**Predator's alternative food sources do not support ecoepidemics with two-strains-diseased prey**

*Article*

# Predator's alternative food sources do not support ecoepidemics with two-strains-diseased prey

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**Abstract**
An ecoepidemic model is presented, in which two diseases affect the prey. Predators are allowed to have other food sources. Equilibria are analyzed for feasibility and stability. The most striking result is that in these conditions the two strains cannot both survive in the system, contrary to what is possible to obtain, under suitable assumptions, in standard epidemic models.

**Keywords** ecoepidemics; epidemics; predator-prey; two strains.

## 1 Introduction

Real life systems are always very complicated. Mathematical models represent a tool for understanding their behavior, but very often they tend to oversimplify the real systems, in the tentative to analytically extract some information on their possible future evolution (Rai et al., 2011; Elsadany, 2012; Elsadany et al., 2012; Ivanchikov and Nedorezov, 2011, 2012; Nedorezov, 2011, 2012; Nedorezov and Sadykov, 2012; Visser et al., 2012). Networks are recently becoming essential to more adequately model the complexity of interactions among several system components (Zhang, 2011a). In ecology, they can be used to set up the interactions mechanisms among the various entities that one wants to take into account (Dormann, 2011). The network approach is a very versatile instrument, as it can be used at all levels, from modeling the function of molecular components (Martínez-Antonio, 2011) to the higher ecological level (Zhang, 2011b) and human population level, with human artifacts modifying the environment (Ferrarini, 2011).

An important issue in mathematical biology is represented by ecology. Research in this field has been carried out with applications in the management of public resources and crop and harvesting of renewable resources, like for instance in fisheries. On the other hand, starting almost a century ago (Kermack and McKendrick, 1927) mathematics has successfully been applied also to fighting epidemics spread. Some diseases that have affected humanity since centuries have now been put under control. In 1980 indeed, thanks also to results due to mathematical modelling, WHO has declared smallpox worldwide eradicated. But new epidemics nowadays loom and renewed efforts are needed from researchers and scientists.

Diseases affect the animal populations too, and especially for animals that are raised in farms, their economic burden can be relevant. One can just mention the cases of foot and mouth disease that affected UK in the past years with the negative impact for horse raising. Among wild animal communities diseases also play an important role and might even threaten the species survival. But in such situations the interplay among

different populations too is to be considered, as well as the possibility that the disease gets transmitted into the other population.

In the biomathematical literature, models for the study of these complex interactions have been proposed since about twenty years (Hadeler and Freedman, 1989). Later on, this field of research has received a name, ecoepidemiology. A brief account of its development can be found in Venturino (2007).

Ecoepidemic models thus describe general systems in which two or more populations interact, but are also subject to diseases. Scientists have begun to study them systematically since the early nineties of the past century (Beltrami and Carroll, 1994; Venturino, 1994; Venturino, 1995, Chattopadhyay and Arino, 1999). A fair amount of literature in the subject has then appeared, focusing mainly on a disease spreading among the prey (Arino et al., 2004). Diseases among the predators have also been considered (Venturino, 2002), and infections spreading among the interacting species are of more recent vintage (Venturino, 2006; Hsieh and Hsiao, 2008). A more detailed account of some of these more recent results can be found in Chapter 7 of Malchow, Petrovskii and Venturino (2008).

While all these models are represented using mainly ordinary differential equations as mathematical tool, and the number of dependent variables is usually kept low, so that the dynamical system can be analytically investigated, it is not difficult to conceive more intricated situations in which more than just two diseased populations interact, i.e. one can have several species in a food web for instance, but also several diseases at hand that need to be represented in the model description. The various connections between interacting populations and the various diseases affecting them are then easily described via a network graph. However, the basic features of a complex model might at times be captured also via minimal models. In this perspective, on the way to consider the nontrivial interactions of several diseases on intermigling populations, we want at first to understand the outcomes possible when two different strains affect the very same population in a predator-prey system.

Much more recently than previous works in ecoepidemiology indeed, in this context the authors have proposed and investigated a simple ecoepidemic model with two diseases in the prey (Elena et al., 2011). A similar model with two diseases affecting the predators has also recently appeared (Roman et al., 2011). Common results of these investigations is the striking result that the minimal models so conceived do not support the two diseases affecting both at the same time the infected population, whether be it a the higher or the lower trophical level.

In this paper we consider again a minimal network, in which predators and prey interact, and two diseases affect the very same species. We thus modify the model with diseases in the prey, in the attempt to obtain coexistence of the two strains. The modification accounts for other possible food sources available for the predators. Our findings are however still negative. This is surprising, since for standard epidemic models, i.e. with the diseases spreading in an isolated population, that does not interact with other ones, the two strains are sustainable by suitable “ecological” assumptions (Ackleh and Allen, 2003; Andreasen et al., 1997; Castillo-Chavez et al., 1999). In ecoepidemic models, one could presume that these extra assumptions would come from the interaction with the other population. Based on our findings so far, this does not seem to be the case.

The model we present here assumes, contrary to (Elena et al., 2011), that predators in absence of the prey considered in the system, evolve toward the carrying capacity allowed by the environment in which they live, this being possible in view of other food resources not explicitly modeled in the system.

The paper is organized as follows. In the next section we present the model. The following section contains the analysis of the system’s equilibria. Their stability is investigated in the subsequent Section. We then report some simulations on the system behavior. A final discussion and interpretation of the results concludes the paper.

## 2 New Model

Let $S$ denote the healthy prey, $I$ the diseased prey of type A and $Y$ those of type B, $P$ be their predators. We consider here the situation in which available for the predators are also other food sources, which are not explicitly modeled.

