Effects of a disease affecting a predator on the dynamics of a predator–prey system

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A B S T R A C T
We study the effects of a disease affecting a predator on the dynamics of a predator–prey system. We couple an SIRS model applied to the predator population, to a Lotka–Volterra model. The SIRS model describes the spread of the disease in a predator population subdivided into susceptible, infected and removed individuals. The Lotka–Volterra model describes the predator–prey interactions. We consider two time scales, a fast one for the disease and a comparatively slow one for predator–prey interactions and for predator mortality. We use the classical “aggregation method” in order to obtain a reduced equivalent model. We show that there are two possible asymptotic behaviors: either the predator population dies out and the prey tends to its carrying capacity, or the predator and prey coexist. In this latter case, the predator population tends either to a “disease-free” or to a “disease-endemic” state. Moreover, the total predator density in the disease-endemic state is greater than the predator density in the “disease-free” equilibrium (DFE).

1. Introduction

Ecology and epidemiology are two major and distinct fields of study. However there are situations where some diseases which are responsible for epidemics, impact heavily on predator–prey systems. For instance, Hethcote et al. (2004) show how the presence of parasites can change the demographic behavior of a population. Such diseases regulate the host population density (Getz and Pickering, 1983) and sometimes help the co-existence of species (Bairagi et al., 2007). Eco-epidemiology which is a relatively new branch of study in theoretical biology, tackles such situations by dealing with both ecological and epidemiological issues. It can be viewed as the coupling of an ecological predator–prey (or competition) model and an epidemiological SI, SIS or SIRS model. Following Anderson and May (1982) who were the first to propose an eco-epidemiological model by merging the ecological predator–prey model introduced by Lotka and Volterra, and the epidemiological SIR model introduced by Kermack and McKendrick, many works have been devoted to the study of the effects of a disease on a predator–prey system (Beltrami and Coarrol, 1994; Beretta and Kuang, 1998; Ebert et al., 2000; Freedman, 1990; Hadeler and Freedman, 1989). Most models study predator–prey–parasite systems with microparasitic infections (Chattopadhyay and Arino, 1999; Chattopadhyay and Bairagi, 2001; Chattopadhyay and Pal, 2002; Freedman, 1990; Hadeler and Freedman, 1989; Venturino, 2002), while a few ones consider macroparasitic infections (Hudson et al., 1992; Packer et al., 2003). In most microparasitic infection predator–prey models, the parasites infect only the prey population (Chattopadhyay and Arino, 1999; Chattopadhyay and Bairagi, 2001; Chattopadhyay and Pal, 2002; Chattopadhyay et al., 2003; Hethcote et al., 2004; Hudson et al., 1992; Xiao and Chen, 2001). In such circumstances, a prey epidemics which would have evolved towards an endemic state without predators, can die out because of the high rate of predation on infectious and hence more vulnerable prey. Another hypothesis was considered by Bairagi et al. (2007), who suggested that the predator may avoid the infected prey and predate only the susceptible ones, leading to prey extinction. In some other cases, the parasites originally
hosted by the prey, are able to infect the predator and cross the species barrier (Haderle and Freedman, 1989; Venturino, 2002; Anderson and May, 1982). This happens for instance in Haderle and Freedman (1989) where they consider the possibility of an infection spreading into the predator population, following the predation of an infected prey by a susceptible predator.

Among the numerous studies devoted to the effects of a disease on a predator–prey system (Beltrami and Coarroll, 1994; Chattopadhyay et al., 2003; Chattopadhyay and Bairagi, 2001; Chattopadhyay and Arino, 1999; Ebert et al., 2000; Freedman, 1990; Haderle and Freedman, 1989; Hethcote et al., 2004; Hudson et al., 1992; Michich et al., 2007; Packer et al., 2003; Xiao and Chen, 2001), most contributions consider epidemics affecting the prey, and comparatively less attention has been paid to the effects of a disease affecting the predator (Han et al., 2001; Haque and Venturino, 2007; Hethcote et al., 2004; Venturino, 2002). This contrasts with the empirical evidence that a disease spreading in a population of predators can have unexpected effects (Wilmers, 2006). The aim of this work is to propose a predator–prey model in which predators can become infected by a disease. We consider two time scales, a fast one for the disease and a comparatively slow one for predator–prey interactions and for predator mortality.

