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Role of transmissible disease in an infected prey-dependent predator–prey system

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The role of disease in ecological systems is a very important issue from both mathematical and ecological points of view. This paper deals with the qualitative analysis of a prey-dependent predator–prey system in which a disease is spreading among the prey species only. We have analysed the behaviour of the system around each equilibrium and obtained conditions for global stability of the system around an equilibrium by using suitable Lypunov functions. We have also worked out the region of parametric space under which the system enters a Hopf bifurcation and a transcritical bifurcation but does not experience either saddle-node bifurcations or pitchfork bifurcations around the disease-free equilibrium E_2. Finally, we have given an example of a real ecological situation with experimental data simulations.

Keywords: Eco-epidemiology; Local stability; Global stability; Saddle-node bifurcation; Transcritical bifurcation; Pitchfork bifurcation; Hopf bifurcation

1. Introduction

The use of mathematical models allows us to identify key parameters that determine the dynamics of the biological system. Prey-dependent models have a major use in theoretical ecology. Prey-dependent predator–prey models have been studied extensively, for example by Murray [1] and Freedman [2], and the references therein. The classical prey-dependent predator–prey model exhibits not only the well-known 'paradox of enrichment' formulated by Hariston *et al.* [3] and Rosenzweig [4] but also the so-called 'biological control paradox', which was brought into discussion by Luck [5]. Again, models with a prey-dependent functional response have been facing challenges from both biological and physiological researchers [6–9]. Although prey-dependent and ratio-dependent models are extreme or limiting cases, prey-dependent models

focusing entirely on the daily energy balance of approach have proved more fertile, since their 'paradoxes' seem to be quite realistic where the premises for their existence are found.

In developing a quantitative theory for interactions of predator and prey, mathematical ecology is also an important factor, along with experimental ecology. The importance of transmissible disease in an ecological situation has now become a major field of study in its own right. The ecological literature has increasingly emphasized the importance of parasites in shaping the dynamics of both plant and animal communities [10–12]. Nowadays, it has been observed that viral, bacterial, fungal, and metazoan parasites can mediate host vulnerability to predation [13,14] and herbivores [15,16]. Similarly epidemiological models have also received much attention after the seminal model of Kermac–McKendric on SIRS (susceptible-infective-removal-susceptible) systems.

Although studies on ecology and epidemiology are now challenging and important issues from an ecological point of view, there are several common features between these systems. Very few studies have been performed in this direction [17–25], and very little attention has been paid so far to merge these two important areas of research.

Freedman [2] and May [26] proposed and analysed a prey-dependent predator–prey model. We have already mentioned that the disease factor plays an important role in predator–prey dynamics. From this viewpoint, we modify their models with an extra factor representing the disease spreading among the prey population. The main objective of this article is to observe whether or not the viral disease may act as a biological control agent in such a system. We should also emphasize that the force of infection has an important contribution to the dynamics of the eco-epidemiological system.

2. Basic assumptions, mathematical model, and preliminary results

A Lotka–Voltera-type predator–prey model with a Michaelis–Menten type of functional response [2,26] is given by

$$\left.\begin{aligned}\frac{dx}{dt} &= ax\left(1-\frac{x}{K}\right)-\frac{cxy}{m+x}\\ \frac{dy}{dt} &= y\left(\frac{fx}{m+x}-d\right)\end{aligned}\right\}, \tag{1}$$

$$x(0)=x_0>0, \qquad y(0)=y_0>0.$$

We shall now modify the above model with an introduction of transmissible disease in the prey species. We make the following basic assumptions:

(1) In the absence of transmissible disease, the prey population grows according to a logistic law with carrying capacity K ($K \in R_+$) and an intrinsic birth-rate constant a ($a \in R_+$).
(2) In the presence of the virus, the prey population is divided into two classes, namely susceptible prey denoted by $S(t)$ and infected prey denoted by $I(t)$. Therefore, at time t the total population is

$$x(t)=S(t)+I(t).$$

(3) Results from Berthier *et al.* [27] show that a mass-action incidence assumption is more appropriate than that of a proportional mixing one in describing the dynamics of direct transmission. So, here we have assumed that the mode of disease transmission follows the simple law of mass action as in Chattopadhyay and Arino [20].

(4) We assume that only susceptible prey are capable of reproducing and contribute to the carrying capacity. We also assume that the infected prey do not grow, recover, and reproduce. The model of Hamilton *et al.* [28] showed that no infected individuals contribute in the reproduction process. The infection rather reduces the remaining capacity due to an inability to compete for resources.

With the above assumptions, model (1) reduces to the following set of autonomous non-linear differential equations:

$$\left.\begin{aligned}\frac{dS}{dt} &= aS\left(1-\frac{S}{K}\right)-\frac{c_1SP}{m_1+S}-\lambda SI\\ \frac{dI}{dt} &= \lambda SI-\frac{c_2IP}{m_2+I}-\gamma I\\ \frac{dP}{dt} &= P\left(\frac{f_1S}{m_1+S}-\frac{f_2I}{m_2+I}-d\right)\end{aligned}\right\}, \tag{2}$$

where c_1, c_2, f_1, and d are the search rate of susceptible prey, search rate of infected prey, predator growth rate due to predation of susceptible prey, and natural death rate of infected prey, respectively. m_1 and m_2 are both half-saturation constants for susceptible and infected prey populations, respectively; f_2 is the death rate of the predator population due to predation of infected prey; and $\gamma=\gamma_1+\gamma_2$ is the natural death rate of prey + the natural death rate due to infection. λ ($\lambda \in R_+$) is the force of infection.

We consider ecological meaningful initial conditions:

$$S(0)=S_0>0, \quad I(0)=I_0>0, \quad P(0)=P_0>0.$$

The purpose of the mathematical study of system (2) is to describe the qualitative behaviour of the system around positive equilibria. We are also interested to find out the region for which a predator population will be saved from extinction.

If J is the Jacobian matrix of system (2), we cannot determine a diagonal matrix M such that $MJ+J^TM=0$, i.e. system (2) is not conservative. Again, due to the boundedness of the functional responses, we observe that

$$lim_{(S,I,P)\to(0,0,0)}\frac{dS}{dt}=lim_{(S,I,P)\to(0,0,0)}\frac{dI}{dt}=lim_{(S,I,P)\to(0,0,0)}\frac{dP}{dt}=0.$$

Hence, if we assume $\frac{dS}{dt}(0,0,0)=\frac{dI}{dt}(0,0,0)=\frac{dP}{dt}(0,0,0)=0$, then these functions are continuous in $R_+^3[(S,I,P): S\geq 0, I\geq 0, P\geq 0]$. Indeed, straightforward computation shows that they are Lipschitzian on R_+^3. Hence, solutions of equation (2) with non-negative conditions exist and are unique.

