A ratio-dependent eco-epidemiological model of the Salton Sea

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SUMMARY

Ratio-dependent models set up a challenging issue for their rich dynamics incomparison to prey-dependent models. Little attention has been paid so far to describe the importance of transmissible disease in ecological situation by considering ratio-dependent models. In this paper, by assuming the predator response function as ratio-dependent, we consider a model of a system of three non-linear differential equations describing the time evolution of susceptible and infected Tilapia fish population and their predator, the Pelican. Existence and stability analysis of different equilibria of the system lead to different realistic thresholds in terms of system parameters. The condition for extinction of the species is also worked out. Our analytical and numerical studies may be helpful to chalk out suitable control strategies for minimizing the extinction of the Pelicans. We also suggest that supply of alternative food source for predator population may be used as a possible solution to save the predator from their extinction.

KEY WORDS: Tilapia; Pelican; infection; functional response; ratio-dependence; control

1. INTRODUCTION

Continuous predator–prey models have been studied mathematically since publication of the Lotka–Volterra equations. In an ecological model to describe a predator–prey relationship, it is necessary to specify the rate of prey consumption by an average predator. This functional response largely determines dynamic stability, responses to environmental influences and the nature of indirect effects in the food web containing the predator–prey pair. Nevertheless, measurements of functional responses in nature are quite rare. Recently, much work has been devoted to compare two idealized forms of the functional response: prey dependent

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and ratio dependent. Prey-dependent functional responses describe the relationship between an individualist rate of consumption and food density. The behavioural response in which the quantity of food per predator, or the prey/predator ratio, is substituted for prey density in the equation is called 'ratio dependent' functional response. Most traditional predator–prey models rest, mechanistically, on the law of mass action—the random encounter between particles in a homogenously mixed gas or liquid. Under this assumption, the *per capita* growth rate of a predator population is determined entirely, by the density of prey. In contrast, ratio-dependent theory rests, mechanistically, on the law of diminishing returns—the increasing difficulty a consumer has in meeting its energy demands as its population density rises. Under this assumption, the *per capita* growth rate of predator is determined by the demand/supply, or predator/prey ratio, a fact that can be demonstrated from geometric models of random search in a finite environment [1]. Royama [2] recognized two basic kind of attack functions, which is called 'instantaneous' and 'overall' hunting equations. The former are defined by the general relationship (in Royama's symbolism)

$$n = f(X)Yt$$

where n is the total number of prey killed during time t. X is the density of prey and Y is the density of predators. The equation holds if and only if X and Y are fixed during t, and so only applies as t approaches zero. The above equation is Lotka–Volterra model when f is linear and Watt–Ivlev–Gause or Monod–Michaelis–Menten–Holling equation when f is appropriate non-linear predator satiation function. The mass-action predator–prey models contain instantaneous functional responses.

Overall hunting equations, on the other hand, are captured by the general relationship (again in Royama's formalism)

$$z = F(x_0, Yt)$$

where z is the number of prey killed when prey density is not fixed during t, x_0 is the initial density of prey, and Y is the fixed density of predators during t. This equation includes the effect of diminishing returns because prey density can be reduced during t by predator attacks. A special case of above equation is the Arditi–Ginzburg [3] formulation

$$z = F\left(\frac{x_0}{Y}\right)$$

which is simple and parsimonious way of introducing diminishing returns into the hunting equation. Because ratio-dependent models capture the effect of diminishing returns in the prey/predator ratio, they can usually be considered, in Royama's terms, as overall hunting equations.

Royama [2] clearly demonstrates that many predator–prey models are interrelated and differ mainly in their generality, time scales and purpose. The only question is, which form is more appropriate for modelling a particular situation? Mass action models apply only over an instant of time, which may not be a serious restriction for systems in continuous motion, like planets or chemostat. However, for systems in which the objects of interest sleep, hibernate, or reproduce synchronously in response to diurnal and seasonal rhythms, the restriction may cause serious problems. In such case it may be more appropriate to use a model that integrates over the confounding temporal patterns. Hence the overall hunting equation, or the integrated form of instantaneous equation, may be more appropriate for modelling natural ecosystem.

Ecology and epidemiology are major field of studies in their own right, but there are some common features between these systems. The system which includes both ecology and epidemiology are now termed as eco-epidemiology (see, Reference [4]). Prey-dependent models on such eco-epidemiological situations exist, but ratio-dependent models in the eco-epidemiology system are less or none.

Since mid-August of 1996, a bacterial outbreak of *Vibrio vulnificus* in the Salton Sea among the Tilapia has led to massive deaths not only among the fish themselves, but also in the pelican population. Studies have indicated that the bacterial infection contributes to low oxygen levels in the tissues of the infected fish. The shortage of oxygen causes the fish to seek oxygen from the sea surface and leads to a favourable environment for botulism to grow in the tissues of the infected fish. When pelicans prey upon these vulnerable fishes, it is likely that they ingest the botulism toxins that eventually contribute to the development of *Avian botulism*. Avian botulism is a debilitating neurological disease which usually inflicts death upon its host. Chattopadhyay and Bairagi [5] proposed a mathematical model on this problem by assuming that the predator population preys only the infected prey population. They obtained the conditions for which the system around the positive equilibrium is stable. Their conditions depend on the search rate of the predator. Chattopadhyay *et al.* [6] modified the above model by taking into consideration that the predator population consumes both susceptible and infected fish population. They assumed that the functional response due to predation of susceptible fish population and infected fish population follows Holling type I and Holling type II functional responses, respectively. In the modified model they obtained the conditions for which the system will be disease free. As the predator population have to search the susceptible fish due to scarcity, the ratio-dependent functional response would be an appropriate choice.

In this paper, we consider a model of three non-linear differential equations consists of susceptible Tilapia fish, infected Tilapia fish and their predator, the Pelican. In our paper we assume that pelicans will consume whatever is available, be it infected or susceptible fish to describe the natural dynamics. We also consider that the functional response for preying the susceptible fish is ratio-dependent. We develop these assumptions into a model and then find and classify its equilibrium points and have shown that this model is capable of producing richer and more reasonable dynamics. We have also shown that when the functional response due to predation of susceptible prey population has been taken as ratio-dependent the three population can coexist with stable population distribution for some threshold value of the force of infection. Numerical experiments are then carried out to confirm and visualize our analytical findings. Moreover, we try to understand the changes in the dynamics of system in this situation.