We use the “aggregation method” in order to obtain a reduced equivalent model with variables \((n, p)\), where \(n\) is the prey density and \(p\) is the predator density. We show that this aggregated model always has a globally asymptotically stable equilibrium \((n^*, p^*)\). Depending on the values of the parameters, this equilibrium is either predator free, i.e. \(p^* = 0\), positive and disease free, i.e. \((n^*_1, p^*_1)\), with \(n^*_1 > 0\), \(p^*_1 > 0\), \(p^*_1 = S_1^* + I_1^* + R_1^*\) and \(I_1^* = 0\), or positive and endemic, i.e. \((n^*_2, p^*_2)\), with \(n^*_2 > 0\), \(p^*_2 > 0\), \(p^*_2 = S_2^* + I_2^* + R_2^*\) and \(I_2^* > 0\). Moreover, if \(\beta\) is the infection rate and \(\delta\) is the rate at which infected predators enter into compartment \(R\), then the inequality \(p^*_1 < \delta/\beta < p^*_2\), holds. In other words, the predator density at endemic equilibrium is always greater than the predator density at disease-free equilibrium (DFE). All these results are obtained under the hypothesis that the transmission of the disease follows the classical mass action law \(SIR\), where \(S\) and \(I\) are densities such as population numbers per unit area or unit volume. More complex models of pathogen transmission as functions of the densities \(S\) and \(I\) can be considered (McCallum et al., 2001), for instance, the asymptotic law of incidence (Anderson and May, 1978; Heesterbeek and Metz, 1993; McCallum et al., 2001; Roberts, 1996) has been proposed for populations of phytoplankton contaminated by viruses. In Section 5, we analyze a modified eco-epidemiological model where the “true mass action” (de Jong et al., 1995; McCullum et al., 2001), since \(S\) and \(I\) represent densities, rather than numbers. This transmission law has been used in Beretta and Kuang (1998), Chattopadhyay and Pal (2002), Diekmann and Kretzschmar (1991), Haderle and Freedman (1989), Han et al. (2001), Xiao and Chen (2001) and is common to numerous models in the literature.

As assumed above, the disease dynamics is fast whereas the mortality and the recruitment of predators are comparatively slow.

For instance, in the fourth equation, the contribution of the epidemiological process in the time scale \(t\) is \(dR/dt = (\gamma I - \gamma R)\), whereas the contribution of predator mortality and recruitment in the time scale \(t\) is \(dR/dt = [-\mu R + bnR]\), or equivalently \(dR/dt = dR/dt \cdot dR/dt = \varepsilon(\mu R + bnR)\), and the fourth equation follows:

\[
\begin{align*}
\frac{dn}{dt} &= \left[\frac{m(1 - n)}{R} - an(S + I + R)\right], \\
\frac{ds}{dt} &= \left[\gamma R - \beta SI\right] + \varepsilon(-\mu S + bnS)], \\
\frac{dl}{dt} &= \left[\beta SI - \delta I\right] + \varepsilon(-\mu l + bnI)], \\
\frac{dr}{dt} &= \left(\delta I - \gamma R\right) + \varepsilon(-\mu R + bnR].
\end{align*}
\]

The predator–prey parameters of the model are as follows: \(r\) is the prey intrinsic growth rate, \(K\) is the prey carrying capacity, \(a\) is the capture rate and \(b\) is a parameter related to predator recruitment as a consequence of predator–prey interactions. It is usually assumed that \(b = ea\) where \(e\) is a conversion parameter from prey to predator density.

The epidemiological parameters \(\gamma, \beta\) and \(\delta\) represent, respectively, the rate at which predators loss immunity, the infection rate and the rate at which infected predators become immune.

The predator mortality rate is \(\mu\). We assume that, even if the disease is a mild one, it has a long term impact on the predator longevity. This extra-mortality of infected predators happens at rate \(\mu'\).
Examples of such diseases are infections with asymptomatic carriers, i.e. diseases where infected individuals have no apparent symptoms. The behavior of such infected individuals is not modified, but in the long term their immune system is affected and becomes less and less efficient. This causes a decrease of the average longevity. A first example of such a disease is the toxoplasmosis for feline species. Feline are the only definitive hosts for this disease which is caused by a single-celled parasite called Toxoplasma gondii. Most infected feline do not show any symptom, but the presence of antibodies to T. gondii in lynx and bobcats suggests that this organism is widespread (Labelle et al., 2001). The predation of hares by lynx is therefore a good example illustrating the study carried out in this paper. A second example is avian malaria (Cosgrove et al., 2006; Merino et al., 1997) where birds serve as definitive hosts. Indeed, Plasmodium can be pathogenic to penguins, domestic poultry, ducks, canaries, falcons and pigeons, but it is most commonly carried asymptotically by passerine birds which are predators for some insect species.

