ECOLOGICAL MODELLING

Ecological Modelling 167 (2003) 199–211

Pelicans at risk in Salton Sea—an eco-epidemiological model-II

J. Chattopadhyay [a,*], P.D.N. Srinivasu [b], N. Bairagi [c]

[a] *Embryology Research Unit, Indian Statistical Institute, 203 B.T. Road, Calcutta 700035, India*
[b] *Department of Mathematics, Andhra University, Visakhapatnam 530003, India*
[c] *Jadavpur University, Kolkata 700032, India*

Received 12 June 2002; received in revised form 18 February 2003; accepted 7 April 2003

Abstract

The Salton Sea which is located in the Southeast desert of California is becoming a dangerous habitat for birds. It is supposed that elevated salinity, accelerated eutrophication, blooms of Avian botulism and dramatic water quality fluctuation are the key factors for massive die-off of Tilapia (prey) and Pelican (predator) in the Salton sea. Chattopadhyay and Bairagi [Ecol. Model. 136 (2001) 103] proposed and analyzed a three-component eco-epidemiological model consisting of susceptible fish population, infected fish population and their predator, the Pelican population. We modify their model from more biologically realistic point of view and then analyze it. The main objective of the work is to find out conditions for which the modified system becomes disease free. Numerical simulations for a hypothetical set of parameter values are presented to illustrate the analytical findings. It is observed that if the initial value of the system is contained in the invariant set which contain the disease-free equilibrium, the solution will approach the disease-free equilibrium under suitable parametric conditions. If the initial value of the system is not in the invariant set, impulsive harvesting strategies can be used to change the initial state to the desired disease-free equilibrium state.

Keywords: Avian botulism; Susceptible Tilapia; Infected Tilapia; Pelican; Local stability

1. Introduction

The Salton Sea has become a dangerous habitat of wild migratory birds. Each year millions of birds are paralyzed or 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 it grow. When it dies oxygen is pulled from the sea water to help the algae decay causing oxygen depletion in the Sea water. This usually happens during the late summer when there is little dissolved oxygen in the water and becomes a suitable medium for botulism bacteria to grow and produce toxin (for details, see

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 off every year. The Tilapia which are infected by the disease develop some oxygen-free portions in their body, making good habitat for botulism. As fish affected with vivrio tend to rot from the inside out while they are alive. Frank Shipley, Director of Northwest Biological Science Center in Seattle, remarked that the Tilapia while dying of vivrio infections, would also be harboring fatal doses of botulism when they were eaten alive by the Pelican. As the fish struggle in their death they tend to rise to the surface of the sea and become more vulnerable as well as attractive to fish-eating birds, like Pelican. Thus, a unique interaction occurs between Pelican and sick Tilapia having 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.

Chattopadhyay and Bairagi (2001) proposed and analyzed a three dimensional eco-epidemiological model consisting of susceptible fish population, infected fish population and their predator the Pelican population. Where they assumed that the predator population preys infected fish population only. 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 predator is low, the system around the positive interior equilibrium is stable. But the instability sets in with the increase of search rate level of predator. Sarkar et al. (2001) modified the model of Chattopadhyay and Bairagi (2001) by introducing an additive color noise in the infected Tilapia population and studied the dynamical behavior of the system. They also concluded that for the persistence of Tilapia and Pelican in the Salton Sea, reduction of Tilapia population in considerable amount is required and hence a suitable harvesting strategy should be implemented.

In this paper, we first modify the eco-epidemiological model proposed by Chattopadhyay and Bairagi (2001). We assume that Pelican feeds not only on infected fish but on susceptible fish also. Feeding on infected fish enhances the death rate of Pelican and is considered to contribute negative growth where as feeding on susceptible fish enhances their growth rate and is considered to contribute positive growth. We believe that above assumptions make the model biologically more realistic. One of the basic goals for the study of eco-epidemiological model is to find conditions for which the system becomes disease free. So we have tried to establish conditions for which the modified system eventually becomes disease free. Numerical solutions for a hypothetical set of parameter values are presented to illustrate the analytical findings.

The organization of the paper is as follows: Motivation of the present study has been given in Section 2, Section 3 deals with the basic model and some basic results. Local stability analysis of the system around various equilibrium points are studied in Section 4, the geometric configuration of the model has been discussed in Section 5 and finally a discussion in Section 6.

2. Motivation of the present study

Ecology and epidemiology are major fields of study in their own right. But there are some common features between these two fields. Unfortunately, little attention has been paid so far to see the effect of epidemiological parameters in ecological systems. To the best of our knowledge, Hadeler and Freedman (1989) were the first who described a predator–prey model where the prey is infected by a parasite, and the prey in turn infects the predator with the parasite. They dealt with two different, though closely related problems, persistence of a parasite in a given prey–predator system and parasite mediated coexistence of prey and predator. But the paper was mainly theoretical and did not address any specific situation. Beltrami and Carroll (1994) observed the roll of

viral disease in recurrent phytoplankton blooms by proposing a three species model consisting of susceptible phytoplankton, infected phytoplankton and their grazer. They showed that introduction of virus contaminated cells, even in very small numbers, has the effect of destabilizing. They also observed that the model displays aperiodic or even chaotic fluctuation in bloom outbreaks levels from year to year that mimics the recurrent pattern seen for Noctiluca scitillans in a remarkable data set of cell counts over a consecutive twenty-one years span. Venturino (1995) proposed a two-dimensional prey–predator model and studied how the presence of the disease among the prey affects the behavior of the model. They concluded that under suitable assumptions the disease acts as a control for the system. Chattopadhyay and Arino (1999) 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 populations, 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. Xiao and Chen (2001) used the model of Chattopadhyay and Arino. They considered only the susceptible prey population is capable of reproducing. Incidence and interaction rates between prey and predator follow the simple law of mass action and the predator mainly eats the infected prey. They also assumed the reproduction of predator after predating the prey would not be instantaneous, but mediated by some discrete time lag required for gestation of period. They conclude for large time delay the otherwise unstable positive equilibrium can be stable again. Chattopadhyay and Pal (2002) considered a model by merging the model of Venturino (1995) and Beltrami and Carroll (1994). They observed that there is a possibility for the coexistence of the system when the contact rate follows the law of mass action. This observation is similar to Venturino. But if the contact rate follows the law of standard incidence a minute amount of infection can destabilize the system. This result is similar to Beltrami and Carroll (1994). They concluded that the behavior of such systems is very much model dependent and progresses of this burning issue depends on the responsibility of the researchers.

