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Predator-prey populations with parasitic infection

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Abstract. A predator-prey model, where both species are subjected to parasitism, is developed and analyzed. For the case where there is coexistence of the predator with the uninfected prey, an epidemic threshold theorem is proved. It is shown that in the case where the uninfected predator cannot survive only on uninfected prey, the parasitization could lead to persistence of the predator provided a certain threshold of transmission is surpassed.

Key words: Predator-prey — Parasite-mediated persistence — Epidemic threshold

1. Introduction

Many macroparasite species exhibit a regular alternation between two host species. The transition from one host to the other is accomplished either by releasing eggs or larvae into the environment where they are taken up by one of the hosts or by a prey-predator relation between the two host species. In some cases more sophisticated life cycles with even three hosts and complicated transmission techniques have evolved (e.g., in the snail-ant-sheep-system of *Dicrocoelium lanceolatum*; see Holmes and Bethel 1972 for further references). A mathematical model for the parasite-prey-predator interaction contains a description of the prey-predator relation as well as of the host-parasite-interaction. For both components of the system one can use well-established models. However the complete model has a higher degree of complexity and requires more detailed investigation.

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The interaction of one-prey and one-predator populations is reasonably well understood. After the early and basic work of Lotka, Volterra, Kolmogorov and others, major breakthroughs were obtained by the systematic investigations of response functions (Holling 1959) and by the discovery of the phenomenon of destabilization by enrichment (Rosenzweig 1971; Freedman 1987; Freedman and Wolkowicz 1986). Mathematically, the investigation of these models is simplified by the fact that they are two-dimensional and thus the Poincaré–Bendixson theorem applies.

Models for the interaction of more than two species have been studied in recent years. Then more complex phenomena arise, making the mathematical problems of a complete qualitative analysis, in some cases, insurmountable. On the other hand, a complete analysis is often not appropriate to answer the underlying biological questions. Hence many recent investigations have been concentrated on persistence problems. In biological terms, persistence says that asymptotically the density of each species remains above a positive bound independent of the initial conditions, i.e. all species stay away from extinction. Mathematically, this may be stated in terms of behavior of solutions of the models representing the biological phenomena, termed uniform persistence or permanence (Freedman and Waltman 1985; Hofbauer and Sigmund 1987).

Most models for the transmission of infectious diseases descend from the classical SIR model of Kermack and McKendrick (1927). Susceptibles become infectious by contact with infectious individuals. Later they may recover and join the group of immune (or dead) individuals. Also the Kermack–McKendrick model and many of its extensions are well investigated. Whereas the SIR model describes only the spread of the disease, some extended versions show the demographic impact of the disease (Hadeler 1984).

.Although many parasite species have two or more stages which live in different hosts, for some important parasitic diseases of vertebrates (in particular humans, e.g., malaria, schistosomyasis, or onchocercasis), the secondary host does not play an essential role in the ecology of the primary host, except for the transmission of the disease.

On the other hand, there are quite a number of examples where the two hosts are in a prey-predator relation and where the predation itself transmits the parasite from the prey to the predator, whereas the transmission from the predator to the prey typically is far less directed. In many cases the parasite modifies the external features or the behavior of the prey so as to make infected individuals more vulnerable to predation. In a review article Holmes and Bethel (1972) have collected many examples of such modification. In simple cases the infected individuals are less active and can be caught more easily. In more subtle cases the behavior of the prey individuals is modified such that they live in parts of the habitat which are accessible to the predator (fish and aquatic snails staying close to water surface, snails staying on top of the vegetation rather then under the plant cover), or in extreme cases, the body of the prey becomes more conspicuous. Dobson (1988) has provided further examples (mainly from the group of Acanthocephales) and has studied several sets of model equations. Peterson (Peterson and Page 1987; 1988; Mech et al. 1987) has indicated that

wolf attacks on moose are more often successful if the moose is heavily infected by *Echinococcus granulosus*. The moose is an intermediate host and the wolf is a terminal host of the parasite.

From a biological perspective, there are two rather different situations. A parasite population can invade an existing predator-prey relation if it manages to satisfy the two basic requirements for a successful parasite, namely to contact and invade the host with sufficient reliability, and to restrict the resulting increase in host mortality. In the situation of the predator-prey system, the parasite has to satisfy these conditions with respect to both species. The conditions for persistence of the parasite in the prey-predator system can be formulated as a threshold condition for the transmission rates or as a condition for the basic reproduction rate of the parasite ("net reproductive value" in a more general context introduced by R. A. Fisher 1930, "basic reproduction rate z_0" introduced by G. Macdonald 1952, 1955 for malaria, "reproduction rate R" of K. Dietz 1976 and Aron and May 1982). In simple epidemics the basic reproduction rate may be evaluated at the uninfected equilibrium of the host population. However, in prey-predator models the uninfected equilibrium may be unstable, and the threshold conditions have to be computed at a periodic solution (see Sect. 3 below). As stated earlier, a successful and well-adapted parasite ensures its persistence by establishing a reliable transmission and by restricting the damage inferred upon the host. As Curio (1988) has observed the situation is more complex with respect to an intermediate host which is the prey in a prey-predator relation. Here increased mortality due to predation is favoured in order to guarantee transmission from the intermediate to the definite host.

In the second situation only the presence of the parasite makes the predator-prey relation possible. In the absence of the parasite the predator can prey upon the prey species but not to such an extent as to sustain a stable population. On the other hand, infected individuals are more easily caught by the predator. A certain level of infection with the prey species is required for persistence of the predator on this particular prey.

In this second approach the following evolutionary question poses itself. How could the parasite adapt itself to the predator-prey-situation if that relation never existed without the parasite? This problem is also reflected in the mathematical results. It turns out that the prey-only stationary point never becomes unstable, even at high transmission rates, and the prey-predator-parasite population is not persistent in the usual orthant. However, at high transmission rates a new stable stationary point appears together with a saddle-point. In this situation there is a threshold phenomenon: if there are just a few predators, then the predator population becomes extinct, whereas if there are many (infected) predators then the system evolves towards the stable predator-prey-parasite state.

In modelling this situation we do not introduce the parasites as an interacting species, but we consider populations structured by the level of infection (Hadeler and Dietz 1983, 1984; Kretzschmar 1988; Waldstätter et al. 1988). We consider the most simple situation, we do not consider multiple infection or sexual reproduction in the host. Hence our model is a system of four ordinary differential equations for four types (noninfected and infected prey and

predators, respectively). Our approach is different from that of Dobson (1988) who uses the variables eggs, density of parasites in intermediate hosts and in definite hosts, number of intermediate hosts, whereas the number of definite hosts is assumed constant. In our model we retain the typical properties of the prey-predator model (extinction of the predator if the carrying capacity of the prey is decreased, destabilization by enrichment). Also we believe that the structured population model gives a better description of what really happens at the level of the individual hosts. On the other hand, in our approach multiple infections (clumping of parasites, Anderson and May 1979) can only be described with several classes of infected hosts. We do not think it necessary to model the compartment "eggs" by a separate variable. The "loss" of eggs or larvae due to successful infection can be neglected.

Anderson and May (1986) have studied five prey-predator-parasite interactions (invasion of a pathogen into an existing host-pathogen relation, introduction of a new host into such relation, invasion of a pathogen into a system of competing hosts, infection of either the prey or the predator by a parasite). Our problem is different because the prey is the intermediate host.

It is well known, even for three-dimensional problems, that the persistence problem becomes difficult if the faces of $\mathbb{R}^n_+$ contain periodic orbits. Here we overcome this difficulty by an appropriate application of the Perron–Frobenius theorem.

