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INSTITUTE OF PHYSICS PUBLISHING NONLINEARITY

Nonlinearity **17** (2004) 1101–1116 PII: S0951-7715(04)67126-X

Infection in prey population may act as a biological control in ratio-dependent predator–prey models

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Received 28 July 2003, in final form 30 January 2004
Published 2 April 2004
Online at stacks.iop.org/Non/17/1101
DOI: 10.1088/0951-7715/17/3/018

Recommended by J A Glazier

Abstract

A ratio-dependent predator–prey model with infection in prey population is proposed and analysed. The behaviour of the system near the biological feasible equilibria is observed. The conditions for which no trajectory can reach the origin following any fixed direction or spirally are worked out. We investigate the criteria for which the system will persist. It is observed that the introduction of an infected population in the classical ratio-dependent predator–prey model may act as a biological control to save the population from extinction.

Mathematics Subject Classification: 34D05, 34K20, 92D25

1. Introduction

After the pioneering studies of Alfred James Lotka and Vito Volterra in the mid-1920s for predator–prey interactions, prey-dependent predator–prey models were studied extensively (see, e.g., Freedman (1980), Murray (1989) and references therein). Similarly, epidemiological models have also received much attention after the seminal model of Kermack–McKendrick on SIRS (susceptible–infective–removed–susceptible) systems. There are so many references in this context that we are unable to cite all of them (see, e.g., Bailey (1975), Anderson (1991) and references therein).

[4] The authors are extremely sorry to announce that the first author of this paper, Professor Ovide Arino, passed away and this paper is dedicated to his memory.
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Ecology and epidemiology are major fields of study in their own right, but there are some common features between these systems. It is interesting and important from the biological point of view to study ecological systems under the influence of epidemiological factors. Quite a good number of studies has already been performed in eco-epidemiological systems (see, e.g., Hadeler and Freedman (1989), Beltrami and Carroll (1995), Venturino (1995), Chattopadhyay and Arino (1999), Chattopadhyay and Bairagi (2001), Chattopadhyay and Pal (2002)). Most of the studies on such eco-epidemiological systems are based on prey-dependent models. As far as our knowledge goes, no work has been carried out on such systems with a ratio-dependent functional response. This paper deals with an eco-epidemiological model with disease in the prey population and functional responses following the law of ratio-dependent theory.

Before introducing the model, we would like to present a brief historical account of the biological relevance of the classical prey-dependent model and the controversial (Abrams 1994, Abrams and Ginzburg 2000) ratio-dependent model.

The classical prey-dependent predator–prey model exhibits not only the well known 'paradox of enrichment' formulated by Hairston *et al* (1960) and Rosenzweig (1969) but also the so-called 'biological control paradox', which was recently discussed by Luck (1990). The analysis of the ratio-dependent predator–prey model shows that it will produce neither the paradox of enrichment nor the biological control paradox (see Hsu *et al* (2001a, b)). It also allows mutual extinction as a possible outcome of a predator–prey interaction (Kuang and Beretta 1998, Jost *et al* 1999). Ratio-dependent models require high population densities for both prey and predator while the most interesting dynamics is near the origin (see, e.g., Xiao and Ruan (2001)). The ratio-dependent theory has been successfully used by Hsu *et al* (2003) in the food chain model.

The 'paradox of enrichment' states that enriching a predator–prey system (increasing the carrying capacity) will cause an increase in the equilibrium density of the predator but not in that of the prey, and will finally destabilize the positive equilibrium. As a result, it increases the possibility of stochastic extinction of the predator. But what is observed in nature is that enriching the system increases the prey density, does not destabilize a stable steady state and fails to increase the amplitude of oscillations in systems that already cycle (Abrams and Walter 1996).

The so-called 'biological control paradox' states that we cannot have a low and stable prey equilibrium density, which contradicts many examples of successful biological controls where the prey is maintained at low densities compared with its carrying capacity (Arditi and Berryman 1991). A further example is Cactoblastis-Opuntia in Australia, where the crucial factor seems to be Pseudointerference (see May (1981)); here, biological control has worked and resulted in low and stable pest densities. So this paradox is a pure artefact, created by simplifying assumptions on functional response. For the rest, the paradox of enrichment exists, but only in systems where one predator–prey pair exists in isolation, and the predictions radically change when they are embedded in a simple food chain model (see Oksanen and Oksanen (2000)). Most natural systems are indeed much more complex than this, since predators are exposed to various degrees of facultative secondary carnivory (intraguild predation) and interacting with $(n+1)$ other predator–prey systems in various ways. This clearly indicates that the paradox of biological control is not intrinsic to predator–prey interaction.

When we have good data on simple and fairly isolated predator–prey systems, like Mary Power's catfish–alga system in Panamanian streams (Oksanen *et al* 1995), we see that more resources for the prey at equilibrium will, indeed, mean more predators but no more prey. In systems where the Holling disc is a reasonable assumption for predators (e.g. the weasel–vole system found in boreal Europe, in which voles have no hiding places where weasels could not follow them and weasels have no significant alternative resources), we do indeed find large

