



Effects of Diseased Top Predators in Food Chains

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Abstract

Ecoepidemiology studies spreading diseases among interacting populations. Food webs occur everywhere in nature. In this paper we investigate a dynamical system for an epidemic affecting the top predators in a three-trophic level food chain. The feasible model equilibria are identified and their stability is assessed, showing transcritical bifurcations relating some of them, and analytically establishing the impossibility of Hopf bifurcations, with the exception for the coexistence equilibrium. Simulations reveal indeed that all subpopulations can thrive together by sustained periodic oscillations.

This investigation supplements other parallel studies on other tri-trophic ecoepidemic food chains. The general conclusions support earlier findings that purely demographic models are not an adequate description of real environments, if possible disease effects are not suitably accounted for in the model formulation.

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1 Introduction

Interactions between populations are ubiquitous in nature. In particular competition for resources and predation for feeding purposes represent very common phenomena. It is often the case that several such interactions occur at the same time between different populations, giving rise to what are called food webs. A subset of these is made by food chains, in which there is a top-down order of the populations involved, with a top predator feeding on a second population, which itself hunts a third population, that in turn needs another one to survive, and so on to arrive at the prey at the lowest level.

Ecoepidemiology is the branch of biomathematics dealing with the spread of diseases among intermingling populations, see Chapter 7 of [1] for a brief introduction. It envisions a closer-to-reality model representation of communities living in natural environments, because in addition to

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their demographic interactions, it considers also the possible effects of diseases affecting one or more of the populations under scrutiny. An example is given in [2], where diseases in aquatic systems are considered. In this context generally the scope of mathematical modeling has been the consideration of simple systems involving two populations with an endemic disease. Note that here the word “simple” refers to the structure of the interactions, while the mathematical framework can be rather sophisticated and attains quite intricate consequences, see for instance [3,4,5]. More recently however, also food chains have been considered [6,7].

In this paper we consider a simple three-trophic levels food chain affected by a disease. The epidemics is assumed to affect only the top predators. The study completes parallel investigations in which the models contained the disease strains at the other trophic levels [6,7].

The paper is organized as follows. In the next section we present some biological antefacts, section 3 contains the model, then some particular cases are investigated. The model is analysed in section 5, the results are interpreted in section 6 and a final conclusion summarizes the findings.

2 Biological Background

The interactions among several populations in different natural contexts always aroused the interest of many researchers, see e.g. [8,9,10]. More recently, also the influences of diseases in these ecosystems have been elucidated, for instance in [11,12,13].

In [13] it is observed that stream fishes largely feed on crickets affected by nematomorph parasites. Attempting to understand how this behavior affects the ecosystem, the experiments show that when crickets abound, predatory fish eat fewer other invertebrates, that in turn decrease the amount of algae in the water. Thus the ecosystem function can be substantially altered by the presence and the action of a parasite within its host. Similar conclusions are suggested also in other ecosystems by the presence of many other parasites.

The influence of the wheather can heavily affect host-pathogen relationships. It could then be possible that several infections can occur simultaneously, with heavier consequences for the affected populations. The data reported in [12] indicate how unfavourable climatic conditions can trigger coinfections with higher mortalities than normal. Two specific examples are pointed out involving the canine distemper virus (CDV): the 1994 CDV epidemic in Serengeti lions (*Panthera leo*) and the 2001 CDV epidemic in the nearby Ngorongoro Crater lion. Data from earlier times show that CDV is not usually fatal, but it becomes so when coupled with hemoparasitism. The chain reaction that led to these extremely virulent epidemics was prompted by droughts that caused high Cape buffalo (*Syncerus caffer*) mortality, followed by heavy rains that favored the infestation of the buffalos by ticks. As a consequence, coinfections due to extremely high Babesia populations, coupled by the immunosuppressive effects of CDV coinfections severely affected the lions.