2.1 *Equilibria*

The positive equilibria of system (2) are (1) $E_0(0, 0, 0)$, (2) $E_1(S_1, 0, 0)$, (3) $E_2(S_2, 0, P_2)$, (4) $E_3(S_3, I_3, 0)$, and (5) $E_4(S_4, I_4, P_4)$, where $S_1 = K$, $S_2 = \frac{m_1 d}{f_1 - d}$, $P_2 = \frac{a}{c_1}(1 - S_2/K)(m_1 + S_2)$, $S_3 = \gamma/\lambda$, $I_3 = \frac{a}{\lambda}(1 - S_3/K)$, $I_4 = \frac{m_2(f_1 - d)S_4 - m_1 m_2 d}{S_4(f_2 - f_1 + d) + m_1(f_2 + d)}$, $P_4 = (\lambda S_4 - \gamma)\frac{m_2 + I_4}{c_2}$, and S_4 is a real positive root of the cubic

$$a_z x^3 + 3b_z x^2 + 3c_z x + d_z = 0, \tag{3}$$

where $a_z = a(f_2 - f_1 + d)$, $3b_z = am_1(f_2 - f_1 + d) + am_1(f_2 + d) + \lambda K m_2(f_1 - d) - aK(f_2 - f_1 + d) + \lambda K c_1 m_2 f_2$, $3c_z = am_1^2(f_2 + d) - aKm_1(f_2 - f_1 + d) - aKm_1(f_2 + d) + \lambda K c_1 m_1 m_2 f_2 - \gamma K c_1 m_2 f_2$, $d_z = -[aKm_1 m_2(f_2 + d) + \gamma K c_1 m_1 m_2 f_2 + \lambda m_1^2 m_2 K d]$.

Equation (3) has at least one real positive root if and only if $f_1 < f_2 + d$, and it has exactly one real positive root if $G^2 + 4H^3 > 0$, where $G = a_z^2 d_z - 3a_z b_z c_z + 2(b_z)^3$, $H = a_z c_z - b_z^2$, and using Cardan's method, we obtain the root as $\frac{1}{a_z}(p - \frac{H}{p} - b_z)$, where p denotes one of the three values of $\left[\frac{1}{2}(-G + \sqrt{G^2 + 4H^3})^{\frac{1}{3}}\right]$. Here, we note that $I_4 < \frac{m_2 f_1 S_4}{S_4(f_2 - f_1 + d) + m_1(f_2 + d)} = \phi(say)$ and $P_4 < \frac{\lambda S_4(m_2 + I_4)}{c_2} = \psi(say)$.

2.2 *Existence conditions*

The equilibria E_0 and E_1 always exist. The equilibrium E_2 exists if $\frac{f_1 K}{m_1 + K} < d < f_1$. The equilibrium E_3 exists when $\lambda > \frac{\gamma}{K}$ holds, and the existence conditions for the equilibrium E_4 are (1) $G^2 + 4H^3 > 0$, (2) $f_2 + d > f_1$ and (3) $S_4 > max\{\frac{\gamma}{\lambda}, \frac{m_1 d}{f_1 - d}\} = 0(say)$.

2.3 *Condition for epidemic*

We observe that $\frac{dI}{dt} < I(\lambda K - \gamma)$. So, $\frac{dI}{dt} < 0$, if $\frac{\lambda K}{\gamma} < 1$. This may be deemed to be a threshold phenomenon. Let $R_0 = \frac{\lambda K}{\gamma}$. Then, the infection will spread if $R_0 \geq 1$. This threshold phenomenon is closely related to the 'basic reproductive ratio R_0' of epidemic theory.

2.4 *Boundedness of the system*

The question of boundedness in a predator–prey system is not only interesting for mathematical reasons but also important from an ecological point of view. We shall now show that system (2) is uniformly bounded.

Lemma 1 *All solutions of system* (1) *which initiate in* R_+^3 *are bounded.*

Proof We define a function

$$\Omega = S + I + P. \tag{4}$$

The time derivative of equation (4) along the solutions of equation (2) is

$$\frac{d\Omega}{dt} = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dP}{dt}.$$

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

$$\frac{d\Omega}{dt} + \mu\Omega = S\left\{\mu + a\left(1 - \frac{S}{K}\right)\right\} - \frac{(c_1 - f_1)SP}{m_1 + S} - \frac{(f_2 + c_2)IP}{(m_2 + I)} - (\gamma - \mu)I - (d - \mu)P$$

$$\leq S\left\{\mu + a\left(1 - \frac{S}{K}\right)\right\} - (\gamma - \mu)I - (d - \mu)P[As\ f_1 < c_1]$$

$$\leq \frac{K(\mu + a)^2}{4a} - (\gamma - \mu)I - (d - \mu)P.$$

Now, if we take $\mu < min(\gamma, d)$, then the right-hand side of the above inequality is bounded. Then, we can find $\varphi > 0$ such that

$$\frac{d\Omega}{dt} + \mu\Omega = \varphi.$$

From the above equation we have $\frac{d\Omega}{dt} \leq -\mu\Omega + \varphi$, which implies that

$$\Omega(t) \leq e^{-\mu t}\Omega(0) + \frac{\varphi}{\mu}(1 - e^{-\mu t}) \leq max\left(\Omega(0), \frac{\varphi}{\mu}\right).$$

Moreover, we have lim sup $\Omega(t) \leq \frac{\varphi}{\mu} = \bar{\Theta}(say)$ as $t \to \infty$, which is independent of the initial condition. □

2.5 *Remark*

It can be easily shown that

$$lim_{t\to\infty}\, sup\, S(t) \leq K.$$

Again from the second equation of (2):

$$\frac{dI}{dt} = I\left(\lambda S - \frac{c_2 P}{m_2 + I} - \gamma\right) \leq I\left(\frac{\lambda K m_2 + \lambda K I - m_2\gamma - \gamma I}{m_2 + I}\right)$$

since $S(t) \leq K$ for all t, otherwise all of the three species will eventually go to extinction.

A standard comparison argument shows that

$$lim_{t\to\infty}\, sup\, I(t) \leq \frac{m_2(\lambda K - \gamma)}{(\gamma - \lambda K)} = K_2(say).$$

Also, from the third equation, we obtain $\frac{dP}{dt} \leq (f_1 - d)P$. Thus, $\lim_{t\to\infty} P(t) \leq 0$ if $d > f_1$. Thus, the system is not permanent as $K_2 < 0$. So, we can determine a region of parametric space such that the disease dies out from the ecological system.

Now, we are in a position to perform the stability analysis of the system. The system (2) can be written in the form $\dot{X} = F(X, \lambda)$, where $X = (S, I, P)^T$, and the Jacobian matrix of the system $J \equiv DF(X, \lambda) = (b_{ij})_{3\times 3}$, where:

$$b_{11} = a - \frac{2aS}{K} - \frac{c_1 m_1 P}{(m_1 + S)^2} - \lambda I, \quad b_{12} = -\lambda S, \quad b_{13} = -\frac{c_1 S}{m_1 + S}$$

$$b_{21} = \lambda I, \quad b_{22} = \lambda S - \frac{c_2 m_2 P}{(m_2 + I)^2} - \gamma, \quad b_{23} = -\frac{c_2 I}{m_2 + I}$$

$$b_{31} = \frac{f_1 m_1 P}{(m_1 + S)^2}, \quad b_{32} = -\frac{f_2 m_2 P}{(m_2 + I)^2}, \quad b_{33} = \frac{f_1 S}{m_1 + S} - \frac{f_2 I}{m_2 + I} - d.$$

We denote $J_k = J$ *at* E_k and $b_{ij}^{[k]} = b_{ij}$ *at* E_k, $i = 1, 2, 3, j = 1, 2, 3, k = 1, 2, 3, 4$. We again denote determinant $J_k = \det J$, trace $J_k = \mathrm{tr}\, J$, $M_k =$ sum of the second-order principal minor of J_k and $C_k = tr\ J_k \times M_k - det\ J_k$.