2. BASIC MATHEMATICAL MODEL

We have two populations

(a) The fishes, Tilapia, whose population is denoted by N, number of Tilapia per unit designated area.
(b) The pelican birds, whose population is denoted by p, number of birds per unit designated area.

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

(1) In the absence of bacterial infection, the fish population grows according to a logistic fashion with carrying capacity k and an intrinsic birth rate constant r such that

$$\frac{\mathrm{d}N}{\mathrm{d}t} = rN\left(1 - \frac{N}{k}\right)$$

(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 $N(t) = s(t) + i(t)$.

(3) We assume that only susceptible fish populations, s, are capable of reproducing with logistic law (1) and infective fish population, i, dies before having the capability of reproducing. However the infective fish, i, still contribute with s to population growth towards the carrying capacity.

(4) The mode of disease transmission follows the simple law of mass action. λ is the rate of transmission (or force of infection).

(5) The disease is not genetically inherited. The infected population does not recover or become immune. The predator (bird) population preys both susceptible and infected prey population. The death rate of infected prey population (not due to predation) is μ. The natural death rate of predator is denoted by d.

(6) Here we assume that due to easy availability of infected fish, functional response (infected prey eaten per predator per unit of time) follows the Holling type I functional response whereas due to scarcity of susceptible fish the predator have to search food, the functional response (susceptible prey eaten per predator per unit time) has been taken as ratio-dependent.

From the above assumptions, we can now write down the following differential equations:

$$\left.\begin{aligned} \frac{\mathrm{d}s}{\mathrm{d}t} &= rs\left(1 - \frac{s+i}{k}\right) - \lambda is - \frac{m_1 sp}{ap+s} \\ \frac{\mathrm{d}i}{\mathrm{d}t} &= \lambda is - m_2 ip - \mu i \\ \frac{\mathrm{d}p}{\mathrm{d}t} &= \frac{\theta_1 sp}{ap+s} + \theta_2 ip - \mathrm{d}p \end{aligned}\right\} \tag{1}$$

as our model.

Here m_1 is the search rate of susceptible prey, a is the half saturation coefficient and θ_1 is the conversion factor due to predation of susceptible prey, m_2 is the search rate of infected prey, θ_2 is the conversion factor due to predation of infected prey.

For simplicity, we non-dimensionalize system (1) with the following scaling:

$$S = \frac{s}{k}, \quad I = \frac{i}{k}, \quad P = \frac{ap}{k}, \quad rt = \tau$$

with these quantities the system is transformed into a dimensionless form as follows:

$$\left.\begin{aligned}\frac{dS}{d\tau} &= S(1-S-I)-\alpha IS-\frac{\gamma_1 SP}{P+S}=F_1(S,I,P)\\ \frac{dI}{d\tau} &= \alpha IS-\xi_1 IP-\delta I=F_2(S,I,P)\\ \frac{dP}{d\tau} &= \frac{\gamma_2 SP}{P+S}+\xi_2 IP-\eta P=F_3(S,I,P)\end{aligned}\right\} \tag{2}$$

Clearly the system is not defined at $(0,0,0)$ so following [7] the system (2) is redefined as

$$\begin{aligned}\frac{dS}{d\tau} &= S(1-S-I)-\alpha IS-\frac{\gamma_1 SP}{P+S}=F_1(S,I,P)\\ \frac{dI}{d\tau} &= \alpha IS-\xi_1 IP-\delta I=F_2(S,I,P)\\ \frac{dP}{d\tau} &= \frac{\gamma_2 SP}{P+S}+\xi_2 IP-\eta P=F_3(S,I,P)\end{aligned} \tag{3a}$$

$$F_1(0,0,0)=0 \quad F_2(0,0,0)=0 \quad F_3(0,0,0)=0 \tag{3b}$$

where $\alpha=\lambda k/r$, $\gamma_1=m_1/ar$, $\xi_1=m_2k/ar$, $\xi_2=\theta_2 k/r$, $\delta=\mu/r$, $\eta=d/r$, $\gamma_2=\theta_1/r$. For convenience, in the following, time τ is replaced by t as the dimensionless time. Initial condition for system of Equation (2) is given by, $S(0)=S_0\geqslant 0$, $I(0)=I_0\geqslant 0$ and $P(0)=P_0\geqslant 0$ which are biologically meaningful.

3. BOUNDEDNESS

Due to the boundedness of the functional responses, we see that

$$\lim_{(S,I,P)\to(0,0,0)} F_1(S,I,P)=\lim_{(S,I,P)\to(0,0,0)} F_2(S,I,P)=\lim_{(S,I,P)\to(0,0,0)} F_3(S,I,P)=0$$

Using Equation (2) we can conclude that the functions $F_1(S,I,P)$, $F_2(S,I,P)$ and $F_3(S,I,P)$ are continuous functions on $\Re_3^+=[(S,I,P): S\geqslant 0,\ I\geqslant 0,\ P\geqslant 0]$ [8,9]. Straightforward computation shows that they are Lipschizian on $\Re_3^+$. Hence the solutions of (2) with non-negative initial condition exist and are unique. It is also easy to see that these solutions exist and stay non-negative for all $t>0$. In fact, if $S(0)=S_0>0$, then $S(t)>0$ for all $t>0$. Same argument is valid for I and P-component. Hence, the interior of $\Re_3^+$ is an invariant set for the model system (2). Our next task is to consider the boundedness of the solutions of system (2).

Lemma 3.1
All the solutions which initiate in $\Re_3^+$ are uniformly bounded if the condition $(\gamma_1/\gamma_2)<(\xi_1/\xi_2)$ is satisfied.

Proof
We define $W=\gamma_2 S+\gamma_2 I+\gamma_1 P$.