2.1. Aggregated model

We are now going to build an equivalent aggregated system based on the slow time scale, by assuming that the complete system is permanently in a state corresponding to the fast equilibrium.

2.1.1. Case 1: \( \mathcal{R}_0 < 1 \)

When \( \mathcal{R}_0 < 1 \), which is equivalent to \( p < \delta / \beta \), the unique nonnegative fast equilibrium is \( (p, 0, 0) \), hence \( l = 0 \). The aggregated system is

\[
\begin{align*}
\frac{dn}{dt} &= n \left[ r \left( 1 - \frac{n}{K} \right) - ap \right], \\
\frac{dp}{dt} &= p (-\mu + bn).
\end{align*}
\]

(2)

2.1.2. Case 2: \( \mathcal{R}_0 > 1 \)

When \( \mathcal{R}_0 > 1 \), which is equivalent to \( p > \delta / \beta \), the unique fast equilibrium is the one exhibited in Section 3.1. This equilibrium is endemic and the corresponding aggregated system is

\[
\begin{align*}
\frac{dn}{dt} &= n \left[ r \left( 1 - \frac{n}{K} \right) - ap \right], \\
\frac{dp}{dt} &= -\mu p - \frac{\mu' \gamma}{\gamma + \delta} \left( p - \frac{\delta}{\beta} \right) + bn.
\end{align*}
\]

(3)

This shows that, depending on \( p \), the aggregated model is either system (2) or system (3). Let us now subdivide the positive quadrant \( (n, p) \) into two sub-domains:

\[ D_1 = [0, +\infty) \times \left[ 0, \frac{\delta}{\beta} \right] \quad \text{and} \quad D_2 = [0, +\infty) \times \left[ \frac{\delta}{\beta}, +\infty \right]. \]

In sub-domain \( D_1 \) the aggregated system (2) is valid, whereas in subdomain \( D_2 \) system (3) holds. This latter system looks like a Lotka–Volterra system, but with a constant immigration in the predator population.

2.1.3. Dynamics of the aggregated system

Let \( X_1(n, p) \) be the vector field associated with (2) on \( \mathbb{R}^2 \), and let \( X_2(n, p) \) be the vector field associated with (3) on \( \mathbb{R}^2 \). The aggregated system is defined on the nonnegative orthant \( \mathbb{R}^2_+ \) by a vector field which is equal to \( X_1 \) on the closure of \( D_1 \) and is equal to \( X_2 \) on \( D_2 \). To summarize, let us denote \( X(n, p) \) the aggregated vector field defined by the following equation:

\[
\begin{align*}
X(n, p) &= \begin{cases} 
X_1(n, p) = \left[ \frac{n[r(1 - \frac{n}{K}) - ap]}{p(-\mu + bn)} \right] & \text{if } (n, p) \in \text{closure}(D_1), \\
X(n, p) = \begin{cases} 
\frac{n[r(1 - \frac{n}{K}) - ap]}{-\mu p - \frac{\mu' \gamma}{\gamma + \delta} (p - \frac{\delta}{\beta}) + bn} & \text{if } (n, p) \in D_2.
\end{cases}
\end{cases}
\]

(4)

It is straightforward to check that this vector field is continuous on the entire positive orthant, and is \( \mathcal{C}^1 \) on the subsets \( D_1 \) and \( D_2 \). Moreover, starting from any point of the boundary between \( D_1 \) and \( D_2 \), i.e. if initially \( \mathcal{R}_0 = 1 \) or equivalently \( p = \delta / \beta \), the trajectory issued from this vector field leaves instantaneously the boundary.

It follows from classical results on dynamical systems, that this vector field has a unique solution for any initial condition in the orthant.

2.2. Equilibria and stability

We are now going to compute the “slow” equilibria of the dynamical system associated with \( X \) on the positive orthant. Then we will analyze their stability.

On \( \mathbb{R}^2 \) the vector field \( X_1 \) has there equilibria: \( (0, 0), (K, 0) \) and \( (n_1^*, p_1^*) \) where \( n_1^* = \mu / b \) and \( p_1^* = r / a (1 - \mu / b K) \).