3. Stability analysis

3.1 *Dynamics of the system about* E_0

E_0 is a hyperbolic saddle point with unstable manifold orthogonal to the $I - P$ coordinate plane.

3.2 *Dynamics of the system about* E_1

The behaviour of system (2) about E_1 is given in the following theorem.

Theorem 1

(1) *The hyperbolic equilibrium* E_1 *will be a sink if and only if* $\gamma > \gamma_*$ *and* $d > d_*$ *where* $\gamma_* = \lambda K$, $d_* = f_1 K/(m_1 + K)$, *it will be an unstable saddle point when* $\gamma < \gamma_*$ *or* $d < d_*$, *and it can never be a source.*

(2) *The local asymptotic stability of system* (2) *ensures its global stability about* E_1.

Proof

(1) Since $b_{11}^{[1]} = -a < 0$, $b_{22}^{[1]} < 0$ if and only if $\gamma > \gamma_*$ and $b_{33}^{[1]} < 0$ if and only if $d > d_*$, all the eigenvalues are negative, and thus the proof follows from the Routh–Hurwitz criterion.

(2) Let $R_S^3 = [(S, I, P) \in R_+^3, S > 0, I \geq 0, P \geq 0]$ and consider the scalar function

$$L_1 : R_S^3 \to R$$

$$L_1(t) = S + K log \frac{K}{S}. \tag{5}$$

The derivative of (5) along the solutions of system (2) is

$$\frac{dL_1}{dt} = -(K - S)\frac{\dot{S}}{S} \leq 0 \quad (see\ Appendix\ C), \tag{6}$$

and $\frac{dL_1}{dt} = 0$ when $(S, I, P) = (K, 0, 0)$. The proof follows from equation (6) and the Lyapunov-LaSalle invariance principle [29]. □

3.3 *Dynamics of the system about* E_2

The stability analysis of system (2) about E_2 is given in the following theorem.

Theorem 2

(1) *If* $m_1 < K$, *then the necessary and sufficient condition for* E_2 *to be a sink is* $f_1 < f_1^*$ *and* $\lambda < \lambda^*$, where $f_1^* = \frac{d(K+m_1)}{(K-m_1)}$, $\lambda^* = \frac{\gamma(f_1-d)}{m_1 d} + \frac{ac_2 f_1}{c_1 m_2 d}\left(1 - \frac{m_1 d}{K(f_1-d)}\right)$.

(2) *When* $\lambda = \lambda^*$, *then the equilibrium* E_2 *will be transformed into a non-hyperbolic equilibrium, and the system attains neither a saddle-node bifurcation nor a pitchfork bifurcation, but experiences a transcritical bifurcation.*

(3) *If the inequality*

$$\left[\frac{2am_1 d}{K(f_1-d)} + \frac{d}{f_1}\left(a - \frac{m_1}{K}\right)\right]^2 \geq \frac{4ad}{f_1}\left[(f_1 - d) - \frac{m_1 d}{K}\right] \tag{7}$$

holds true along with $f_1 > f_1^*$, *then the system near* E_2 *enters into a Hopf bifurcation at* $\lambda = \lambda_{hb}$, *where* $\lambda_{hb} = \lambda^* + \theta_1$ *and* $\theta_1 = -\frac{b_{11}^{[2]}}{2S_2} + \frac{1}{2S_2}\sqrt{(b_{11}^{[2]})^2 + 4b_{13}^{[2]}b_{31}^{[2]}}$, $(\theta_1 > 0)$.

Proof (1) We obtain, $\operatorname{tr} J_2 = b_{11}^{[2]} + b_{22}^{[2]}$, $\det J_2 = -b_{13}^{[2]}b_{22}^{[2]}b_{31}^{[2]}$, $C_2 = M_2 \times tr\ J_2 - det\ J_2 = (b_{11}^{[2]} + b_{22}^{[2]})\, b_{11}^{[2]}b_{22}^{[2]} - b_{11}^{[2]}b_{13}^{[2]}b_{31}^{[2]}$. Now, $b_{11}^{[2]} < 0$ if and only if $f_1 > f_1^*$ and $b_{22}^{[2]} < 0$ if and only if $\lambda < \lambda^*$, $b_{13}^{[2]} = \frac{c_1 d}{f_1}$ and $b_{31}^{[2]}$ is always positive, and f_1^* will be positive if $m_1 < K$. Thus, when $f_1 > f_1^*$, $m_1 < K$ and $\lambda < \lambda^*$, the Routh–Hurwitz necessary and sufficient conditions are satisfied. Hence, the proof. For the proofs of (2) and (3), see Appendix A. □

3.4 *Dynamics of the system about* E_3

The behaviour of system (2) about E_3 is given in the following theorem.

Theorem 3

(1) *The solution of system* (2) *about the hyperbolic equilibrium* E_3 *is locally asymptotically stable if* $f_2 > f_2^*$, *where* $f_2^* = [\lambda^2 K m_2 + a(\lambda K - \gamma)][(f_1 - d)\gamma - m_2\lambda d]/[a(\lambda K - \gamma)(m_2\lambda + \gamma)]$.

(2) *When* $f_2 < f_2^*$, E_3 *will be a saddle point with unstable manifold locally orthogonal to the* $S-I$ *coordinate plane, and it will never be a source.*

Proof

(1) It is easy to see that $b_{11}^{[3]} = -\frac{a\gamma}{\lambda K} < 0$, $b_{12}^{[3]} = -\lambda S_3$, $b_{21}^{[3]} = \lambda I_3 > 0$ and $b_{33}^{[3]} < 0$ if and only if $f_2 > f_2^*$. Again, $\operatorname{tr} J_3 = b_{11}^{[3]} + b_{33}^{[3]}$, $\det J_3 = -b_{12}^{[3]}b_{21}^{[3]}b_{33}^{[3]}$ and $C_3 = b_{11}^{[3]}(b_{11}^{[3]}b_{33}^{[3]} - b_{12}^{[3]}b_{21}^{[3]}) + b_{11}^{[3]}(b_{33}^{[3]})^2$. So, if $f_2 < f_2^*$, then the Routh–Hurwitz necessary and sufficient conditions are satisfied. Hence, the proof.

(2) We can observe that if $f_2 < f_2^*$, then $b_{33}^{[3]} > 0$, which in turn implies that $\det J_3 = -b_{12}^{[3]}b_{21}^{[3]}b_{33}^{[3]} > 0$, hence the proof. □

3.5 *Dynamics of the system about* E_4

The behaviour of system (2) about E_4 is given in the following theorem.

Theorem 4

(1) *The solution of system (2) about the positive interior equilibrium* E_4 *is locally asymptotically stable if the following inequalities hold true:*

(a) $\frac{a(1+m_1+K)}{m_1+\theta}+\frac{\lambda K}{m_2}<\frac{a\theta}{K}$

(b) $\frac{a}{m_1+\theta}<\frac{a\theta}{K}$

(c) $\lambda K\left(\frac{m_2+1}{m_2}\right)<\frac{f_1 m_1\theta}{(m_1+K)^2}$

(d) $\frac{ac_2f_2m_2}{(m_2+\phi)^2}>\lambda\left[\frac{m_1f_1c_2K}{m_1+\theta}+\frac{c_2f_2\phi}{m_2}+\frac{c_1f_2K}{m_1}\right]+\frac{c_1c_2\psi}{m_1m_2}(Kf_1+f_2)+\frac{2ac_2f_2}{m_2}]$.