The time derivative of W along the solutions of (2) is

$$\frac{dW}{dt} = \gamma_2 S(1 - S - I) - \gamma_2 \xi_1 IP - \gamma_2 \delta I + \gamma_1 \xi_2 IP - \gamma_1 \eta P$$

Now for each $\Omega > 0$ and for the condition $(\gamma_1/\gamma_2) < (\xi_2/\xi_1)$ we have

$$\frac{dW}{dt} + \Omega W \leqslant ((1 - S) + \Omega)\gamma_2 S + (\Omega - \delta)\gamma_2 I + (\Omega - \eta)\gamma_1 P$$

Now, if we take $\Omega < \min(\delta, \eta)$ then the right-hand side of the above equation is bounded and we can define a constant $l = (\gamma_2(1 + \Omega)^2/4)(> 0)$ such that $(dw/dt) + \Omega W \leqslant l$.

Thus applying the theory of differential inequality [10], we obtain

$$0 < W(S, I, P) < \frac{1 - e^{-\Omega t}}{\Omega} + W(S(0), I(0), P(0))e^{-\Omega t}$$

and for $t \to \infty$ we have

$$0 < W < \frac{1}{\Omega}$$

Hence all the solutions of the above system (2) that initiate in $\Re^3_+$ are confined in the region

$$\left(B = S, I, P \in \Re^3_+ : W = \frac{1}{\Omega} + \zeta, \text{ for any } \zeta > 0\right)$$

4. EQUILIBRIA AND THEIR EXISTENCE

For population models in deterministic environments, with the environmental parameters are all well-defined constants, it is a natural curiosity to find out the community equilibria where all the populations have time independent values, that is where all net growth rates are zero. Classical three species predator–prey models always possess at least four equilibrium points: (i) trivial equilibrium, (ii) axial equilibrium, (iii) plannar equilibrium, (iv) positive interior equilibrium [11]. As observed by Freedman and Mathsen [12], Kuang and Beretta [13] and Jost *et al.* [14], ratio-dependent models are not well defined at the origin. Hence the system (2) is not well-defined at the origin $(0, 0, 0)$ and thus cannot be linearized around $(0, 0, 0)$. This is responsible for the ratio-dependent predator–prey model system to have very rich and complicated dynamics around $(0, 0, 0)$. The system of Equations (2) has five equilibria, one is $E_0(0, 0, 0)$ (though system (2) cannot be linearized around $E_0(0, 0, 0)$) and the second is $E_1(1, 0, 0)$, the third one is $E_2((\delta/\alpha), (\alpha - \delta/\alpha(1 + \alpha)), 0)$, the fourth one is $E_3((\gamma_2(1 - \gamma_1) + \gamma_1\eta)/\gamma_2, 0, ((\gamma_2 - \eta)(\gamma_2(1 - \gamma_1) + \gamma_1\eta)/\eta\gamma_2))$. The fifth and most interesting equilibrium point (from biological point of view) is $E^*(S^*, I^*, P^*)$ where S^*, I^* and P^* are non-zero positive solutions of the equations $F_1(S, I, P) = F_2(S, I, P) = F_3(S, I, P) = 0$, where S^* is the real positive root of the quadratic equation

$$f(S) = (\alpha\xi_2 + \xi_1\xi_2)S^2 - (\alpha\xi_2 + \xi_1\xi_2 + \delta\xi_2 + \xi_1\gamma_2 - \alpha\eta - \xi_1\eta + \alpha\xi_1\gamma_2 - \alpha^2\eta$$

$$- \alpha\xi_1\eta - \gamma_1\xi_2\alpha)S + (\delta\xi_2 - \delta\eta - \alpha\delta\eta - \gamma_1\xi_2\delta) = 0$$

and is given by $S^* = (k_1 + A)/B$ where $k_1 = (\alpha\xi_2 + \xi_1\xi_2 + \delta\xi_2 + \xi_1\gamma_2 - \alpha\eta - \xi_1\eta + \alpha\xi_1\gamma_2 - \alpha^2\eta - \alpha\xi_1\eta - \gamma_1\xi_2\alpha)$

$$A = [k_1^2 - 4k_2(\alpha\xi_2 + \xi_1\xi_2)]^{1/2}$$

$$k_2 = \delta\xi_2 - \delta\eta - \alpha\delta\eta - \gamma_1\xi_2\delta$$

$$B = 2(\alpha\xi_2 + \xi_1\xi_2)$$

I^* and P^* are given by $I^* = ((\alpha\eta s^* + \xi_1\eta s^* - \xi_1\gamma_2 s^* - \delta\eta)/\xi_2(\alpha s^* - \delta + \xi_1 s^*))$, $P^* = (\alpha S^* - \delta)/\xi_1$, respectively.

Remark 4.1
It is easy to see that equilibria E_0 and E_1 exists for all parameter values. The plannar equilibria E_2 and E_3 exist if $\alpha > \delta$ and $\gamma_2 > \eta$, $\gamma_1 < (\gamma_2/(\gamma_2 - \eta))$, respectively.

Remark 4.2
If the condition $\xi_2 < (\eta(1 + \alpha)/(1 - \gamma_1))$ is satisfied then there exists an unique positive real root S^* of the equation $f(S) = 0$. In order to have $P^* > 0$ we must have $S^* > \delta/\alpha$. Moreover, $I^* > 0$ implies $S^* > (\delta\eta/(\alpha\eta + \xi_1\eta - \xi_1\gamma_2))$. Therefore the interior equilibrium $E^*(S^*, I^*, P^*)$ exists if the conditions (i) $\xi_2 < (\eta(1 + \alpha)/(1 - \gamma_1))$, (ii) $S^* > \max((\delta/\alpha), (\delta\eta/(\alpha\eta + \xi_1\eta - \xi_1\gamma_2)))$ are satisfied.

5. CONDITIONS FOR EXTINCTION

At the trivial equilibrium E_0, the Jacobian matrix is not defined. Let us now for a moment, consider in a general context, that is to say we consider a system in $\Re^N$

$$\frac{dX}{dt} = H(X(t)) + Q(X(t)) \tag{4}$$

in which H is C^1 outside the origin, is continuous and homogenous of degree 1.

$$H(sX) = sH(X)$$

for all $s \geqslant 0$, $X \in \Re^N$, and Q is a C^1 function such that

$$Q(X) = o(X)$$

in the vicinity of the origin.