In Sect. 2 we derive and discuss the model equations, in Sect. 3 we prove the epidemic threshold theorems, Sect. 4 is devoted to a short discussion of the persistence problem. In Sect. 5 we derive results on parasite-mediated coexistence of prey and predator, in Sect. 6 we show the results of numerical simulations, Sect. 7 contains a concluding discussion of the results and their biological implications, in the Appendix, Sect. 8, we present those features of the Perron–Frobenius theory which are needed in the proofs.

2. Formulation of the model

The four population densities are the noninfected prey x_0, the infected prey x_1, the noninfected predator y_0, and the infected predator y_1. For convenience we introduce the total densities of prey and predator, respectively,

$$x = x_0 + x_1, \qquad y = y_0 + y_1.$$

Since our models will consist of systems of autonomous ordinary differential equations, we will assume throughout this paper that all functions are sufficiently smooth so that solutions to initial value problems exist and are unique.

In the absence of the parasite the two species follow a Rosenzweig predator-prey dynamics

$$\begin{aligned} \dot{x} &= B(x) - D(x) - q(x)y, \\ \dot{y} &= -\gamma(y) + cq(x)y, \end{aligned} \tag{2.1}$$

where B, D are the birth and death functions of the prey, γ is the death rate of the predator, q is the response function, and c is the conversion rate of prey biomass into predator biomass. These functions are assumed to satisfy the following hypotheses:

$$\begin{gathered} B(0) = D(0) = 0; \qquad B(x) > 0, \qquad D(x) > 0 \quad \text{for } x > 0; \\ B'(x) > 0, \qquad D'(x) > 0 \quad \text{for } x > 0; \qquad B'(0) > D'(0) \geqslant 0; \\ \text{there exists a unique } K > 0 \text{ such that } B(K) = D(K); \\ B'(K) < D'(K). \end{gathered} \tag{2.2}$$

Hypotheses (2.2) imply that birth and death rates increase with increasing populations. Initially the birth rate must be greater than the death rate for the prey population to survive, but at some positive population density K (the carrying capacity of the environment), birth rate equals death rate. For larger densities, death rates exceed birth rates.

$$q(0) = 0, \qquad q'(x) > 0. \tag{2.3}$$

Hypotheses (2.3) imply that the predator functional response is an increasing function of prey density.

$$\gamma(0) = 0, \qquad \gamma'(y) > 0. \tag{2.4}$$

$\gamma(y)$ is the density-dependent predator death rate.

The response function of the predator with respect to partially infected prey is

$$p(x_0, x_1) = p_0(x_0, x_1) + p_1(x_0, x_1), \tag{2.5}$$

where $p_0(x_0, x_1) \geqslant 0$ is the consumed noninfected prey, and $p_1(x_0, x_1) \geqslant 0$ is the consumed infected prey. We assume $p_0(x_0, 0) = q(x_0)$,

$$p_0(x_0, x_1) + p_1(x_0, x_1) \geqslant q(x), \tag{2.6}$$

and

$$p(x_0, x_1) \leqslant kx \quad \text{with some } k > 0. \tag{2.7}$$

Most important are the laws for the transmission of the parasite between the two species. The parasites are spread into the habitat by the infected predators and are taken up by the prey. We assume that the noninfected prey are infected in proportion to their own number and in proportion to the density of infected predators. The predators become infected by feeding on infected prey. Thus it appears to be a natural assumption that the noninfected predators become infected in proportion to the consumed infected prey. Then the transmission function is

$$\kappa p_1(x_0, x_1) y_0. \tag{2.8}$$

Thus the full model becomes

$$\begin{aligned}
\dot{x}_0 &= B(x) - \frac{x_0}{x} D(x) - p_0(x_0, x_1)y - \beta x_0 y_1, \\
\dot{y}_0 &= -\gamma(y)\frac{y_0}{y} + c(p_0(x_0, x_1) + p_1(x_0, x_1))y - \kappa p_1(x_0, x_1)y_0, \\
\dot{x}_1 &= \beta y_1 x_0 - \frac{x_1}{x} D(x) - p_1(x_0, x_1)y, \\
\dot{y}_1 &= -\gamma(y)\frac{y_1}{y} + \kappa p_1(x_0, x_1)y_0.
\end{aligned} \tag{2.9}$$

Here β and κ are constants representing the rate of conversion of uninfected populations into infected populations.

Of course these equations are considered in $\mathbb{R}^4_+$. The orthant $\mathbb{R}^4_+$ is positively invariant. Note that the solutions of the system (2.9) exist for all $t > 0$ in view of the obvious inequalities

$$\begin{aligned}
\dot{x} &\leqslant B(x) - D(x), \\
\dot{y} &\leqslant -\gamma(y) + kcxy,
\end{aligned}$$

and the assumed conditions on the coefficient functions.

Of the 15 faces of the cone $\mathbb{R}^4_+$ only few contain invariant subsystems. There are no proper three-dimensional invariant subsystems and the only two-dimensional invariant subsystem is the noninfected predator-prey system.

The classical Rosenzweig model (for uninfected populations) is conveniently written

$$\begin{aligned}
\dot{x} &= ax\left(1 - \frac{x}{K}\right) - \frac{x}{x + A}y, \\
\dot{y} &= c\left(\frac{x}{x + A} - \frac{B}{B + A}\right)y.
\end{aligned} \tag{2.10}$$

Then the $\dot{y} = 0$ isocline is just the straight line $x = B$, and the three major cases of interest are characterised by

$K < B$: no coexistence,

$(K + A)/2 < B < K$: stable coexistence equilibrium,

$0 < B < (K + A)/2$: stable periodic orbit of coexistence.

Note that in this case

$$\begin{aligned}
&B(x) = ax, \qquad D(x) = \frac{ax^2}{K}, \\
&q(x) = \frac{x}{x + A}, \qquad \gamma(y) = \gamma \cdot y, \qquad \gamma = c\frac{B}{B + A} < c.
\end{aligned} \tag{2.11}$$

Here we assume that the level of saturation does not depend on whether the prey is infected, i.e. we assume

$$p_0(x_0, x_1) = \frac{x_0}{A + x_0 + \varrho x_1}, \qquad p_1(x_0, x_1) = \frac{\varrho x_1}{A + x_0 + \varrho x_1}, \tag{2.12}$$

where $\varrho > 1$ is a constant. Thus in the Rosenzweig case the full model reads

$$\begin{aligned}
\dot{x}_0 &= ax - \frac{ax_0 x}{K} - \frac{x_0}{A + x_0 + \varrho x_1} y - \beta x_0 y_1, \\
\dot{y}_0 &= -c \frac{B}{B + A} y_0 + c \frac{x_0 + \varrho x_1}{A + x_0 + \varrho x_1} y - \kappa \frac{\varrho x_1}{A + x_0 + \varrho x_1} y_0, \\
\dot{x}_1 &= \beta y_1 x_0 - \frac{ax_1 x}{K} - \frac{\varrho x_1}{A + x_0 + \varrho x_1} y, \\
\dot{y}_1 &= -c \frac{B}{B + A} y_1 + \kappa \frac{\varrho x_1}{A + x_0 + \varrho x_1} y_0.
\end{aligned} \tag{2.13}$$