(2) *The positive interior hyperbolic equilibrium* E_4 *will be unstable if* $\theta > K/2$.

Proof (1) To show the asymptotic stability of the equilibrium E_4, we use the method of first approximation. For rigorous calculations and mathematical justification of this technique, see Li *et al.* [30] and the references therein. In this method, we use the following Lemma given in Li *et al.* [30] (P. 200). □

Lemma 2 *Let A be an* $m \times m$ *matrix with real entries. For A to be stable, it is necessary and sufficient that:*

(1) *the second computed matrix* $A^{[2]}$ *is stable;*
(2) $(-1)^m\, det(A) > 0$.

Therefore, in our case, we have to show (1) the second compound matrix $J^{[2]}(E_4)$ (see Appendix B) of J_4 is stable and (2) $det(J_4) < 0$.

Now, for $E_4=(S_4,I_4,P_4)$ and the diagonal matrix $D=diag(P_4,I_4,S_4)$, the matrix $J^{[2]}(E_4)$ is similar to $DJ^{[2]}(E_4)D^{-1}=(a_{ij})_{3\times 3}$, where $a_{11}=\bar{b}_{11}$, $a_{12}=\bar{b}_{12}\frac{P_4}{I_4}$, $a_{13}=\bar{b}_{13}\frac{P_4}{S_4}$, $a_{21}=\bar{b}_{21}\frac{I_4}{P_4}$, $a_{22}=\bar{b}_{22}$, $a_{23}=\bar{b}_{23}\frac{I_4}{S_4}$, $a_{31}=\bar{b}_{31}\frac{S_4}{P_4}$, $a_{32}=\bar{b}_{32}\frac{S_4}{I_4}$, and $a_{33}=\bar{b}_{33}$. The matrix $J^{[2]}(E_4)$ is stable if and only if $DJ^{[2]}(E_4)D^{-1}$ is stable. Since the diagonal elements of the matrix $DJ^{[2]}(E_4)D^{-1}$ are negative, an easy argument using Gersgorin's discs shows that it is stable if it is diagonally dominant in rows. Set $\mu^*=max\{g_1, g_2, g_3\}$, where

$$
\begin{aligned}
g_1 = a_{11}+a_{12}+a_{13} &= \frac{(1+m_1+S)c_1P}{(m_1+S)^2}+\frac{c_2P}{(m_2+I)^2}-\frac{aS}{K}-\frac{c_2P}{m_2+I}\\
&= \frac{(1+m_1+S)}{m_1+S}\left[a\left(1-\frac{S}{K}\right)-\lambda I\right]+\frac{1}{m_2+I}(\lambda S-\gamma)-\frac{aS}{K}-\frac{c_2P}{m_2+I}\\
&\le \frac{a(1+m_1+S)}{m_1+S}+\frac{\lambda S}{m_2+I}-\frac{aS}{K}\\
&\le \frac{a(1+m_1+K)}{m_1+\theta}+\frac{\lambda K}{m_2}-\frac{a\theta}{K},
\end{aligned}
$$

$$
\begin{aligned}
g_2 &= a_{21} + a_{22} + a_{23} \\
&= \frac{c_1 P}{(m_1 + S)^2} - \frac{f_2 m_2 IP}{(m_2 + I)^2} - \frac{aS}{K} - \lambda I \\
&\leq \frac{c_1 P}{(m_1 + S)^2} - \frac{aS}{K} \\
&= \frac{1}{m_1 + S}\left[a\left(1 - \frac{S}{K}\right) - \lambda I\right] - \frac{aS}{K} \\
&\leq \frac{a}{m_1 + S} - \frac{aS}{K} \leq \frac{a}{m_1 + \theta} - \frac{a\theta}{K},
\end{aligned}
$$

$$
\begin{aligned}
g_3 &= a_{31} + a_{32} + a_{33} \\
&= \lambda S + \frac{c_2 P}{(m_2 + I)^2} - \frac{f_1 m_1 S}{(m_1 + S)^2} \\
&= \lambda S + \frac{1}{m_2 + I}[\lambda S - \gamma] - \frac{f_1 m_1 S}{(m_1 + S)^2} \\
&\leq \lambda S + \frac{\lambda S}{m_2 + I} - \frac{f_1 m_1 S}{(m_1 + S)^2} \\
&\leq \lambda K + \frac{\lambda K}{m_2} - \frac{f_1 m_1 \theta}{(m_1 + K)^2} = \lambda K\left(\frac{m_2 + 1}{m_2}\right) - \frac{f_1 m_1 \theta}{(m_1 + K)^2}.
\end{aligned}
$$

Now, when the above conditions hold true, then $\mu^* < 0$, which implies the diagonal dominance and thus verifies the first condition.

Again

$$
\begin{aligned}
det\ J_4 &= b_{23}^{[4]} b_{12}^{[4]} b_{31}^{[4]} - b_{22}^{[4]} b_{13}^{[4]} b_{31}^{[4]} - b_{23}^{[4]} b_{11}^{[4]} b_{32}^{[4]} + b_{13}^{[4]} b_{21}^{[4]} b_{32}^{[4]} \\
&= \left[\frac{\lambda m_1 f_1 c_2 S}{(m_1 + S)^2} + \frac{c_1 c_2 f_1 m_1 SP}{(m_2 + I)(m_1 + S)^2} - \frac{ac_2 f_2 m_2}{(m_2 + I)^2} + \frac{2ac_2 m_2 f_2 S}{K(m_2 + I)^2}\right. \\
&\quad \left. + \frac{c_1 c_2 m_1 m_2 f_2 P}{(m_2 + I)^2 (m_1 + S)^2} + \frac{\lambda c_2 m_2 f_2 I}{(m_2 + I)^2} + \frac{\lambda c_1 f_2 m_2 S}{(m_1 + S)(m_2 + I)}\right] \frac{IP}{(m_2 + I)} \\
&\leq \left[\frac{\lambda m_1 f_1 c_2 K}{(m_1 + \theta)^2} + \frac{c_1 c_2 f_1 m_1 K\psi}{m_2 (m_1)^2} - \frac{ac_2 f_2 m_2}{(m_2 + \phi)^2} + \frac{2ac_2 m_2 f_2 K}{K(m_2)^2}\right. \\
&\quad \left. + \frac{c_1 c_2 m_1 m_2 f_2 \psi}{(m_2)^2 (m_1)^2} + \frac{\lambda c_2 m_2 f_2 \phi}{(m_2)^2} + \frac{\lambda c_1 f_2 m_2 K}{m_1 m_2}\right] \frac{IP}{(m_2 + I)} \\
&< 0.
\end{aligned}
$$

This verifies the second condition and completes the proof.