Throughout the section, $\|.\|$ denotes the Euclidean norm on $\Re^N$ and $(.,.)$ the associated inner product. In the case of our model, $N = 3$

$$X = (x_1, x_2, x_3) = (S, I, P)$$

$$H(X) = (H_1(X), H_2(X), H_3(X))$$

$$Q(X) = (Q_1(X), Q_2(X), Q_3(X))$$

The function H_i and Q_i $(i=1,2,3)$ are given by

$$H_1(X) = x_1 - \frac{\gamma_1 x_1 x_3}{x_1 + x_3}$$

$$H_2(X) = -\delta x_2$$

$$H_3(X) = -\eta x_3 + \frac{\gamma_2 x_1 x_3}{x_1 + x_3}$$

$$Q_1(X) = -x_1^2 - x_1 x_2 - \alpha x_1 x_2$$

$$Q_2(X) = \alpha x_1 x_2 - \xi_1 x_2 x_3$$

$$Q_3(X) = \xi_2 x_2 x_3$$

Let $X(t)$ be a solution of system (4). Assume that $\liminf_{t\longrightarrow\infty} \|X(t)\| = 0$, and X is bounded. One can extract from the family $(X(t+.))_{t\geqslant 0}$ sequences $X(t_n+.)$, $t_n \longrightarrow \infty$, such that $X(t_n+.) \longrightarrow 0$ locally uniformly on $s \in \Re$.

Define

$$y_n(s) = \frac{X(t_n+s)}{\|X(t_n+s)\|} \tag{5}$$

Recall that

$$Q(X) = o(X)$$

in the vicinity of the origin. We can then write Q as

$$Q(X) = (\|X\|)^2 0(1) \tag{6}$$

We have

$$\frac{\mathrm{d}X(t_n+s)}{\mathrm{d}s} = H(X(t_n+s)) + Q(X(t_n+s)) \tag{7}$$

From (5), we have

$$X(t_n+s) = y_n(s)\|X(t_n+s)\| = y_n(s)\langle X(t_n+s), X(t_n+s)\rangle^{1/2} \tag{8}$$

Now using the derivative of $\langle X(t_n+s), X(t_n+s)\rangle$ with respect to s

$$\frac{\mathrm{d}}{\mathrm{d}s}(\langle X(t_n+s), X(t_n+s)\rangle) = 2\left\langle X(t_n+s), \frac{\mathrm{d}X(t_n+s)}{\mathrm{d}s}\right\rangle$$

in (8), we obtain

$$\frac{\mathrm{d}(X(t_n+s))}{\mathrm{d}s} = \frac{\mathrm{d}y_n(s)}{\mathrm{d}s}(\|X(t_n+s)\|) + \frac{y_n(s)}{\|X(t_n+s)\|}\left\langle X(t_n+s), \frac{\mathrm{d}X(t_n+s)}{\mathrm{d}s}\right\rangle$$

Therefore, we have

$$H(X(t_n+s))+Q(X(t_n+s))=\frac{dy_n(s)}{ds}\|X(t_n+s)\|+\frac{y_n(s)}{\|X(t_n+s)\|}\langle X(t_n+s),H(X(t_n+s))+Q(X(t_n+s))\rangle$$

Now dividing by $\|X(t_n+s)\|$ and replacing $X(t_n+s)/\|X(t_n+s)\|$ by $y_n(s)$, we obtain

$$\frac{dy_n(s)}{ds}=H(y_n(s))-\langle y_n(s),H(y_n(s))\rangle y_n(s)+\|X(t_n+s)\|\left\{\frac{1}{\|X(t_n+s)\|^2}Q(X(t_n+s))-\left\langle y_n(s),\frac{1}{\|X(t_n+s)\|^2}Q(X(t_n+s))\right\rangle y_n(s)\right\}$$

which is equivalent to

$$\frac{dy_n}{ds}=[H(y_n(s))-(y_n(s),H(y_n(s)))y_n(s)]+\|X(t_n+s)\|[Q(y_n(s))-(y_n(s),Q(y_n(s)))y_n(s)]$$

Clearly, y_n is bounded, $\|y_n(s)\|=1$ for any s and $(dy_n/ds)=1$ is bounded too. So applying the Ascoli–Arzela theorem (see, e.g. Reference [15]), one can extract from y_n a subsequence—also denoted by y_n—which converges locally uniformly on $\Re$ towards some function y such that $\|X(t_n+s)\|[Q(y_n(s))-(y_n(s),Q(y_n(s)))y_n(s)]_{t_n\to\infty}\to 0$ and y satisfies the following system:

$$\frac{dy}{dt}=H(y(t))-(y(t),H(y(t)))y(t),\quad \|y(t)\|=1\ \forall t \tag{9}$$

Equation (9) is defined for all $t\in\Re$.

Let us, for a moment, focus on the study of Equation (9). The steady states of H are vectors V satisfying

$$H(V)=(V,H(V))V$$

This is the so-called non-linear eigenvalue. Note that the equation can be alternatively written as

$$H(V)=\phi V$$

with $\|V\|=1$; it then holds that $\phi=(V,H(V))$.

These stationary solutions correspond to fixed direction that the trajectories of Equation (9) may reach asymptotically

$$[(\phi-1)v_1+(\phi-1+\gamma_1)v_3]v_1=0 \tag{10}$$

$$[(\phi+\delta)v_2]v_2=0 \tag{11}$$

$$[(\phi+\eta-\gamma_2)v_1+(\phi+\eta)v_3]v_3=0 \tag{12}$$

Now, we are in a position to discuss in detail the possibility of reaching the origin following fixed direction.

Case 1: $v_2 = 0$

(a) $v_1 = 0$ and $v_3 \neq 0$.
In this case, there is a possibility of reaching the origin following the P-axis with $\phi = -\eta$
(b) $v_1 \neq 0$ and $v_3 = 0$.
In this case, there is no possibility to reach the origin following S-axis.
(c) $v_1 \neq 0$ and $v_3 \neq 0$.
In this case, there are two subcases

Case i: If $\gamma_2 + \gamma_1\eta < \gamma_1\gamma_2$ there is no possibility of going to the origin following a fixed direction that is contained in the positive octant.
Case ii: If $\gamma_2 + \gamma_1\eta > \gamma_1\gamma_2$ the trajectories may follow a fixed direction that is contained in the positive octant.