This is reflected in the last equation of the following system,

$$\begin{aligned}
\frac{dS}{dt} &= r\left(1-\frac{S+I+Y}{k}\right)S-\lambda IS-\beta YS-aSP+\gamma I+\varphi Y \qquad (1)\\
\frac{dI}{dt} &= \lambda IS-\mu I-\gamma I\\
\frac{dY}{dt} &= \beta YS-\nu Y-\varphi Y\\
\frac{dP}{dt} &= n\left(1-\frac{P}{H}\right)P+eSP.
\end{aligned}$$

More specifically, the older model proposed in (Elena et al., 2011) differs essentially only in the last equation for the predators, in which instead of the logistic growth, they experience mortality in absence of the prey modeled in the system. Explicitly, the former, alternative version of the model contains the very same first three equations of (1), while the last one is replaced by

$$\frac{dP}{dt} = -mP+eSP. \qquad (2)$$

In the predators' evolution equation, fourth equation of (1), the last term gives the reward predators have from hunting. It is assumed here that only sound prey are captured. Thus, infected are identifiable, and avoided by the predators who do not feed on them.

The first three equations of (1) instead describe the prey dynamics. In the first one, sound prey reproduce logistically, experiencing the total prey population intraspecific pressure, can get infected with either one of the two strains, and are hunted by the predators. The final two terms express the fact that both diseases are assumed to be recoverable.

The next assumption furthermore is very important as it concerns the mutual relationship of the two types of disease. We consider strains that cannot both simultaneously affect the same individual. Further, any individual infected by any one of them cannot contract the other strain, nor can the latter replace the former. Thus, neither co-infection nor super-infection are possible here.

The second and third equations of (1) contain the epidemics dynamics. Individuals enter these classes via successful contacts among susceptibles and infected of the "right" strain, and leave them by natural plus disease related mortalities, respectively $\mu$ and $\nu$, or by recovering, at respective rates $\gamma$ and $\varphi$.

For later purposes, it is necessary to have the Jacobian of (1) at hand. It is reported below.

$$J^* = \begin{pmatrix} J^*_{11} & -\frac{r}{k}S-\lambda S+\gamma & -\frac{r}{k}S-\beta S+\varphi & -aS \\ \lambda I & \lambda S-\mu-\gamma & 0 & 0 \\ \beta Y & 0 & \beta S-\nu-\varphi & 0 \\ eP & 0 & 0 & n-2\frac{n}{H}P+eS \end{pmatrix} \qquad (3)$$

where

$$J_{11}^{*} = r - \frac{2r}{k}S - \frac{r}{k}I - \frac{r}{k}Y - \lambda I - \beta Y - aP.$$

Note that boundedness for (1) is immediately obtained from standard arguments like those used in the previous model, (Elena et. al., 2011), since the only change in this new model affects just the last equation and for it, easily, we obtain the estimate

$\limsup_{t\to\infty} P = H$,

thus an upper bound on $P$ is always ensured.

**3 Equilibria**

We now turn to the investigation of the equilibria $Q_i = (S_i,I_i,Y_i,P_i)$, $i = 0,...,7$ of (1). Four of the former equilibria of (Elena et al., 2011) are again easily found in this case, since in all of them the predators are absent. In fact they are again the origin $Q_0 \equiv E_0$, then $Q_1 \equiv E_1 = (k,0,0,0)$. Also the points in which one disease is endemic coincide, namely $Q_3 = E_3$ and $Q_4 = E_4$, with the same feasibility conditions $\upsilon+\varphi<k\beta$ and $\mu+\gamma<k\lambda$. Note instead that in place of $E_2$ we find now the equilibrium

$$Q_2 = \left( kn\frac{r-aH}{rn+aeHk}, 0, 0, rH\frac{ek+n}{rn+aeHk} \right).$$

feasible if

$$1 > \frac{aH}{r}. \tag{4}$$

In the modified model (1), however, new equilibria arise with respect to those of (Elena et al., 2011). First, the point $Q_5 = (0,0,0,H)$ is found, which is always feasible. We then find $Q_6$ whose population values are

$$\left( \frac{\nu+\varphi}{\beta}, 0, \frac{\nu+\varphi}{\beta n}\frac{nr(k\beta-\nu-\varphi)-aHk(e\nu+e\varphi+\beta n)}{r(\nu+\varphi)+\varphi\beta}, \frac{e\nu+e\varphi+n\beta}{n\beta}H \right)$$

Defining

$$R_1 = \frac{r}{aH}\frac{k\beta-\nu-\varphi}{(\nu+\varphi)e+\beta n}$$

the point is feasible if

$$R_1 > 1. \tag{5}$$

Finally, we have $Q_7$ with components

$$\left( \frac{\mu+\gamma}{\lambda}, \frac{\mu+\gamma}{n\lambda}\frac{nr(k\lambda-\mu-\gamma)-aHk(e\mu+e\gamma+nH\lambda)}{r(\mu+\gamma)+k\gamma\lambda}, 0, \frac{e\mu+e\gamma+n\lambda}{n\lambda}H \right)$$

Defining

$$R_2 = \frac{r}{aH}\frac{k\lambda-\mu-\gamma}{(\mu+\gamma)e+\lambda n}$$

the feasibility condition of the latter is

$$R_2 > 1. \tag{6}$$

However, also in this situation the coexistence equilibrium does not exist, since solving the equilibrium equations we are led to the conditions

$$S = \frac{\mu+\gamma}{\lambda}, \quad S = \frac{\nu+\varphi}{\beta}, \quad S = \frac{m}{e},$$

which for generic parameter values cannot be all satisfied by the same value of the susceptible population. Therefore also this modified model does not allow the infected population to simultaneously sustain the two strains.

**4 Stability**

We now turn to the local stability analysis for the equilibria found in the previous Section.

At the origin the eigenvalues are $r$, $-(\mu+\gamma)$, $-(\nu+\varphi)$, $n$, showing that it is an unstable saddle.

At $Q_1$ the eigenvalues are $-r$, $\lambda k-\mu-\gamma$, $\beta k-\nu-\varphi$, $n+ek$, showing that also this point is an unstable saddle.