(2) Now, $\det J_4 > 0$ if $b_{11}^{[4]} < 0$, and $b_{22}^{[4]} < 0$. Again, $b_{22}^{[4]} = \lambda S - \frac{c_2 m_2 P}{(m_1 + I)^2} - \gamma = \frac{c_2 IP}{(m_2 + I)^2} > 0$, $b_{11}^{[4]} \leq a - \frac{2aS}{K} \leq a - \frac{2a\theta}{K} < 0$, if the condition given in the above theorem holds. □

4. Experimental data simulations

In this section, we have performed the numerical simulation with the help of experimental data given in Beltrami and Carroll [31] for a phytoplankton–zooplankton system. The object of this section is to study the global structure of system (2) for the disease-free equilibria $E_1(S_1, 0, 0)$ and $E_2(S_2, 0, P_2)$. Among these two equilibria, $E_2(S_2, 0, P_2)$ is very desirable, as in this case the predator population is present. Beltrami and Carroll [31] have taken the following range of parameters values: $a = 9$; $25 < K < 35$; $d = .4$; $0.1 \leq \gamma \leq 0.8$. They did not consider the conversion factors, but we do, so we assume $c_1 = 2$ and $c_2 = 3$. We have also numerically experimented with the effects of parameter changes in the proposed model with the initial conditions $S_0 = 2.5$, $I_0 = 2$ and $P_0 = 5$, for all figures. The results are reported below. Figure 1 has been obtained for the following parameter values, created using MATLAB®: $a = 9$; $K = 25$; $c_1 = 2$; $m_1 = 27$; $\lambda = .015$; $c_2 = 3$; $m_2 = 20$; $\gamma = .8$; $f_1 = .7$; $f_2 = .5$; $d = .4$. Figure 1 demonstrates the global stability of system (2) about E_1, where susceptible prey $S(t)$ (upper line) is going towards its carrying capacity, and infected prey $I(t)$ and predator $P(t)$ are going towards zero. This is a disease-free equilibrium, but here the predator population becomes extinct along with the infected prey population.

There is an another disease-free equilibrium E_2, which is most desirable, as here the predator population is present. Figure 2 shows the global stability of the system about equilibrium E_2 with the same parameter values as in figure 1, except that $f_1 = .8$ and $K = 30$.

Here, we also observe that, as in the previous case, the parameters values satisfy the local stability conditions (namely $m_1 < K$, $f_1 =< f_1^* = 7.6$ and $\lambda < \lambda^* = .1646$).

In figure 2, the upper line indicates the susceptible prey $S(t)$, the middle line the predator population $P(t)$, and the lowermost line infected prey $I(t)$, which is with the zero line.

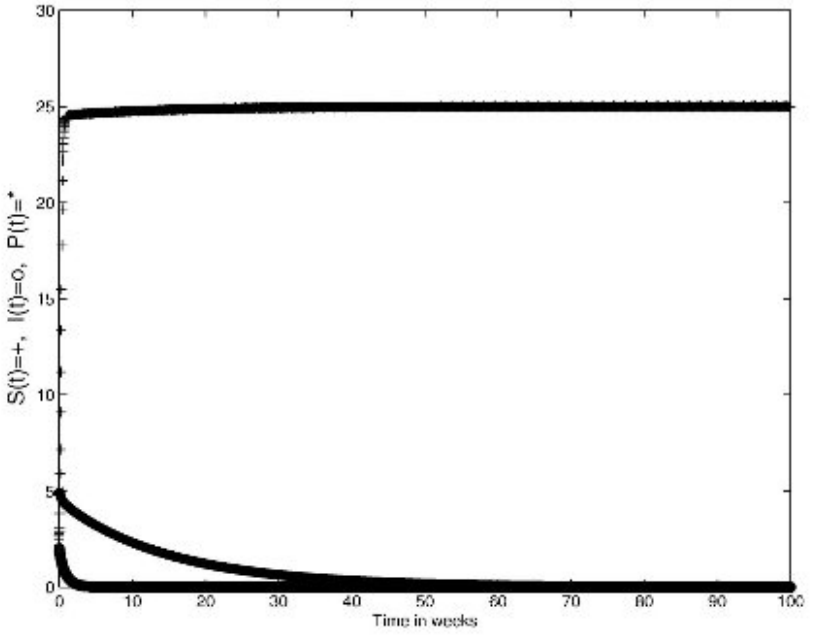

Figure 1. Global stability of system (2) about E_1. Here, $d > d_* = .3365$, $\gamma > \gamma_* = .3750$, and $R_0 = .4688 < 1$.

Note that similar results are obtained for other values of the parameters, not shown here due to lack of space. By changing the parameter values, we can show that system (2) enters a Hopf bifurcation about E_2. For instance, if we choose $m_1 = 17$, $\lambda = .05$, $m_2 = 16$, $c_1 = 5$, $c_2 = 6$, $f_1 = 3$, and $f_2 = 2$, and the other parameters remain the same as in figure 1. This situation is shown in figure 3.

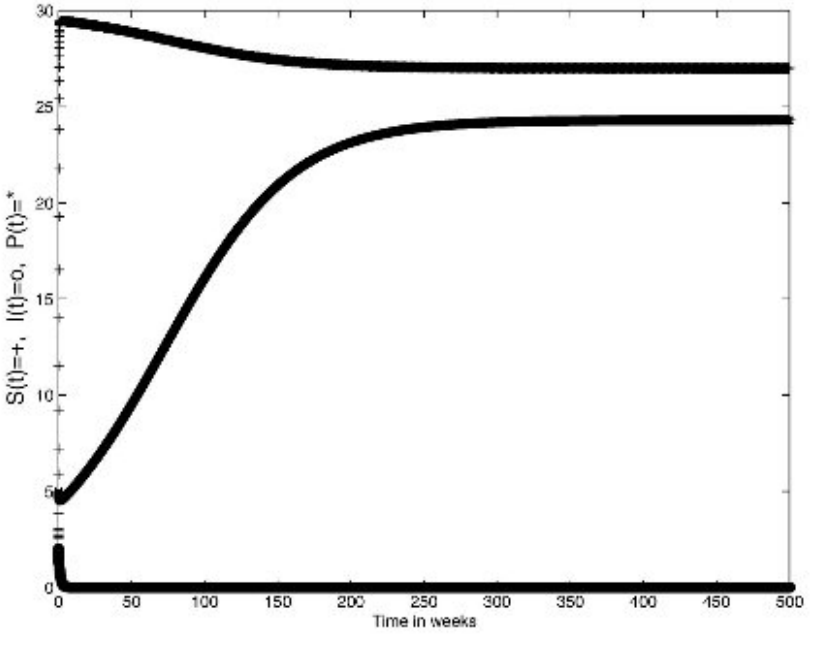

Figure 2. Extinction of the disease with $R_0 = 0.5625 < 1$.

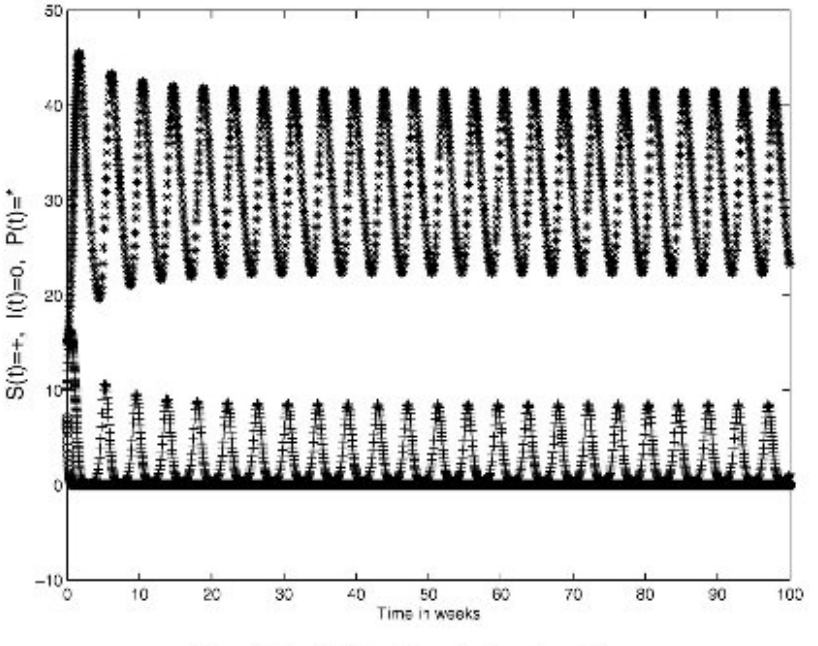

Figure 3. Hopf bifurcation situation about E_2.