Use of viruses as biological control agents has not been tested in the field or in the laboratory due to insufficient information on viral-host interactions. Environmental concerns by the public and regulatory agencies presently limit the potential application of the control method. Reviewing all such studies, we may conclude in this stage, it is quite impossible to draw a general conclusion whether the virus/parasite acts as a biological control. As such we feel that researchers should concentrate on how the overall system can be made disease free. In this study we have tried to establish conditions for which the considered prey–predator system with disease in the prey species will eventually become disease free. We have observed that to achieve this goal suitable impulsive harvesting strategies may play a crucial role (see Sections 5 and 6 for details). We do not want to claim that our model is the best one, but somehow is nearer to the reality.

3. The basic assumptions and the mathematical model

We have two populations:

1. Tilapia fish whose population is denoted by N([N]: number of Tilapia per unit designated area).
2. Pelican 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.

Assumption 1. In the absence of 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

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

Assumption 2. In the presence of bacterial infection we assume that the total fish population N is divided into two classes, namely, susceptible fish population, denoted by S, and infected fish population, denoted by I. Therefore, at any time t the total number of fish population is

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

Assumption 3. We assume that only susceptible fish population, S, is capable of reproducing with logistic law (Eq. (1)) and the infective fish population, I, does not reproduce. However, the infective fish, I, still contributes with S to population growth towards the carrying capacity.

Assumption 4. The mode of disease transmission follows the simple law of mass action. Therefore, the evolution equation for the susceptible fish population, S, according to Eq. (1) and Assumptions (3) and (4), 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).

Assumption 5. The disease is spread among the prey population only and the disease is not genetically inherited. The infected population does not recover or become immune. Since prey population are infected by a (lethal) disease: infected preys are weakened and become easier to catch. Also they are present in the Salton Sea in considerable number, so we assume that predator's functional response to the infective prey follows simple law of much action and included in the predator's growth equation with a negative sign. While susceptible preys easily escape and predation becomes difficult, so we assume that predator's functional response to the susceptible prey follows Holling type II predation form which is included in the predator's growth equation with a positive sign (Holling, 1965). From the above assumptions, we can write down the following modified set of differential equations as our model:

$$\begin{aligned}
\frac{dS}{dt} &= rS\left(1 - \frac{S+I}{K}\right) - \lambda IS - \frac{\theta_1 SP}{a+S},\\
\frac{dI}{dt} &= \lambda IS - m_1 IP - \mu I,\\
\frac{dP}{dt} &= \frac{\theta_2 SP}{a+S} - m_2 IP - \delta P,
\end{aligned} \tag{4}$$

where θ_1, m_1 are the search rates, $\theta_2(\leq\theta_1)$ represents the conversion factor, m_2 represents the death rate of the predator per unit consumption of the infected fish, a is the half saturation constant, μ and δ are the natural death rates of infected prey population and predator population, respectively.

We observe that the right hand-side of Eq. (4) is a smooth function of the variables (S, I, P) and the parameters, as long as these quantities are non-negative, so local existence, uniqueness and continuation properties hold in the positive octant.

Lemma 1. *All the solutions of (4) which initiate in R^3_+ are uniformly bounded.*

Proof. We define a function

$$\omega = S + I + \frac{\theta_1}{\theta_2}P. \tag{5}$$

The time derivative of (5) along the solutions of (4) is

$$\begin{aligned}
\frac{d\omega}{dt} &= rS\left(1 - \frac{S+I}{K}\right) - m_1 IP - \mu I\\
&\quad - \frac{\theta_1 m_2}{\theta_2}IP - \frac{\theta_1 \delta P}{\theta_2}.
\end{aligned} \tag{6}$$

For each $\eta > 0$, the following inequality holds:

$$\begin{aligned}
\frac{d\omega}{dt} + \eta\omega &\leq \left(r\left(1 - \frac{S}{K}\right) + \eta\right)S + (\eta - \mu)I\\
&\quad + (\eta - \delta)\frac{\theta_1}{\theta_2}P \leq K\frac{(r+\eta)^2}{4r}\\
&\quad + (\eta - \mu)I + (\eta - \delta)\frac{\theta_1 P}{\theta_2}.
\end{aligned}$$

Now if we take $\eta < \min(\mu, \delta)$ then the right hand side of (6) is bounded. Then we can find a constant $l > 0$ (say), such that

$$\frac{d\omega}{dt} + \eta\omega < l.$$

Applying a theory of differential inequality (Birkhoff and Rota, 1982), we obtain

$$\begin{aligned}
0 < \omega(S, I, P) &< \frac{l}{\eta}(1 - e^{-\eta t})\\
&\quad + \omega(S(0), I(0), P(0))e^{-\eta t},
\end{aligned}$$

and for $t \to \infty$, we have

$$0 < \omega < \frac{l}{\eta}. \tag{7}$$

Hence, all the solutions $(S(t), I(t), P(t))$ of (4) that initiate at $(S(0), I(0), P(0)) \in R^3_+$ are confined in the region

$$B = \{(S, I, P) \in R^3_+ : \omega = \frac{l}{\eta} + \zeta, \quad \text{for any } \zeta > 0\}, \tag{8}$$

for all $t \geq T$, where T depends on the initial values $(S(0), I(0), P(0))$. Thus, the set B is an invariant set which contains the Ω-limit set of all the paths of the system (4) that initiate in the positive octant. □

4. Existence of equilibria and variational analysis

The following observations are necessary before we investigate the existence and nature of admissible equilibrium of system (4). As mentioned in Section 3, in the absence of the predator and infection in the ecosystem, the susceptible population follows the logistic growth with carrying capacity K. Hence, from Lakshmikantham and Leela (1969), we have $0 < S(t) \leq K$ for the system (4) and without loss of generality we assume that $S(0) \leq K$. Thus, the infected population $I(t)$ decreases to 0 for any initial value $I(0) > 0$ if

$$\lambda K - \mu < 0. \tag{9}$$

Similarly the predator population also approaches 0 if

$$\frac{\theta_2 K}{a + K} - \delta < 0. \tag{10}$$

Thus, we observe that if the parameters of the system (4) satisfy the condition (9) then the infection automatically gets washed away from the ecosystem and only the susceptible fish population and the predator survive in the environment eventually. Similarly if the parameters satisfy the relation (10), the predator population goes to extinction leaving only the susceptible and infected fish population in the ecosystem eventually. Now we shall examine the dynamics of the system restricted to the boundary planes of the domain of the system. That is we wish to study the dynamics of the state when the considered ecosystem is free from one of the species, viz. susceptible, infective or predator. This study reveals the nature and dynamics of the paths of the system (4) if the initial value of one of the components of the state (i.e. $S(0)$, $I(0)$ or $P(0)$) is zero. Considering the ecosystem which is free from infection we have,

$$\frac{\mathrm{d}S}{\mathrm{d}t} = rS\left(1 - \frac{S}{K}\right) - \frac{\theta_1 SP}{a + S}, \quad \frac{\mathrm{d}P}{\mathrm{d}t} = \frac{\theta_2 SP}{a + S} - \delta P.$$