At $Q_2 = (S_2,0,0,P_2)$ we find the eigenvalues and the roots of the quadratic

$$\beta S_2 - \nu - \varphi, \quad \lambda S_2 - \nu - \gamma,$$

with

$$\Lambda^2 - F_1\Lambda + F_0 = 0,$$

$$F_1 = n + r + \frac{1}{k}(ek-2r)S_2 - \frac{1}{H}(aH+2n)P_2,$$

$$F_0 = \left[r\left(1-\frac{2}{k}S_2\right) - aP_2\right]\left[n\left(1-\frac{2}{H}P_2\right) + eS_2\right] + aeP_2S_2.$$

The Routh-Hurwitz conditions require $F_0 > 0$, which reduces to

$$2aH < ek + r + n, \tag{7}$$

and $F_1 > 0$ as well, which gives

$$aH(n^2r + ae^2Hk^2 + aeHkn) < r(aeHkn + ae^2Hk^2 + eknr + nr) \tag{8}$$

and in addition the negativity of the other eigenvalues, namely

$$kn\frac{r-aH}{aeHk+nr} < \min\left\{\frac{\nu+\varphi}{\beta}, \frac{\nu+\gamma}{\lambda}\right\}. \tag{9}$$

At $Q_3$ and $Q_4$ two eigenvalues are explicitly found, and one is common to both equilibria, namely $n+eS^*$. Clearly the value of $S^*$ changes in the two cases, but what counts is that the eigenvalue is positive, thereby implying that both equilibria are unstable.

Here an interesting remark can be made. On comparing $Q_3$ and $Q_4$ with $E_3$ and $E_4$ of (Elena et al., 2011), we observe both $Q_3$ and $Q_4$ are never stable, while instead $E_3$ and $E_4$ are stabilized respectively by

$$\frac{\nu+\varphi}{\beta} < \min\left\{\frac{m}{e}, \frac{\mu+\gamma}{\lambda}\right\}, \quad r > \frac{k\beta}{\varphi+\nu}\frac{(\varphi+\nu)(\varphi+\nu-k\beta)+k\beta\nu}{\varphi+\nu+2k\beta}.$$

and by

$$\frac{\mu+\gamma}{\lambda} < \min\left\{\frac{m}{e}, \frac{\nu+\varphi}{\beta}\right\}, \quad r > \frac{k\lambda}{\gamma+\mu}\frac{(\mu+\gamma)(\mu+\gamma-k\lambda)+k\lambda\mu}{\mu+\gamma+2k\lambda}.$$

For $Q_5$ the eigenvalues are $-n$, $-\mu-\gamma$, $-\nu-\varphi$, $r-aH$, so that its stability condition reduces to

$$H > \frac{r}{a}. \tag{10}$$

For the equilibria $Q_6$ and $Q_7$, stability is very hard to be analytically investigated; we therefore study it via numerical methods.

**5 Simulations**

First of all we exhibit the behavior of the system without additional food sources. Figures 1 and 2 show the stable behavior of the system settling to the equilibria $E_1 \equiv Q_1$ which is common to both models, and $E_2$, which differs from $Q_2$. The latter is shown instead in Fig. 5. The stable equilibria $E_3 \equiv Q_3$, $E_4 \equiv Q_4$, which are once again common to both models, are shown in Figures 3 and 4 for the model (2). These equilibria for (1) are unstable, and therefore not shown.

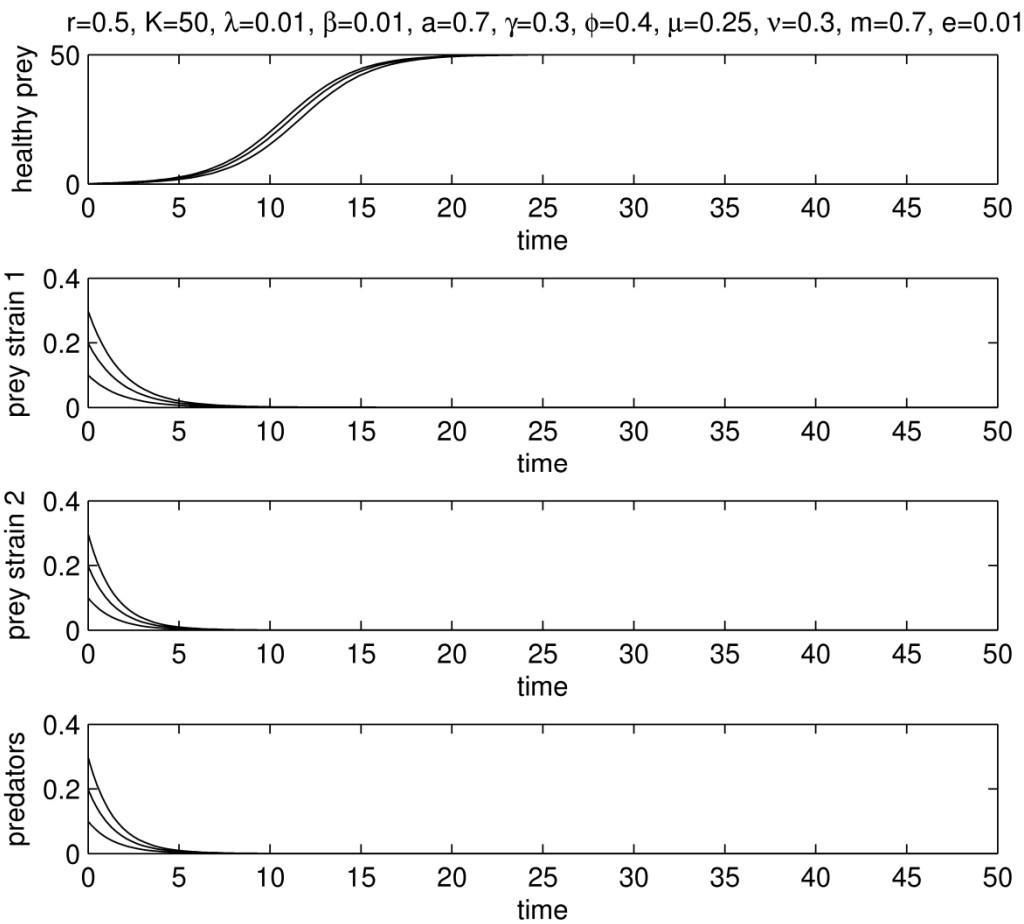


**Fig. 1** Equilibrium $E_1$ obtained for the parameter values: r=0.5, k=50, λ=0.01, β=0.01, a=0.7, γ=0.3, φ=0.4, μ=0.25, ν=0.3, m=0.7, e=0.01.