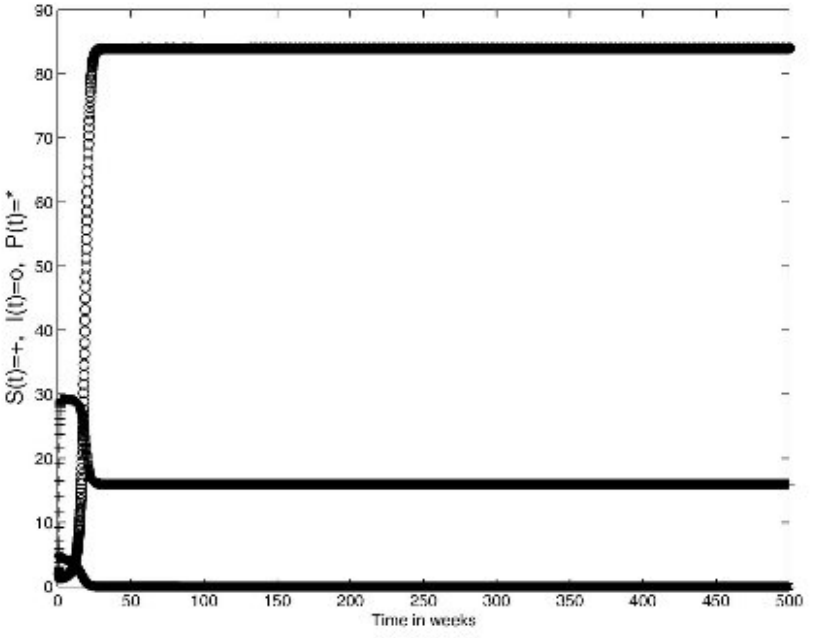

Figure 4. Persistence of the disease with healthy prey. Here, $R_0 = 1.8750 > 1$.

Finally, by increasing the value of λ, we obtain the situation in figure 4, which represents the disease endemic situation at $\lambda = .05$ with the other parameters remaining the same as in figure 2. In figure 4, the upper line represents the infected prey $I(t)$, the middle line indicates the susceptible prey $S(t)$, and the lowermost line represents the predator population $P(t)$.

Our numerical simulations confirm the analytical results and emphasize that the disease transmission coefficient λ has an important contribution to the dynamics of the eco-epidemiological system.

5. Discussion

Our mathematical model consists of three non-linear autonomous ordinary differential equations for three different populations, namely susceptible prey, infected prey, and predators. We have obtained conditions for the boundedness of the solutions and the existence and stability of the system equilibria.

It has been observed that to spread the infection, the initial number of susceptible prey must exceed a threshold value. This value will be determined from the ratio of the total death rate of the infected prey and the force of infection. This threshold property may be related to the 'basic reproductive ratio R_0' of epidemic theory [32].

It may easily be verified that system (1) has three equilibria, namely (1) $\hat{E}_0(0,0)$, (2) $\hat{E}_1(K,0)$, and (3) $\hat{E}^*(x^*, y^*)$, where $x^* = \frac{md}{f-d}$, $y^* = \frac{a}{c}(1 - \frac{x^*}{K})(m + x^*)$. Using the Routh–Huritz criteria, it can be easily shown that $\hat{E}_0$ is always unstable, $\hat{E}_1$ is stable if $d > \frac{fK}{m+K}$, and $\hat{E}^*$ will be stable if $f < \frac{d(K+m)}{K-m}$.

It should be noted here that $E_0(0, 0, 0)$ and $E_1(0, 0, 0)$ for system (2) are analogous to that of $\hat{E}_0$ and $\hat{E}_1$ of system (1). The Dynamical behaviours of both systems about $\hat{E}_0$ (0, 0, 0) and $\hat{E}_0$ are the same.

However, for local stability of $\hat{E}_1$ an additional condition ($\lambda K - \gamma < 0$) is required. Thus, we may conclude that the dynamics of system (2) are influenced by the force of infection. Again, when $d > \frac{fK}{m+K}$, $\hat{E}_1$ is stable, but $E_1(K, 0, 0)$ is unstable if we choose $\lambda > \frac{\gamma}{K}$. So, introduction of a disease in the disease-free predator–prey model is enough to destabilize the otherwise stable equilibrium.

The equilibrium $E_2(S_2, 0, P_2)$ is again analogous to that of $\hat{E}^*$, but the stability analysis differs in several ways. The feasibility conditions for both the equilibria are the same, but the stability of E_2 demands an additional condition $\lambda < \lambda^*$ (see Theorem 2(1)). In this case also, we can destabilize the otherwise stable equilibrium $\hat{E}^*$ by taking $\lambda > \lambda^*$. Thus, the infection has a relevant influence on the ecosystem.

Again, we find that the predator–prey system considered by Jost *et al.* [33] and Kuang and Beretta [35] can go to total extinction under certain parametric conditions. It is also noted here that our system (2) is equivalent to their system for $I = 0$. But in our system, if the conditions (1) given in Theorem 2 are true, total extinction of the populations is not possible. Hence, we can conclude that the introduction of infected prey to the predator–prey system can prevent total extinction and may act as a *biological control*.

It is also interesting to note that conditions (1) of Theorem 2 give us the stability of the disease-free equilibrium. In this case, the predator will act as a *system saver* if we can monitor three system parameters, namely, the growth rates of susceptible prey and predators, and the disease-transmission coefficient λ.

Before ending this article, we should point out that most of the conditions obtained throughout the paper suggest to us that the the disease-transmission coefficient λ plays an important role in the persistence of the populations. We hope these observations will help experimental ecologists with their experimental setups, and, as a result might enhance the development of theoretical ecology.

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References

[1] Murray, J.D., 1989, *Mathematical Biology* (New York: Springer).
[2] Freedman, H.I., 1980, *Deterministic Mathematical Models in Population Ecology* (New York: Dekker).
[3] Hariston, N.G., Smith, F.E., Slobodkin, L.B., 1960, Community structure, population control and competition. *American Naturalist*, **94**, 4221.
[4] Rosenzweig, M.L., 1971, Paradox of environment, destabilization of explosion systems in ecological time. *Science*, **171**, 385–387.
[5] Luck, R.F., 1990, Evaluation of natural enemies for biological control: a behavior approach. *Trends in Ecology and Evolution*, **5**, 196–199.
[6] Arditi, R., Ginzburg, L.R., 1989, Coupling in the predator–prey dynamics; ratio-dependence. *Journal of Theoretical Biology*, **139**, 311–326.
[7] Arditi, R., Ginzburg, L.R., Akcakaya, H.R., 1991, Variation in the plankton densities among lakes; a case for ratio-dependent predation models. *American Naturalist*, **138**, 1287–1296.