amplitude population cycles, which have the signature of a carnivore–herbivore cycle (see Oksanen *et al* (2000, 2001)). As predicted by the paradox of enrichment, these cycles are found in relatively productive taiga areas and in the most productive parts of the tundra, while voles inhabiting less productive tundra areas are relatively stable (Oksanen *et al* 1999). In high alpine barrens, where predators are practically absent, violent cycles are found again, but now with rodent time trajectories showing the fingerprints of a predator (Turchin *et al* 2000). The shift in the dynamic position of herbivores along productivity gradients thus corroborates the predictions of simple food chain models (see Oksanen and Oksanen (2000)). Recently, models with such a prey-dependent response function have been facing challenges both from biological and physiological researchers (see, e.g., Arditi and Ginzburg (1989), Arditi and Berryman (1991), Akçakaya (1992), Gutierrez (1992)). The main criticism which has been raised against the ratio-dependent approach is that its proponents have made things too easy by providing easy explanations of positive correlations found in nature in an *ad hoc* manner, without having a credible mechanism that could produce ratio-dependence (the time scale argument initially used is pathetic as it tacitly presumes that current reproductive output of a predator would depend on future prey density). It also seems to fail to account for the cases where the paradoxes of prey dependence are actually found. Indeed, prey-dependent and ratio-dependent models are extremes or limiting cases; prey-dependent models focusing entirely on the daily energy balance of predators, ratio-dependent models presupposing that prey are easy to find and that predator dynamics is, in essence, governed by direct density dependence, with prey densities determining the sizes of defended territories. In nature, both aspects probably influence predator–prey dynamics, and the question regarding which of the two extremes is closer to reality in which systems is wide open. Moreover, pursuit of the prey-dependent approach has proved more fertile, since its 'paradoxes' seem to be quite realistic where the premises for their existence are found, but here opinions may differ and there is no reason to close one door or another. Thus, it is definitely good to develop both approaches towards greater realism by including other interactions. Based on biological and physiological evidence, some researchers argue that the functional response in a predator–prey model should be based on the ratio-dependent theory, especially when predators have to search for food, and the per capita predator growth rate should be a function of the ratio of prey to predator abundance.

Arditi and Ginzburg (1989) first proposed the following Michaelis–Menten type, ratio-dependent predator–prey system, which was then studied extensively by a number of authors (see, e.g., Kuang and Beretta (1998), Jost *et al* (1999), Hsu *et al* (2001a, b))

$$\begin{aligned} \frac{\mathrm{d}x}{\mathrm{d}t} &= rx\left(1-\frac{x}{K}\right)-\alpha\frac{xP}{k_1P+x} \\ \frac{\mathrm{d}P}{\mathrm{d}t} &= P\left(-\delta_2+\frac{fx}{k_1P+x}\right) \end{aligned} \qquad x(0)>0,\ P(0)>0. \tag{1}$$

Disease in ecological systems cannot be ignored. From this fact, we now modify the above model (1) by introducing a transmissible disease in the prey population. We make the following assumptions:

(A1) In the absence of disease the prey population grows in logistic fashion with carrying capacity $K > 0$ and an intrinsic growth rate constant $r > 0$.

(A2) In the presence of disease the prey population is divided into two parts, the susceptible prey ($S(t)$) and the infected prey ($I(t)$). Therefore, at time t the total prey population is

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

(A3) We assume that only the susceptible prey population is capable of reproducing and contributing to its carrying capacity. We also assume that the infected prey do not grow, recover and reproduce. The experiment on dinoflagellate *Noctiluca scintillans* (miliaris) in the German Bight by Uhlig and Sahling (1992) indicated that the cells become damaged, and they neither feed anymore nor reproduce. The model of Hamilton *et al* (1990) showed that no infected individuals contribute in the reproduction process; infection rather reduces the remaining capacity due to the inability to compete for resources. Beltrami and Carroll (1995) also used this theory to establish the role of viral disease in recurrent phytoplankton blooms. Thus, one can assume that the growth term of the susceptible population follows only the law of logistic growth.

(A4) We also assume that the disease transmission follows the simple law of mass-action.

With the above assumptions, model (1) leads to the following set of ordinary differential equations

$$\begin{aligned}
\frac{\mathrm{d}S}{\mathrm{d}t} &= rS\left(1-\frac{S}{K}\right)-\alpha_S\frac{SP}{k_1P+S+I}-\lambda SI\\
\frac{\mathrm{d}I}{\mathrm{d}t} &= \lambda SI-\alpha_I\frac{IP}{k_1P+S+I}-\delta_1 I \qquad\qquad S(0)>0,\ I(0)>0,\ P(0)>0. \qquad (2)\\
\frac{\mathrm{d}P}{\mathrm{d}t} &= \frac{k(\alpha_S S+\alpha_I I)P}{k_1P+S+I}-\delta_2 P
\end{aligned}$$

Here, α_S and α_I are the searching efficiency constants or the predation rate on the susceptible and infective prey, respectively. It is observed in nature that α_S can be less or, in contrast, larger than α_I depending on the type of parasitism. At this point, we would like to mention some relevant books: e.g. Dawkins '*The Extended Phenotype*' (1982), Combes '*Les associations du vivant*' (2001a) and Combes '*Parasitism, The Ecology and Evolution of Intimate Interactions*' (2001b). α_S/k_1 and α_I/k_1 are the maximum per capita capturing rate for predator on susceptible prey and infected prey, respectively. λ is the force of infection, δ_1 and δ_2 are the death rates of infected prey and predator, respectively, and k is a conversion rate.

This work can be seen as a continuation of the work done by two of the authors (Chattopadhyay and Arino 1997a) on a predator–prey system with disease in the prey. The main modification here is that we take into account the ratio-dependent theory, which has some important features for the dynamics of the system.

Kuang and Beretta (1998), Jost *et al* (1999), Xiao and Ruan (2001), observed that the dynamics of system (1) near the origin is more complicated since the vector field is not well defined at that point and cannot be linearized around this point. There exist numerous kinds of topological structures in the vicinity of the origin (see, e.g., Berezovskaya *et al* (2001), Xiao and Ruan (2001)). This is the main reason for ratio-dependent models possibly to have complicated rich dynamics. Kuang and Beretta (1998) proved that total extinction is also possible. Jost *et al* (1999) proved that the origin can be a saddle-point or an attractor. Xiao and Ruan (2001) analysed a situation where solutions reach the origin following a fixed direction. The results obtained in the three above-mentioned papers are roughly complementary to each other. We would like to mention that the diseased population cannot be ignored in such models and has some influence on the dynamics. We study the dynamics of the zero equilibrium starting from the first positive quadrant, and, especially, carry out a thorough study near the origin in the sense that we have studied the possibilities of reaching this critical point following any fixed direction in the domain of interest. In this paper, we use a reduction principle which allows us to reduce the system to a two-dimensional system where the Poincaré–Bendixson result can be applied. We find suitable conditions on the parameters such that we cannot reach the origin

spirally. We have also pointed out that total extinction can be controlled by the diseased prey population and that under some conditions, the model is persistent.

