameters see main text.

a biological point of view as vivrio is passed from one infected fish to another susceptible fish; the more infected Tilapia is in the sea the more chance of the susceptible Tilapia to be infected in a large scale by the disease and instability conditions sets in. Thus, if the infected fishes are allowed to spread infection by means of introduction of delay in the predation term, there is a high possibility of an epidemic outbreak in the fish population and eventually in the Pelican population. In other words, the chances of stability of the system around the positive interior equilibrium will increase by decreasing τ. Thus, control of the disease may be achieved by controlling the value of λ and τ. Therefore, for sustaining Tilapia and Pelican in the Sea, the infected fishes must be reduced as early as possible. We may finally conclude that for sustainability of Tilapia and Pelican from explosive epidemic in the Salton Sea, reduction of the Tilapia population (specially infected Tilapia) in a considerable amount is essential and hence a suitable harvesting policy should be implemented. Steve Horvitz pointed out in his report *Salton sea 101* that for prevention of Salton Sea the number of the Tilapia population should be reduced by thousand of tons of fish each year. These findings are also supported by Gonzalez et al. [1] from experimental view point and by Chattopadhyay and Bairagi [5] from modelling point of view.

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APPENDIX

Calculation for $\lambda < \gamma$ in Lemma 1.

We observe that, if $A + B_1$ and $A - B_1$ are both positive then $K_1 > 0$. Now

$$A + B_1 = \lambda I^* - \frac{mP^*}{a + I^*} + \frac{maP^*}{(a + I^*)^2} = I^* \frac{[\lambda(a + I^*)^2 - mP^*]}{(a + I^*)^2}.$$

Again, if $\lambda > \frac{mP^*}{(a+I^*)^2}$ then $(A + B_1) > 0$ and in system parameters this condition can be written as $\lambda < \frac{\mu(\theta-d)}{(K\theta-Kd-ad-a\theta)}$.

Similarly,

$$A - B_1 = \lambda I^* - \frac{mP^*}{a + I^*} - \frac{maP^*}{(a + I^*)^2} = \frac{\lambda I^*(a + I^*)^2 - mP^*(2a + I^*)}{(a + I^*)^2}.$$

So, $A - B_1 > 0$ if $\lambda < \frac{\mu(\theta-d)(2\theta-d)}{(K\theta-Kd-ad-a\theta)}$.
Therefore, $K_1 > 0$ if

$$\lambda < \min\left[\frac{\mu(\theta - d)}{(K\theta - Kd - ad - a\theta)}, \frac{\mu(\theta - d)(2\theta - d)}{(K\theta - Kd - ad - a\theta)}\right]$$

That is if

$$\lambda < \frac{\mu(\theta - d)(2\theta - d)}{(K\theta - Kd - ad - a\theta)} = \gamma(say).$$