3. Epidemic threshold theorems

In this section the threshold theorems are derived. The Jacobian at an arbitrary point can be seen as a block matrix

$$J = \begin{pmatrix} \mathscr{A} & \mathscr{B} \\ \mathscr{C} & \mathscr{D} \end{pmatrix}, \tag{3.1}$$

where the four 2×2 blocks are given by

$$\mathscr{A} = \left[\begin{array}{c|c} B'(x) - x_0 D'(x)/x - x_1 D(x)/x^2 & -p_0(x_0, x_1) \\ -\beta y_1 - p_{00}(x_0, x_1) y & \\ \hline c(p_{00}(x_0, x_1) + p_{10}(x_0, x_1)) y & -(\gamma'(y) y y_0 + \gamma(y) y_1)/y^2 \\ -\kappa p_{10}(x_0, x_1) y_0 & cp(x_0, x_1) - \kappa p_1(x_0, x_1) \end{array}\right], \tag{3.2}$$

$$\mathscr{B} = \left[\begin{array}{c|c} B'(x) + x_0(D(x) - xD'(x))/x^2 & -p_0(x_0, x_1) - \beta x_0 \\ + p_{01}(x_0, x_1) y & \\ \hline c(p_{01}(x_0, x_1) + p_{11}(x_0, x_1)) y & (-\gamma'(y) y + \gamma(y)) y_0/y^2 \\ -\kappa p_{11}(x_0, x_1) y_0 & cp(x_0, x_1) \end{array}\right], \tag{3.3}$$

$$\mathscr{C} = \left[\begin{array}{c|c} \beta y_1 + x_1(D(x) - xD'(x))/x^2 & -p_1(x_0, x_1) \\ -p_{10}(x_0, x_1) y & \\ \hline \kappa p_{10}(x_0, x_1) y_0 & (-\gamma'(y) y + \gamma(y)) y_1/y^2 \\ & + \kappa p_1(x_0, x_1) \end{array}\right], \tag{3.4}$$

$$\mathscr{D} = \left(\begin{array}{c|c} -(x_0 D(x) + x_1 x D'(x))/x^2 & \beta x_0 - p_1(x_0, x_1) \\ -p_{11}(x_0, x_1) y & \\ \hline \kappa p_{11}(x_0, x_1) y_0 & -(\gamma'(y) y y_1 + \gamma(y) y_0)/y^2 \end{array}\right). \tag{3.5}$$

Here the symbol p_{jk} denotes the partial derivative of p_j with respect to x_k, $j, k = 0, 1$. Then the equations $x_1 = 0$, $y_1 = 0$ define an invariant manifold (the noninfected manifold). At points of this manifold the lower left block $\mathscr{C}$ vanishes. The upper left block $\mathscr{A}$ is related to the noninfected manifold. It describes the action of J on its invariant subspace $\{x_1 = y_1 = 0\}$. The lower right block $\mathscr{D}$ is also related to a two-dimensional invariant subspace. However, this subspace is not constant, i.e. it varies with the point where the Jacobian is formed. The block $\mathscr{D}$ describes the stability of the disease-free situation (stationary point or limit cycle) with respect to infection.

Along the noninfected manifold the lower right block $\mathscr{D}$ has the form

$$\mathscr{D} = \left(\begin{array}{c|c} -(D(x_0)/x_0 + y_0 p_{11}(x_0, 0)) & \beta x_0 \\ \hline \kappa y_0 p_{11}(x_0, 0) & -\gamma(y_0)/y_0 \end{array}\right). \tag{3.6}$$

This block describes the invasibility of the disease free situation (stationary state or periodic orbit). The off-diagonal elements of this matrix are nonnegative, hence the Perron–Frobenius theory applies (here for continuous semigroups). The spectral bound (largest real part of any eigenvalue of $\mathscr{D}$) is itself an eigenvalue (the leading eigenvalue, see Sect. 8), corresponding to this eigenvalue there is a nonnegative eigenvector (of $\mathscr{D}$). In the actual proof the off-diagonal elements are positive ($x_0 > 0$, $y_0 > 0$), then the spectral bound is a simple eigenvalue. The other eigenvalue is (real and) strictly smaller and there is a positive eigenvector. Of course, in this simple case the eigenvalues can be explicitly computed from a quadratic equation and the statements can be checked directly. However we need the general approach for the proof of Proposition 2. Our first result is

Proposition 1. *If the (two-dimensional) noninfected model has a stable equilibrium* $(\bar{x}_0, \bar{y}_0)$ *with* $\bar{x}_0, \bar{y}_0 > 0$, *and if this equilibrium is considered as an equilibrium* $(\bar{x}_0, \bar{y}_0, 0, 0)$ *of the four-dimensional system then there is a bifurcation towards an epidemic equilibrium at* $\beta\kappa = \tau_0$, *where*

$$\tau_0 = \frac{D(\bar{x}_0)/\bar{x}_0 + p_{11}(\bar{x}_0, 0)\bar{y}_0}{\bar{x}_0 p_{11}(\bar{x}_0, 0)\bar{y}_0} \cdot \frac{\gamma(\bar{y}_0)}{\bar{y}_0}. \tag{3.7}$$

For $\beta\kappa < \tau_0$, *the noninfected equilibrium is locally stable. For* $\beta\kappa > \tau_0$, *the noninfected equilibrium loses its stability, and for* $\beta\kappa - \tau_0 > 0$ *small there is a locally stable epidemic equilibrium* $(\tilde{x}_0, \tilde{y}_0, \tilde{x}_1, \tilde{y}_1)$ *with all components positive.*

Proof. Let $\bar{E}(\bar{x}_0, \bar{y}_0, 0, 0)$ denote the equilibrium in question. Let $\bar{J}, \bar{\mathscr{A}}, \bar{\mathscr{B}}, \bar{\mathscr{D}}$ be the matrices $J, \mathscr{A}, \mathscr{B}, \mathscr{D}$ respectively evaluated at $\bar{E}$. The eigenvalues of J are precisely the eigenvalues of the two matrices $\bar{\mathscr{A}}$ and $\bar{\mathscr{D}}$. The eigenvalues of $\bar{\mathscr{A}}$, by assumption, both have negative real parts. The eigenvalues of $\bar{\mathscr{D}}$ are both real. One of them is negative since the trace of $\bar{\mathscr{D}}$ is negative. Hence the sign of the determinant of $\bar{\mathscr{D}}$ (or of $\bar{J}$) determines whether the leading eigenvalue is positive or negative.

The two right eigenvectors $\bar{z}_1, \bar{z}_2$ of $\bar{\mathscr{D}}$ can be extended to eigenvectors $\hat{z}_1, \hat{z}_2$ of $\bar{J}$. Thus $\bar{J}$ has the eigenvector $\hat{z}_1$ for which the last two components are positive and the eigenvector $\hat{z}_2$ for which the last two components have opposite sign.

Thus, if the leading root is positive then the flow near the noninfected equilibrium is inward with respect to $\mathbb{R}^4_+$ in the direction of $\hat{z}_1$.

By the usual argument one can establish a bifurcation which leads locally to a new stable equilibrium and an exchange of stability at $\beta\kappa = \tau_0$. At this moment nothing can be said about the global stability of this epidemic equilibrium or about its existence or stability for $\beta\kappa - \tau_0$ large. □

The threshold condition $\beta\kappa < \tau_0$ can be written in the form

$$\frac{\beta\bar{x}_0\bar{y}_0}{\gamma(\bar{y}_0)} \cdot \frac{\kappa\bar{x}_0 p_{11}(\bar{x}_0, 0)\bar{y}_0}{D(\bar{x}_0) + x_0 p_{11}(\bar{x}_0, 0)\bar{y}_0} < 1, \tag{3.8}$$

which has a direct interpretation as $R_1 R_2 < 1$ where R_1 and R_2 are the number of infections in preys per infectious predator and the number of infected predators per infected prey, respectively, in a totally susceptible population. This proposition is the appropriate analogue of the Kermack–McKendrick epidemic threshold theorem. Here two species are involved, and what is important is the *product* of the two transmission rates. If this product is small then the parasite cannot persist, if it is above a certain threshold τ_0 then an epidemic situation occurs. We discuss how the threshold condition depends on the other parameters, for the special case of the Rosenzweig dynamics, where

$$\tau_0 = \frac{B + \varrho(K - B)}{B\varrho(K - B)} \cdot \gamma = \frac{B + \varrho(K - B)}{B\varrho(K - B)} \cdot c\frac{B}{A + B}. \tag{3.9}$$

The threshold for the product of the transmission rates is increasing, if the predator mortality γ increases. The threshold decreases, if the carrying capacity K increases. Of course, this behavior of the model agrees with biological intuition. Furthermore, for fixed γ, the threshold increases with increasing equilibrium prey density. But in this Rosenzweig model, as long as $(K - A)/2 < B < K$, increasing prey density is coupled to decreasing predator density. If γ is fixed, then τ_0 is an increasing function of B, as it should be.