This paper is organized as follows: section 2 gives preliminary results; different equilibria are given in section 3; the behaviour of the system around $E_0(0, 0, 0)$ is discussed in section 4. In section 5, we show that the infected prey may act as a system saver. Persistence results are presented in section 6. Section 7 deals with the problem of finding some suitable conditions for which there is no periodic solution around the positive equilibrium and this paper ends with a conclusion.

2. Preliminary results

We first observe that the right-hand side of system (2) is a smooth function of the variables (S, I, P) and the parameters in the positive octant, as long as the sum of these quantities is non-zero. So, local existence and uniqueness properties hold in the positive octant.

From system (2), it follows that $S = 0$ (resp. $I = 0$, $P = 0$) is an invariant subset, that is, $S \equiv 0$ (resp. $I \equiv 0$, $P \equiv 0$) if and only if $S(t) = 0$ (resp. $I(t) = 0$, $P(t) = 0$) for some t. Thus, $S(t) > 0$ (resp. $I(t) > 0$, $P(t) > 0$) for all t if $S(0) > 0$ (resp. $I(0) > 0$, $P(0) > 0$).

So, if $I = 0$, which corresponds to a system without disease, then the system reduces to a two-dimensional one which has been studied extensively by several authors (see, e.g., Kuang and Beretta (1998), Jost *et al* (1999), Xiao and Ruan (2001)).

We first prove the boundedness of system (2).

Lemma 1. *All the solutions of system (2) which initiate in $\mathbb{R}^3_+$ are bounded, with ultimate bound.*

Proof. We define a function

$$W(t) = kS(t) + kI(t) + P(t). \tag{3}$$

Taking the time derivative of W along the solutions of (2), we have

$$\frac{\mathrm{d}W}{\mathrm{d}t}(t) = rkS(t)\left(1 - \frac{S(t)}{K}\right) - k\delta_1 I(t) - \delta_2 P(t).$$

For any positive constant μ (>0), we have

$$\frac{\mathrm{d}W}{\mathrm{d}t} + \mu W = S\left\{rk + \mu - rk\frac{S}{K}\right\} + (\mu - k\delta_1)I + (\mu - \delta_2)P.$$

If we take μ such that $0 < \mu < \min(k\delta_1, \delta_2)$, then, we obtain

$$\frac{\mathrm{d}W}{\mathrm{d}t} + \mu W \leqslant \frac{K}{rk}\left(\frac{rk + \mu}{2}\right)^2 = M \tag{4}$$

where M is the maximum value of the function $S\{rk + \mu - rk(S/K)\}$.

From (4) we have

$$\frac{\mathrm{d}W}{\mathrm{d}t} \leqslant -\mu W + M$$

which implies that

$$W(t) \leqslant \mathrm{e}^{-\mu t}W(0) + \frac{M}{\mu}(1 - \mathrm{e}^{-\mu t}) \leqslant \max\left(W(0), \frac{M}{\mu}\right).$$

Moreover, we have

$$\limsup_{t\to\infty} W(t) \leqslant \frac{M}{\mu}$$

which is independent of the initial condition. □

3. Equilibria

System (2) has the following equilibria

$E_0(0, 0, 0)$, $E_1(K, 0, 0)$, $E_2(S_2, 0, P_2)$

with

$$S_2 = K\left(1 - \frac{k\alpha_S - \delta_2}{kk_1 r}\right) \quad \text{and} \quad P_2 = r\left(1 - \frac{S_2}{K}\right)\frac{kS_2}{\delta_2}$$

$E_3(S_3, I_3, 0)$

with

$$S_3 = \frac{\delta_1}{\lambda} \quad \text{and} \quad I_3 = \frac{r}{\lambda}\left(1 - \frac{\delta_1}{\lambda K}\right)$$

and an interior equilibrium $E^*(S^*, I^*, P^*)$ where

$$I^* = -\left(\frac{r}{\lambda K} + \frac{\alpha_S}{\alpha_I}\right)S^* + \left(\frac{r}{\lambda} + \frac{\alpha_S\delta_1}{\lambda\alpha_I}\right), \qquad P^* = \frac{(k\alpha_S - \delta_2)S^* + (k\alpha_I - \delta_2)I^*}{\delta_2 K}$$

and S^* satisfies the following equation

$$AS^2 + BS + C = 0$$

with

$$\begin{aligned} A &= kk_1 r\alpha_I\lambda \\ B &= -k\{r\alpha_I(\alpha_I + k_1\delta_1) + k_1K(r\alpha_I + \alpha_S\delta_1)\lambda + \delta_2(-r\alpha_I + k\lambda(\alpha_I - \alpha_S))\} \\ C &= K(r\alpha_I + \alpha_S\delta_1)\{k(\alpha_I + k_1\delta_1) - \delta_2\}. \end{aligned}$$

It is easy to see that a necessary and sufficient condition for the existence of E_2 is

$$0 < k\alpha_S - \delta_2 < kk_1 r.$$

From the expression of I_3, it is clear that a necessary and sufficient condition for the existence of E_3 is

$$\lambda K - \delta_1 > 0.$$

It can be easily shown that E^* exists and is unique if the following set of inequalities hold simultaneously

$$k < 1, \qquad \delta_1 < K\lambda < \frac{r\alpha_I(1-k)}{k(\alpha_I + \alpha_S)}, \qquad \delta_2 < \min\left(k\alpha_I, k\alpha_S, \frac{r(k\alpha_I + \alpha_S)(K\lambda - \delta_1)}{r\alpha_I + \alpha_S\lambda K}\right)$$
$$r > \frac{k\alpha_S\delta_1 + \alpha_I(\delta_2 + k\delta_1)}{\alpha_I(1-k)}.$$

4. Behaviour of the system around $E_0(0, 0, 0)$

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

$$\frac{\mathrm{d}X}{\mathrm{d}t} = H(X(t)) + Q(X(t)) \tag{5}$$

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

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

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

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

in the vicinity of the origin.