This system admits three equilibrium points, viz. (0, 0), $(K, 0)$ and an interior equilibrium point $(\delta a/(\theta_2 - \delta))$, $(r/\theta_1 K)(K - (\delta a/(\theta_2 - \delta))(a + \delta a/(\theta_2 - \delta)))$ in the SP-plane. Here points (0, 0) and $(K, 0)$ are saddles and the interior equilibrium point may be globally stable or this system may admit a unique stable limit cycle around the interior equilibrium point in the interior of the SP-plane (Kuang and Freedman, 1998; Srinivasu et al., 2001).

Now considering the dynamics of the ecosystem in the absence of the predator we have

$$\frac{\mathrm{d}S}{\mathrm{d}t} = rS\left(1 - \frac{S + I}{K}\right) - \lambda IS, \quad \frac{\mathrm{d}I}{\mathrm{d}t} = \lambda IS - \mu I.$$

This system also admits three equilibrium points with (0, 0) and $(K, 0)$ on the boundary and (μ/λ), $r(K - (\mu/\lambda)/(r + \lambda K))$ in the interior of the SI-plane. It can be verified that the interior equilibrium point is globally stable while the other two boundary equilibrium points are saddles.

The system (4) restricted to PI-plane is given by

$$\frac{\mathrm{d}I}{\mathrm{d}t} = -m_1 IP - \mu I, \quad \frac{\mathrm{d}P}{\mathrm{d}t} = -m_2 IP - \delta P.$$

Here it is clear that (0, 0) is the only asymptotically stable equilibrium point. And all paths initiating in the PI-plane will approach (0, 0).

Now we investigate the existence of equilibrium points admitted by the system (4) in the positive octant R^3_+. $E_0(0, 0, 0)$ is the trivial equilibrium, $E_1(K, 0, 0)$ is the axial equilibrium, $E_2(\mu/\lambda, r(\lambda K - \mu)/\lambda(r + \lambda K), 0)$ and $E_3(\bar{S}, 0, \bar{P})$ where $\bar{S} = \delta a/(\theta_2 - \delta)$, and $\bar{P} = (r/\theta_1 K)(K - \delta a/(\theta_2 - \delta))(a + \delta a/(\theta_2 - \delta))$ are the boundary equilibria. A unique interior equilibrium is $E^*(S^*, I^*, P^*)$ where

$$I^* = \frac{1}{m_2}\left(\frac{\theta_2 S^*}{a + S^*} - \delta\right), \quad P^* = \frac{\lambda S^* - \mu}{m_1},$$

and S^* is the positive root of

$$rm_2S^{*2} - \left[m_2Kr - am_2r - \theta_2 r + \delta r - K\lambda\theta_2 + \delta K\lambda - \frac{\theta_1 m_2 K\lambda}{m_1}\right]S^* - \left(m_2Kra + \delta ra + \delta aK\lambda + \frac{\theta_1 m_2 K\mu}{m_1}\right) = 0, \quad (11)$$

and must satisfy the inequality

$$S^* + \frac{1}{m_2}\left(\frac{\theta_2 S^*}{a+S^*} - \delta\right) \le K. \quad (12)$$

The condition (12) is due to the fact that $S(t)+I(t) \le K$ for all $t > 0$.

We see that equilibria E_0, E_1 always exist. Also observe that the existence of the interior equilibrium point E^* guarantees the existence of E_2 and E_3. Existence of E_2 or E_3 or both implies the instability of E_1. These facts can be observed by studying the variational matrices of the system evaluated at each of the above equilibrium points.

It is to be noted here that if $K = \mu/\lambda$ and $K = \delta a/(\theta_2 - \delta)$ then E_2 and E_3 will approach E_1 which biologically means eventually eradication of infected Tilapia and Pelican population. But to preserve the ecosystem we require the stability of E_3 which biologically means coexistence of susceptible fish population and Pelican population. So we concentrate more on the stabilization of the disease-free equilibrium E_3.

The variational matrix of system (4) around E_0 is

$$V_0 = \begin{bmatrix} r & 0 & 0 \\ 0 & -\mu & 0 \\ 0 & 0 & -\delta \end{bmatrix}.$$

The eigen values of the variational matrix are $\xi_1^0 = r > 0, \xi_2^0 = -\mu < 0$ and $\xi_3^0 = -\delta < 0$. Therefore, E_0 is a saddle with S-axis as its unstable manifold and IP-plane as its stable manifold. Thus, no path initiating in the positive octant can approach E_0.

The variational matrix of system (4) at E_1 is

$$V_1 = \begin{bmatrix} -r & -r-\lambda K & -\dfrac{\theta_1 K}{a+K} \\ 0 & \lambda K - \mu & 0 \\ 0 & 0 & \dfrac{\theta_2 K}{a+K} - \delta \end{bmatrix}.$$

The eigen values of the variational matrix are $\xi_1^1 = -r, \xi_2^1 = \lambda K - \mu$ and $\xi_3^1 = \theta_2 K/(a+K) - \delta$. Hence, the solution E_1 is stable if

$$0 < K < \min\left(\frac{\mu}{\lambda}, \frac{a\delta}{\theta_2 - \delta}\right), \quad (13)$$

i.e. $\xi_2^1 < 0$ and $\xi_3^1 < 0$. Infact, in this case the E_1 becomes globally stable. For, condition (13) eliminates the existence of E_2, E_3 and E^*. All solutions initiating on the IP-plane approach E_0 and all other solutions with initial values in $R_+^3 \setminus \{IP\text{-plane}\}$ will approach E_1 as we have $dI/dt < 0$ and $dP/dt < 0$ whenever (13) holds. Thus, local stability of E_1 implies its global stability.

Suppose $\xi_2^1 > 0$ and $\xi_3^1 < 0$. In this case the system (4) admits E_0, E_1 and E_2 as its equilibrium points. Clearly, E_0 and E_1 are saddles with SP-plane and I-axis as stable and unstable manifolds of E_1.