Increasing the response parameter ϱ decreases the threshold and thus enhances transmission. Of course, increasing ϱ also increases prey mortality (cf. our earlier remark). Since predation cannot extinguish the prey, for the parasite the beneficiary effect dominates.

In the proof the stability of the equilibrium $\bar{E}(\bar{x}_0, \bar{y}_0, 0, 0)$ has not been used, only the fact that the eigenvalues of the matix $\mathcal{A}$ are different from zero. Hence there is a bifurcation towards an infected stationary point also in the case where $\bar{E}$ is unstable. But in that case the new stationary point is unstable.

In the Rosenzweig model the stationary point may lose its stability to a stable limit cycle. It is known by the results of Liu and Cheng 1988 (see also Kuang and Freedman 1988) that this limit cycle is unique. For this situation we prove the next proposition.

Proposition 2. *Suppose the (two-dimensional) noninfected system has a stable limit cycle* $(\bar{x}_0(t), \bar{y}_0(t))$. *Consider this orbit as a limit cycle* $(\bar{x}_0(t), \bar{y}_0(t), 0, 0)$ *of the four-dimensional system. Then there is a unique critical level* τ_1 *depending only on the noninfected system with the following properties. If the product* $\beta\kappa$ *is less than*

τ_1 then the limit cycle, considered as a periodic orbit of the four-dimensional system, is stable. For $\beta\kappa > \tau_1$ the limit cycle (in the four-dimensional system) has a multiplier greater than 1 and thus is unstable. The corresponding eigenvector of the Poincaré map is pointing inward with respect to $\mathbb{R}^4_+$. For $\beta\kappa - \tau_1 > 0$, but small, there is a locally stable limit cycle with $x_1(t), y_1(t) > 0$ throughout.

For the proof we need the following lemma:

Lemma 3. *Consider the linear equation*

$$\dot{u}(t) = \tilde{A}(t)u(t) + \lambda\tilde{B}(t)u(t) \tag{3.10}$$

where the matrices $\tilde{A}$ and $\tilde{B}$ are periodic with period $\omega > 0$, and

$$\begin{aligned} a_{jk}(t) &\geqslant 0, \quad j \neq k, \\ b_{jk}(t) &\geqslant 0, \quad \text{for all } j, k. \end{aligned} \tag{3.11}$$

Then the leading multiplier μ_1 is a nondecreasing function of λ.

Proof. Consider the fundamental matrix $U(t)$ defined as a solution of

$$\dot{U}(t) = \tilde{A}(t)U(t) + \lambda\tilde{B}(t)U(t), \qquad U(0) = I, \tag{3.12}$$

and its derivative $V(t) = \partial U(t)/\partial\lambda$ which satisfies

$$\dot{V}(t) = (\tilde{A}(t) + \lambda\tilde{B}(t))V(t) + \tilde{B}(t)U(t), \qquad V(0) = 0, \tag{3.13}$$

and thus, by the variation of constants formula,

$$V(t) = \int_0^t U(t)U(s)^{-1}\tilde{B}(s)U(s)\,ds. \tag{3.14}$$

In particular, the monodromy matrix is $U(\omega)$, and its derivative with respect to λ is $V(\omega)$.

Now, in view of (3.11), $U(s)$ is a nonnegative matrix, and for $t \geqslant s$, the matrix $U(t)U(s)^{-1}$ is nonnegative. The matrix $\tilde{B}(s)$ is nonnegative by assumption. Hence $U(\omega)$ and $V(\omega)$ are both nonnegative. Hence we can apply the classical Perron–Frobenius theorem (see Sect. 8) to the matrix $U(\omega)$. The maximum of the absolute values of the Floquet multipliers is itself a multiplier (the leading multiplier).

Now it is well known that the spectral radius of a nonnegative matrix depends monotonely on the matrix elements. Hence the leading multiplier is a nondecreasing function of λ. □

Now assume that the off-diagonal elements of $\tilde{B}$ are strictly positive. Then, for $\lambda > 0$, the off-diagonal elements of $\tilde{A} + \lambda\tilde{B}$ are strictly positive. Then $U(s)$ and $U^{-1}(t)U(s)$ are strictly positive for $0 \leqslant s \leqslant t$. Thus $\tilde{B}(s)U(s)$ is a positive matrix, and hence $V(t)$ is positive for $t > 0$. Therefore $V(\omega)$ is positive and thus the leading multiplier is strictly increasing.

Since the off-diagonal elements of $B(t)$ have a positive uniform lower bound, the leading multiplier goes to $+\infty$ for $\lambda \to +\infty$.

Proof of Proposition 2. By trivial change of coordinates, we obtain for the system $\dot{u} = \mathscr{D}u$ (determining the multipliers related to the x_1, y_1-directions) the form

$$\dot{u}(t) = \tilde{A}(t)u(t) + \sqrt{\beta\kappa}\tilde{B}(t)u(t), \tag{3.15}$$

where

$$\tilde{A}(t) = \left(\begin{array}{c|c} -(D(x_0(t)/x_0(t) + y_0(t)p_{11}(x_0(t), 0)) & 0 \\ \hline 0 & -\gamma(y_0(t))/y_0(t) \end{array}\right), \tag{3.16}$$

$$\tilde{B}(t) = \left(\begin{array}{c|c} 0 & x_0(t) \\ \hline y_0(t)p_{11}(x_0(t), 0) & 0 \end{array}\right). \tag{3.17}$$

Since the periodic orbit of the noninfected system is a compact subset of the open set $x_0 > 0, y_0 > 0$, the off-diagonal elements of $B(t)$ have a positive uniform lower bound. Hence Proposition 2 is proved. □

In the situation where the noninfected system has a stable periodic orbit the noninfected equilibrium is a repeller (of the two-dimensional system). This repeller, considered as an equilibrium of the four-dimensional system, for increasing $\beta\kappa$, also undergoes the transition described in Proposition 1. There is a number τ_0 such that for $\beta\kappa < \tau_0$ the two additional eigenvalues have negative real parts, for $\beta\kappa > \tau_0$ the leading additional eigenvalue is positive.

In general there will be no relation between the two numbers τ_0 and τ_1. The two cases $\tau_0 < \tau_1$ and $\tau_0 > \tau_1$ lead to rather different phase space patterns. We characterize these patterns by their phase diagrams. A circle indicates the existence of a stationary point or limit cycle. A black circle indicates an attractor, arrows indicate trajectories (at least one trajectory) going from one object (singular element) to another.