Throughout the section, $\|.\|$ denotes the Euclidian norm on $\mathbb{R}^N$ 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 functions H_i and Q_i $(i = 1, 2, 3)$ are given by

$$H_1(X) = rx_1 - \alpha_S \frac{x_1 x_3}{k_1 x_3 + x_1 + x_2}, \qquad H_2(X) = -\alpha_I \frac{x_2 x_3}{k_1 x_3 + x_1 + x_2} - \delta_1 x_2$$
$$H_3(X) = \frac{k(\alpha_S x_1 + \alpha_I x_2) x_3}{k_1 x_3 + x_1 + x_2} - \delta_2 x_3$$
$$Q_1(X) = -r\frac{x_1^2}{K} - \lambda x_1 x_2, \qquad Q_2(X) = \lambda x_1 x_2, \qquad Q_3(X) = 0.$$

Let $X(t)$ be a solution of system (5). Assume that $\liminf_{t\to\infty} \|X(t)\| = 0$, and X is bounded. One can extract from the family $(X(t + .))_{t\geqslant 0}$ sequences $X(t_n + .)$, $t_n \to \infty$, such that $X(t_n + .) \to 0$ locally uniformly on $s \in \mathbb{R}$.

Define

$$y_n(s) = \frac{X(t_n + s)}{\|X(t_n + s)\|}. \tag{6}$$

Recall that

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

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

$$Q(X) = \|X\|^2 O(1). \tag{7}$$

We have

$$\frac{dX(t_n + s)}{ds} = H(X(t_n + s)) + Q(X(t_n + s)). \tag{8}$$

From (6), we have

$$X(t_n + s) = y_n(s)\|X(t_n + s)\| = y_n(s) \cdot \langle X(t_n + s), X(t_n + s)\rangle^{1/2}. \tag{9}$$

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

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

in (9), we obtain

$$\frac{dX(t_n + s)}{ds} = \frac{dy_n(s)}{ds}\|X(t_n + s)\| + \frac{y_n(s)}{\|X(t_n + s)\|}\left\langle X(t_n + s), \frac{dX(t_n + s)}{ds}\right\rangle.$$

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

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

which is equivalent to

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

Clearly, y_n is bounded, $\|y_n(s)\| = 1$, $\forall s$, and $\mathrm{d}y_n/\mathrm{d}s$ is bounded too. So, applying the Ascoli–Arzela theorem (see, e.g., Brezis (1983)), one can extract from y_n a subsequence—also denoted by y_n—which converges locally uniformly on $\mathbb{R}$ towards some function y, such that

$$\|X(t_n+s)\|[Q(y_n(s)) - (y_n(s), Q(y_n(s)))y_n(s)] \underset{t_n\to\infty}{\to} 0$$

and y satisfies the following system:

$$\frac{\mathrm{d}y}{\mathrm{d}t} = H(y(t)) - (y(t), H(y(t)))y(t), \qquad |y(t)\| = 1, \quad \forall t. \tag{10}$$

Equation (10) is defined for all $t \in \mathbb{R}$.

Let us, for a moment, focus on the study of equation (10). The steady states of H are vectors V satisfying

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

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

$$H(V) = \mu V \tag{11}$$

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

These stationary solutions correspond to fixed directions that the trajectories of equation (10) may reach asymptotically.

Equation (11) can be written as

$$[(\mu - r)v_1 + (\mu - r)v_2 + (\alpha_S + k_1\mu - k_1 r)v_3]v_1 = 0 \tag{12}$$

$$[(\mu + \delta_1)v_1 + (\mu + \delta_1)v_2 + (\alpha_I + k_1\mu + k_1\delta_1)v_3]v_2 = 0 \tag{13}$$

$$[(\mu + \delta_2 - k\alpha_S)v_1 + (\mu + \delta_2 - k\alpha_I)v_2 + k_1(\mu + \delta_3)v_3]v_3 = 0. \tag{14}$$

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

Case 1. $v_1 = 0$

(a) $v_2 = 0$ and $v_3 \neq 0$
In this case, there is a possibility of reaching the origin following the P-axis, with $\mu = -\delta_3$.
(b) $v_2 \neq 0$ and $v_3 = 0$
In this case also, there is a possibility of reaching zero following the I-axis, with $\mu = -\delta_1$.
(c) $v_2 \neq 0$ and $v_3 \neq 0$
In this case, we obtain different results depending on the parameters.

Subcase 1. If $kk_1 < 1$ then

(i) We reach the origin if $kk_1\delta_1 + k\alpha_I - \delta_2 < 0$.
(ii) We cannot reach the origin if $kk_1\delta_1 + k\alpha_I - \delta_2 > 0$.

Subcase 2. If $kk_1 > 1$ then the conclusion is the reverse of subcase 1.

Case 2. $v_1 \neq 0$

(a) $v_2 = 0$ and $v_3 = 0$
In this case, we cannot reach the origin following the S-axis; that is to say that the S-axis is not a fixed direction that the trajectories can follow to reach zero.

(b) $v_2 = 0$ and $v_3 \neq 0$
In this case, we have two possibilities

(i) If E_2 exists and $kk_1 < 1$ then the SP-plane can be followed by the trajectories to reach the origin.

(ii) If $kk_1 > 1$ then there is no possibility of reaching the origin following the SP-plane.

(c) $v_2 \neq 0$ and $v_3 = 0$
In this case, there is no possibility of reaching the origin following the SI-plane.