Reduced biodiversity affects the transmission of diseases in the animal as well as in the vegetative realm, in general increasing it [11]. But also the reverse could occur. Indeed, [11] also address the question of biodiversity within the body of an individual host, or “microbiome”. Some interesting experiments show that an increase in the microbial diversity proves beneficial against infections, by preventing invasion of pathogens. A large degree of biodiversity could constitute instead a source, or the reserve, of new pathogenic agents. Thus the preservation of pristine ecosystems would be the best way of fighting diseases. Essentially, biodiversity can affect disease transmission in several ways. In an environment with several populations the successful contacts of disease carrier or parasites are reduced, as not every individual may constitute a favorable host for the pathogenic agent, or the latter could find competitor or predatory organisms to keep it in check. But on the other hand a diverse environment could foster a higher dynamics of the disease carrier, and therefore increase disease prevalence. A larger set of species that are not all possible hosts for a specific pathogen may reduce the prevalence, but at the same time in a larger diversity of species more vectors for a specific disease may be harbored.

Evidently, from all these considerations, it is clear that purely qualitative statements of opposite nature can be derived. These considerations emphasize also the need of mathematical modelling, to better assess quantitatively the outcome of the system.

3 The Model

The three populations of concern here are the top predator, which is affected by a disease and therefore divided into two subpopulations of susceptibles S and infected I , the intermediate predator P and the prey Q at the bottom. The top predators cannot survive without the presence of the intermediate population. The susceptibles migrate into the class of infected when they come in contact with individuals of this class, at rate β . Their mortality rate is denoted by m . They hunt the population P and produce healthy offsprings at rate a . The infected instead are subject to a natural plus disease-related mortality γ , and hunt the population P , giving birth to infected offsprings at rate $b \neq a$ in general. Therefore, we assume that the disease is vertically transmitted. New infected of course come also from the class S as mentioned before, at rate β . The intermediate population has a mortality rate v , is captured at rates $l > a$ and $e > b$ respectively by susceptible and infective top predators, and predate upon the bottom population Q , converting them into newborns at rate c . The prey Q instead are assumed to have enough resources in the environment, so as to be able to reproduce at rate r and reach a carrying capacity K in the absence of disturbances. They are however captured at rate $h > c$ by individuals of the population P . The model is thus formulated as follows

$$\begin{aligned} \frac{dS}{dt} &= -\beta SI - mS + aSP \\ \frac{dI}{dt} &= \beta SI + bIP - \gamma I \\ \frac{dP}{dt} &= -lSP - eIP - vP + cPQ \\ \frac{dQ}{dt} &= rQ\left(1 - \frac{Q}{K}\right) - hPQ. \end{aligned} \tag{3.1}$$

Note in particular that due to the presence of the term bIP in the infected equation, as already stated above, here we have also vertical transmission, which makes this model sensibly differ from both the systems considered in [6] and [7].

4 Special Situations

4.1 The model in the absence of epidemics

Although this particular case is examined in [7], we briefly present here a summary of the main findings for the convenience of the reader.

Here, the first two equations of (3.1) need to be replaced by an equation for the top predator total population $U = S + I$. In their place, we thus obtain the equation

$$\frac{dU}{dt} = -mU + aUP.$$

In the model (3.1) one needs to delete the terms containing I in the equation for the population P at the intermediate trophic level. Also, the Jacobian becomes a 3×3 matrix.

The system has only three meaningful equilibria, since the origin is unconditionally unstable. The bottom prey-only-equilibrium $A_1 = (0, 0, K)$, the top-predator-free equilibrium $\bar{A} = (0, \bar{P}, \bar{Q})$,

$$\bar{P} = \frac{r}{h} \left(1 - \frac{v}{cK}\right), \quad \bar{Q} = \frac{v}{c}$$

and the coexistence equilibrium $A^* = (U^*, P^*, Q^*)$, with

$$U^* = \frac{1}{l} \left[cK \left(1 - \frac{hm}{ar} \right) - v \right], \quad P^* = \frac{m}{a}, \quad Q^* = K \left(1 - \frac{hm}{ar} \right).$$