Case 2: $v_2 \neq 0$. In this case the trajectory always follow the P-axis to reach the origin.

Under these conditions, discussed above, it is possible to reach the trivial equilibrium point $E_0(0,0,0)$ and hence E_0 is an attractor for the model system (2).

6. BEHAVIOUR AROUND THE OTHER EQUILIBRIA

Theorem 6.1
The axial equilibrium $E_1(1,0,0)$ is unstable saddle along the S-axis if $\alpha > \delta$ (which is the condition of existence of E_2). Existence condition of E_2 itself shows that it is unstable saddle. The plannar equilibrium E_3 is unstable saddle if $(a + \gamma - \lambda)(e\gamma + h(\lambda - \gamma)) > bd\gamma$.

Proof
In order to find the stability of the other equilibria we have to calculate the Jacobian matrix $J(S,I,P)$ for system (2) at any point (S,I,P) within the first quadrant of SIP-plane except origin, and is given by

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

where $a_{11} = (1-S-I) - \alpha I - (\gamma_1 P^2/(P+S)^2)$, $a_{12} = -(1+\alpha)S$, $a_{13} = (-\gamma_1 S^2/(P+S)^2)$, $a_{21} = \alpha I$, $a_{22} = \alpha S - \xi_1 P - \delta$, $a_{23} = -\xi_1 I$, $a_{31} = (\gamma_2 P^2/(P+s)^2)$, $a_{32} = \xi_2 P$, $a_{33} = (\gamma_2 S^2/(P+S)^2) + \xi_2 I - \eta$.
The eigenvalues of the variational matrix for the equilibrium $E_1(1,0,0)$ are given by -1, $(\alpha - \delta)$, $(\gamma_2 - \eta)$. Therefore, it is easy to say that if the equilibrium E_2 exists then automatically E_1 is unstable saddle in nature.
Further the eigenvalues of the variational matrix for the equilibrium E_2 are ρ_1' and ρ_2' which are the roots of the equation

$$\rho^2 + \frac{\delta}{\alpha}\rho + \frac{\delta'}{\quad} \quad \frac{\quad}{\quad}$$

and clearly ρ_1' and ρ_2' have negative real parts (as $\alpha>\delta$ for the existence of E_2). Now if $\rho_3'>0$ i.e. if $(\gamma_2-\eta)+(\xi_2(\alpha-\delta)/\alpha(1+\alpha))>0$ then E_2 is a unstable saddle. Now $\rho_3'>0$ if the conditions $(\alpha>\delta)$ and $(\gamma_2>\eta)$ (which is the existence condition of E_3) are satisfied. Therefore, we can say that existence conditions of E_2 and E_3 imply the unstable saddle nature of the plannar equilibrium E_2.

Further the eigenvalues of the variational matrix for the equilibrium E_3 are ρ_1, ρ_2 which are the roots of the equation

$$\rho^2 - B\rho + C = 0$$

where $B=(1-2S_3-(\gamma_1P_3^2/(P_3+S_3)^2)+(\gamma_2S_3^2/(P_3+S_3)^2)-\eta)$, $C=(1-2S_3)((\gamma_2S_3^2/(P_3+S_3)^2)-\eta)+(\eta\gamma_1P_3^2/(P_3+S_3)^2)$.

Here $S_3=(\gamma_2(1-\gamma_1)+\gamma_1\eta/\gamma_2)$, $P_3=((\gamma_2-\eta)S_3/\eta)$ and $\rho_3=\alpha S_3-\xi_1P_3-\delta$.

Now if the condition $\rho_3>0$, i.e. $((\gamma_2(1-\gamma_1)+\gamma_1\eta/\gamma_2))((\alpha\eta+\xi_1\eta-\xi_1\gamma_2/\eta))>\delta$ is satisfied then E_3 is a unstable saddle in nature.

Next we assume that the interior equilibrium exists and study its local stability. This will yield some analytic and computational conditions for stable coexistence of all three species.

Theorem 6.2

If the conditions

(i) $S^*>\gamma_1/2$.

(ii) $\gamma_2>(\gamma_1\alpha\xi_2S^*/(1+\alpha)\xi_1P^*)$ then the interior equilibrium E^* is locally asymptotically stable.

Proof

The variational matrix of system (2) around the positive equilibrium $E_*(S^*,I^*,P^*)$ is

$$V=\begin{bmatrix} A_{11} & A_{12} & A_{13} \\ A_{21} & A_{22} & A_{23} \\ A_{31} & A_{32} & A_{33} \end{bmatrix}$$

where $A_{11}=(\gamma_1S^*P^*/(P^*+S^*)^2)-S^*$, $A_{12}=-(1+\alpha)S^*$, $A_{13}=(-\gamma_1S^{*2}/(P^*+S^*)^2)$, $A_{21}=\alpha I^*$, $A_{22}=0$, $A_{23}=-\xi_1I^*$, $A_{31}=(\gamma_2P^{*2}/(P^*+s^*)^2)$, $A_{32}=\xi_2P^*$, $A_{33}=(-\gamma_2S^*P^*/(P^*+S^*)^2)$.

For positive equilibrium $E^*(S^*,I^*,P^*)$, the characteristic equation is given by

$$X^3+\sigma_1X^2+\sigma_2X+\sigma_3=0$$

where the coefficients $\sigma_I, I=1,2,3$ are

$$\sigma_1 = -(A_{11}+A_{33})$$

$$\sigma_2 = (A_{11}A_{33}-A_{12}A_{21}-A_{23}A_{32}-A_{13}A_{31})$$

$$\sigma_3 = (A_{11}A_{23}A_{32}+A_{12}A_{21}A_{33}-A_{12}A_{23}A_{31}-A_{13}A_{21}A_{32})$$

From Routh–Hurwitz criterion, E^* is locally asymptotically stable if and only if $\sigma_1 > 0, \sigma_3 > 0$ and $\sigma_1\sigma_2 > \sigma_3$.