(d) $v_2 \neq 0$ and $v_3 \neq 0$
In this case, there are two subcases

(i) $\alpha_S \leqslant \alpha_I$
In this case, there is no possibility of going to the origin following a fixed direction that is contained in the positive octant.

(ii) $\alpha_S > \alpha_I$
Under this condition, the trajectories may follow a fixed direction that is contained in the positive octant.

Now, we would like to show that under some suitable conditions, no orbit of system (2) tends to the critical point spirally. To do that, we return to equation (10).

Proposition 2. *If we assume that*

$$H_i(X) = X_i \bar{H}_i(X)$$

for all i, then equation (10) preserves positiveness. So, every non-zero solution of equation (10) with closure at a positive distance from at least one of the $(N-1)$-coordinate hyperplanes can in fact be seen as a solution of an o.d.e. in $\mathbb{R}^{N-1}$.

Proof. Let y be a non-zero solution of

$$\frac{\mathrm{d}y}{\mathrm{d}t} = H(y(t)) - (H(y(t)), y(t))y(t).$$

With no loss of generality, assume that $y_N(t) \geqslant m > 0$; then, we may write

$$y_N = \sqrt{1 - y_1^2 - \cdots - y_{N-1}^2}$$

$\tilde{y} = (y_1, \ldots, y_{N-1})$ satisfies a system of o.d.e of the form

$$\frac{\mathrm{d}\tilde{y}}{\mathrm{d}t}(t) = \tilde{G}(\tilde{y}(t))$$

in an open subset of $\mathbb{R}_+^{N-1}$ in which $\tilde{G}$ is C^1. □

Corollary 3. *In the case when $N = 3$, the reduced system is a planar o.d.e. to which the Poincaré–Bendixson theorem applies.*

We now return to the equation under study with y being the limit of a sequence $X(s+t_n)/\|X(s+t_n)\|$.

Let us investigate the asymptotic behaviour of $y(t)$. Either there exists $j \in \{1,2,3\}$ such that $y_j(t) \geqslant m > 0 \ \forall t$. In this case, the equation can be reduced to its planar projection onto (y_i, y_k), $i, k \neq j$, or $\liminf_{t\to\infty} y_i(t) = 0$, for $i = 1, 2, 3$.

We have

$$\frac{\mathrm{d}y}{\mathrm{d}t}(t) = H(y(t)) - (y(t), H(y(t)))y(t) \qquad \|y(t)\| = 1.$$

If we put $y = (y_1, y_2, y_3)$ and $\tilde{y} = (y_1, y_2)$, then we can write $y_3 = \sqrt{1 - y_1^2 - y_2^2}$ and reduce the system to the following two-dimensional system:

$$\tilde{y}'(t) = \bar{H}(\tilde{y}(t)) - \tilde{y}(t)[(\tilde{y}(t), \bar{H}(\tilde{y}(t))) + \sqrt{1-\tilde{y}^2(t)}\bar{H}_3(\tilde{y}(t))] \qquad \|\tilde{y}(t)\| < 1 \tag{15}$$

with

$$\bar{H}((y_1, y_2)) = (H_1, H_2)\Big(y_1, y_2, \sqrt{1 - y_1^2 - y_2^2}\Big)$$

$$\bar{H}_3((y_1, y_2)) = H_3\Big(y_1, y_2, \sqrt{1 - y_1^2 - y_2^2}\Big).$$

Now, we will use the Poincaré–Bendixson criteria to show that under some suitable conditions, if a solution of (15) tends to the origin then it must tend to it along a fixed direction.

We define

$$f_1((y_1, y_2)) = H_1\Big(y_1, y_2, \sqrt{1 - y_1^2 - y_2^2}\Big) - y_1 g((y_1, y_2))$$

$$f_2((y_1, y_2)) = H_2\Big(y_1, y_2, \sqrt{1 - y_1^2 - y_2^2}\Big) - y_2 g((y_1, y_2))$$

$$g((y_1, y_2)) \ = ((y_1, y_2), \bar{H}((y_1, y_2))) + \sqrt{1-\tilde{y}^2(t)}\bar{H}_3(\tilde{y}(t)).$$

We have

$$y_1(t)\frac{\mathrm{d}y_2}{\mathrm{d}t}(t) - y_2(t)\frac{\mathrm{d}y_1}{\mathrm{d}t}(t) = y_1(t)H_2\Big(\tilde{y}(t), \sqrt{1-\tilde{y}^2(t)}\Big) - y_2(t)H_1\Big(\tilde{y}(t), \sqrt{1-\tilde{y}^2(t)}\Big).$$

Using the expressions for H_1 and H_2, we find that

$$\frac{\mathrm{d}}{\mathrm{d}t}\left[\ln\left(\frac{y_2(t)}{y_1(t)}\right)\right] = (\alpha_S - \alpha_I)\frac{\sqrt{1-\tilde{y}^2(t)}}{y_1 + y_2 + k\sqrt{1-\tilde{y}^2(t)}} - (\delta_1 + r). \tag{16}$$

From equation (16), we can conclude that if $\alpha_S \leqslant \alpha_I$ then we cannot have a periodic solution because $(\mathrm{d}/\mathrm{d}t)[\ln(y_2(t)/y_1(t))]$ cannot change signs. In the other case, when $\alpha_S > \alpha_I$, suppose that $\tilde{y}(t)$ is a periodic solution, then, from equation (16), we can see that a necessary condition is that on the minimum point; the quantity $(\mathrm{d}/\mathrm{d}t)[\ln(y_2(t)/y_1(t))]$ vanishes and is less than the maximum value of the right-hand side of equation (16), which implies that

$$0 \leqslant \frac{(\alpha_S - \alpha_I)}{k} - (\delta_1 + r). \tag{17}$$

We conclude that if $\alpha_I > \alpha_S - k(\delta_1 + r)$, then we cannot reach the origin spirally.