Now, A_1 is stable if

$$\frac{cK}{v} < 1. \tag{4.1}$$

Instead, \bar{A} is feasible for the opposite condition

$$\frac{cK}{v} > 1, \tag{4.2}$$

showing thus a transcritical bifurcation. For \bar{A} the stability condition is

$$\frac{m}{a} > \frac{r}{h} \left(1 - \frac{v}{cK} \right). \tag{4.3}$$

A^* is feasible for the opposite condition, namely

$$\frac{m}{a} < \frac{r}{h} \left(1 - \frac{v}{cK} \right), \tag{4.4}$$

thus providing another transcritical bifurcation. The characteristic equation in this case is $\sum_{k=0}^3 A_k X^k$, with $A_3 = 1$,

$$A_2 = \frac{r}{K} Q^* > 0, \quad A_0 = al \frac{r}{K} P^* Q^* U^* > 0, \quad A_1 = al P^* U^* + ch P^* U^* > 0.$$

Further, the remaining Routh-Hurwitz condition becomes

$$\frac{r}{K} Q^* (al U^* + ch P^*) > al \frac{r}{K} P^* Q^* U^*,$$

which upon simplification is clearly seen to be always true. Thus from the latter A^* is unconditionally stable, nor can Hopf bifurcations arise.

In fact, these equilibria can be proven to be globally asymptotically stable, see [7] for details.

4.2 The top two trophic levels ecoepidemic subsystem

Note that the formulation of the model (3.1) implies that the (top) predators are specialists, in other words in the absence of the intermediate population P they would starve to death since they only feed on the P population. A more general situation could be envisaged if they had other food sources or in case of the food web in which they could also hunt the bottom prey Q .

As a consequence of the above remark, It is easily observed that by disregarding the Q population and dropping the related fourth equation in (3.1), the resulting system admits only the trivial equilibrium. In fact no other stationary point can exist, since the dynamics for the intermediate population P is bound to drive the population itself to extinction. Since this is the only resource for the top predators, they are bound to disappear in turn as well. This argument can be made rigorous, but we however avoid this formality here.

In order to examine a meaningful version of the particular subsystem of the food chain composed by the highest two trophic levels with specialist top predators, we would be forced to assume that the population at the intermediate trophic level has other food sources. Mathematically, we would then need to replace the mortality term $-vP$ by a logistic correction, similar to the one appearing in the fourth equation. However, a system of this type has already been formerly investigated. It turns out to be in fact an ecoepidemic system with disease in the predators, [14]. In fact the published system is even more general, since the predators are generalists. In other words, the model in [14] allows for other food sources for the predator population. Additionally, intraspecific competition is more complex,

as both healthy and infected predators resent of the influence of all their similar, independently of the infective class to which they belong. Also from the disease viewpoint it is a more general model that the subsystem that we consider here. In fact, in [14] the disease is recoverable, while here it is not.

Hence, the subsystem at hand here can be considered as a particular case of the model in [14], and will not further be investigated, referring the reader to that paper for the results of the analysis.

5 Model Analysis

5.1 Boundedness

We now show that all trajectories are bounded. By setting $\psi(t) = S(t) + I(t) + P(t) + Q(t)$ we can calculate the derivative of ψ along the solution trajectories of (3.1). Summing the equations in (3.1) we have:

$$\frac{d\psi}{dt} = SP(a-l) + IP(b-e) + PQ(c-h) - mS - \gamma I - vP + rQ\left(1 - \frac{Q}{K}\right);$$

in view of the assumptions $a < l, b < e, c < h$, we find

$$\frac{d\psi}{dt} \leq -mS - \gamma I - vP + rQ\left(1 - \frac{Q}{K}\right). \quad (5.1)$$

Taking now a suitable $\eta > 0$, we rewrite the former inequality (5.1) as follows

$$\frac{d\psi}{dt} + \eta\psi \leq (-m + \eta)S + (-\gamma + \eta)I + (-v + \eta)P + (r + \eta)Q - \frac{rQ^2}{K}.$$