Now $\sigma_1 = (S^* + (\gamma_2 S^* P^*/(P^* + S^*)^2) - (\gamma_1 S^* P^*/(P^* + S^*)^2))$.

The condition for $\sigma_1 > 0$ is $S^* > (\gamma_1 S^* P^*/(P^* + S^*)^2)$. Simplifying we get if $S^* > \gamma_1/2$ then $\sigma_1 > 0$.

Here $\sigma_3 = (S^* - (\gamma_1 S^* P^*/(S^* + P^*)^2)) + \alpha(1+\alpha)S^* I^*(\gamma_2 S^* P^*/(P^* + S^*)^2) - \xi_1(1+\alpha)S^* I^* (\gamma_2 P^{*2}/(P^* + S^*)^2) + \alpha\xi_2 I^* P^*(\gamma_1 S^{*2}/(P^* + S^*)^2)$.

The condition for $\sigma_3 > 0$ is $((1+\alpha)S^* I^* P^*/(P^* + S^*)^2)(\alpha\gamma_2 S^* - \xi_1\gamma_2 P^*) > 0$, i.e. if $\alpha S^* > \xi_1 P^*$, i.e. if $\alpha S^* > (\alpha S^* - \delta)$ (putting the value of P^*) which is obvious.

After tedious computations, we have $\sigma_1\sigma_2 > \sigma_3$ if the condition $\gamma_2(1+\alpha)\xi_1 P^* > \gamma_1\alpha\xi_2 S^*$ is satisfied.

7. RESULTS AND DISCUSSION

In this section we use numerical experiments to confirm and visualize our analytical findings. The dynamics of system (2) around the positive interior steady state has been numerically simulated for a wide range of parameter values. We consider the hypothetical set of parameter values as $r = 0.9$/day, $k = 50$ tonnes, $m_1 = 0.4$/day, $a = 1.2$ tonnes, $\lambda = 0.25$/day, $m_2 = 0.08$/day, $\mu = 0.02$/day, $\theta_1 = 0.25$/day, $\theta_2 = 0.01$/day, $d = 0.06$/day. These satisfy the existence condition and stability condition. The specific growth rate r of the susceptible prey S and the force of infection λ are the two parameters that directly influence the population density of the preys.

7.1. Dynamics of the system for increasing rate of infection λ

The dynamics of the three populations (for $r = 0.9$), around E^*, for increasing λ is shown in the following figures:

(a) The infected prey population does not persist below a minimum strength (threshold) of infection ($\lambda_{\min} = 0.21$), and hence, the disease does not spread in the prey population. For $\lambda > 0.21$, there is a range of $(0.21 < \lambda < 0.343)$ where both the susceptible and infected fish population co-exist at equilibrium with their predator population.

(b) Increasing λ (when $\lambda > 0.342 = \lambda_{\max}$) further induces instability in the system. If the force of infection exceeds its threshold value ($\lambda > 0.342$) we observe that predator population tends to extinction whereas both susceptible and infected fish population oscillate with fluctuating amplitude. This is not as usual observation, perhaps some chaotic dynamics may observe in such situation. As this part is not our interest for this paper, we are leaving this for our future study.

Figure 1 also shows that the response of the susceptible prey population and therefore the predator population for increasing force of infection is highly non-linear. These threshold phenomena for the force of infection may be used as a control parameter for monitoring the system.