On \( \mathbb{R}^2 \) the vector field \( X_2 \) has two equilibria: \( (0, \bar{p}) \in D_1 \) with

\[
\bar{p} = \frac{1}{1 + \frac{\mu' \gamma}{\gamma + \delta} p} \quad \text{and} \quad (n_2^*, p_2^*) \text{ with}
\]

\[
p_2^* = \frac{r}{a} \left( -\frac{\delta (\delta + \gamma)}{2b \beta K (\delta + \gamma)} + \sqrt{A} \right) > 0
\]

and

\[
n_2^* = 1 - \frac{a}{r} p_2^*.
\]

where

\[
A = \frac{\beta^2 (\delta + \gamma) (\mu - b K) + \mu' \gamma^2}{r} \frac{4abK \beta \delta (\delta + \gamma)}{r} > 0.
\]

For each equilibrium \( (n_i^*, p_i^*) \), \( i = 1, 2 \), we have a corresponding basic reproduction number

\[
\mathcal{R}_0^* = \frac{\bar{p}}{a} p_i^*.
\]

Theorem 2.1. With the preceding notations, the following properties hold:

- The trivial equilibrium \( (0, 0) \) is a saddle point.
- If \( p_1^* < 0 \), then the PFE \( (K, 0) \) is GAS.
- If \( 0 < p_2^* < \delta / \beta \) or equivalently \( \mathcal{R}_0^* < 1 \), then the equilibrium state \( (n_1^*, p_1^*) \in D_1 \) is GAS on the positive orthant.
- If \( \delta / \beta < p_2^* \) or equivalently \( \mathcal{R}_0^* > 1 \), then the equilibrium state \( (n_2^*, p_2^*) \in D_2 \) is GAS on the positive orthant.

Proof. See Appendix A.

2.3. Numerical results

In this section we present some simulations which illustrate Theorem 2.1. Hereafter, blue lines are trajectories of the system,
black lines are isoclines and a red line, the line \( p = \frac{\delta}{\beta} \), is the separation between \( D_1 \) and \( D_2 \):

- In Fig. 1 there is no positive equilibrium point. Indeed, the predator population dies out and the prey population tends to its carrying capacity.
- In Fig. 2, \( (n_1^*, p_1^*) \) is the unique equilibrium point. It is GAS and it belongs to \( D_1 \) since it is under the red horizontal line of equation \( p = \frac{\delta}{\beta} = 4 \).
- In Fig. 3, \( (n_2^*, p_2^*) \) is the only equilibrium point. It is GAS and it belongs to \( D_2 \) since it is above the red horizontal line of equation \( p = \frac{\delta}{\beta} = 1.6 \).

Let us now compare the aggregated model to the complete model. In Figs. 4–6, the original model corresponds to red dots whereas the aggregated model correspond to blue dots. It can be seen that the two phase plane trajectories coincide for small values of \( \epsilon \).

3. Comments

1. The case \( p_1^* < 0 \), occurs when there is no positive stable equilibrium in the positive orthant. In this case, \((K, 0)\) is the unique stable equilibrium. Fig. 1 illustrates this case of predator extinction and stabilization of the prey at its carrying capacity.
2. illustrate the case of a unique positive stable equilibrium either in \( D_1 \) or in \( D_2 \). In the long term, the total predator density tends to an equilibrium value \( p^* \) whereas the prey density tends to an equilibrium value \( n^* \). In Fig. 2, \( p_1 < \frac{\delta}{\beta} \) and the equilibrium point \( (n_1^*, p_1^*) \) is disease free, whereas in Fig. 3, \( p_2 \geq \frac{\delta}{\beta} \) and the equilibrium point \( (n_2^*, p_2^*) \) is endemic.
3. The predator density at endemic equilibrium is always larger than the predator density at DFE.