From it, by imposing $\eta \leq \min\{m, \gamma, v\}$, we find

$$\frac{d\psi}{dt} + \eta\psi \leq (r + \eta)Q - \frac{rQ^2}{K} \leq \frac{(r + \eta)^2 K}{4r} \equiv W,$$

where W is the maximum of the parabola $f(Q) = (r + \eta)Q - rQ^2 K^{-1}$. From the theory of differential inequalities, we then obtain

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

and for $t \rightarrow +\infty$, it follows $0 < \psi(S, I, P, Q) < W\eta^{-1} := W^*$, hence all solutions of (1) starting in the first orthant are confined in the region $G = \{(S, I, P, Q)^T \in \mathbb{R}_+^4 : \psi = W^* + \theta\}$, for any $\theta > 0$ and for all $t > T_*$, where T_* depends on the initial values $(S(0), I(0), P(0), Q(0))^T$. Thus the whole population in the ecosystem is bounded, and therefore also each subpopulation is.

5.2 Equilibria

Model (3.1) admits the following points as equilibria in the $SIPQ$ phase space. The origin, $E_0 = (0, 0, 0, 0)$, the prey-only equilibrium $E_1 = (0, 0, 0, K)$, the equilibrium in which only the infected survive among the top predators,

$$E_2 = \left(0, \frac{rbcK - \gamma cKh - rbv}{rbe}, \frac{\gamma}{b}, \frac{K(rb - h\gamma)}{rb}\right),$$

the top-predator-free subsystem,

$$E_3 = \left(0, 0, \frac{r(-v + cK)}{cKh}, \frac{v}{c}\right),$$

the disease-free environment,

$$E_4 = \left(\frac{cKra - vra - cKhm}{alr}, 0, \frac{m}{a}, \frac{K(ra - hm)}{ra} \right),$$

and finally the coexistence equilibrium $E_5 = (S_5, I_5, P_5, Q_5)$ with population levels given by

$$S_5 = \frac{\beta vbr - \beta cKbr + \beta cKh\gamma - mebr + aer\gamma}{\beta\Delta}, \quad I_5 = \frac{-\beta cKhm + mblr - \beta vra + \beta cKra - ral\gamma}{\beta\Delta},$$

$$P_5 = \frac{r(-\beta v + \beta cK + me - l\gamma)}{\Delta}, \quad Q_5 = \frac{K(aer - blr + h\beta v - hme + hl\gamma)}{\Delta},$$

where we have set $\Delta := \beta cKh + aer - blr$. The first two equilibria are always feasible. For the remaining ones, we have for E_2 the feasibility condition

$$rb - h\gamma > \frac{rbv}{cK}, \tag{5.2}$$

for E_3 instead

$$cK > v, \tag{5.3}$$

and finally for E_4 we find

$$ra - hm > \frac{vra}{cK}. \tag{5.4}$$

To study E_5 we set

$$z = -r\beta v + \beta cKr - rl\gamma, \quad y = -r\beta v + \beta cKr + rme.$$

We then need either one of the following two sets of conditions, namely

$$\Delta > 0,$$

$$z > \max \left\{ -rem, \frac{-mblr + \beta cKhm}{a} \right\},$$

$$y < \frac{\beta cKh\gamma + aer\gamma}{b},$$

$$r > -\frac{h(\beta v - me + \gamma l)}{ae - bl},$$
(5.5)

or

$$\Delta < 0,$$

$$z < \min \left(-rem, \frac{-mblr + \beta cKhm}{a} \right),$$

$$y > \frac{\beta cKh\gamma + aer\gamma}{b},$$

$$r < -\frac{h(\beta v - me + \gamma l)}{ae - bl}.$$
(5.6)

These conditions will be investigated numerically. The simulations show that their solution sets are nonempty.