Now we are in a position to summarize the above results in the following two theorems.

Theorem 4. *If the predation rate on the infected prey is higher than or equal to that of the susceptible prey, then any trajectory can reach the origin from the interior following a fixed direction.*

Theorem 5. *If the difference of the predation rate has an upper threshold value, given by*

$$\alpha_S - \alpha_I < k(\delta_2 + r)$$

then any trajectory can reach the origin spirally from the interior.

Remark 6. In the paper of Jost *et al* (1999), $(0, 0, 0)$ is a global attractor if $Q + R < S$ (see proposition 4.1, p 27), which in terms of our model parameter corresponds to $\alpha_S > k_1(r + \delta_2)$.

We find that if $\alpha_S \leqslant \alpha_I$ then no trajectory can reach $E_0(0, 0, 0)$. So, if we set

$$k_1(r + \delta_2) < \alpha_S \leqslant \alpha_I$$

then total extinction is not possible with our model while it is possible with Jost *et al*'s model.

Xiao and Ruan (2000) studied the behaviour of zero equilibrium as $t \to +\infty$ or $t \to -\infty$ depending on the parameters in the interior of the first octant, but they did not discuss the behaviour of this equilibrium starting from the rest of the domain. Our analysis takes this into account. So, our results include the whole topological structure near $(0, 0, 0)$ starting from any position of the domain of interest. (In Ruan's paper, the parameters b/a, resp. c, m, d and f, correspond to K, resp. α_S, k_1, δ_2 and k.)

Kuang and Beretta (see theorem 2.6) proved that under some conditions, the origin is globally asymptotically stable; that is to say, the system goes to total extinction. Their conditions can be formulated in terms of our system parameters as follows:

$$k \geqslant \frac{\delta_2}{\alpha_S - k_1 r} \qquad \text{and} \qquad \alpha_S > k_1 r.$$

5. Role of infected population

In the remark of section 4 we find that the predator–prey system considered by Jost *et al* (1999) and Kuang and Beretta (1998) goes to total extinction under some parametric conditions. It is to be noted here that our system (1) is equivalent to their system, considered in the above-mentioned papers, for $I = 0$.

In the following theorem we show that introduction of infected prey into the predator–prey system prevents total extinction and may act as a biological control.

Theorem 7. *Assume that* $\lambda < \lambda^*$, $kk_1 > 1$ *and*

$$\delta_2 > \frac{k\alpha_S}{kk_1 - 1}$$

then the boundary steady state E_2 *is locally asymptotically stable (LAS).*

Proof. Local stability of E_2 depends on the sign of a_{22} and the eigenvalues of the following matrix

$$V_2^0 = \begin{pmatrix} a_{11} & a_{13} \\ a_{31} & a_{33} \end{pmatrix}.$$

We have

$$\det(V_2) = a_{22}\left[\frac{\delta(k\alpha_S - \delta_2)(kk_1 r - k\alpha_S + \delta_2)}{k_1 k^2 \alpha_S}\right]$$

$$\operatorname{tr}(V_2) \quad = a_{22} - r - \frac{(k\alpha_S - \delta_2)\,[(kk_1 - 1)\delta_2 - k\alpha_S]}{k_1 k^2 \alpha_S}.$$

Existence of E_2 implies that $\det(V_2)$ has the same sign as that of a_{22}.

Now if $\lambda < \lambda^*$, then $a_{22} < 0$. In this case, the stability of E_2 depends on the eigenvalues of V_2^0.

We have

$$\det(V_2^0) = \frac{1}{a_{22}} \det(V_2)$$

$$\operatorname{tr}(V_2^0) = \frac{1}{a_{22}} \operatorname{tr}(V_2).$$

We know that $\det(V_2^0) > 0$. Sufficient conditions to have $\mathrm{tr}(V_2^0) < 0$ are

$$kk_1 > 1$$
$$\delta_2 > \frac{k\alpha_S}{kk_1 - 1}.$$

□

Remark. E_2 is a disease-free equilibrium, and if the conditions given in theorem 7 are true, then this disease-free equilibrium is stable and cannot reach $(0, 0, 0)$. This means that under these conditions total extinction is not possible and hence the introduction of disease into the system may act as a biological control to save the population from extinction.

6. Persistence results

We have already proved that system (2) is uniformly bounded. Now, in order to prove the persistence of the system, we will first show that all the boundary equilibria are repellers.

Theorem 8. *If the following conditions hold*

(i) $\alpha_I \geqslant \alpha_S$
(ii) $0 < k\alpha_S - \delta_2 < kk_1 r$
(iii) $\lambda > \lambda^*$

where λ^* *is given in (18), then the system is persistent.*

Proof. After computing the variational matrix associated with $E_1(K, 0, 0)$, we find the following eigenvalues: $\mu_1 = -r$, $\mu_2 = \lambda K - \delta_1$ and $\mu_3 = k\alpha_S - \delta_2$.

We conclude that existence of E_2 or E_3 implies that E_1 is unstable.