Further, the new discovered equilibria that pertain only to the system (1) are pictured in Figures 6-8.

All these simulations are in agreement with our theoretical analysis, showing both existence and stability of the relevant equilibria.

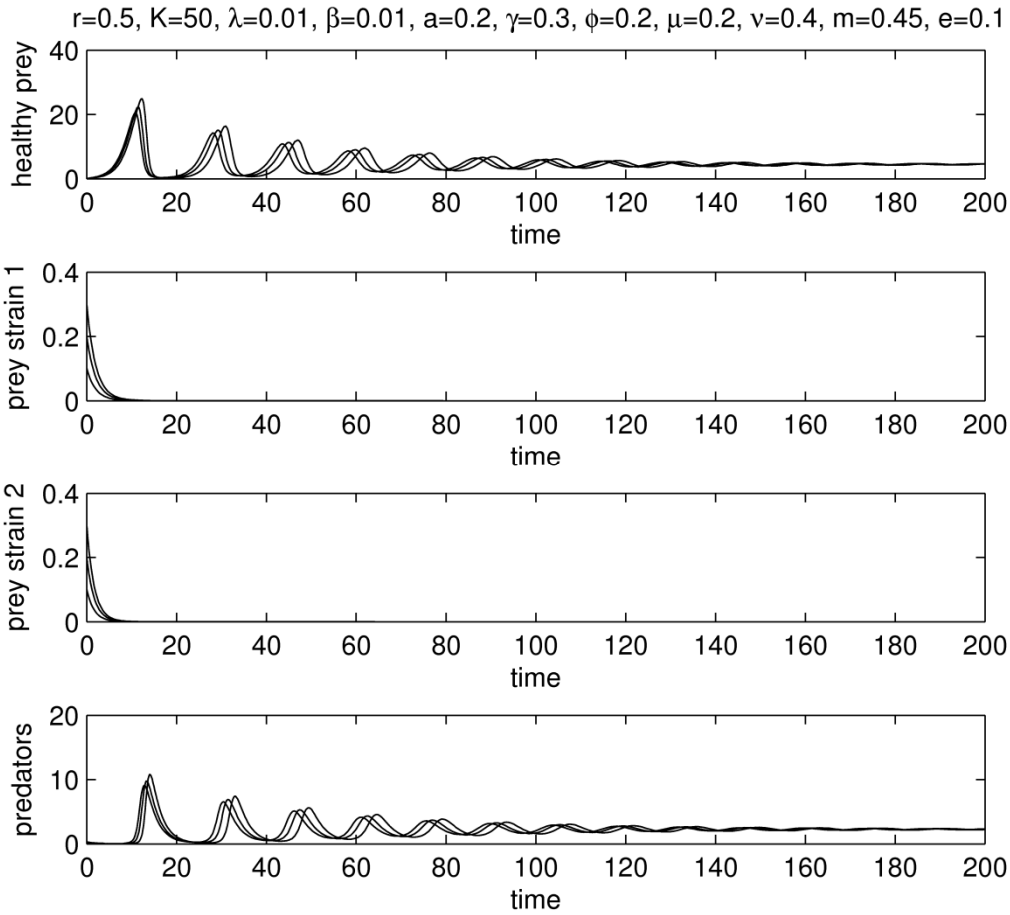


**Fig. 2** Equilibrium $E_2$ obtained for the parameter values: r=0.5, k=50, λ=0.01, β=0.01, a=0.2, γ=0.3, φ=0.2, μ=0.2, ν=0.4, m=0.45, e=0.1.