5.3 Stability

To investigate stability, we need the sign of the eigenvalues of the Jacobian evaluated at the critical points. We write down the Jacobian of the system (3.1).

$$J = \begin{bmatrix} J_{11} & -\beta S & aS & 0 \\ \beta I & J_{22} & bI & 0 \\ -Pl & -Pe & J_{33} & cP \\ 0 & 0 & -Qh & J_{44} \end{bmatrix} \tag{5.7}$$

where $J_{11} = -\beta I - m + aP$, $J_{22} = \beta S + bP - \gamma$, $J_{33} = -lS - eI - v + cQ$, $J_{44} = r\left(1 - \frac{Q}{K}\right) - \frac{rQ}{K} - hP$.

At E_0 the eigenvalues are $-m$, $-\gamma$, $-v$ and r showing that it is always unstable. At E_1 also we find explicitly $-m$, $-\gamma$, $-v + cK$ and $-r$ giving the stability condition

$$cK < v. \tag{5.8}$$

The eigenvalues of (5.7) at E_3 are

$$\lambda_{1,2} = \frac{1 - vr \pm \sqrt{v^2 r^2 + 4v^2 rcK - 4vrc^2 K^2}}{2cK} \tag{5.9}$$

$$\lambda_3 = \frac{vra + cKra - cKhm}{cKh}, \quad \lambda_4 = -\frac{rbv - rbcK + \gamma cKh}{cKh}.$$

Now λ_1 and λ_2 are complex conjugate when $v^2 r^2 + 4v^2 rcK - 4vrc^2 K^2 < 0$; in this case they have negative real part. If instead $v^2 r^2 + 4v^2 rcK - 4vrc^2 K^2 \geq 0$, these eigenvalues are real and negative, trivially so if the previous condition is an equality. Otherwise observing that the strict positivity implies that $\lambda_1 < 0$, letting $vr = s$ leads to

$$s^2 + 4vscK - 4sc^2 K^2 > 0, \quad s > 0, \quad s^2 + 4vscK - 4sc^2 K^2 < s^2.$$

The first two inequalities are always satisfied, the last one holds in view of the feasibility condition (5.3). Also, clearly, $\lambda_2 < 0$ is always satisfied. Thus stability of E_3 hinges on the signs of λ_3 and λ_4 , and specifically when

$$cK - v < \frac{chK}{r} \min\left\{\frac{m}{a}, \frac{\gamma}{b}\right\}. \tag{5.10}$$

For E_3 the stability can thus be achieved with suitable parameter choices. We omit the relative simulation, since this in fact is the equilibrium of the classical Lotka-Volterra predator-prey system with logistic correction for the prey.

For the equilibria E_2 and E_4 the characteristic equation factorizes and one eigenvalue is easily found. In fact, for the former we have

$$\lambda = \frac{q}{rbe}.$$

where the numerator coincides with the value of the numerator of S_5 , i.e.

$$q = \beta vbr - \beta cKbr + \beta cKh\gamma - mebr + aer\gamma. \tag{5.11}$$

The remaining characteristic equation is the cubic

$$\lambda^3 + \frac{r}{K} Q_2 \lambda^2 + P_2 [beI_2 + chQ_2] \lambda + be \frac{r}{K} I_2 P_2 Q_2 = 0.$$

It is easily seen that the Routh-Hurwitz stability conditions are always satisfied, namely

$$\frac{r}{K} Q_2 > 0, \quad be \frac{r}{K} I_2 P_2 Q_2 > 0, \quad beI_2 + chQ_2 > beI_2. \tag{5.12}$$

For stability we must impose just that the first eigenvalue be negative,

$$\beta vbr + \beta cKh\gamma + aer\gamma < \beta cKbr + mebr. \tag{5.13}$$

Also for one eigenvalue of the Jacobian $J(E_4)$ we find the numerator of I_5 , namely letting

$$\tilde{q} = -\beta cKhm + mblr - \beta vra + \beta cKra - ral\gamma, \tag{5.14}$$

we have

$$\lambda = \frac{\tilde{q}}{ral}.$$

Again the remaining cubic simplifies to

$$\lambda^3 + \frac{r}{K}Q_4\lambda^2 + P_4[alS_4 + chQ_4]\lambda + al\frac{r}{K}P_4Q_4S_4 = 0.$$