$\tau_0 < \tau_1$: If $\kappa\beta < \tau_0$ then the stationary point and the limit cycle attract trajectories from the interior. If $\kappa\beta$ passes through τ_0 then the stationary point undergoes a pitchfork bifurcation. There is a new stationary point in $\mathbb{R}^4_+$ which is stable in the x_1, y_1-direction but unstable in the x_0, y_0-direction. The periodic orbit is stable, as long as $\beta\kappa < \tau_1$. If $\beta\kappa > \tau_1$ then there is new stable periodic orbit. Hence locally the phase diagram looks like the following:

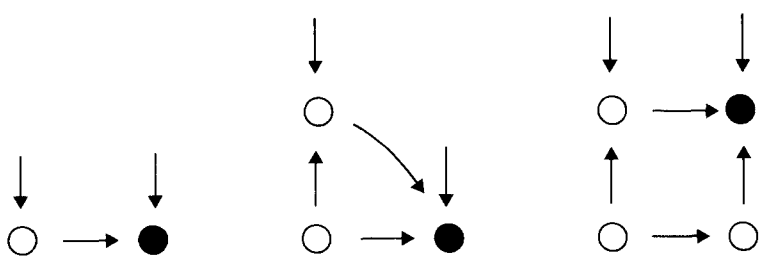

Fig. 1. Phase diagrams for $\tau_0 < \tau_1$

Thus the essential threshold, from the epidemiological viewpoint, is τ_1.

$\tau_0 > \tau_1$: If $\beta\kappa < \tau_1$ then both the unstable stationary point and the periodic orbit attract trajectories from the interior. If $\beta\kappa$ passes through τ_1 then a new stable periodic orbit appears, and if $\beta\kappa > \tau_0$ then also the stationary point repels trajectories from the inside. In this case the phase diagrams are:

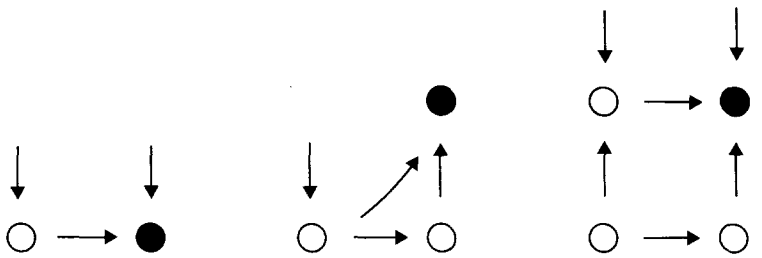

Fig. 2. Phase diagrams for $\tau_0 > \tau_1$

Also in this case the essential epidemic threshold is τ_1. Thus we can collect the results obtained so far in the following theorem:

Theorem 4. *There is a well-defined threshold τ with the following property. If the product of transmission rates $\beta\kappa$ is less than τ then the uninfected stable attractor (with respect to the uninfected system, stationary point or periodic orbit) is also stable against parasitic infection. If the product of transmission rates passes through the threshold then the uninfected attractor loses its stability and a stable infected attractor appears.*

The case $\tau_1 < \beta\kappa < \tau_0$ seems quite interesting. There is a nice stable periodic attractor representing an infected population. Nevertheless the system is not persistent with respect to any of the usual definitions since the stationary point has a two-dimensional stable manifold. This situation has been called almost persistence (G. J. Butler, private communication).

For completeness we consider the stationary solution $(K, 0, 0, 0)$. Here

$$\mathscr{D} = \left(\begin{array}{c|c} -D(K)/K & \beta K \\ 0 & -\gamma'(K) \end{array}\right).$$

Naturally this situation is stable against infection, but of course it is not stable against the immigration of predators.

4. A nonpersistence result

In this section we consider the question as to whether or not a predator-prey system, where the predator is unable to persist on the prey in the absence of

parasites will become a persistent system with respect to the nonnegative cone when parasites are present. First we must define persistence and nonpersistence.

We adopt the notions of persistence defined in Butler et al. (1986).

Let X be a locally compact metric space with metric ϱ and $G \subset X$ have nonempty interior. Let ∂G denote the boundary of G, and G° the interior of G. Let $u \in X$ and let $f(u, t)$ be a flow given by a dynamical system defined on X ($f(u, 0) = u, f(f(u, t), t_\alpha) = f(u, t + t_\alpha), f(u, t)$ is continuous). Then we say that $f(u, t)$ exhibits *persistence* with respect to G if

$$u \in G^\circ \Rightarrow \liminf_{t \to \infty} \varrho(f(u, t), \partial G) > 0.$$

We further say that $f(u, t)$ exhibits *uniform persistence* or *permanence* with respect to G if

$$\liminf_{t \to \infty} \varrho(f(G^\circ, t), \partial G) \geqslant \delta > 0, \quad \text{where } f(G^\circ, t) = \{f(u, t) : u \in G^\circ\}.$$

If persistence does not occur, we say that we have *nonpersistence*. In Freedman and Waltman (1984, 1985), $X = \mathbb{R}^3$ and $G = \mathbb{R}^3_+$ are chosen. There, persistence criteria were developed in that context for predator-prey and for competitive systems. A nice survey article on permanence is Hofbauer and Sigmund (1987).

The predator-prey dynamics in the uninfected case is given by the invariant flow in the x_0, y_0 plane. There are three possible equilibria in this palne, $E_0(0, 0, 0, 0)$, $E_K(K, 0, 0, 0)$ and $\bar{E}(\bar{x}_0, \bar{y}_0, 0, 0)$. E_0 and E_K always exist (given our hypotheses). It is well known (Freedman 1987) that $\bar{E}$ exists if and only if y_0 persists, i.e. $\liminf_{t \to \infty} y_0(t) > 0$.

Computing the eigenvalues of the Jacobian matrices λ_{0i} and λ_{Ki}, $i = 1, \ldots, 4$, about the equilibria E_0 and E_K, respectively, we get from the previous section

$$\lambda_{01} = B'(0) - D'(0), \quad \lambda_{02} = -\gamma'(0), \quad \lambda_{03} = -D'(0), \quad \lambda_{04} = -\gamma'(0), \tag{4.1}$$

$$\lambda_{K1} = B'(K) - D'(K), \qquad \lambda_{K2} = -\gamma'(K) + cp_0(K, 0),$$
$$\lambda_{K3} = -\frac{D(K)}{K}, \qquad \lambda_{K4} = -\gamma'(K). \tag{4.2}$$

Since $\lambda_{01} > 0$, $\lambda_{0i} < 0$, $i = 2, 3, 4$, E_0 is a saddle point. Further, $\lambda_{K1} < 0$, $\lambda_{K3} < 0$, $\lambda_{K4} < 0$. In the two-dimensional case, we know that y_0 persists if and only if $\lambda_{K2} > 0$.

Suppose now that y_0 does not survive on x_0 in the uninfected predator-prey dynamics, i.e.

$$-\gamma'(0) + cp_0(K, 0) < 0 \tag{4.3}$$

(and hence $\bar{E}$ does not exist). Then E_K is asymptotically stable, and hence there are solutions to system (2.6) with positive initial conditions which approach E_K as $t \to \infty$. This implies nonpersistence with respect to $\mathbb{R}^4_+$.

An interpretation of this result is that this system could not evolve from an uninfected predator-prey system in which the predator species is not able to survive solely on this particular prey species.

In the next section, we show that persistence with respect to a set $G \subset \mathbb{R}^4_+$, where part of the boundary of G is a certain separatrix surface intersecting the positive cone, may be possible.

5. Parasite-mediated coexistence

For the discussion of the parasite-mediated coexistence it is necessary to discuss the stationary points in detail. Since we are mainly interested in the behavior of the system near the prey-only equilibrium we shall not consider periodic orbits. This discussion also throws some new light on the bifurcations in the threshold theorem. Here we assume the Rosenzweig dynamics. Our approach is to reduce the set of nonlinear equations to a single equation for the variable $x = x_0 + x_1$.