[8] Akcakaya, H.R., 1992, Population cycles of mammals; evidence for a ratio-dependent predator–prey hypothesis. *Ecological Monographs*, **62**, 119–142.
[9] Gutierrez, A.P., 1992, The physiological basis of the ratio-dependent predator–prey theory; a metabolic pool model of Nicholson's blowfly as an example, *Ecology*, **73**, 1552–1563.
[10] Minchella, D.J. and Scott, M.E., 1991, Parasitism a tropic determinant of animal community structure. *Trends in Ecology and Evolution*, **6**, 250–254.
[11] Gibson, C.C. and Watkinson, A.R., 1992, The role of the hemi parasite annual *Rhinanthus minor* in determining grass land community structure. *Oecologia*, **89**, 62–68.
[12] Dobson, A. and Crawley, M., 1994, Pathogens and the structure of plant communities. *Trends in Ecology and Evolution*, **9**, 393–398.
[13] Temple, S.A., 1987, Do predators always capture subtended individuals disproportionately from prey population? *Ecology*, **68**, 669–674.
[14] Hudson, P.J., Dobson, A.P. and Newborn, D., 1992, Do the parasites make prey vulnerable to predation? Redgrouse and parasites. *Journal of Animal Ecology*, **61**, 681–692.
[15] Hammond, A.M. and Hardy, T.N., 1989, In: E.A. Heinrichs (Ed.) *Plant Stress Insect Interactions* (New York: Wiley).
[16] Linhart, Y.B, 1991, Disease, parasitism and herbivore multidimensional challenges in plant evolution. *Trends in Ecology and Evolution*, **6**, 392–396.
[17] Hadeler, K.P. and Freedman, H.I., 1989, Predator–prey population with parasitic infection. *Journal of Mathematical Biology*, **27**, 609–631.
[18] Freedman, H.I., 1990, A model of predator–prey dynamics as modified by the action of parasite. *Mathematical Biosciences*, **99**, 143–155.
[19] Venturino, E., 1995, Epidemics in prey-predator models: Disease in the prey. In: O. Arino, D. Axelrod, M. Kimmel and M. Langlais (Eds) *Mathematical Population Dynamics: Analysis of Heterogeneity, Volume One: Theory of Epidemics* (Winnipeg, Canada: Wuertz Publishing Ltd.), pp. 381–393.
[20] Chattopadhyay, J. and Arino, O., 1999, A predator prey model with disease in prey. *Nonlinear Analysis*, **36**, 747–766.
[21] Xiao, Y. and Chen, L., 2001, Modelling and analysis of a predator–prey model with disease in prey. *Mathematical Biosciences*, **171**, 59–82.
[22] Chattopadhyay, J. and Bairagi, N., 2001, Pelican at risk in Salton Sea: an eco-epidemiological model. *Ecological Modelling*, **136**, 103–112.
[23] Bairagi and Chattopadhyay, 2002, Pelican at risk in Salton sea—a delay induced eco-epidemiological model. *Mathematical and Computer Modelling of Dynamical Systems*, **8**, 257–272.
[24] Haque, M. and Chattopadhyay, J., 2003, Influences of non-linear incidence rate in an eco-epidemiological model of the Salton Sea. *Nonlinear Studies*, **10**, 273–388.
[25] Jin, Z. and Haque M., (in press), Global stability analysis of an eco-epidemiological model of the Salton Sea. *Journal of Biological Systems*.
[26] May, R.M., 1974, *Stability and Complexity in Model Ecosystems* (Princeton, NJ: Princeton University Press).
[27] Berthier, K., Langlasis, M., Auger, P. and Pontier, D., 2000, Dynamics of a feline virus with two transmission models with exponentially growing host populations. *Proceedings of the Royal Society of London. Series B: Biological Sciences*, **267**, 2049–2056.
[28] Hamilton, W.D., Axelrod, R. and Tanese, R., 1990, Sexual reproduction as an adaptation to resist parasite (a review). *Proceedings of the National Academy of Sciences of the United States of America*, **87**, 3566–3573.
[29] Hale, J., 1989, *Ordinary Differential Equations* (Malabar, FL: Krieger).
[30] Li, Y.M., Graef, J.R., Wang, L. and Karsai, J., 1999, Global dynamics of a SEIR model with varying total population size. *Mathematical Biosciences*, **160**, 191–213.
[31] Beltrami, E. and Carroll, T.O., 1994, Modelling the role of viral disease in recurrent phytoplankton blooms. *Journal of Mathematical Biology*, **32**, 857–863.
[32] Anderson, R.M. and May, R.M., 1991, *Infectious Diseases of Humans: Dynamics and Control* (Oxford: Oxford University Press).
[33] Jost, C., Arino, O. and Arditi, R., 1999, About ratio-dependent predator–prey models. *Bulletin of Mathematical Biology*, **61**, 19–32.
[34] Kuang, Y. and Beretta, E., 1998, Global qualitative analysis of a ratio-dependent predator–prey system. *Journal of Mathematical Biology*, **36**, 389–406.
[35] Sotomayor, J., 1973, In: M.M. Peixoto (ED.), *Dynamical Systems* (New York: Academic Press).

Appendix A

Proof of Theorem 2 (2) One of the eigenvalues of J_2 will be zero if and only if $\det J_2 = b_{13}{}^{[2]} b_{22}{}^{[2]} b_{31}{}^{[2]} = 0$, i.e. $b_{22}{}^{[2]} = 0$, since $(b_{13}{}^{[2]}, b_{31}{}^{[2]}) \neq (0, 0)$. This gives $\lambda = \lambda^*$, and the other two eigenvalues are given by

$$\varsigma_2, \varsigma_3 = \frac{b_{11}{}^{[2]}}{2} \pm \frac{1}{2}\sqrt{(b_{11}{}^{[2]})^2 + 4 b_{13}{}^{[2]} b_{31}{}^{[2]}}.$$

Since $b_{13}{}^{[2]} < 0$ and $b_{31}{}^{[2]} > 0$, the real parts of ς_2, ς_3 will be same sign as the sign of $b_{11}{}^{[2]}$. Now, if $f_1 < f_1{}^*$ then $b_{11}{}^{[2]} > 0$ and two eigenvalues of J_2 will be positive; hence the proof follows from Sotomayor [35]. Again if $f_1 > f_1{}^*$ then $b_{11}{}^{[2]} < 0$. In this case, $\mho = (\theta_1, \theta_2, \theta_3)^T$, $\Upsilon = (0, \hbar_2, 0)^T$, where $\mho$, Υ are the eigenvectors corresponding to the eigenvalue $\varsigma_1 = 0$ of the matrices J_2 and $J_2{}^T$, respectively, and $\theta_1 = -\frac{b_{32}{}^{[2]}}{b_{31}{}^{[2]}}\theta_2$, $\theta_3 = -\frac{b_{11}{}^{[2]}}{b_{13}{}^{[2]}}\theta_1 - \frac{b_{12}{}^{[2]}}{b_{13}{}^{[2]}}\theta_2$ and θ_2, $\hbar_2$ are any two non-zero real numbers. Now, $\Upsilon^T[F_\lambda(X_2, \lambda^*)] = 0$, so the system does not experience any saddle-node bifurcations. Again, $\Upsilon^T[DF_\lambda(X_2, \lambda^*)\mho] = S_2 \hbar_2 \theta_2 \neq 0$ and $\Upsilon^T[D^2F(X_2, \lambda^*)(\mho, \mho)] \neq 0$, where $[DF_\lambda(X_2, \lambda^*)] = (\zeta_{ij})_{3\times 3}$ and $\zeta_{11} = 0$, $\zeta_{12} = -S_2$, $\zeta_{13} = 0$, $\zeta_{21} = 0$, $\zeta_{22} = S_2$, $\zeta_{23} = 0$, $\zeta_{31} = 0$, $\zeta_{32} = 0$, $\zeta_{33} = 0$, and $D^2F(X_2, \lambda^*)$ is a $3 \times 3 \times 3$ tensor. Thus, by the same theorem [35] the system possesses a transcritical bifurcation. Again, $\Upsilon^T[D^2F(X_2, \lambda^*)(\mho, \mho)] \neq 0$. Therefore, the system does not experience a pitchfork bifurcation. □