Once more the Routh-Hurwitz conditions hold unconditionally,

$$\frac{r}{K}Q_4 > 0, \quad be\frac{r}{K}P_4Q_4S_4 > 0, \quad alS_4 + chQ_4 > alS_4, \quad (5.15)$$

so that stability of E_4 is ensured just by

$$mblr + \beta cKra < ral\gamma + \beta cKhm + \beta vra. \quad (5.16)$$

Numerical simulations confirm this analysis, but they are omitted, since this point coincides with the coexistence equilibrium of the classical 3-trophic level food chain.

For E_5 , we note that the characteristic equation has one coefficient easily found, namely

$$a_1 = \frac{r(aer - blr + h\beta v - hme + hl\gamma)}{\beta cKh + aer - blr} = \frac{K}{r}Q_5 > 0, \quad (5.17)$$

so that one of the Routh-Hurwitz conditions holds. But the remaining ones are too involved and we investigate coexistence only numerically. In fact the endemic equilibrium is seen to be stable with the following parameter choice

$$\beta = 0.2, \quad \mu = 0.1, \quad a = 0.8, \quad b = 0.3, \quad \gamma = 1.5, \quad l = 4, \\ e = 3.5, \quad \alpha = 0.2, \quad c = 2, \quad h = 6, \quad r = 12, \quad K = 5,$$

see Figure 1.

Figure 1: Coexistence equilibrium attained at a stable state for $\beta = 0.2, \mu = 0.1, a = 0.8, b = 0.3, \gamma = 1.5, l = 4, e = 3.5, \alpha = 0.2, c = 2, h = 6, r = 12, K = 5$. Top to bottom the populations S, I, P, Q as functions of time.

5.4 Bifurcations

Comparing the feasibility and stability conditions it is easy to establish that E_3 is feasible if and only if E_1 is unstable, as well as the stability of E_1 entails the infeasibility of both E_2 and E_4 . These results indicate the existence of transcritical bifurcations between the named pairs of equilibria.

Indeed, for the last claim, observe that from the stability of E_1 we find $cK < v$, from which

$$\frac{rav}{cK} > ra.$$

Assuming feasibility for E_4 , we have

$$ra - hm > \frac{vra}{cK}$$

and combining with the previous inequality, we find

$$ra - hm > \frac{vra}{cK} > ra$$

from which $-hm > 0$, which is impossible. In a similar way the infeasibility of E_2 can be obtained.

Thus, comparing the conditions (5.10) with the feasibility conditions of E_2 and E_4 , namely (5.2) and (5.4), we have found transcritical bifurcations. Specifically, E_3 stable implies that E_2 and E_4 are both not feasible. Conversely, if E_2 or E_4 are feasible, then E_3 is unstable.

In case $\Delta > 0$, we can prove that stability of E_1 implies the infeasibility of E_5 . In fact, from (5.8) we have $cK < v$. Then from P_5 we obtain

$$r(-\beta v + \beta cK + me - l\gamma) < r(-\beta v + \beta v + me - l\gamma) = r(me - l\gamma) < r(\gamma l - l\gamma) = 0,$$

the last inequalities following from the biological assumptions on the parameters, i.e. $m < \gamma$, $e < l$.

Finally, considering that for both the equilibria E_2 and E_4 the population values are related to those of the coexistence equilibrium E_5 , it follows that if the denominator Δ of the populations S_5 and I_5 of E_5 is positive, and if E_5 is feasible, then E_2 and E_4 are both unstable.

Since the equilibria E_0 and E_1 have only real eigenvalues, Hopf bifurcations cannot arise at these points.