At a stationary point we have

$$ax - \frac{ax_0 x}{K} - \frac{x_0}{A + x_0 + \varrho x_1} y - \beta x_0 y_1 = 0, \tag{5.1a}$$

$$\beta x_0 y_1 - \frac{ax_1 x}{K} - \frac{\varrho x_1}{A + x_0 + \varrho x_1} y = 0, \tag{5.1b}$$

$$-c\frac{B}{B+A} y_0 + c\frac{x_0 + \varrho x_1}{A + x_0 + \varrho x_1} y - \frac{\kappa \varrho x_1}{A + x_0 + \varrho x_1} y_0 = 0, \tag{5.1c}$$

$$-c\frac{B}{B+A} y_1 + \frac{\kappa \varrho x_1}{A + x_0 + \varrho x_1} y_0 = 0. \tag{5.1d}$$

Adding Eqs. (5.1c,d) gives for $y \neq 0$

$$\frac{B}{B+A} = \frac{x_0 + \varrho x_1}{A + x_0 + \varrho x_1}, \tag{5.2}$$

from which

$$x_0 + \varrho x_1 = B, \tag{5.3}$$

and

$$x_0 = \frac{\varrho x - B}{\varrho - 1} \quad \text{and} \quad x_1 = \frac{B - x}{\varrho - 1}. \tag{5.4}$$

Adding Eqs. (5.1a,b) we obtain

$$ax\left(1 - \frac{x}{K}\right) = \frac{x_0 + \varrho x_1}{A + x_0 + \varrho x_1} y. \tag{5.5}$$

From Eq. (5.1d) and $y_0 + y_1 = y$ it follows that

$$y_1 = \frac{\kappa \varrho x_1}{Bc + \kappa \varrho x_1} y. \tag{5.6}$$

We introduce the expressions obtained for x_0, y_1 into Eq. (5.1b),

$$\beta \frac{\kappa\varrho x_1}{Bc + \kappa\varrho x_1} \cdot \frac{\varrho x - B}{\varrho - 1} y - \frac{axx_1}{K} - \frac{\varrho x_1}{A + B} y = 0. \tag{5.7}$$

This equation is satisfied for $x_1 = 0$. We assume $x_1 \neq 0$, and replace x_1 and y. Then we arrive at

$$\beta \frac{\kappa\varrho(A + B)(\varrho x - B)}{Bc(\varrho - 1) + \kappa\varrho(B - x)} = \frac{B}{K - x} + \varrho. \tag{5.8}$$

The equation is equivalent to a quadratic equation, hence there are at most two real roots. The equation assumes the form

$$\beta f(x) = g(x) \tag{5.9}$$

where

$$f(x) = \frac{\kappa\varrho(A + B)(\varrho x - B)}{Bc(\varrho - 1) + \kappa\varrho(B - x)}, \tag{5.10}$$

$$g(x) = \frac{B}{K - x} + \varrho. \tag{5.11}$$

Both the graphs of f and g are hyperbolas. The function f has a zero at B/ϱ and a pole at

$$x^* = B + \frac{\varrho - 1}{\varrho} \frac{Bc}{\kappa} > B. \tag{5.12}$$

The function g has a pole at K and a zero at $K + B/\varrho$. We distinguish four cases.

Case 1: $B < x^* < K$. For small β there is one root in (B, x^*). For increasing β the root decreases, and at

$$\beta_0 = \frac{B}{K - B} \cdot \frac{c}{(A + B)\kappa\varrho} \tag{5.13}$$

it enters the interval $(B/\varrho, B)$. Hence at $\beta = \beta_0$ there is a transcritical bifurcation.

Case 2: $B < K < x^*$. For small β there are no roots. For sufficiently large β there are two roots ξ, η in $(B/\varrho, K)$. Since $f(B) = \kappa\varrho(A + B)/c$ and $g(B) = B/(K - B) + \varrho$, the critical β is again given by (5.13). The two roots satisfy $\xi < B < \eta$ for $\beta > \beta_0$. There is a pitchfork bifurcation at $\beta = \beta_0$.

Case 3: $B/\varrho < K < B$. For small β there are no roots, for sufficiently large β there are two roots $\xi_1 < \xi_2$ in the interval $(B/\varrho, K) \subset (B/\varrho, B)$. There is a saddle-node bifurcation for some β. It may not be worth the effort to give an explicit formula for the critical β.

We do not perform a formal stability analysis, but it is plausible that the lower root ξ_1 which corresponds to low prey density and large predator density, is stable, whereas the upper root (low predator density) corresponds to a saddle point with a one-dimensional unstable manifold.

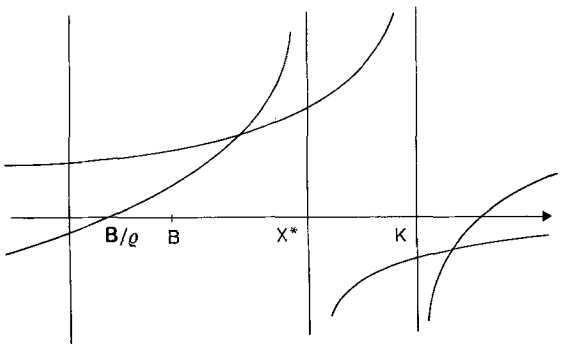

Fig. 3. The two hyperbolas in Case 1

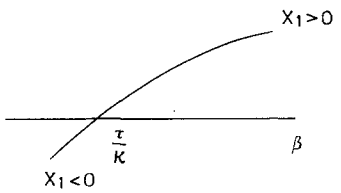

Fig. 4. Pitchfork bifurcation in Case 2

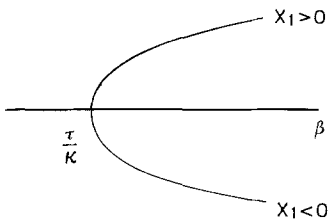

Fig. 5. Transcritical bifurcation in Case 1

Case 4: $K < B/\varrho$. In view of (5.4) we are looking for $x \in (B/\varrho, B)$. In this interval we have $f(x) > 0$. But $g(x)$ may assume positive values. Hence there may be solutions x of (5.9) for small β. Since $K < B/\varrho$, these solutions lead to $y < 0$ in view of (5.5). There are no feasible solutions in this case.

Now we discuss the biological significance of these bifurcation phenomena.

In Cases 1 and 2 the predator can survive on the uninfected prey. For $\beta > \beta_0$ the established prey-predator system loses its stability against infection by the

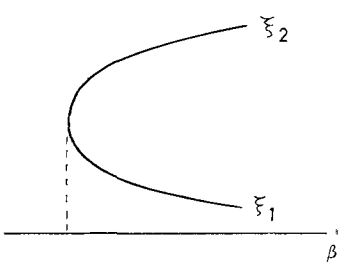

Fig. 6. Saddle-node bifurcation in Case 3

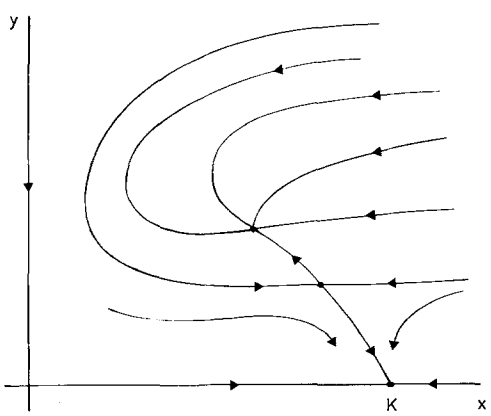

Fig. 7. Projected phase space in the case $B > K$ for high transmission rates: parasite mediated coexistence

parasite, an infected stationary situation appears. The distinction between the transcritical and the pitchfork situation has no great biological significance. In the pitchfork case the level of infection increases more rapidly once β exceeds the threshold.