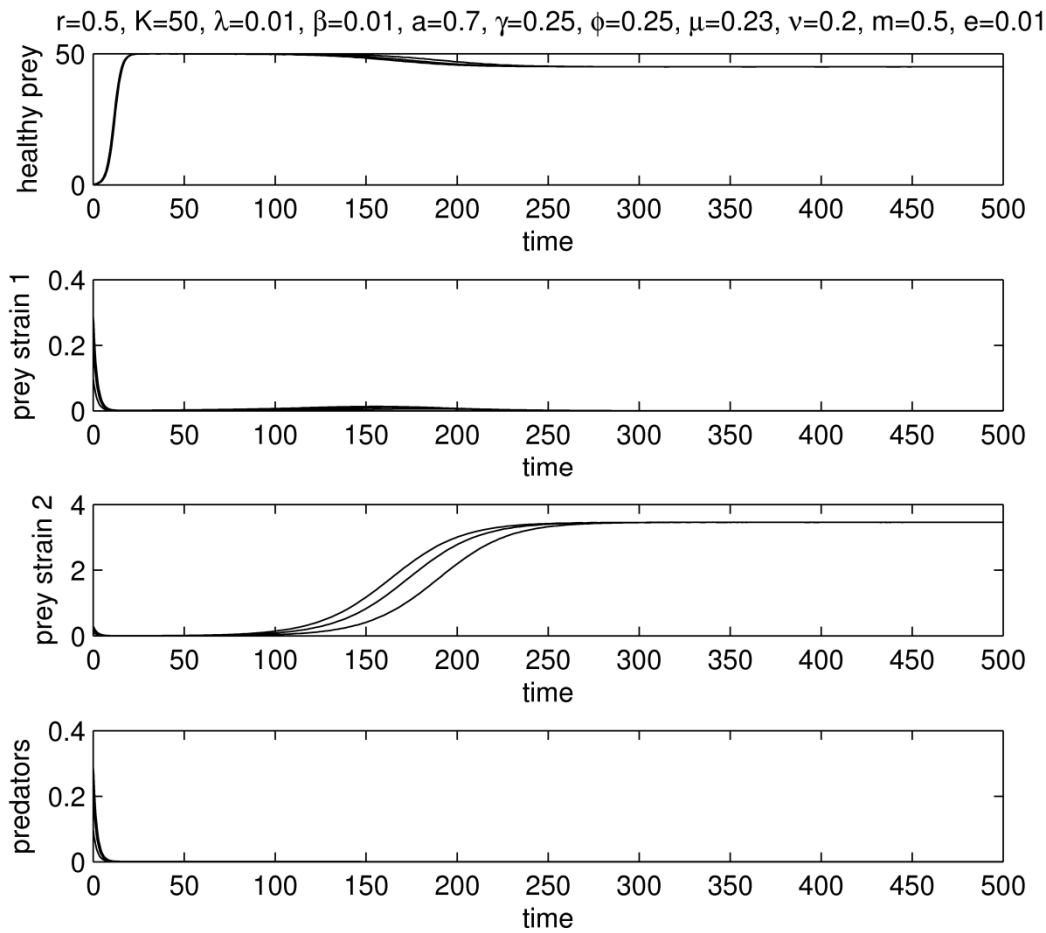


**Fig. 3** Equilibrium $E_3$ obtained for the parameter values: r=0.5, k=50, λ=0.01, β=0.01, a=0.7, γ=0.25, φ=0.25, μ=0.23, ν=0.2, m=0.5, e=0.01.

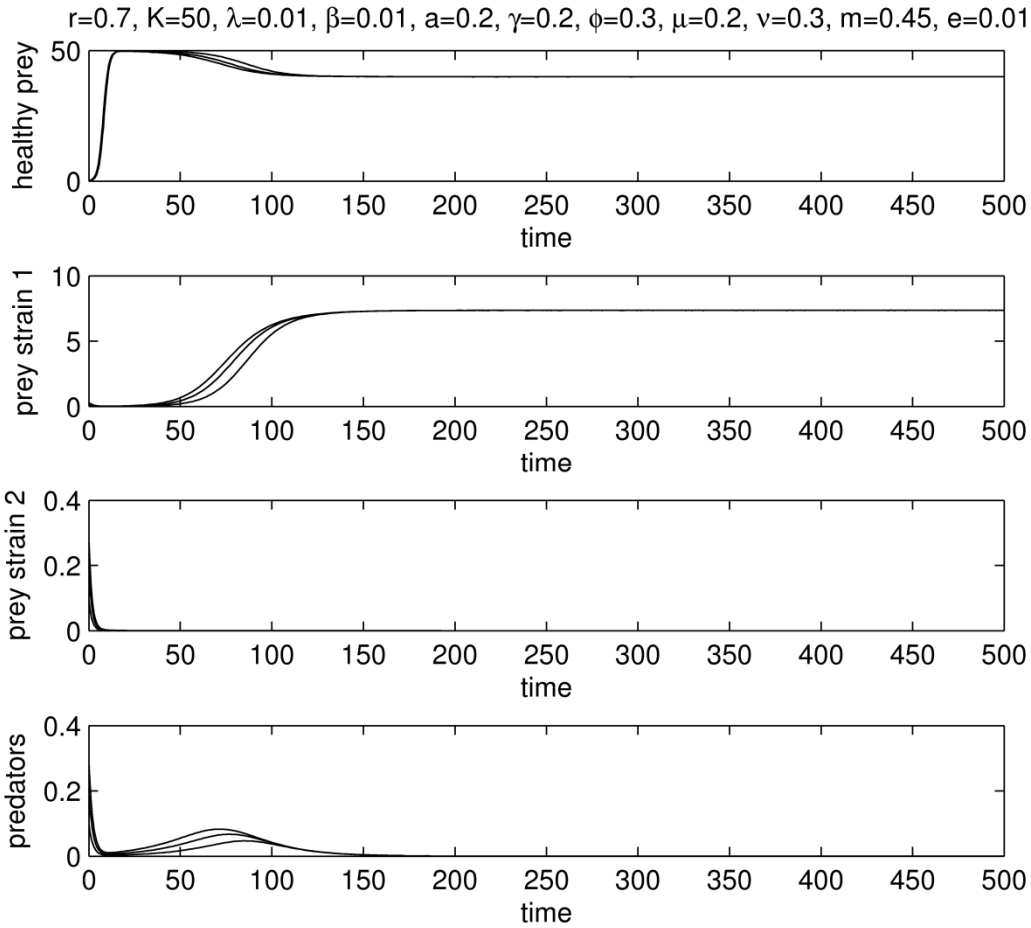


**Fig. 4** Equilibrium $E_4$ obtained for the parameter values: r=0.7, k=50, λ=0.01, β=0.01, a=0.2, γ=0.2, φ=0.3, μ=0.2, ν=0.3, m=0.45, e=0.01.