At E_3 two eigenvalues are real, while λ_1 and λ_2 can never become pure imaginary, since their real parts cannot vanish. Thus Hopf bifurcations are forbidden also around this equilibrium point.

At E_2 one eigenvalue is real, and the remaining ones are roots of a cubic equation. In order that a Hopf bifurcation arises, we need the last Routh-Hurwitz condition (5.12) to become an equality. But in view of the strict inequality of the expression, that can never occur. Hence Hopf bifurcations are forbidden at E_2 as well.

Completely similar considerations hold for E_4 , considering the strict inequality in the last condition (5.15).

For the remaining coexistence equilibrium E_5 , the analysis is too difficult. But numerical simulations show that a careful choice of the model parameters leads to sustained oscillations, Figure 2, obtained for the following set of parameter values:

$$\begin{aligned} \beta = 2, \quad m = 0.1, \quad a = 3.95, \quad b = 0.1, \quad \gamma = 1.5, \quad l = 4, \\ e = 3.5, \quad v = 0.2, \quad c = 2, \quad h = 3.5, \quad r = 9.5, \quad K = 1.7. \end{aligned}$$

Figure 2: Coexistence through persistent oscillations is obtained for the parameter values $\beta = 2$, $m = 0.1$, $a = 3.95$, $b = 0.1$, $\gamma = 1.5$, $l = 4$, $e = 3.5$, $v = 0.2$, $c = 2$, $h = 3.5$, $r = 9.5$, $K = 1.7$. Top to bottom the populations S , I , P , Q as functions of time.

5.5 Bistability

We now turn the attention to the analysis of possible bistability situations. These are important as in such case the phase space would be partitioned into several domains of attractions, one for each one such equilibrium. Very recently, algorithms for the accurate computation of the separatrix surfaces of these basins of attractions have begun to be studied, [15]. Taking into consideration the results of Sections 5.3 and 5.4, only the following pairs of equilibria can be simultaneously stable:

$$E_1 - E_5, \quad E_2 - E_5, \quad E_3 - E_5, \quad E_4 - E_5.$$

We note that the coexistence equilibrium is always involved in these pairs. Also, the first, second and fourth cases are possible only if the denominator of the coordinates of E_5 is negative. For each of the previous pairs we realised several numerical simulations looking for sets of parameters satisfying bistability, but we never found them.

These negative results, coupled with the findings on transcritical bifurcations among the system's equilibria, support the conjecture that the various equilibria, when feasible and locally asymptotically stable, are also globally asymptotically stable in the case of this ecoepidemic tri-trophic level food chain with disease in the top predator too.

5.6 Malthus Growth for the Bottom Prey

We consider now the Malthus variant of model (3.1), assuming that for the bottom prey abundant food is available. Mathematically, the last equation of (3.1) gets replaced by

$$\frac{dQ}{dt} = rQ - hPQ,$$

which can be thought of as letting $K \rightarrow \infty$ in the last equation of (3.1). In this way, it is also seen that the equilibria E_1, E_2, E_4 shoot off to infinity, while equilibria $E_0^M \equiv E_0$ and $E_5^M \equiv E_5$ are still possible, together with E_3^M which now attains the population levels

$$P_3^M = \frac{r}{h}, \quad Q_3^M = \frac{v}{c}.$$

In a similar way, coexistence now settles at the population values $P_5^M = rh^{-1}$,

$$S_5^M = \frac{h\gamma - br}{\beta h}, \quad I_5^M = \frac{ra - hm}{\beta h}, \quad Q_5^M = \frac{aer -blr + h\beta v - hme + hl\gamma}{\beta ch}.$$

The change in the Jacobian involves only the entry $J_{44}^M = r - hP$, so that the equilibrium E_3 now becomes a center. This can be seen from the eigenvalues (5.9): the first two become in this case $\pm\sqrt{vri}$, while in order that the system trajectories be attracted toward the PQ plane, for the remaining two we need to require

$$ra < hm, \quad br < h\gamma. \tag{5.18}$$

The origin E_0^M retains its instability. The change in the Jacobian for E_5^M entails that now $-\text{tr}(J(E_5^M)) = 0$, so that the coexistence equilibrium in the Malthus version of the model is unconditionally unstable.