4. Let us now go back to the original system (1). Since $p = S + I + R$, this system can be written

$$\begin{align*}
\frac{dn}{dt} &= \varepsilon \left[ r \left( 1 - \frac{n}{R} \right) - an(S + I + R) \right], \\
\frac{dp}{dt} &= \varepsilon \left[ p(-\mu + bn) - \varepsilon \mu l \right].
\end{align*}$$

When compared to the classical Lotka–Volterra equations with logistic growth for the prey, this system differs only in the term $-\mu l$ in the second equation. Moreover, it corresponds to the vector field $\mathbf{X}_1$ of Section 3.2.3, modified by the term $-\mu l$ in its second component. It is classical (see Appendix A) that $X_1$ admits either a coexistence positive equilibrium which is GAS, or a GAS predator-free equilibrium. With this global property for $X_1$, there exists a positively invariant compact set $\Omega$ containing a subset $[0, N] \times [0, P]$ for $N$ and $P$ sufficiently large. $\Omega$ is an absorbing set (Li and Muldowney, 1996). This means that for any initial condition, the system ultimately enters into the interior of $\Omega$. Using continuity arguments, it can be shown that there exists, for a sufficiently small values of $\varepsilon$, a set which satisfies the same conditions for system (5). The global asymptotic stability of (1) is then reduced to the GAS on a compact positively invariant set. Since the “slow” manifold is attracting in a positively compact set, a reduction principle holds (Seibert, 1997; Seibert and Florio, 1995): the GAS of the aggregated system ensures the GAS of the initial system.

4. Modified model with asymptotic transmission

The mode of transmission is crucial, because it has a great impact on the evolution of a system where a pathogen is introduced in a susceptible population. The most used transmission law is undoubtedly the law of mass action. However, several more complex functions relating disease transmission to the densities of susceptible and infected hosts have also been proposed: frequency-dependent transmission, power relationship, negative binomial, Holling like, asymptotic transmission, etc.

In this section, we will analyze the dynamics of our model when the disease transmission follows an asymptotic transmission (Anderson and May, 1978; Heesterbeek and Metz, 1993; McCallum et al., 2001; Roberts, 1996). This law of transmission has been considered in a predator–prey model with infection of the prey (Belltrami and Cao, 1994).

Under such a modification the model becomes

$$\begin{align*}
\frac{dn}{dt} &= \varepsilon \left[ r \left( 1 - \frac{n}{R} \right) - an(S + I + R) \right], \\
\frac{ds}{dt} &= \left( \gamma R - \beta \frac{SI}{S+I} \right) + \varepsilon [-\mu S + bnS], \\
\frac{dl}{dt} &= \left( \beta \frac{SI}{S+I} - \delta l \right) + \varepsilon [-\mu l - \mu l + bnI], \\
\frac{dr}{dt} &= (\delta l - \gamma R) + \varepsilon [-\mu R + bnR].
\end{align*}$$

As in the preceding section, we will take advantage of the presence of two different time scales by first computing the fast equilibrium, and then analyzing the equivalent aggregated model obtained by substituting this fast equilibrium to the corresponding variables in the complete system.

4.1. Fast equilibrium

Lemma 4.1.

- If $\beta > \delta$ then the fast equilibrium point is endemic where $S^* = \gamma \delta p / \gamma \beta + (\beta - \delta) \delta$, $I^* = \gamma (\beta - \delta) p / (\gamma \beta + (\beta - \delta) \delta)$, $R^* = \delta (\beta - \delta) p / (\gamma \beta + (\beta - \delta) \delta)$ is asymptotically stable.
- If $\beta < \delta$ then the fast equilibrium point is disease free where $S^* = p$, $I^* = 0$, $R^* = 0$ is also asymptotically stable.

4.2. Aggregated model

The aggregated model is

$$\begin{align*}
\frac{dn}{dt} &= r n \left( 1 - \frac{n}{R} \right) - anp, \\
\frac{dp}{dt} &= -\bar{\mu} p + bnp,
\end{align*}$$

where

$$\begin{align*}
\bar{\mu} &= \mu, \\
\bar{\mu} &= \mu + \gamma (\beta - \delta) \delta, \\
\bar{\mu} &= \mu + \gamma (\beta - \delta) \delta, \\
\bar{\mu} &= \mu + \gamma (\beta - \delta) \delta, \\
\bar{\mu} &= \mu + \gamma (\beta - \delta) \delta.
\end{align*}$$
4.3. Equilibrium points and stability analysis of the aggregated model. There are three possible equilibrium points for the aggregated model (7):

- \((0,0)\) which is a always saddle point.
- \((K,0)\) which is a stable equilibrium if \(K < \bar{m}/\bar{b}\) and saddle point otherwise.
- \((n^*, p^*)\) where \(n^* = \bar{m}/\bar{b}\) and \(p^* = r/\alpha(1 - \bar{m}/bK)\). This last equilibrium is stable if \(K > \bar{m}/\bar{b}\) and is a saddle point otherwise.