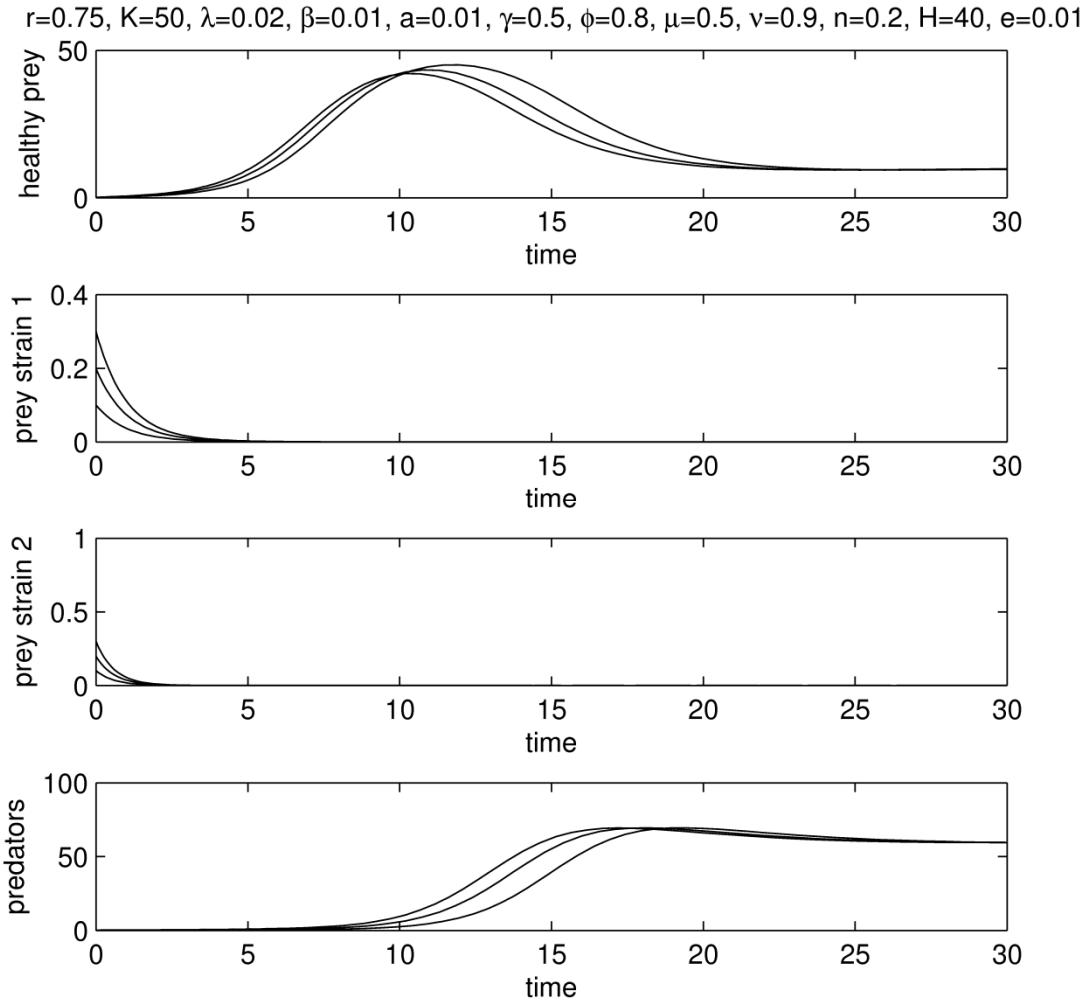


**Fig. 5** Equilibrium $Q_2$ obtained for the parameter values: r=0.75, k=50, λ=0.02, β=0.01, a=0.01, γ=0.5, φ=0.8, μ=0.5, ν=0.9, n=0.2, e=0.01, H=40.

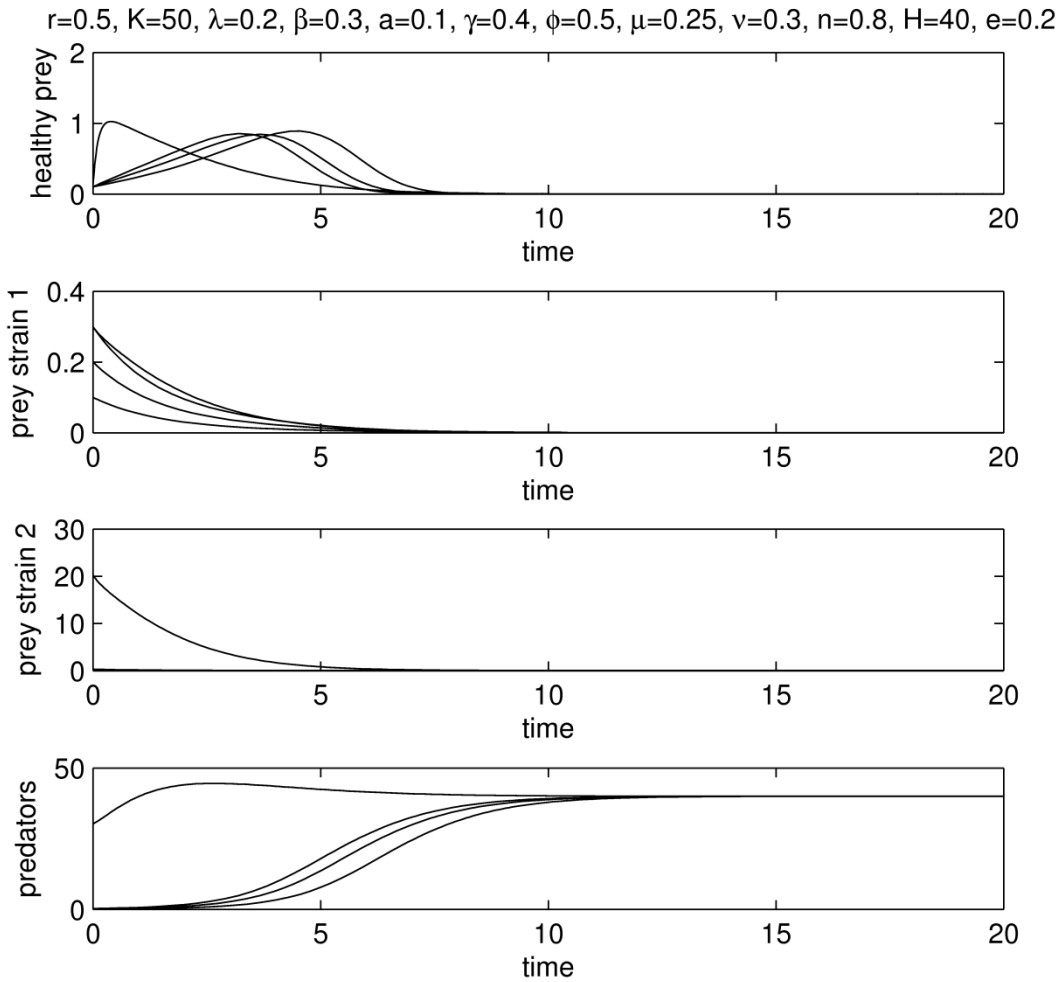


**Fig. 6** Equilibrium $Q_5$ obtained for the parameter values: r=0.5, k=50, λ=0.2, β=0.3, a=0.1, γ=0.4, φ=0.5, μ=0.25, ν=0.3, n=0.8, e=0.2, H=40.