6 Discussion

The model trajectories have been shown to be bounded, a biologically reasonable condition in view of the limited amount of resources to steadily support the system growth. Also the ecosystem cannot disappear, since the origin is always unstable, a good result from the biological point of view.

The analysis of the equilibria shows that essentially only one of them can be feasible and stable at each time. Starting from the bottom prey only equilibrium E_1 , the intermediate trophic level can permanently settle into the system whenever their mortality becomes low enough, compare (5.3). This equilibrium E_3 can then disappear by allowing into the system the top predators. They can enter in three different ways: either just as infected, equilibrium E_2 , or just as healthy E_4 , or both healthy and infected, point E_5 , in which case all the system (sub)populations coexist. Note that the possibility of having just infected top predators thriving together with the two lowermost trophic layers is due to the fundamental assumption that the disease is vertically transmitted. This assumption is not contained, for instance, in the model considered in [7], but in that case since the disease affects only the bottom prey, the fact that the disease can be transmitted only horizontally has a less relevant role in the general system behavior. Here instead equilibrium E_2 would not exist in case of a pure horizontal transmission of the epidemics.

Note that E_3 bifurcates into E_4 whenever the first stability condition (5.10) is violated, namely when the natural plus disease-related mortality γ undergoes a certain threshold, while it becomes equilibrium E_2 in case the second condition (5.10) fails. For the latter situation to occur, it is instead the healthy top predator mortality that has to become low enough. When both stability conditions (5.10) do not hold, the top predators invade the system with their disease being endemic. This can occur at a stable level, i.e. the equilibrium E_5 , or coexistence can be ensured with variable populations, leading to persistent oscillations as shown by our numerical experiments. The comparison with the subsystems investigated in the subsection 4 shows that the disease-free model does not present these very features exhibited by the ecoepidemic food chain.

Instead, some of these temporal pattern forming structures are found also in similar models that have been investigated recently, [7], in which the disease lies at the bottom trophic level, and [6], containing an epidemics affecting the intermediate level population.

The fact that in this model the disease lies at the top trophic level implies that it can be present only when all the other populations thrive. This conclusion matches the one found for the case in which the epidemics affects the intermediate population, [6].

Thus, the presence of the disease markedly affects the behavior of food chains. This remark further substantiates an analogous claim already made for simpler interacting population models, that the occurrence of diseases in nature is a fact that cannot be easily overlooked in ecological situations, [16]. Thus in general purely demographic models are not a good and a complete description of natural environments and their conclusions could be different should the effects of diseases be taken into account.

7 Conclusions

This paper has examined a simple ecoepidemiological model in which three trophic levels are involved, and the disease affects only the topmost layer. The major findings of the study can be summarized as follows.

- a** In the section (3), the model is presented and analysed.
- b** The system trajectories cannot shoot off to infinity, subsection (5.1), which is biologically meaningful, as an infinite amount of resources is not available.
- c** Subsection (5.2), contains the equilibria analysis for feasibility, while their stability is examined in the subsection (5.3).
- d** The relationships between the various equilibria are elucidated in the subsection (5.4), showing that in most situations only one such feasible equilibrium is possible, because it then implies the instability of the remaining ones. This fact is expressed by the various transcritical bifurcations existing between suitable pairs of equilibria.
- e** The coexistence equilibrium experimentally shows Hopf bifurcations, subsection (5.4).
- f** We also conjecture about the global stability of these equilibria, basing our consideration on experimental simulations for the bistability results, subsection (5.5).
- g** The coexistence equilibrium of the Malthus particular case becomes always unstable.
- h** These results are finally interpreted in the section (6).

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Competing Interests

The authors declare that no competing interests exist.

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