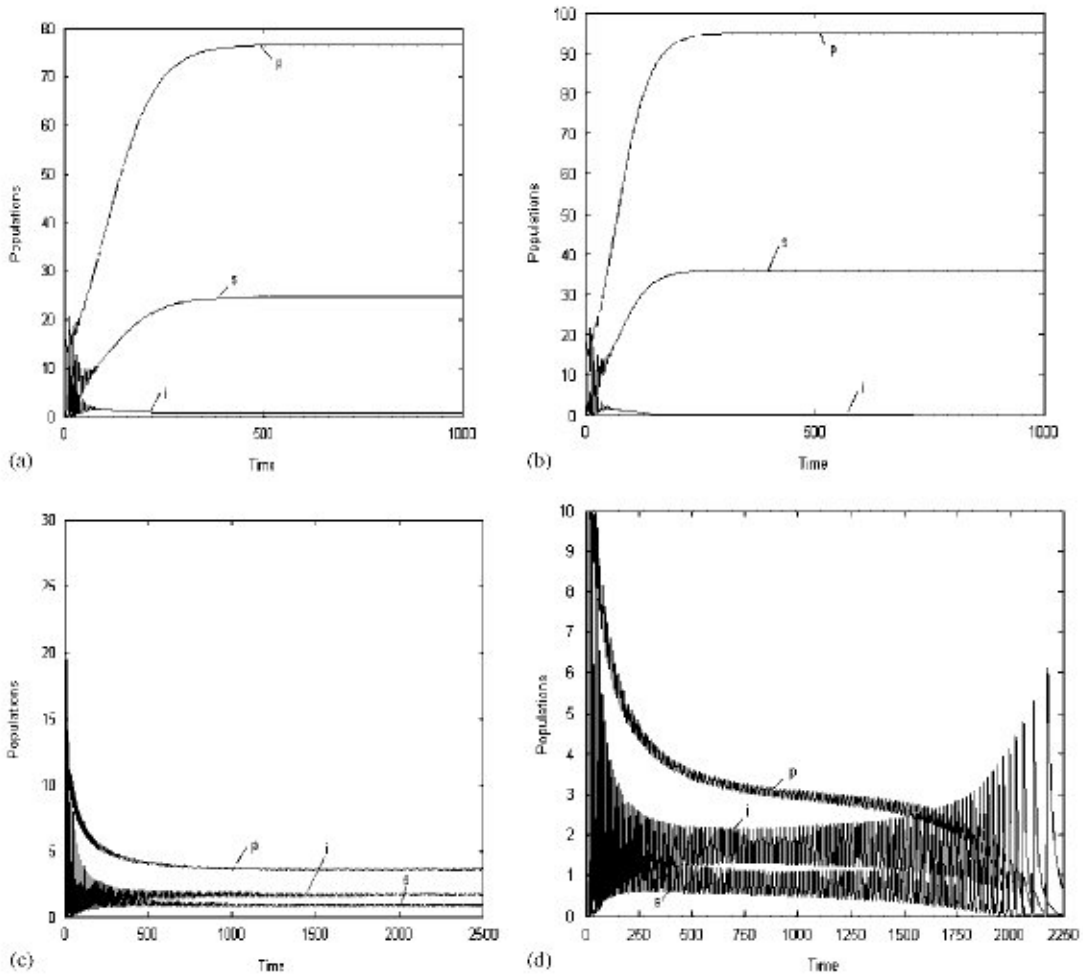

Figure 1. (a) The figure depicts the stable population distribution of three species around the interior equilibrium point for $\lambda = 0.25$; (b) the figure depicts the extinction of infected prey population for $\lambda = 0.21$; (c) the figure depicts the coexistence and stable population distribution around E_* for $\lambda = 0.342$; and (d) the figure depicts the fluctuating oscillations of susceptible and infected fish population whereas the predator population tends to extinction.

7.2. *Population stability in* $(\lambda - r)$ *parameter space*

Figure 2 shows the stability of system equation (2) obtained through linear stability analysis, for variation in both the force of infection (λ) and the specific growth rate (r) of the susceptible prey. Here we observe that λ_{max} is the increasing functions of the growth rate r of the susceptible prey but the growth rate r does not affect the value λ_{min}. Thus, this system (susceptible and infected fish and their predator Pelican) co-exist for a larger range of values of the force of infection λ for prey species with higher growth rates.

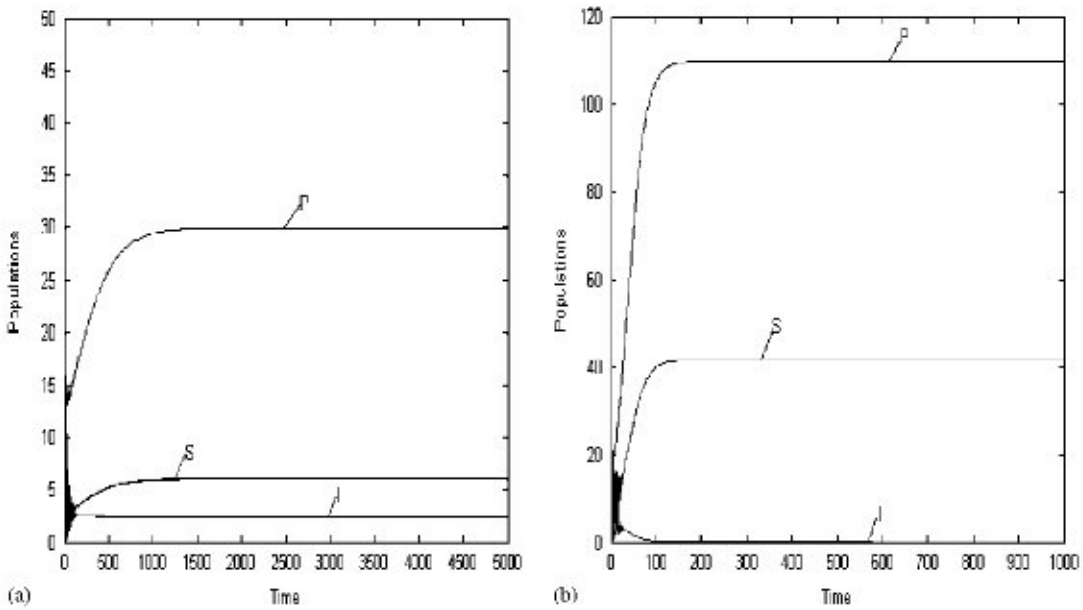

Figure 2. (a) The figure depicts the stable population distribution for $r = 1.5$ and $\lambda = 0.4$; and (b) the figure depicts the extinction of infective population for $\lambda = 0.21$.

Since $\lambda_{\min}$ is fixed, the infected prey $I > 0$ for $\lambda > \lambda_{\min} = 0.21$ indicating that there is a minimum threshold of the force of infection, below which the infected population does not persist and hence the disease does not spread.

7.3. Effect of rate of infection (λ) when infected population has negative effect on the growth rate of predator

Next we go through the regions where there are interesting dynamics and shed new light on their physical meaning. We consider that the conversion factor due to predation of infected prey of the predator is negative. By taking the hypothetical set of parametric values $m_1 = 0.4$/day, $\lambda = 0.15$/day, $m_2 = 0.08$/day, $\theta_1 = 0.3$/day, $\theta_2 = 0.01$/day, and taking the other parameters fixed we observe that all the three population show the stable coexistence:

(a) The infected prey population does not persist below a minimum strength (threshold) of infection ($\lambda_{\min} = 0.148$), and hence, the disease does not spread in the prey population. For $\lambda > 0.148$, there is a small range of λ, $(0.148 < \lambda < 0.165)$ where both the susceptible and infected fish population co-exist at equilibrium with their predator population. Increasing λ (when $\lambda > 0.165$) further induces instability in the system. If λ exceeds the value 0.165 it has been shown that the susceptible prey population as well as predator population goes to extinction.

(b) If predation rate of taking infected prey is larger than predation rate of taking susceptible prey (keeping the other parameter fixed varying $m_1 = 0.1$/day, $m_2 = 0.25$/day, $\theta_1 = 0.08$/day, $\theta_2 = 0.09$/day) the infected prey population does not persist below

