I. Troch, F. Breitenecker, eds. ISBN 978-3-901608-35-3

INTERMINGLING OF DISEASE-AFFECTED POPULATIONS

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Abstract. Population models date back to the Malthus model of the early nineteenth century. Verhulst corrected it a few decades later to avoid its only two possible outcomes, either exponential growth or decay. His logistic correction allows the population to tend to a constant value representing the carrying capacity of the environment for this population. The early twentieth century saw the predator-prey works of Volterra prompted by the data on fishing in the Adriatic sea collected by the biologist D'Ancona. The now called Lotka-Volterra model contains an interior equilibrium point which exhibits neutral stability. It has been later modified to avoid this drawback and to make it more realistic, by including quadratic and other nonlinear models, accounting for logistic growth or feeding saturation. These models have also been generalized to food webs, with a top predator feeding on some other populations, this possibly going down to several levels.

Mathematical epidemiology was founded by Kermac and McKendric who studied an SIRS (Susceptible-Infected-Removed-Susceptible) system. A disease spreads by contact in a population of fixed size. The major aim consists in searchig for strategies to control the disease, possibly by devising suitable vaccination policies. The population is divided into classes of susceptibles, infected and removed, i.e. quarantined, individuals. The mathematical analysis sheds light on the epidemics spread, remarking that the problem is given by the infectives, which are not identified until they show the disease symptoms, but in the while they have been able to infect other individuals. The most important success of this discipline has been the decision of the WHO in 1980, by which smallpox has been declared worldwide eradicated, and consequently compulsory smallpox vaccination has been discontinued. This decision has been made on the basis of a suitably validated mathematical epidemics model. Specifically, it is the basic reproduction number that tells whether a disease will ultimately propagate or not. A variety of other models in this field of study are also possible.

Ecoepidemiology is a rather new subject of study, in that it considers interacting populations in which an epidemic is spreading. It therefore merges characterists belonging to both types of models described above. In most of the work so far undertaken by several researchers, various types of interactions between the populations have been considered. The disease has always been taken to spread only in one population, either the prey or the predators, or one of the two competing or symbiotic species described by the underlying demographic model. Only fairly recently epidemics in both populations have been considered.

We consider a rather complicated ecosystem in which two populations thrive, and both are disease-affected. The epidemics can pass from one species to the other one by contact. We analytically and numerically investigate the feasibility and stability conditions of the equilibria of the system. We also study possible interesting behaviors of special cases of the proposed model. Some relevant findings are outlined below.

In the proposed ecoepidemic model the disease cannot be eradicated while at the same time preserving both populations, thus the two sound populations cannot thrive together, while this currently happens in the corresponding purely demographic models.

The predators' presence in the model destabilizes the prey endemic equilibrium population, although it is not a priori easy to determine the outcome of a possible predator introduction into the environment. The system will tend to one of its equilibria, where the disease for the prey may not or may be present, but perhaps in such case even affect them more.

The introduction of the disease in the prey may save the sound predators from extinction; in fact predators may go to extinction in the ecoepidemic model should the prey be disease-free.

In the ecoepidemic model considered here the disease cannot disappear just from the predators. It can vanish from the prey leaving only the sound prey as the sole subpopulation thriving in the system. Otherwise, the disease remains endemic in the ecosystem.

1 The state of the art

Population models date back to the Malthus model of the early nineteenth century. Verhulst corrected it a few decades later to avoid its only two possible outcomes, either exponential growth or decay. His logistic correction

allows the population to tend to a constant value representing the carrying capacity of the environment for this population. The early twentieth century saw the predator-prey works of Volterra prompted by the data on fishing in the Adriatic sea collected by the biologist D'Ancona. The now called Lotka-Volterra model contains an interior equilibrium point which exhibits neutral stability. It has been later modified to avoid this drawback and to make it more realistic, by including quadratic and other nonlinear models, accounting for logistic growth or feeding saturation. These models have also been generalized to food webs, with a top predator feeding on some other populations, this possibly going down to several levels.

Mathematical epidemiology was founded by Kermac and McKendric [15], who studied an SIRS (Susceptible-Infected-Removed-Susceptible) system. A disease spreads by contact in a population of fixed size. The major aim consists in searchig for strategies to control the disease, possibly by devising suitable vaccination policies. The population is divided into classes of susceptibles, infected and removed, i.e. quarantined, individuals. The mathematical analysis sheds light on the epidemics spread, remarking that the problem is given by the infectives, which are not identified until they show the disease symptoms, but in the while they have been able to infect other individuals. The most important success of this discipline has been the decision of the WHO in 1980, by which smallpox has been declared worldwide eradicated, and consequently compulsory smallpox vaccination has been discontinued. This decision has been made on the basis of a suitably validated mathematical epidemics model. Specifically, it is the basic reproduction number that tells whether a disease will ultimately propagate or not, [3]. The fairly recent review [10] contains the description also of a variety of other possible models in this field of study.