The interior equilibrium point shows that the predator density in the DFE is larger than the predator density in the endemic equilibrium.

In this model, the comparison of the predator densities of disease free and endemic equilibria, lead to a conclusion opposite to what we found in the model with the law of mass action.

5. Conclusion

In this paper, we have studied the effects of a disease affecting a predator, on the dynamics of a predator–prey system. The SIRS model with simple mass action law was first used to describe the spread of the disease in the predator population. In this model, the population is divided into three compartments, namely susceptible, infected and removed individuals. On the other hand, the predator–prey interactions were modeled by the classical Lotka–Volterra equations.

It was assumed that there are two time scales, a fast one for the disease and a comparatively slow one for predator–prey interactions and for predator mortality. The “aggregation method” was used in order to obtain a reduced equivalent system with parameters \((n, p)\), where \(n\) is the fast equilibrium value for the prey population and \(n\) is the fast equilibrium value for the predator population. Since there are two possible fast equilibria which are stable, the aggregated model uses a vector field \(X_1\) if \(p < \delta/\beta\), and another vector field \(X_2\) otherwise. The analysis carried out here shows that the aggregated system has three possible equilibria which are GAS: a predator-free equilibrium \((K,0)\), a disease-free equilibrium \((n_1, p_1)\), with \(p_1 < \delta/\beta\), and a positive endemic equilibrium \((n_2, p_2)\), with \(p_2 > \delta/\beta\).

The fact that the predator population \(p_2\) at endemic equilibrium is greater than the predator population \(p_1\) at disease-free equilibrium is consistent with the fact, in SIRS models with standard mass action law, the basic reproduction number \(R_0\) is an increasing function of the total density. Then we have studied the dynamics of the system when the disease transmission follows an asymptotic incidence law. In this case, the predator population at DFE is greater than the predator population at endemic equilibrium.

We have therefore considered two transmission laws which lead to opposite conclusions with respect to the predator densities at equilibrium. In practice, the choice of the adequate transmission law is crucial but somewhat difficult. For example de Jong et al. (1995) analyzed data from the Pasteurella muris laboratory epidemics in mice modeled by Anderson and May (1978), and concluded that frequency-dependent (i.e. SI/N) and density-dependent transmission (i.e. mass action) models fitted the data equally well. The same authors remark that the “true mass-action” assumption is valid whenever individuals move randomly within the space in which population lives, with “contacts” corresponding to “collisions”.

All the results presented here are based on the classic SIRS epidemic. Moreover predator–prey interactions are modeled by a Lotka–Volterra system whose parameters are independent of the predator epidemiological status. It may be interesting to see what happens with more complex epidemic models, different transmission laws and other predator response functions such as Holling types II and III.

### Appendix A

In this appendix we give the proof of Theorem 2.1. We use the notation of Section 2.2. To prove the theorem we begin to prove two results on the equilibria of the vector field \(X_1\) and \(X_2\).

**Lemma 6.1.** The assertion \((n_2, p_2) ∈ D_2\) is equivalent to \((n_1', p_1') ∈ D_2\). \((n_2', p_2') ∈ D_1\) is equivalent to \((n_1', p_1') ∈ D_1\).

**Proof.** We recall \(p_1' = (r/\alpha)(1 - \mu/bk)\) and \(p_2' = r[p(\delta + \gamma)(bk - \mu - \mu'\gamma)]/2abK(\delta + \gamma)\) is equivalent to \(\psi = \psi'\).

We can rewrite this expression as

\[
\psi = \frac{r\mu'\gamma}{2abK(\delta + \gamma)}.
\]

Suppose now that \((p_2') ∈ D_2\), which is equivalent to \(p_2' > \delta/\beta\) or equivalently

\[
\left(\frac{p_1'}{2} - \psi\right)^2 + 2\frac{\delta}{\beta} > \left(\frac{\delta}{\beta} + \frac{p_1'}{2} + \psi\right)^2,
\]

which gives \((\delta/\beta)(\delta/\beta - p_1'/2) < 0\), hence \((n_1', p_1') ∈ D_2\). The second equivalence is obtained by an identical proof: By continuity we obtain nonstrict inequalities, the equivalence of negations gives the results. This ends the proof of the lemma.

**Lemma 6.2.** The equilibrium \((n_2, p_2)\) is globally asymptotically stable on the positive orthant for \(X_2\). If \(p_1' > 0\) then \((n_1', p_1')\) is globally asymptotically stable on the positive orthant.