The variational matrix of system (4) at E_2 is

$$V_2 = \begin{bmatrix} \dfrac{-r\mu}{\lambda K} & -\dfrac{\mu(r+\lambda K)}{\lambda K} & -\dfrac{\theta_1\mu}{a\lambda+\mu} \\ \dfrac{r(\lambda K-\mu)}{r+\lambda K} & 0 & -\dfrac{rm_1(\lambda K-\mu)}{\lambda(r+\lambda K)} \\ 0 & 0 & \dfrac{\theta_2\mu}{a\lambda+\mu} - \dfrac{m_2 r(\lambda K-\mu)}{\lambda(r+\lambda K)} - \delta \end{bmatrix}.$$

Observe that the eigen value of the variational matrix in the P-direction is given by

$$\xi_3^2 = \frac{\theta_2\mu}{a\lambda+\mu} - \frac{m_2 r(\lambda K-\mu)}{\lambda(r+\lambda K)} - \delta. \quad (14)$$

and the remaining two eigen values ξ_1^2, ξ_2^2 are the roots of the equation

$$\xi^2 + \frac{r\mu}{\lambda K}\xi + \frac{r\mu}{\lambda K}(\lambda K - \mu) = 0. \quad (15)$$

Clearly both roots of the above equation have negative real parts. Therefore, E_2 is local asymptotically stable

(LAS) if $\xi_3^2 < 0$. But $\xi_2^1 > 0$ and $\xi_3^1 < 0$ implies that $\xi_3^2 < 0$. Thus, in this case E_2 is locally asymptotically stable. Since $\xi_3^1 < 0$ implies that $dP/dt < 0$. Hence, all solutions initiating in the interior of the positive octant will approach SI-plane. Infact E_2 is globally stable in this case.

Suppose $\xi_2^1 < 0$ and $\xi_3^1 > 0$. In this case the system (4) admits E_0, E_1 and E_3 as its equilibrium points. Clearly, E_0 and E_1 are saddles with SI-plane and P-axis as stable and unstable manifolds of E_1. Thus, only solutions with initial values in SI-plane approach E_1 and those with initial values in the interior of the SP-plane will either approach E_3 or a stable limit cycle surrounding E_3 depending on whether E_3 is stable or unstable in the SP-plane.

The variational matrix of system (4) at E_3 is

$$V_3 = \begin{bmatrix} r - \dfrac{2r\bar{S}}{K} - \dfrac{a\theta_1\bar{P}}{(a+\bar{S})^2} & -\dfrac{\bar{S}(r+\lambda K)}{K} & -\dfrac{\theta_1\bar{S}}{a+\bar{S}} \\ 0 & \lambda\bar{S} - m_1\bar{P} - \mu & 0 \\ \dfrac{a\theta_2\bar{P}}{(a+\bar{S})^2} & -m_2\bar{P} & 0 \end{bmatrix}.$$

The eigen values of the above matrix in the I-direction is

$$\xi_2^3 = \frac{\lambda\delta a}{\theta_2 - \delta} - \frac{mr}{K\theta_1}\left(K - \frac{\delta a}{\theta_2 - \delta}\right) \times \left(a + \frac{\delta a}{\theta_2 - \delta}\right) - \mu, \tag{16}$$

and it will be negative if

$$\frac{\delta a}{\theta_2 - \delta} - \frac{\mu}{\lambda} < 0. \tag{17}$$

The other two eigen values ξ_1^3, ξ_3^3 are the roots of the equation

$$\xi^2 - \left[r - \frac{2r\bar{S}}{K} - \frac{a\theta_1\bar{P}}{(a+\bar{S}^2)}\right]\xi + \frac{a\theta_1\theta_2\bar{S}\bar{P}}{(a+\bar{S})^3} = 0. \tag{18}$$

Clearly, real parts of ξ_1^3 and ξ_3^3 have same sign. If this sign is negative then E_3 will be locally asymptotically stable if ξ_2^3 is negative else the SP-plane becomes stable manifold for E_3. If this sign is positive along with $\xi_2^3 > 0$ then E_3 will be unstable. Incase $\xi_2^3 < 0$ then the SP-plane becomes unstable manifold for E_3. In this case the system will admit a unique stable limit cycle on the unstable manifold. Since $\xi_2^1 > 0$ we have $dI/dt < 0$. Hence, all solutions initiating in the interior of the positive octant will be drawn towards SP-plane and eventually approach either E_3 or a stable limit cycle surrounding E_3.

Now let us consider the case $\xi_2^1 > 0$ and $\xi_3^1 > 0$. In this case the system admits all the equilibrium points E_0, E_1, E_2, E_3 and E^*. Clearly, E_0 and E_1 are saddles. From (4), we have

$$\frac{d(S+I)}{dt} = rS\left(1 - \frac{S+I}{K}\right) - m_1IP - \frac{\theta_1 SP}{a+S} - \mu I < r(S+I)\left(1 - \frac{S+I}{K}\right).$$

Hence, from Lakshmikantham and Leela (1969), we have $\lim_{t\to\infty}(S(t) + I(t)) \leq K$. Moreover, if $S(0) + I(0) < K$ then we have $S(t) + I(t) < K$ for all $t > 0$. Thus, we have $S^* + I^* \leq K$. Therefore, condition (12) is always satisfied for our system.

The variational matrix of system (4) around E^* is

$$V^* = \begin{bmatrix} \dfrac{-rS^*}{K} + \dfrac{\theta_1 S^* P^*}{a+S^*} & -\dfrac{S^*(r+\lambda K)}{K} & -\dfrac{\theta_1 S^*}{a+S^*} \\ \lambda I^* & 0 & -m_1 I^* \\ \dfrac{a\theta_2 P^*}{(a+S^*)^2} & -m_2 P^* & 0 \end{bmatrix}.$$

The characteristic equation of the variational matrix is given by

$$-\xi^3 + \left(\frac{-rS^*}{K} + \frac{\theta_1 S^* P^*}{a+S^*}\right)\xi^2 + \left(m_1 m_2 I^* P^* - \frac{\lambda}{K}(r+\lambda K)S^* I^* - \frac{a\theta_1\theta_2 S^* P^*}{(a+S^*)^3}\right)\xi + S^* I^* P^*\left[\frac{am_1\theta_2(r+\lambda K)}{K(a+S^*)^2} + \frac{\theta_1 m_2 \lambda}{a+S^*} - m_1 m_2\left(-\frac{r}{K} + \left(\frac{\theta_1 P^*}{a+S^*}\right)\right)\right] = 0. \tag{19}$$