Case 3 is the most interesting. The predator cannot survive on the uninfected prey. It could however survive if all prey were infected. Hence for large β, when a large proportion of the prey is infected, the predator can survive. There are two stationary points, a saddle and a node/focus. The node/focus is locally stable or it may undergo a Hopf bifurcation and give rise to a locally stable periodic orbit. But in either case the local attractor is not globally stable. The predator goes to extinction, when the initial predator density is too low. This effect is easy to interpret. If the initial predator density is low, then the parasites do not infect sufficient prey for the predator to survive. Then the predator population declines and with it the parasite population. In this case the prey and predator can coexist only in the presence of the parasite.

In Case 4 the predators could not survive even if all prey were infected. Thus coexistence of prey and predator is impossible for arbitrarily high infection rates.

The situation of Case 3 can be described by the following theorem on parasite-mediated coexistence.

Theorem 5. *In the system* (2.13) *assume that* $B > K$, *i.e. that there is no coexistence of the uninfected species. If*

$$\varrho \leqslant \frac{B}{K}$$

then for no choice of $\kappa, \beta > 0$ *the prey and the predator can coexist. If*

$$\varrho > \frac{B}{K}$$

then for every $\kappa > 0$ *there is a* $\beta_1 = \beta_1(\kappa) > 0$ *such that for* $\beta > \beta_1(\kappa)$ *there is an equilibrium of parasite-mediated coexistence.*

In this discussion we have completely ignored periodic orbits. In general periodic orbits will appear if $B/\varrho < (K + A)/2$ and $\beta\kappa$ is large, i.e. in situations where the full system behaves like the two-dimensional Rosenzweig model with B replaced by B/ϱ. Near the value of β where the new stationary point together with the saddle point appears, at least two of the eigenvalues are real. We do not know whether this point will undergo a Hopf bifurcation if the parameters are further changed.

6. Numerical simulation

In order to support the conjectures which we could not prove we have performed some computer simulations. We have used the simple Euler method with constant stepsize. For the simulation we have chosen the fixed parameters $a = 3$, $K = 1$, $b = 0.5$, $c = 1$, $A = 0.5$ and we have varied $B, \varrho, \kappa, \beta$. In Figs. 8–10 we have chosen $\varrho = 2$.

In most plots we have represented the x, y plane, i.e. we have used as coordinates the total number of prey and the total number of predators. Of course the resulting plot is a two-dimensional projection of a four-dimensional phase space. Hence the projected trajectories can intersect.

In the first situation we have a viable predator-prey system infected to variable extent. If $\varrho = 1$ then of course the x, y plane represents the phase plane of the usual Rosenzweig model. For $\varrho > 1$ and large κ, β there are remarkable effects. If the uninfected system has a stable limit cycle then parasitic infection tends to increase the amplitude of the oscillation. This effect is quite plausible. Shifting the predator response from $\varrho = 1$ to $\varrho > 1$ moves the position of the $\dot{y} = 0$ isocline from $x = B$ to $x = B/\varrho$. Hence the system with $\varrho > 1$ instead of $\varrho = 1$ corresponds to a situation of large amplitude oscillation. Similarly, if the uninfected system has a stable coexistence equilibrium the infected system may have a stable periodic orbit. A transition from a stationary state to oscillations has also been observed by Dobson (1988). But we underline that in many cases of interest already the uninfected population performs a stable oscillation, and the infection tends to increase the amplitudes.

Two further remarks are appropriate. Although the shape of the projected periodic orbit in the x, y plane looks very similar to the periodic orbit in the

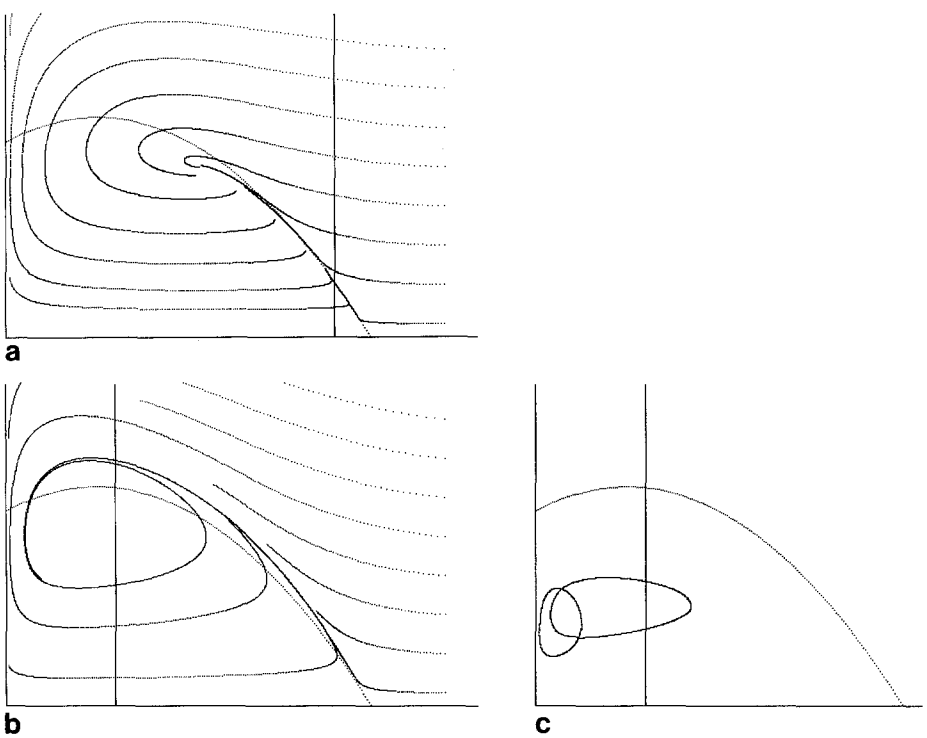

Fig. 8. Hopf bifurcation by decreasing B. **a** x, y-projection, $B = 0.9$, $\kappa = \beta = 2.2$: stable stationary point; **b** x, y-projection, $B = 0.3$, $\kappa = \beta = 2.2$: stable periodic orbit; **c** x_0, y_0 and x_1, y_1-projections, parameters as in **b**

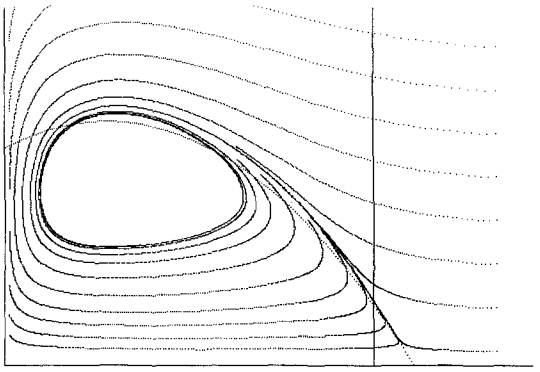

Fig. 9. Hopf bifurcation by increasing the transmission rates. For $B = 0.9$, $\kappa = \beta = 0$ there is a stable stationary point (compare Fig. 8a); for $\kappa = \beta = 4$ a stable periodic orbit occurs

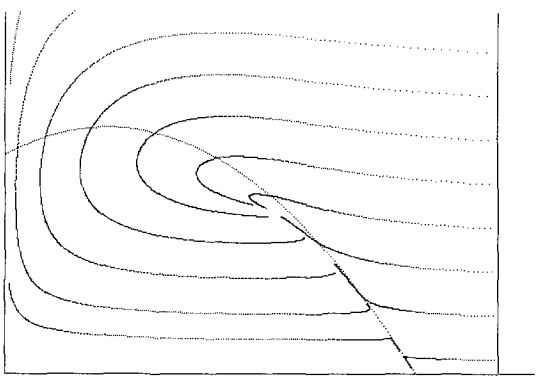

Fig. 10. Parasite-mediated coexistence: saddle-node bifurcation, $B = 1.2$, $\kappa = \beta = 2.2$

uninfected situation, the projections into the x_0, y_0 plane and the x_1, y_1 plane look quite different. In particular the number of infected prey undergoes rapid changes.