For the equilibrium point E_2, the entries of the Jacobian matrix V_2 computed at E_2 are as follows:

$$a_{11} = \alpha_S \frac{S_2 P_2}{(k_1 P_2 + S_2)^2} - \frac{r S_2}{K}, \qquad a_{12} = \alpha_S S_2 P_2 \frac{1}{(k_1 P_2 + S_2)^2} - \lambda S_2,$$
$$a_{13} = -\frac{\alpha_S S_2^2}{(k_1 P_2 + S_2)^2}, \qquad a_{21} = 0, \qquad a_{22} = \lambda S_2 - \frac{\alpha_I P_2}{(k_1 P_2 + S_2)} - \delta_1, \qquad a_{23} = 0,$$
$$a_{31} = \frac{kk_1 \alpha_S P_2^2}{(k_1 P_2 + S_2)^2}, \qquad a_{32} = \frac{kP_2\{\alpha_I k_1 P_2 + (\alpha_I - \alpha_S) S_2\}}{(k_1 P_2 + S_2)^2}, \qquad a_{33} = \frac{k\alpha_S S_2^2}{(k_1 P_2 + S_2)^2} - \delta_2.$$

Since $a_{21} = a_{23} = 0$, we see that a_{22} is an eigenvalue of V_2 and can be written as

$$a_{22} = \frac{K\lambda\alpha_S(kk_1 r - k\alpha_S + \delta_2) - r\alpha_I(k\alpha_S - \delta_2) - kk_1 r\delta_1\alpha_S}{kk_1 r\alpha_S}.$$

We write

$$\lambda^* = \frac{r\alpha_I(k\alpha_S - \delta_2) + kk_1 r\delta_1\alpha_S}{K\alpha_S(kk_1 r - k\alpha_S + \delta_2)}. \tag{18}$$

It is easy to see that if $\lambda > \lambda^*$ then E_2 is unstable (as $a_{22} > 0$).

From the variational matrix computed at E_3 we conclude that if the sufficient condition

$$k \min(\alpha_I, \alpha_S) - \delta_2 > 0$$

is true then E_3 is unstable (because one of the eigenvalues is $(k(\alpha_S S_3 + \alpha_I I_3)/(S_3 + I_3)) - \delta_2 > 0$).

Thus, we see that all the boundary equilibria of system (1) are repellers if the conditions stated in theorem 8 hold. □

7. Conditions for non-existence of periodic solutions around E^*

In this section, we would like to prove that under some suitable conditions, there is no periodic solution of system (2) around the positive equilibrium E^*.

To prove this, the following criteria by Li and Muldowney (1993) can be applied. Consider the general autonomous ordinary differential equation

$$\frac{\mathrm{d}X}{\mathrm{d}t} = F(X(t)) \tag{19}$$

where F is a C^1 function in some open subset of $\mathbb{R}^N$ with values in $\mathbb{R}^N$. Denote by $J = (\partial F/\partial X)$ the Jacobian matrix of (19). Denote by $J^{[2]}$ the $\binom{N}{2} \times \binom{N}{2}$ matrix which is the second additive compound matrix associated with the Jacobian matrix J (see appendix for more details), and recall that if $X \in R^N$ then the corresponding logarithmic norm of $J^{[2]}$ (that we denote by $\mu_\infty(J^{[2]})$) endowed by the vector norm $|X|_\infty = \sup_i |X_i|$ is

$$\mu_\infty(J^{[2]}) = \sup\left\{ \frac{\partial F_r}{\partial x_r} + \frac{\partial F_s}{\partial x_s} + \sum_{j\neq r,s}\left(\left|\frac{\partial F_r}{\partial x_j}\right| + \left|\frac{\partial F_s}{\partial x_j}\right|\right) : 1 \leqslant r < s \leqslant N \right\} \tag{20}$$

where $\mu_\infty(J^{[2]}) < 0$ implies the diagonal dominance by row matrix $J^{[2]}$. Then, the following holds.

Theorem 9. *A simple closed rectifiable curve that is invariant with respect to system (2) cannot exist if* $\mu_\infty(J^{[2]}) < 0$.

Before we find conditions under which there is no periodic solution, we perform some changes of coordinates to lower the number of parameters in system (2).

We set

$$U = \frac{S}{K}, \qquad V = \frac{I}{K}, \qquad W = \frac{P}{K} \qquad \text{and} \qquad \tau = rt \tag{21}$$

and

$$\alpha_S' = \frac{\alpha_S}{r}, \qquad \alpha_I' = \frac{\alpha_I}{r}, \qquad \delta_1' = \frac{\delta_1}{r}, \qquad \delta_2' = \frac{\delta_2}{r}, \qquad \lambda' = \frac{\lambda K}{r}.$$

Then, system (2) preserves the same form but with $r = 1$ and $K = 1$ and the new parameters mentioned in (21) for which we omit the prime to simplify the notation.

Let us now apply Li–Muldowney's criteria in the new coordinates for the non-existence of periodic solutions of system (2). The logarithmic norm μ_∞, endowed by the norm $|X|_\infty$ of the second additive compound matrix $J^{[2]}$, associated with the Jacobian matrix J, computed on E^*, is negative if and only if the suprema of the following functions satisfy

$$1 - (2-\lambda)S - \lambda I - \delta_1 - \frac{\alpha_S P(2k_1P + S + I)}{(k_1P + S + I)^2} + \frac{kP|I(\alpha_S - \alpha_I) + k_1\alpha_S P| + kPS(\alpha_I - \alpha_S) + k_1 P}{(k_1P + S + I)^2} < 0 \tag{22}$$

$$1 - 2S - \lambda I - \delta + \lambda S + \frac{(S+I)k(\alpha_S S + \alpha_I I) - \alpha_S(k_1P + I)}{(k_1P + S + I)^2} + \frac{\alpha_S PS}{(k_1P + S + I)^2} + \frac{\alpha_I kP(k_1P + ((\alpha_S - \alpha_I)/\alpha_I)S)}{(k_1P + S + I)^2} < 0 \tag{23}$$

$$2\lambda S - \alpha_I P\frac{(k_1P + S)}{(k_1P + S + I)^2} - \delta_1 - \delta_2 + \frac{k(\alpha_S S + \alpha_I I)(S+I) + \alpha_S PS + \alpha_S S(S+I)}{(k_1P + S + I)^2} < 0. \tag{24}$$

Sufficient conditions to satisfy (22), (23) and (24) are, respectively,

$$0 < \lambda < 2 \qquad \text{and} \qquad \delta_1 - 1 - \alpha_S > 0 \tag{25}$$

$$0 < \lambda < 2 \qquad \text{and} \qquad \delta_2 - 1 - \alpha_S \left(k + \frac{1}{2k_1} + \frac{k}{k_1} \right) \geqslant 0 \tag{26}$$

$$0 < 2\lambda < \delta_1 + \delta_2 + \frac{\alpha_I}{2k_1} - \alpha_S \left(k + \frac{1}{2k_1} \right) \qquad \text{and} \qquad \delta_1 + \delta_2 + \frac{\alpha_I}{2k_1} > \alpha_S \left(k + \frac{1}{2k_1} \right). \tag{27}$$

A direct application of Li–Muldowney's method shows that under the conditions (25)–(27), there is no periodic solution for system (2).