We know that, if ξ_1^*, ξ_2^* and ξ_3^* represent the three eigen values of the above variational matrix, then we have

$$\xi_1^*\xi_2^*\xi_3^* = S^*I^*P^*\left[\frac{am_1\theta_2(r+\lambda K)}{K(a+S^*)^2} + \frac{\theta_1 m_2\lambda}{a+S^*} - m_1m_2\left(-\frac{r}{K} + \left(\frac{\theta_1 P^*}{a+S^*}\right)\right)\right],$$

$$\xi_1^*\xi_2^* + \xi_2^*\xi_3^* + \xi_1^*\xi_3^* = \left(m_1m_2I^*P^* - \frac{\lambda}{K}(r+\lambda K)S^*I^* - \frac{a\theta_1\theta_2 S^*P^*}{(a+S^*)^3}\right),$$

$$\xi_1^* + \xi_2^* + \xi_3^* = \left(\frac{-rS^*}{K} + \frac{\theta_1 S^*P^*}{a+S^*}\right).$$

We have the following observations. If $\xi_1^*\xi_2^*\xi_3^* < 0$ then Eq. (19) admits one positive real root. The other two roots may be real with both being either positive or negative, or complex with positive real parts or negative real parts. From the above relations we see that if $\xi_1^* + \xi_2^* + \xi_3^* < 0$ then $\xi_1^*\xi_2^*\xi_3^* > 0$.

5. Geometrical configuration of the system

In this section we describe the geometry of the paths of the system (4) under various assumption on the system parameters. The following are the three isocline of the considered system. S-isocline surface is

$$r\left(1 - \frac{S+I}{K}\right) - \lambda I - \frac{\theta_1 P}{a+S} = 0. \tag{20}$$

I-isocline is the plane

$$\lambda S - m_1 P - \mu = 0. \tag{21}$$

P-isocline is the surface

$$\frac{\theta_2 S}{a+S} - m_2 I - \delta = 0. \tag{22}$$

The surfaces (20)–(22) are represented in Fig. 1 by their numbers. S-isocline intersects the SP-, SI-, PI-planes in the curves

$$r\left(1 - \frac{S}{K}\right) - \frac{\theta_1 P}{a+S} = 0, \tag{23}$$

$$S = K - \left(1 + \frac{K\lambda}{r}\right) I, \tag{24}$$

$$r - \left(\frac{r}{K} + \lambda\right) I - \frac{\theta_1 P}{a} = 0, \tag{25}$$

respectively. These isoclines intersect the positive S-, I-, P-axes in the points $(K, 0, 0)$, $(0, rK/(r+\lambda K), 0)$ and $(0, 0, ra/\theta_1)$, respectively. I-isocline intersects SP- and SI-planes in the lines

$$P = \frac{\lambda S - \mu}{m_1}, \tag{26}$$

and

$$S = \frac{\mu}{\lambda}, \tag{27}$$

respectively. The P-isocline intersects SP-plane in the line

$$S = \frac{\delta a}{\theta_2 - \delta}, \tag{28}$$

and intersects SI-plane in the curve

$$\frac{\theta_2 S}{a+S} - m_2 I - \delta = 0. \tag{29}$$

The curves (23)–(29) are represented in Fig. 2 by their numbers. Now let us consider the lines (26) and (28). Their slopes in the SP-plane are

$$0 < \frac{\lambda}{m_1} < \infty \quad \text{and} \, \infty,$$

respectively. Their intersection with the S-axis are $(\mu/\lambda, 0, 0)$ and $(\delta a/(\theta_2 - \delta), 0, 0)$. These lines intersect the curve (23) in the points $(\hat{S}, 0, \hat{P})$ and E_3, respectively, where $\hat{S}$ is the positive root of the equation

$$-\frac{rS^2}{K} + \left(r - \frac{ra}{K} - \frac{\theta_1\lambda}{m_1}\right)S + \frac{\theta_1\mu}{\mu_1} + ra = 0,$$

and

$$\hat{P} = \frac{\lambda\hat{S} - \mu}{m_1}.$$

Consider the curves (27) and (29) on the SI-plane. Slopes of these two curves are given by

$$\infty \quad \text{and} \quad 0 < \frac{a\theta_2}{m_2(a+S)^2} < \infty,$$

respectively. These curves intersect the S-isocline in the points E_2 and $(\tilde{S}, \tilde{I}, 0)$ where $(\tilde{S}, \tilde{I})$ is the intersection of (14) and (29) in the SI-plane. We observe that order and proximity of the values of μ/λ and $\delta a/(\theta_2 - \delta)$ play crucial role in determining the behaviour of the equilibrium points of the system (4).