In the problem of parasite mediated coexistence the simulation shows the desired phenomenon (see Fig. 10). If $B > K$ then the noninfected populations do not coexist, all trajectories converge to $(K, 0, 0, 0)$ in the fashion of a "big bend" which is typical for predator-prey models. If ϱ is sufficiently large and κ, β are increased, then two new stationary points appear, a saddle point and a stable focus. Figure 10 represents trajectories starting from $x_0(0) = y_0(0)$, $x_1(0) = 1.2$ for varying $y_1(0)$, for $B = 1.2$ $\varrho = \kappa = \beta = 2$. Although the projection is not a phase-plane it looks very much like a phase picture of a two-dimensional system. The domain of attraction is tongue-shaped, very low and very large predator densities lead to extinction of the predator.

Of course we cannot exclude that for certain choices of the parameters new phenomena occur but this simulation supports the claim that the local analysis is valid over a certain range of parameters away from the bifurcation point.

7. Discussion

There are several different approaches towards modeling parasitic infections. One can consider host and parasite as interacting species, i.e. the parasite preys upon the host, or one can structure the host population according to the level of infection. The second approach gives a more detailed description of the population. For this reason we have divided both, the prey and the predator population, into classes of infected and of noninfected. Thus we describe the population of three species (prey-predator-parasite) by four variables. We deal with two different, though closely related problems, persistence of a parasite in a given prey-predator system and parasite mediated coexistence of prey and predator. As examples we can choose the european fox tapeworm *Echinococcus multilocularis* and the dog tapeworm *E. granulosus*. The adults of *E. multilocularis* live in the intestinal track of foxes, the eggs are spread in the environment via the feces, and are taken up by mice. The larvae develop in the tissues of mice (and undergo asexual multiplication), they enter the fox via predation. This tapeworm has some medical importance, since the larvae, if taken up by humans, cause an incurable fatal disease. It has been observed that, although the mouse-fox system is widespread, the tapeworm is endemic only in certain areas (at least cases of human infections are restricted to these areas). Hence it seems that the transmission conditions are not satisfied in all habitats. Of course the real situation is more complicated since foxes eat various prey other than mice and mice are decimated by different predators. The adults of *E. granulosus* live in dogs, the intermediate hosts are sheep. In many areas this parasite is becoming rare because of veterinary action. In certain areas of North America such as Western Canada and Alaska, but also on Isle Royale, *E. granulosus* is established on the moose-wolf prey-predator system.

E. multilocularis could be seen as an example of persistence on the mouse-fox prey-predator system. It persists if the transmission conditions are favorable. For this situation our model shows the typical epidemic threshold behavior. Independent of whether the uninfected populations coexist at a stable equilibrium or along a stable limit cycle there is a critical level for the product of the transmission rates, such that an infected equilibrium or an infected periodic solution appears whenever the threshold is exceeded.

In the second approach we assume that a predator cannot survive on a given prey. There is a parasite which can live in the prey and the predator as alternating hosts. The infection makes the prey more easily accessible to the predator, whereas the infection does not change the behavior of the predator. If the predator could not survive on a totally infected prey population, then there is, of course, no coexistence. If, however, the predator can subsist on totally infected prey then large transmission rates lead to a possible coexistence of prey and predator, i.e. a stable equilibrium (or periodic orbit) appears. Nevertheless in this situation the uninfected prey equilibrium remains locally stable. Hence the new coexistence equilibrium is not accessible from low predator levels. This behavior of the system differs from the first case, where coexistence is possible at all levels of infection. There $(K, 0, 0, 0)$ is unstable, and any small number of

predation lead to the coexistence equilibrium. Here the stable coexistence of prey, predators and parasites can only be achieved, if there are sufficiently many predators at the beginning. It has been suggested (Peterson) that some isolated wolf populations can survive on the local moose population only in the presence of *E. granulosus* which provides prey accessible to wolves in the form of heavily infected moose.

Here the positive orthant $\mathbb{R}^4_+$ is not persistent. Culling the predator leads to extinction of the predators and of the parasites. We conjecture, however, that there is a persistence domain bounded by some faces of $\mathbb{R}^4_+$ and the unstable manifold of the saddle point. In biological terms: there is a surface of $\mathbb{R}^4_+$ which separates a domain "many predators, many parasites" from a domain "few predators, or few parasites" such that the first domain is persistent in the sense of Sect. 4. In fact, as the computer simulation shows, an initially very large number of predators also leads to extinction of the predator.

8. Appendix: The Perron–Frobenius theorem

The Perron–Frobenius theorem for nonnegative matrices is well known and can be found in appropriate monographs, e.g., Gantmacher (1959). Here we need the continuous version which is folklore but not even the nomenclature is established (see, e.g., Arendt et al. 1986).

Let $A = (a_{jk})$ be a matrix acting as an operator on $\mathbb{R}^n$. Consider the discrete time evolution equation

$$x_{t+1} = Ax_t, \qquad t = 0, 1, 2, \ldots .$$

The behavior of the solutions is essentially determined by the spectrum of A. The matrix A is called nonnegative (positive) if $a_{jk} \geqslant 0$ (>0) for all j, k. If A is nonnegative then $x_0 \geqslant 0$ implies $x_t \geqslant 0$ for all t. A is called irreducible if A has no proper (i.e. different from $\{0\}$ and $\mathbb{R}^n$) invariant subspace which is the span of coordinate vectors. The essential statement of the Perron–Frobenius theorem is the following. If A is nonnegative and irreducible then its spectral radius (largest modulus of any eigenvalue) is itself an eigenvalue, it is a simple root of the characteristic polynomial, and it has a positive eigenvector. A nonnegative irreducible matrix A is called primitive if the spectral radius is strictly greater in modulus than the other eigenvalues. The matrix A is primitive if and only if A has a positive power. If A is primitive then every solution of the evolution equation starting in $\mathbb{R}^n_+ \setminus \{0\}$ approaches the positive eigenvector (up to scalar factors).

The continuous version of this theorem reads as follows: Consider the evolution equation

$$\dot{x}(t) = Ax(t), \qquad t \in \mathbb{R}.$$

We say that A preserves order if $a_{jk} \geqslant 0$ for $j \neq k$, i.e. $x(0) \geqslant 0$ implies $x(t) \geqslant 0$ for $t \geqslant 0$. If A is irreducible and preserves order than the "spectral bound" (the largest real part of any eigenvalue) is itself an eigenvalue and a simple zero of the

characteristic polynomial with a positive eigenvector. If A is order-preserving and irreducible then the spectral bound is strictly greater than the real part of any other eigenvalue if and only if for some $\alpha \geqslant 0$ the matrix $A + \alpha I$ is nonnegative and has a positive power. This is the case if and only if $\exp(tA)$ is positive for all $t > 0$. Then every solution of the evolution equation starting in $\mathbb{R}^n_+ \setminus \{0\}$ approaches the positive eigenvector (up to scalar factors).

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