8. Conclusion

The classical prey-dependent and ratio-dependent models are well studied. In this paper, we have investigated the dynamical behaviour of a ratio-dependent predator–prey model with infection in the prey population. The proposed model is a modification of the model proposed by Arditi and Ginzburg (1989). The behaviour of the system near the biologically feasible equilibria has been studied. The parametric conditions for which the solutions of the system cannot reach the origin following a fixed direction or spirally have been worked out. All the topological structures near the origin starting from any position of the domain of interest have been taken care of. Moreover, Jost *et al* (1999) and Kuang and Beretta (1998) also studied the model of Arditi and Ginzburg; they obtained the conditions for which the whole population may go to extinction. In contrast, we have observed that introduction of diseased prey into the system may save the population from extinction. Thus, we may conclude that the infected prey population in a classical ratio-dependent predator–prey system may act as a biological control. Moreover, using Muldowney's criteria, persistence conditions have been worked out.

The ratio-dependent functional form in the infection rate is of considerable interest and cannot be ignored. To understand the parasite-induced host extinction, Elbert *et al* (2000) formulated a plausible but *ad hoc* epidemiological macroparasite model and its stochastic variation. Their model fails to explain host deterministic extinction phenomena. Hwang and Kuang (2003) modified their model by taking into consideration that the encounter infection rate makes sense only when it follows the law of ratio-dependence and not the law of simple mass action. They showed that their model exhibits parasite-induced host extinction. This extinction dynamics resembles that by ratio-dependent predator–prey models (many references are given in the introduction). Based on the above observations, we would like to mention that there is much room for the improvement of our model and results in this direction.

Acknowledgments

Part of this work was completed when the author JC was visiting the U R GEODES of the 'Institut de recherche pour le développement (IRD), Bondy, France', under an IRD fellowship.

Appendix

The definition of the second additive compound matrix can be found in the paper of Li and Muldowney (1993).

Let $A = (a_{ij})$ be an $n \times n$ matrix. The *second additive compound* $A^{[2]}$ is the $\binom{n}{2} \times \binom{n}{2}$ matrix defined as follows.

For any integer $i = 1, \ldots, \binom{n}{2}$, let $(i) = (i_1, i_2)$ be the ith member in the lexicographic ordering of integer pairs (i_1, i_2) such that $1 \leqslant i_1 < i_2 \leqslant n$. Then, the element in the

ith row and jth column of $A^{[2]}$ is

$a_{i_1 i_1} + a_{i_2 i_2}$, if $(j) = (i)$

$(-1)^{r+s} a_{i_r j_s}$, if exactly one entry i_r of (i) does not occur in (j) and j_s does not occur in (i)

0, if neither entry from (i) occurs in (j).

For $n = 3$

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

its second additive compound matrix is

$$A^{[2]} = \begin{pmatrix} a_{11} + a_{22} & a_{23} & -a_{13} \\ a_{32} & a_{11} + a_{33} & a_{12} \\ -a_{31} & a_{21} & a_{22} + a_{33} \end{pmatrix}.$$

In this case, $(1) = (1, 2)$, $(2) = (1, 3)$ and $(3) = (2, 3)$.

Theorem (Bendixson's criterion in R^n). A simple closed rectifiable curve that is invariant with respect to (19) cannot exist if any one of the following conditions is satisfied on R^n:

(i) $$\sup \left\{ \frac{\partial F_r}{\partial x_r} + \frac{\partial F_s}{\partial x_s} + \sum_{q \neq r,s} \left(\left| \frac{\partial F_q}{\partial x_r} \right| + \left| \frac{\partial F_q}{\partial x_s} \right| \right) : 1 \leqslant r < s \leqslant n \right\} < 0,$$

(ii) $$\sup \left\{ \frac{\partial F_r}{\partial x_r} + \frac{\partial F_s}{\partial x_s} + \sum_{q \neq r,s} \left(\left| \frac{\partial F_r}{\partial x_q} \right| + \left| \frac{\partial F_s}{\partial x_q} \right| \right) : 1 \leqslant r < s \leqslant n \right\} < 0,$$

(iii) $\lambda_1 + \lambda_2 < 0$,

(iv) $$\inf \left\{ \frac{\partial F_r}{\partial x_r} + \frac{\partial F_s}{\partial x_s} - \sum_{q \neq r,s} \left(\left| \frac{\partial F_q}{\partial x_r} \right| + \left| \frac{\partial F_q}{\partial x_s} \right| \right) : 1 \leqslant r < s \leqslant n \right\} > 0,$$

(v) $$\inf \left\{ \frac{\partial F_r}{\partial x_r} + \frac{\partial F_s}{\partial x_s} - \sum_{q \neq r,s} \left(\left| \frac{\partial F_r}{\partial x_q} \right| + \left| \frac{\partial F_s}{\partial x_q} \right| \right) : 1 \leqslant r < s \leqslant n \right\} > 0,$$

(vi) $\lambda_{n-1} + \lambda_n > 0$.

where $\lambda_1 \geqslant \lambda_2 \geqslant \cdots \geqslant \lambda_n$ are the eigenvalues of $\frac{1}{2}((\partial F/\partial x)^* + (\partial F/\partial x))$ and where $\partial F/\partial x$ is the Jacobian matrix of F, and the asterisk denotes transposition.

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