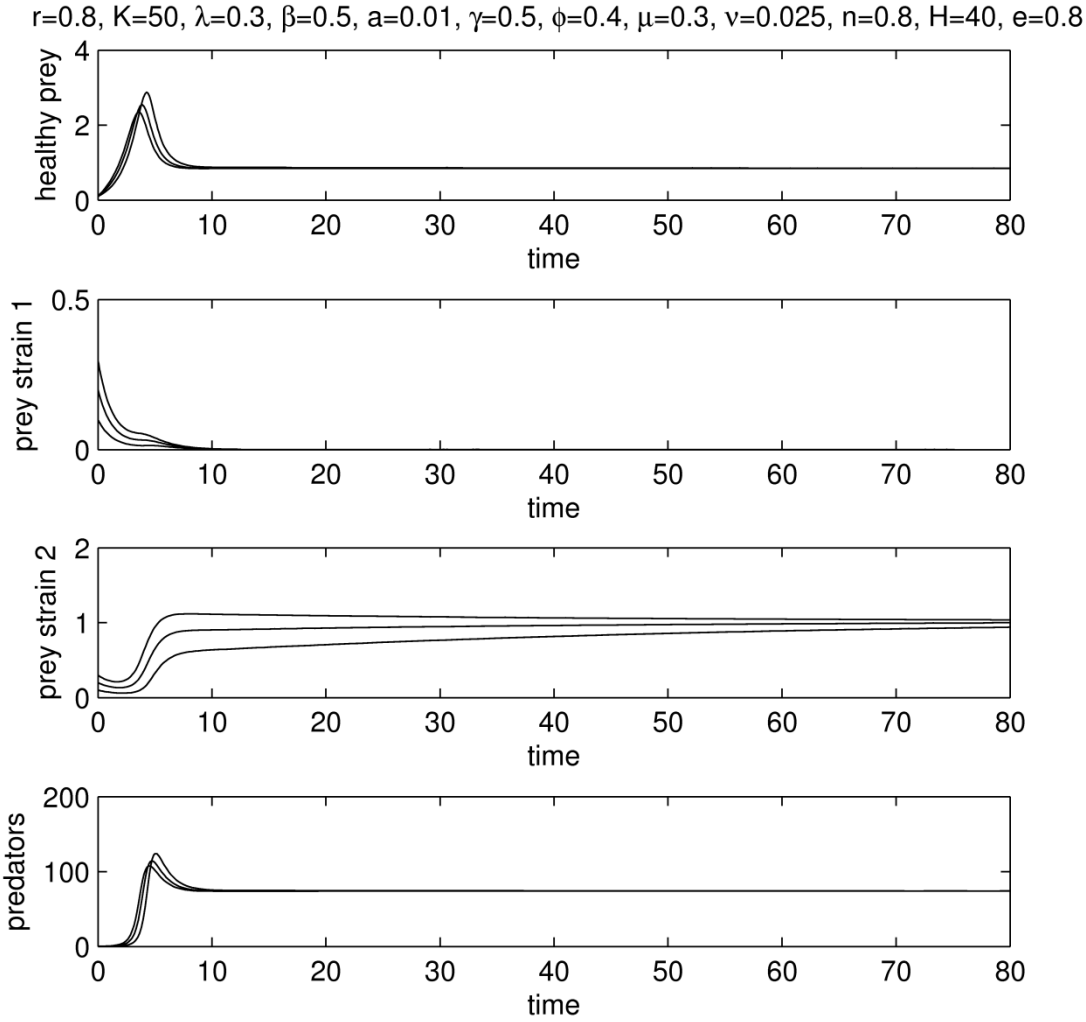


**Fig. 7** Equilibrium $Q_6$ obtained for the parameter values: r=0.8, k=50, λ=0.3, β=0.5, a=0.01, γ=0.5, φ=0.4, μ=0.3, ν=0.025, n=0.8, e=0.8, H=40.