Proof of Theorem 2 (3) We know that if the Jacobian matrix $DF(X_2, \lambda_{hb})$ of the system has a simple pair of purely imaginary eigenvalues and no other eigenvalues with zero real parts, then the implicit function theorem guarantees that for each λ near λ_{hb} there will be a unique equilibrium X_λ (where $X = (S, I, P)^T$) near X_2. However, if the eigenvalues of $DF(X_\lambda, \lambda)$ cross the imaginary axis at $\lambda = \lambda_{hb}$, then the dimensions of the stable and unstable manifolds of X_λ will change, and the local phase portrait of system (2) will also change as λ passes through the bifurcation value λ_{hb}. In the generic case, a Hopf bifurcation occurs where a periodic orbit is created as the stability of the equilibrium X_λ changes.

Thus, necessary and sufficient conditions for Hopf bifurcation to occur are that there exists a $\lambda = \lambda_{hb}$ such that (*a*) $C_2 = \kappa_1(\lambda_{hb})\kappa_2(\lambda_{hb}) - \kappa_3(\lambda_{hb}) = 0$, where $\kappa_1 = -\mathrm{tr}\, J_2$, $\kappa_2 = M_2 =$ sum of the second-order principle minor of J_2 and $\kappa_3 = -$determinant J_2 and (*b*) $\frac{\mathrm{d}}{\mathrm{d}\lambda}(Re(\mu(\lambda)))_{\lambda=\lambda_{hb}} \neq 0$, where μ is given by the characteristic equation of J_2 as follows:

$$\mu^3 + \kappa_1\mu^2 + \kappa_2\mu + \kappa_3 = 0.$$

The condition $\kappa_1\kappa_2 - \kappa_3 = 0$ gives

$$\left[(b_{11}{}^{[2]} + b_{22}{}^{[2]})b_{22}{}^{[2]} - b_{13}{}^{[2]}b_{31}{}^{[2]}\right] = 0,$$

which gives $\lambda = \lambda_{hb}$ (note that λ_{hb} will be real if and only if the inequality (7) holds true). Thus, for $\lambda = \lambda_{hb}$, we have

$$(\mu^2 + \kappa_2)(\mu + \kappa_1) = 0, \tag{8}$$

which has three roots, $\mu_1 = +i\sqrt{\kappa_2}$, $\mu_2 = -i\sqrt{\kappa_2}$, and $\mu_3 = -\kappa_1$. For all values of λ, the roots are in general of the form:

$$\mu_1(\lambda) = \wp_1(\lambda) + i\wp_2(\lambda),\ \mu_2(\lambda) = \wp_1(\lambda) - i\wp_2(\lambda),\ \mu_3(\lambda) = -\kappa_1(\lambda).$$

Now, we shall verify the transversality condition

$$\frac{\mathrm{d}}{\mathrm{d}\lambda}(Re(\mu_j(\lambda)))_{\lambda=\lambda_{hb}} \neq 0,\ j = 1, 2. \tag{9}$$

Substituting $\mu_j(\lambda) = \wp_1(\lambda) + i\wp_2$ into equation (9) and calculating the derivative, we have

$$\Psi(\lambda)\dot{\wp}_1(\lambda) - \Phi(\lambda)\dot{\wp}_2(\lambda) + \Theta(\lambda) = 0, \Phi(\lambda)\dot{\wp}_1(\lambda) + \Psi(\lambda)\dot{\wp}_2(\lambda) + \Gamma(\lambda) = 0,$$

where: $\Psi(\lambda) = 3(\wp_1(\lambda))^2 + 2\kappa_1(\lambda)\wp_1(\lambda) + \kappa_2(\lambda) - 3(\wp_2(\lambda))^2$; $\Phi(\lambda) = 6\wp_1(\lambda)\wp_2(\lambda) + 2\kappa_1(\lambda)\wp_2(\lambda)$; $\Theta(\lambda) = (\wp_1(\lambda))^2\kappa_1(\lambda) + \kappa_2(\lambda)\wp_1(\lambda) + \kappa_3(\lambda) - \kappa_1(\lambda)(\wp_2(\lambda))^2$; and $\Gamma(\lambda) = 2\wp_1(\lambda)\wp_2(\lambda)\kappa_1(\lambda) + \kappa_2(\lambda)\wp_2(\lambda)$.

Since $\Phi(\lambda_{hb})\Gamma(\lambda_{hb}) + \Psi(\lambda_{hb})\Theta(\lambda_{hb}) = -2S_2(b_{22}{}^{[2]})^2[(b_{11}{}^{[2]})^2 - 2(b_{22}{}^{[2]})^2] \neq 0)$, we have

$$\frac{\mathrm{d}}{\mathrm{d}\lambda}(Re(\mu_j(\lambda)))_{\lambda=\lambda_{hb}} = \frac{\Phi\Gamma + \Psi\Theta}{\Psi^2 + \Phi^2}\Big|_{\lambda=\lambda_{hb}} \neq 0,$$

and $\mu_3(\lambda_{hb}) = -\kappa_1(\lambda_{hb}) \neq 0$.

This completes the proof. □

Appendix B

If J is a three-order matrix given by

$$J = \begin{pmatrix} b_{11} & b_{12} & b_{13} \\ b_{21} & b_{22} & b_{23} \\ b_{31} & b_{32} & b_{33} \end{pmatrix},$$

then its second computed matrix is given (see Li *et al.* [30], p.212) by

$$J^{[2]} = \begin{pmatrix} b_{11} + b_{22} & b_{23} & -b_{13} \\ b_{32} & b_{11} + b_{33} & b_{12} \\ -b_{31} & b_{21} & b_{22} + b_{33} \end{pmatrix} = (\bar{b}_{ij})_{3\times 3}(say).$$

Appendix C

From dynamical system (2), it can be easily observed that $\dot{S} \geq 0 \forall$ t; otherwise, if at any time t, $\dot{S} < 0$, then $S(t) \to 0$ as $t \to \infty$, and immediately we have $(I(t), P(t)) \to (0, 0)$ as $t \to \infty$, and the system tends to $E_0(0, 0, 0)$. Thus, the three populations go to extinction, and the system could not attain the axial equilibrium $E_1(K, 0, 0)$. Therefore, if the equilibrium $E_1(K, 0, 0)$ exists, it is clear that $\dot{S} \geq 0 \forall t$. Again, in section 2.5, we have shown that $S(t) \leq K \forall t$.