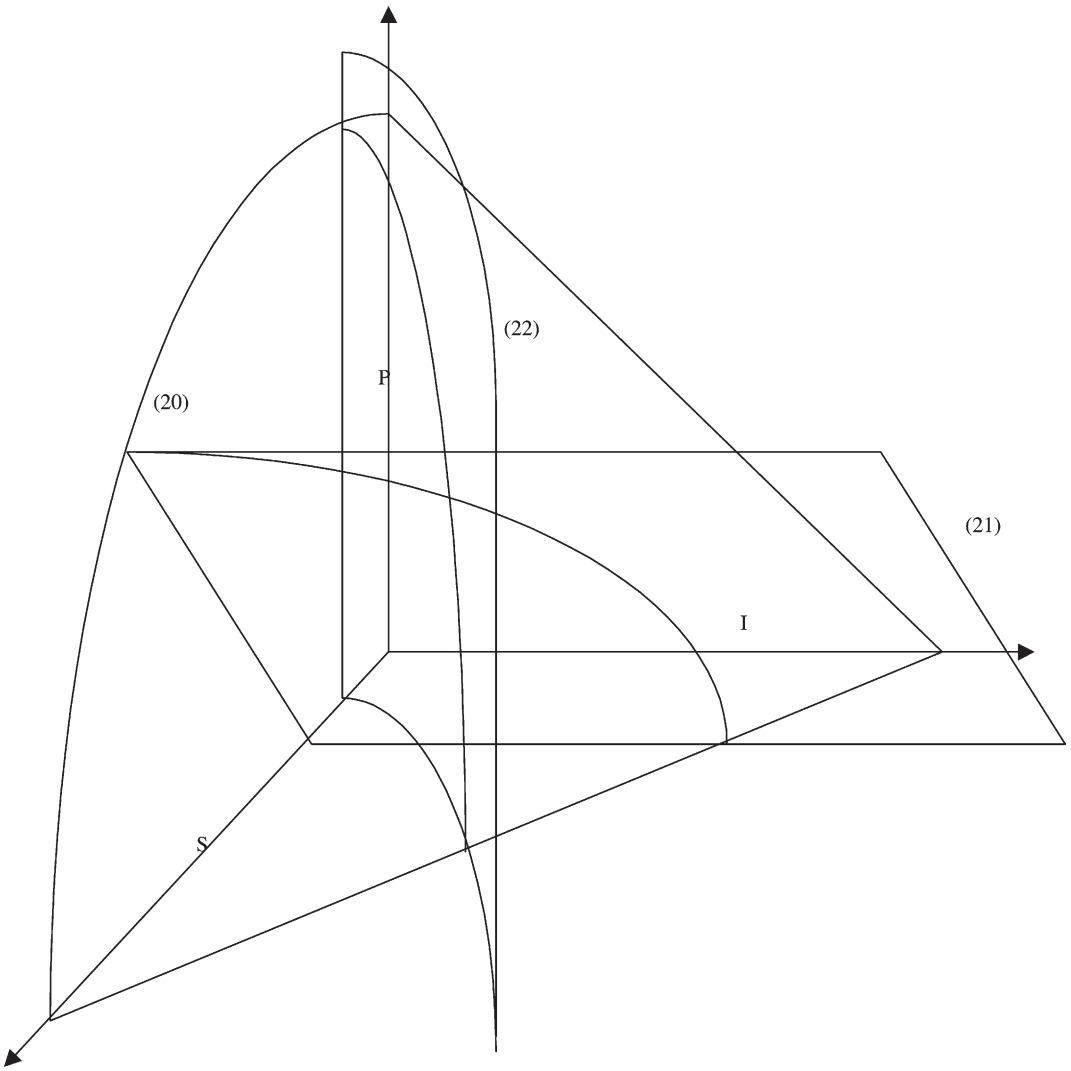

Fig. 1. The figure represents the S-, I-, P-isocline surfaces given by the Eqs. (20)–(22), respectively. Note that the S-isocline touches all boundary planes, viz. SI-, IP-, SP-planes while the I- and P-isoclines touch only SI- and SP-planes in the positive octant of the (S, I, P) space.

Below we illustrate the dependence of the above terms on the existence and nature of the equilibrium points.

Let us consider the case

$$\frac{\delta a}{\theta_2 - \delta} \geq \frac{\mu}{\lambda}. \tag{30}$$

Note that the curves (27) and (29) do not interact in the interior of the SI-plane in this case. We have the following three subcases:

(a) The lines (26) and (28) do not intersect below (23) on SP-plane.
(b) The lines (26) and (28) interact on (23).
(c) The lines (26) and (28) interact below (23).

It is easy to observe that $\xi_3^2 < 0$ holds good in this case. Hence, E_2 is locally stable. Also we observe that ξ_2^3 is positive in case (a), it is zero in case (b) and negative in case (c). Hence, we infer that E_3 is stable in the I-direction and E_2 is locally

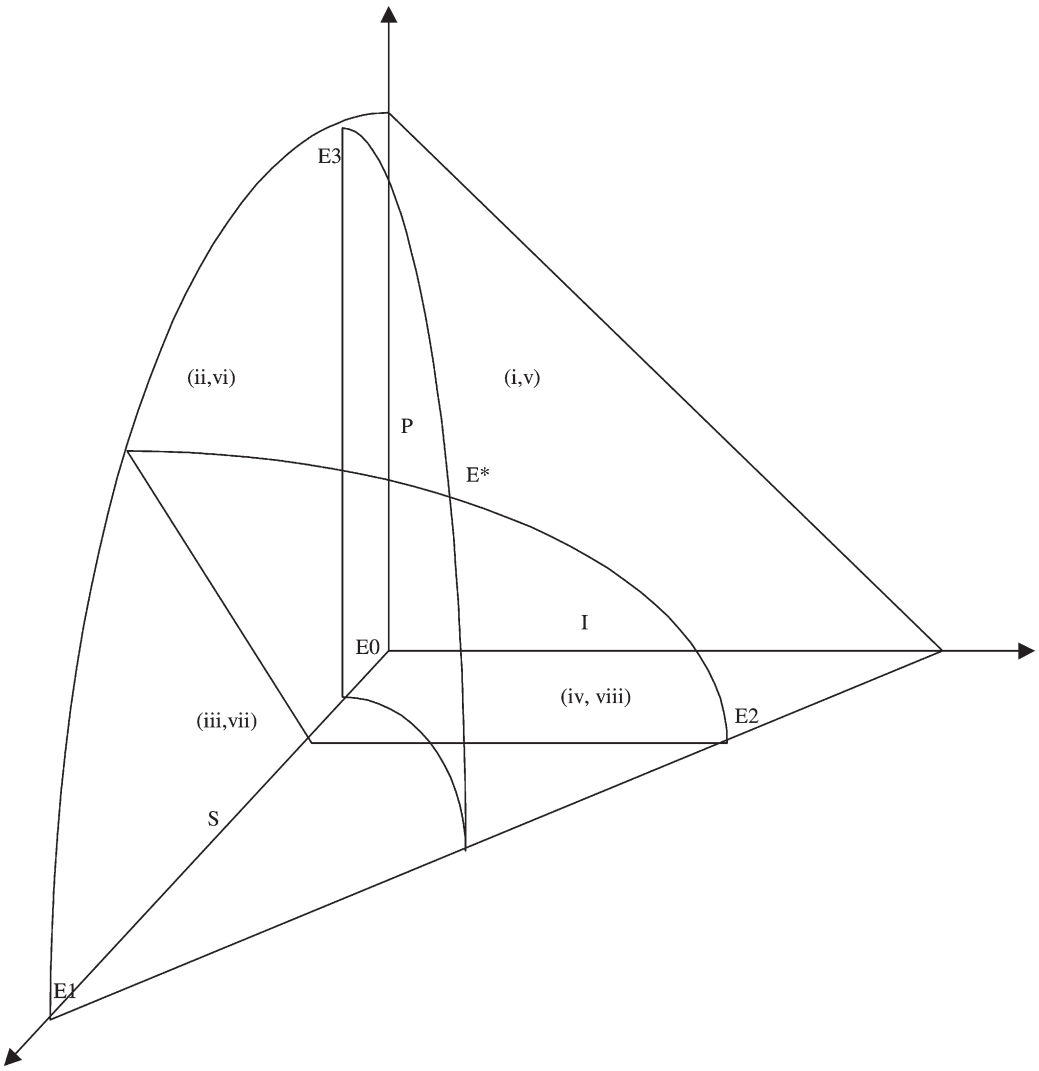

Fig. 2. The figure represents the division of the positive octant of the (S, I, P) space into eight regions by the S-, I-, P-isocline surfaces characterized as (i) $(+,-,-)$, (ii) $(+,-,+)$, (iii) $(+,+,+)$, (iv) $(+,+,-)$, (v) $(-,-,-)$, (vi) $(-,-,+)$, (vii) $(-,+,+)$, and (viii) $(-,+,-)$. Here the first entry in the ordered pairs represents the region below the S-isocline surface and the second one represents the region above the S-isocline surface.