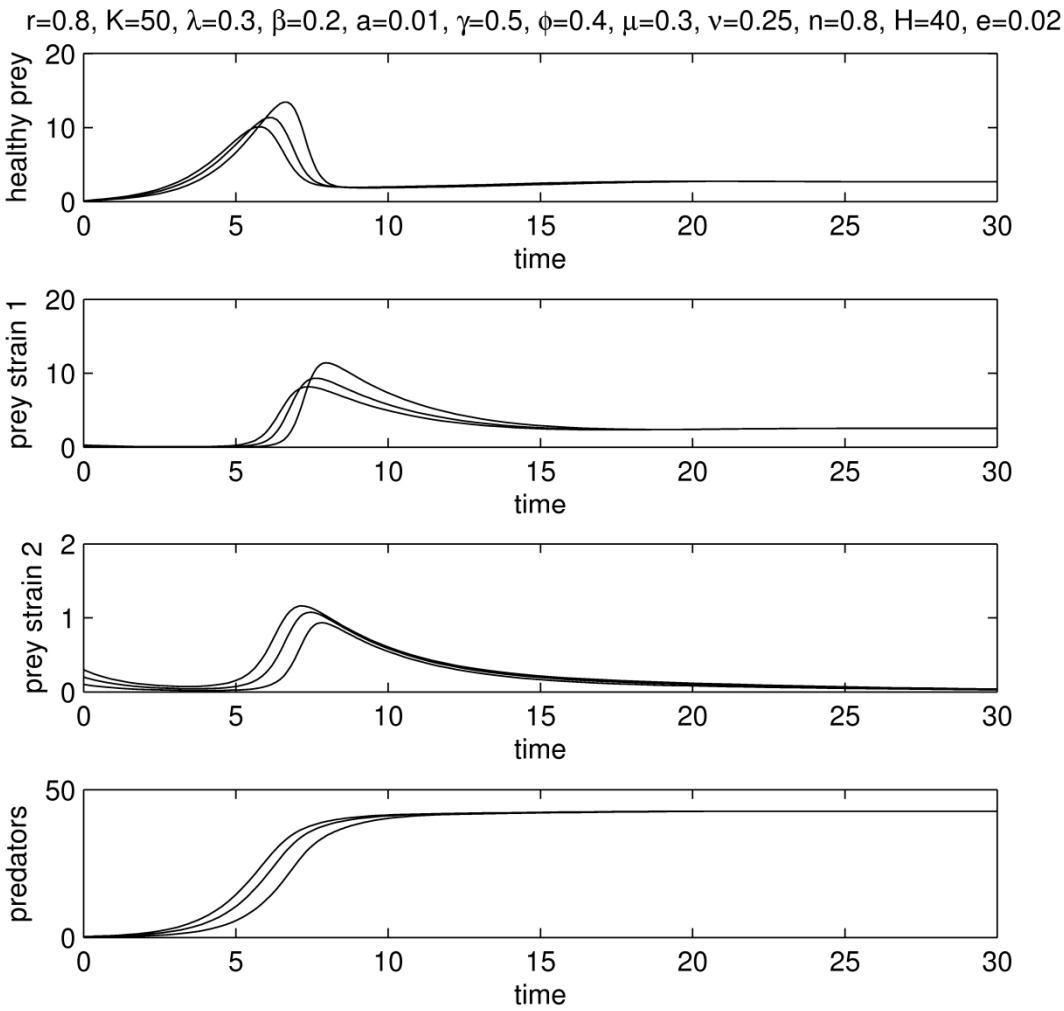


**Fig. 8** Equilibrium $Q_7$ obtained for the parameter values: r=0.8, k=50, λ=0.3, β=0.2, a=0.01, γ=0.5, φ=0.4, μ=0.3, ν=0.25, n=0.8, e=0.02, H=40.

## 6 Conclusions

In this investigation we considered an ecoepidemic system where two diseases affect the prey. The predators can feed also on other sources, which was not the case in the model presented in (Elena et al., 2011).

One positive conclusion in both cases shows that the ecosystem can never disappear, given the unconditional instability of the origin.

Some equilibria of the model with no other food supply for the predators (2) appear also as equilibria of the system in which predators have other resources (1). However, their characteristics differ. We provide a detailed description below.

In fact, the healthy-prey-only equilibrium $E_1 \equiv Q_1$ is conditionally stable for (2), while it cannot be attained in (1), since it is always unstable. In particular, it is stable for the system (2) in which the predators disappear in absence of the prey, if the prey carrying capacity is low enough, since $E_1$ is stable only if the following condition holds,

$$k < \min\left\{\frac{m}{e}, \frac{\nu+\varphi}{\beta}, \frac{\mu+\gamma}{\lambda}\right\},$$

so that if the carrying capacity exceeds the ratio of the predators' mortality with the return that they get from hunting, the latter equilibrium becomes unstable, and the predators enter into the system.

We now compare the disease-free equilibria in the two models, $E_2$ and $Q_2$. We find in fact the feasible equilibrium $E_2$ for (2), stable if the remaining previous stability conditions are still satisfied. This equilibrium is easily seen to originate via a transcritical bifurcation from $E_1$, as mentioned above: compare the stability condition of $E_1$ given above, with the feasibility and stability conditions for $E_2$, namely

$$1 \geq \frac{m}{ek};$$

and

$$\frac{m}{e} < \min\left\{\frac{\mu+\gamma}{\lambda}, \frac{\nu+\varphi}{\beta}\right\}.$$

In the model (1) the corresponding equilibrium $Q_2$ becomes feasible instead if the predators' carrying capacity is larger than the ratio between the prey's reproduction rate and the rate at which they are hunted (4). Both equilibria are conditionally stable, for $Q_2$ the conditions are quite complex, see (7)-(9) compared to the above given feasibility condition for $E_2$. In fact the latter two express the same concept, that the prey population level at the equilibrium must be bounded above by the very same quantities, namely the ratios of the losses the infected classes suffer by mortality and recovery over their recruitment rates. The difference between (9) and the above feasibility condition for $E_2$ lies of course in the level of the prey, which is different in the two models.

The predator-free endemic equilibria with only one strain, $E_3 = Q_3$ and $E_4 = Q_4$, are conditionally stable in the model (2) for which the prey are necessary for the survival of the predators. Instead, if other food sources for the predators are available, the equilibria $Q_3$ and $Q_4$ cannot be attained, given their instability, system (1). From the stability conditions for $E_3$ and for $E_4$ previously given for the model (2), we have shown that for both equilibria no Hopf bifurcations arise.

The model with other resources (1) contains further equilibria. An obvious one is provided by the predators-only point $Q_5$, always feasible but stable for a large enough predators' carrying capacity, see (10). Comparing this with (4), we see that it originates from $Q_2$ via a transcritical bifurcation.

The remaining equilibria, at which predators and prey thrive together, with the latter hosting only one endemic strain, have been shown to be reachable by the system via numerical simulations.

The most striking result is however that in both models (2) and (1) no internal equilibrium exists, nor an equilibrium in which the two diseased populations coexist together, in absence of the predators. Therefore it seems that predators hunting healthy prey, whether be it in presence or absence of additional available resources, do not provide the additional "ecological" conditions found in standard epidemiology, that allow the two strains to be present at the same time in the infected population.

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