**Proof.** The proof use classical Lyapunov functions for Lotka–Volterra systems (Goh, 1976; Harrison, 1979). For the two systems we consider, on the positive orthant

\[
V(x, p) = \frac{b}{a}(n - n_1\ln(n) - n_1'\ln(n_1')) + (p - p_1'\ln(p) - p_1'\ln(p_1')).
\]

Writing \(X_1\) as

\[
X_1(n, p) = \begin{bmatrix}
\frac{an_1'}{bp}(n_1' - n) + (p_1' - p) \\
bp(n - n_1')
\end{bmatrix}.
\]

For \(X_2\) we set \(x = \mu'\gamma(\gamma + \delta)\beta\) so that we can write \(X_2\) as

\[
X_2(n, p) = \begin{bmatrix}
\frac{an_1'}{bp}(n_1' - n) + (p_2' - p) \\
pb(n - n_1'') + \frac{p_2'' - p}{p_2'}
\end{bmatrix}.
\]
It is now straightforward to obtain the derivatives of $V_i$ along the trajectories of $X_i$:

$$V_1 = -\frac{b}{ak}(n - n_i)^2 \leq 0$$

and

$$V_2 = -\frac{b}{ak}(n - n_2)^2 - \frac{(p_2 - p)^2}{\beta_2} \leq 0.$$  

Using Lyapunov theorems for $X_1$ and $X_2$, with LaSalle’s invariance principle for $X_1$, we conclude to the of the equilibria. 

The Jacobian matrix $J_0$ of $X_1$ computed at $(0,0)$ is given by

$$J_0 = \begin{bmatrix} r & 0 \\ 0 & -\mu \end{bmatrix}.$$  

For any value of the parameters the origin is a saddle point. Its stable manifold is the $p$-axis.

The Jacobian matrix $J_1$ of $X_1$ computed at $(n_1^*, p_1^*)$ is given by

$$J_1 = \begin{bmatrix} r - an_1 & 0 \\ bp_1 & 0 \end{bmatrix}.$$  

The Jacobian matrix $J_2$ of $X_1$ computed at $(n_1^*, p_1^*)$ is given by

$$J_2 = \begin{bmatrix} -\frac{an_1^*}{p} & -an_1^* \\ bp_1 & -\frac{\mu + \delta}{\gamma} \end{bmatrix}.$$  

If $p_2 < 0$, from Lemma 6.1, this implies $(n_2^*, p_2^*) \notin D_2$. The Jacobian matrix $J_k$ shows that $(K,0)$ is the unique asymptotically stable equilibrium of the aggregated vector field $X$.

If $0 < p_1^* < \delta/\beta$. Then $(n_1^*, p_1^*) \in D_1$ and $(n_2^*, p_2^*) \in D_1$ by Lemma 6.1. With Lemma 6.2 we deduce that $(n_1^*, p_1^*) \in D_1$ is asymptotically stable for $X$ (at least locally) and that any trajectory of $X$ starting in $D_2$ enters $D_1$.

If $p_2 > \delta/\beta$ then $(n_2^*, p_2^*) \in D_2$ and $(n_1^*, p_1^*) \in D_1$ by Lemma 6.1. With Lemma 6.2 we deduce that $(n_2^*, p_2^*) \in D_2$ is asymptotically stable for $X$ (at least locally) and that any trajectory of $X$ starting in $D_1$ enters $D_2$.

To prove the global stability, it is now sufficient to eliminate the possibility of limit cycles. A theoretical possibility is a trajectory starting from the boundary of $D_1$, entering $D_2$ (e.g. in case 3), and cycling with the first trajectory. With the properties of $X$ we can use Dulac criterion with $B(n,p) = 1/np$. A closed path cannot exist if

$$\frac{\partial}{\partial n}(BP_1) + \frac{\partial}{\partial p}(BQ_1)$$

has the same sign, where $P_1$ and $Q_1$ are, respectively, the first and second components of the vector field $X_1$.

We have

$$\frac{\partial}{\partial n}(BP_1) + \frac{\partial}{\partial p}(BQ_1) = -\frac{r}{K}$$

and

$$\frac{\partial}{\partial n}(BP_2) + \frac{\partial}{\partial p}(BQ_2) = -\frac{r}{K} - \frac{\mu + \gamma}{(\delta + \gamma)p}.$$  

This ends the proof of the theorem. □

References