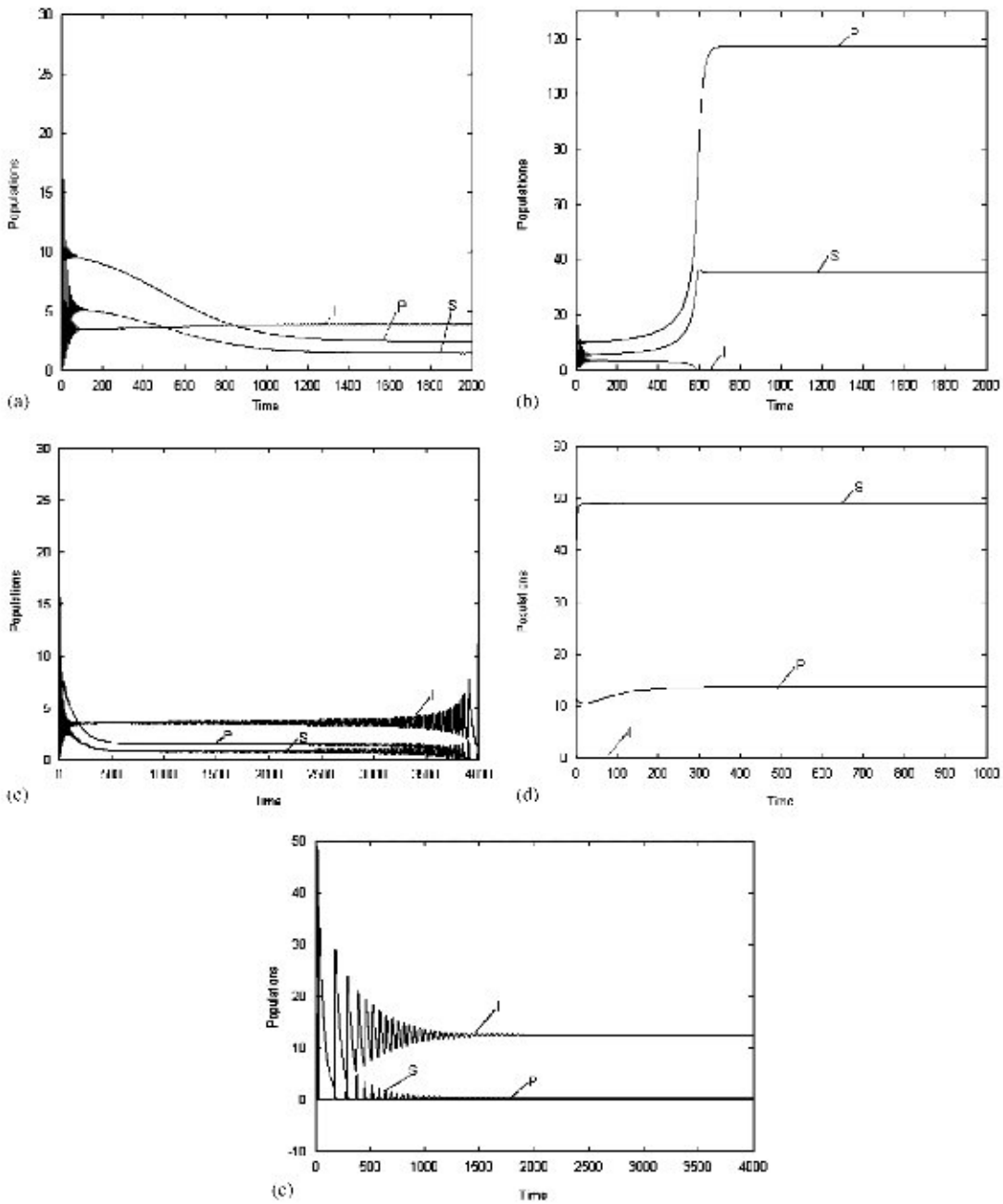

Figure 3. (a) The figure depicts that the stable population distribution around the interior equilibrium for $\lambda = 0.15$; (b) extinction of infective prey population for $\lambda = 0.148$; (c) extinction of susceptible prey as well as predator when $\lambda = 0.166$; (d) extinction of infective population for $m_1 = 0.1$/day, $m_2 = 0.25$/day, $\theta_1 = 0.08$/day, $\theta_2 = 0.09$/day, $\lambda = 0.0534$; and (e) extinction of susceptible prey as well as predator population for $m_1 = 0.1$/day, $m_2 = 0.25$/day, $\theta_1 = 0.08$/day, $\theta_2 = 0.09$/day, $\lambda = 0.055$.

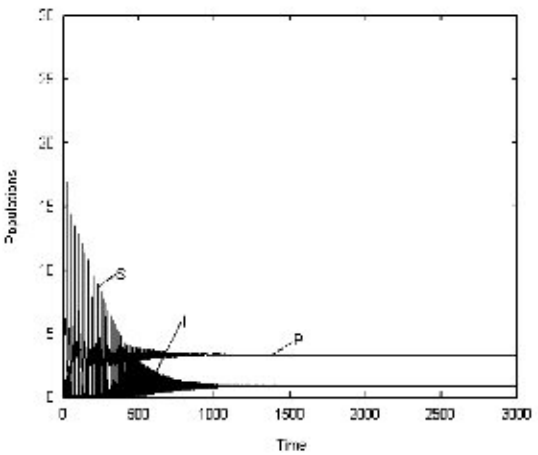

Figure 4. Stable coexistence of three population for $m_1 = 0.1$, $m_2 = 0.25$, $\theta_1 = 0.08$, $\theta_2 = 0.09$, $\lambda = 1$, $r_1 = 0.23$, $k_1 = 20$.

a minimum strength (threshold) of infection ($\lambda = 0.0534$). For $\lambda > 0.0534$, the predator population goes to extinction (Figure 3).

When predation rate of taking infected prey is larger than predation rate of taking susceptible prey then numerical results shows that for a small $\lambda = 0.0534$ we can get an infected prey free region. If $\lambda > 0.0534$ the predator population goes to extinction. So we should think about how the predator can population survive? Is there any way? Our suggestion is that alternative food source may overcome this situation. In such context, model system (1) takes the following form:

$$\frac{ds}{dt} = rs\left(1 - \frac{s+i}{k}\right) - \lambda is - \frac{m_1 sp}{ap+s}$$

$$\frac{di}{dt} = \lambda is - m_2 ip - \mu i$$

$$\frac{dp}{dt} = r_1\left(1 - \frac{p}{k_1}\right) - \frac{\theta_1 sp}{ap+s} + \theta_2 ip$$

where r_1 is the specific growth rate of predator population and k_1 is the carrying capacity of the predator population. We also observed that if $r_1 > 0.23$ then the system exhibits the stable population distribution of three species for large value of λ (Figure 4).

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