asymptotically stable whenever (30) holds and E^* exists.

Now consider the case

$$\frac{\delta a}{\theta_2 - \delta} < \frac{\mu}{\lambda}. \tag{31}$$

Notice that, in this case the curves (26) and (28) do not interact in SP-plane and we have the following three sub cases.

(d) The curves (27) and (29) interact in SI-plane below (24).

(e) The curves (27) and (29) interact on the curve (24).

(f) The curves (27) and (29) interact outside (24).

In this case the sign of ξ_2^3 remains negative. Whereas the sign of ξ_3^2 is negative in case (d), zero in case (e) and positive in case (f). This implies that, E_3 remains stable in I-direction and E_2 also remains

stable if the system parameters satisfy (31) and E^* exists. It becomes unstable in the P-direction if sub case (f) holds.

In the light of these observations let us look at the eigen values of the interior equilibrium E^*. Notice that we always have $\bar{S} < S^*$ and $\bar{P} > P^*$ (refer (Fig. 2)). Hence, we have $\theta_1 \bar{P}/(a+\bar{S}) > \theta_1 P^*/(a+S^*)$. Therefore, from (18), whenever the boundary equilibrium points E_2 and E_3 are such that E_2 is locally stable and E_3 is stable in the I-direction, we have E^* to be a hyperbolic saddle with two-dimensional stable manifold and one-dimensional unstable manifold. This stable manifold divides the positive octant into three invariant regions. Hence, B also gets divided into three invariant sets, say B_1, B_2 and B_3 where $B_3 = \{B \cap$ stable manifold of $E^*\}$. The invariant sets B_1 and B_2 lie on either side of B_3. From the nature of E_2 and E_3 we see that they can not belong to B_3 hence, these two points must belong to $B \setminus B_3$. The unstable manifold of E^* is made up of two (branches) paths of the system (4) who have a common α-limit point as E^*. These paths being off the stable manifold region, move away from the stable manifold monotonically as time progresses (Coddington and Levinson, 1995; Farkas, 1994) but will be completely contained in B. This follows from the invariance of B. Clearly, one branch of the unstable manifold is contained in B_1 and the other is contained in B_2. Thus, these two branches, say Γ_1 and Γ_2 must approach a limit point in the respective invariant regions in which they are lying. Hence, B_1 and B_2 must contain a Ω-limit point each of the system. These points can not be E_0 and E_1 as they can not be approached from interior of the positive quadrant. Hence, the eligible limit points are E_2 and E_3. Therefore, let us assume that $E_2 \in B_1$ and $E_3 \in B_2$. The unstable manifold of E^* connects the points E_2 and E_3 through E^*. The stable manifold intersects the positive octant along S-axis which contains the remaining equilibrium points of the system, E_0 and E_1. It also intersects the PI-plane along a path of the system on PI-plane. Hence, if a path initiates in the set B_1 it will move away from B_3 under the influence of Γ_1 and approach E_2 eventually. If the path is initiated in B_2 it also moves away from B_3 under the influence of Γ_2 and eventually approaches E_3 or a stable limit cycle surrounding E_3 depending on E_3 is stable or unstable in SP-plane, respectively. If the path is initiated in B_3 it eventually approaches E^*. Now we discuss the journey of the paths in the positive octant.

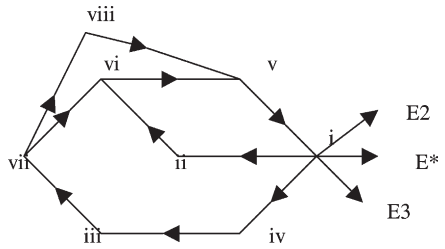

Fig. 3. The figure represents the journey of the state through various regions as time progresses. Note that there are only three alternative ways for any path moving through the positive octant. These alternatives are shown in (32)–(34).

Whenever interior equilibrium is admitted by the system (4), the isocline surfaces given by (20)–(22) divide R^3_+ into eight regions denoted by i, ii, ..., viii. Each region is characterized by a combination of the growth patterns of the state variables S, I and P. (i) $= \{(S, I, P) : S' > 0, I' < 0, P' < 0\}$ where the prime ($'$) denotes the derivative of the state variable it is associated with. We denote this region by (i) $= \{+, -, -\}$. Similarly (ii) $= \{+, -, +\}$, (iii) $= \{+, +, +\}$, (iv) $= \{+, +, -\}$, (v) $= \{-, -, -\}$, (vi) $= \{-, -, +\}$, (vii) $= \{-, +, +\}$, and (viii) $= \{-, +, -\}$. These regions are represented in Fig. 2 using ordered pairs. In each ordered pair the first entry represents the region below the S-isocline and the second one represents the region above the S-isocline surface. The journey of the state as time progresses is described in Fig. 3.

If $S(0), I(0), P(0)$ is in (i), the evolution from this initial value may approach any one of the equilibrium points E_2, E_3, E^* directly as if the equilibrium approached is a node or it may go through the cyclic paths indicated in Fig. 3 and eventually approach the corresponding equilibrium point depending on position of its initial state. The journey through various regions traced out by the paths initiating in B_1, B_2 and B_3 are respectively given by

$$\text{(i)} \to \text{(iv)} \to \text{(iii)} \to \text{(vii)} \to \text{(viii)} \to \text{(v)} \to \text{(i)}, \tag{32}$$

$$\text{(i)} \to \text{(ii)} \to \text{(vi)} \to \text{(v)} \to \text{(i)}, \tag{33}$$

$$\text{(i)} \to \text{(iv)} \to \text{(iii)} \to \text{(vii)} \to \text{(vi)} \to \text{(v)} \to \text{(i)}. \tag{34}$$

6. Discussion

Chattopadhyay and Bairagi (2001) proposed and analyzed an eco-epidemioligical model on Salton Sea. In this paper, we have modified their model by taking into consideration that Pelican population feed on both susceptible and infected fish population and feeding on infected fish enhances the death rate of Pelican. The main objective of this work is to find out the conditions for which considered system will become eventually disease free.

To establish our results we first showed that all the solutions which initiate in R_3^+ are uniformly bounded. System admits four boundary equilibria and one interior equilibrium under some suitable parametric conditions. We observed that under suitable conditions the interior equilibrium becomes a hyperbolic saddle with stable manifold of dimension two. It is also observed that this stable manifold separates the solution space of the system into two disjoint invariant subsets. Hence, if the initial value of the system is contained in the invariant set which contains the equilibrium point E_3, then the solutions will eventually approach E_3 under suitable conditions on system parameters. If the initial value is not contained in the invariant set, then the feedback control can be used to change the initial state to a new state (contained in the invariant set containing E_3), so that the state eventually will approach E_3. This change of the initial state may be achieved by suitable impulsive harvesting strategies which may be positive harvesting or negative harvesting (i.e. adding new population) or combinations of both.

To validate our analytical findings the following hypothetical parameter values have been considered: $r = 3$ per day, $K = 45$ t, $\lambda = 0.006$, $m_1 = 0.05$ per day, $a = 15$ t, $\mu = 0.24$, $d = 0.09$, $\theta_1 = 0.5$, $\theta_2 = 0.2$, $m_2 = 0.04$ per day. With the above parameter values we find that the system admits the following equilibrium points $E_0 = (0, 0, 0)$, $E_1 = (45, 0, 0)$, $E_2 = (40, 4.5871559633, 0)$, E_3=(12.272727273, 0, 119.00826446) and $E^* = (43.351788387, 1.4646923501, 0.40221460646)$. We observed that for these set of parameter values E^* becomes saddle with two-dimensional stable manifold and E_3 unstable in the SP-plane. Also we observed that the states with initial values $d1 = (30, 5, 125)$, $d2 = (9, 5, 125)$, $d3 = (9, 10, 60)$ eventually approach a limit cycle surrounding E_3 in the SP-plane indicating that the initial states are in the required invariant set. While the solution with initial data $d4 = (5, 35, 5)$ approaches the equilibrium point E_2 indicating that the initial state is not in the required invariant set. Time evaluations of the three species and corresponding trajectories for the above initial values are depicted in Fig. 4. Now by taking the value of a to be 50 and retaining the values of the other parameters as above we observed that the system admits the following equilibrium points $E_0 = (0, 0, 0)$, $E_1 = (45, 0, 0)$, $E_2 = (40, 4.5871559633, 0)$, $E_3 = (40.909090909, 0, 49.586776860)$ and $E^* = (44.830214848, 0.11370944219, 0.57962578171)$. In this case we observed that E^* remains saddle with one-dimensional unstable manifold and E_3 locally stable. Moreover, the states with initial values $b1 = (39, 4, 55)$, $b2 = (34, 10, 60)$, $b3 = (5, 20, 55)$ eventually approach E_3 and the solution with initial data $b4 = (5, 35, 5)$ approach E_2. Time evaluations of the three species and corresponding trajectories for the above initial values

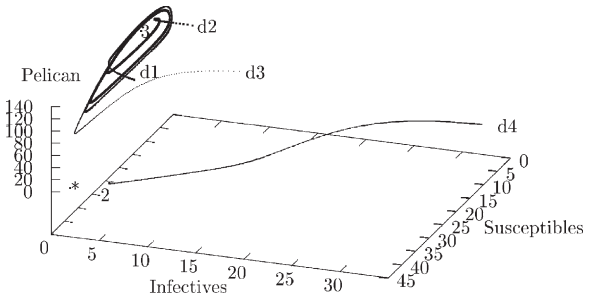

Fig. 4. Labels 2, 3, ∗ represent $E_2 = (40, 4.5871559633, 0)$; $E_3 = (12.272727273, 0, 119.00826446)$; $E^* = (43.351788387, 1.4646923501, 0.40221460646)$, respectively. Solution initiating at $d4$ approaches E_2 and those initiating at $d1$, $d2$, $d3$ approach the limit cycle on the SP-plane surrounding E_3.

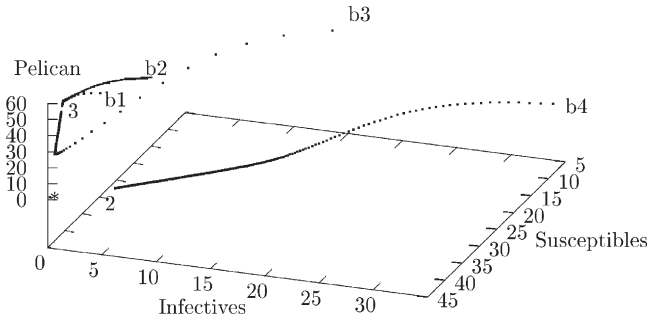

Fig. 5. Labels 2, 3, $*$ represent $E^2 = (40, 4.5871559633, 0)$; $E_3 = (40.909090909, 0, 49.586776860)$; $E_* = (44.830214848, 0.11370944219, 0.57962578181)$, respectively. Solution initiating at $b4$ approaches E_2 and those initiating at $b1$, $b2$, $b3$ approach E_3.

are depicted in Fig. 5. It is also interesting to note that S-value of E^* is greater than the S-value of E_3 while the P-value of E^* is less than the P-value of E_3. The reason is the presence of infected Tilapia population in the Salton Sea. In the case of E^* Pelican population consumes infected population also, resulting in enhancement of death rate of the Pelican population following the dynamic equation of Eq. (5). But for E_3 the P-value is higher than that of E^* as in this case Pelican population consumes only susceptible fish population which enhances their growth rate. In case of E_2 where there is no Pelican in the environment, we observe that the number of infected fish population is much higher than that of susceptible one. This is because the absence of Pelican in the environment, increase the interaction between infective and susceptible resulting in enhancement of infective population. Thus, we may finally conclude that the Pelican population plays a crucial role in keeping the environment healthy and disease free.

Acknowledgements

The authors are very much grateful to the referees and Editor-in-Chief for their criticisms and helpful comments. Research is supported by NBHM/DAE No. 48/2/98-R& D-II/805.

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