Ecoepidemiology is a rather new subject of study, in that it considers interacting populations in which an epidemic is spreading, [5, 1, 19, 20, 21, 22, 2, 11, 12, 6]. It therefore merges characterists belonging to both types of models described above. In most of the work so far undertaken by several researchers, various types of interactions between the populations have been considered. The disease has always been taken to spread only in one population, either the prey or the predators, or one of the two competing or symbiotic species described by the underlying demographic model. Only fairly recently one of the authors [23] and a very recent paper [13] have considered the epidemics in both populations. Here we tackle again this problem considering a generalization of [23].

2 The proposed model

We consider a rather complicated ecosystem in which two populations thrive, and both are disease-affected. The epidemics can pass from one species to the other one by contact.

Some similar models have recently appeared in the literature [23, 13]. The main difference with [13] lies in the fact that here also logistic reproduction of the infected prey is taken into account, as well as infection of prey due to contact with infected predators. In [13] however, predation is modeled via a Michaelis-Menten term, while here we use just a Holling type I term. The predators in [13] become infected by feeding upon infected prey. For us the interaction with infected prey has a dual effect on the predators, positive in that feeding avoids them to starve and thus contributes to their reproduction, and negative in case the contact does not lead to a prey capture, but in the course of it the sound animal possibly gets infected by the diseased one. Mortality of predators is linear in [13], while it is given by the logistic quadratic term here. With respect to [23], note also that the simplifying assumption that a sound prey escapes the attack of an infected predator is here removed.

We therefore consider the following model

$$\begin{aligned}
\dot{P} &= P\left(r_1 - \frac{P+U}{\tilde{K}} - \gamma U - \beta V - b_1(Q+V)\right) \\
\dot{U} &= U\left(r_2 + \gamma P - \frac{P+U}{\tilde{K}} - b_2(Q+V)\right) + \beta PV \\
\dot{Q} &= Q\left(-m - \alpha U - \eta V + e\left(b_1 P + b_2 U\right)\right) \\
\dot{V} &= V\left(-m + \eta Q + e\left(b_1 P + b_2 U\right)\right) + \alpha UQ
\end{aligned} \tag{1}$$

where P represents the sound prey, U the infected prey, Q the sound predators and V the infected predators. The first equation describes the evolution of sound prey, which reproduce logistically, with net growth rate r_1 and with population pressure constant $\tilde{K}^{-1}$. Note that the infected prey do contribute to intraspecific competition for available resources. The third and fourth terms in the first equation describe loss of individuals due to the infection process, by contact with an infected prey and an infected predator respectively. The fifth term instead accounts for predation, by both sound and infected predators at the same rate b_1. Thus we are assuming that the disease does not affect the hunting capabilities of sick predators.

The second equation describes the infected prey evolution. They enter this class via the contacts the sound prey have with infected prey and predators, as seen above at respective rates γ and β, see the second and last term in

the equation, and also they can be born infected, from diseased parents, at rate r_2. They also experience quadratic mortality due to intraspecific competition with the same constant $\tilde{K}^{-1}$ as for sound prey, and are subject to hunt by both sound and diseased predators, again at the very same rate b_2. Note that we consider $b_2 > b_1$, so that the difference in predation rates expresses then the fact that infected prey are more vulnerable than sound ones, while the disease does not affect the hunting of the predators as mentioned above.

The third and fourth equations state that predators in absence of their prey, die with mortality rate m, so that P and U represent their only food source. The parameter e denotes the conversion factor of prey into new predators. Finally the sound predators become infected at rate η via contacts with other infected predators, and with disease incidence α by contacts with infected prey. Therefore hunting infected prey has both positive and negative effects on sound predators.

3 Analysis

3.1 Boundedness

The system can be shown to be bounded, at least under reasonable assumptions, by considering its total environmental population $S(t) = P+U+Q+V$, observing that letting $M = P+U$, $R = Q+V$, it satisfies the equation

$$\dot{S}(t) = r_1 P + r_2 U - mR - \frac{M^2}{\tilde{K}} - (1-e)R(b_1 P + b_2 U).$$

If $e \leq 1$ the following bound follows using Gronwall's inequality

$$0 < S(t) < \frac{1}{\theta}(1-e^{-\theta t})W^* + S(0)e^{-\theta t}, \quad W^* \equiv \frac{1}{4}\tilde{K}(r_1+r_2+\theta)^2,$$

for a suitable θ, with $m > \theta > 0$. Thus for $t \longrightarrow +\infty$, we have $S(t) \longrightarrow \frac{1}{\theta}W^* > 0$.

3.2 Equilibria and their stability

After rescaling, via $p = \theta P$, $u = \phi U$, $q = \psi Q$, $v = \omega V$, $\tau = \sigma t$, with

$$\sigma = m, \quad \phi = \frac{\gamma}{m}, \quad \omega = \frac{\beta}{m}, \quad \theta = \frac{eb_1}{m}, \quad \psi = \frac{b_1}{m}$$

and setting

$$A = \frac{\gamma}{eb_1}, \quad B = \frac{1}{\tilde{K}\gamma}, \quad C = \frac{b_1}{\beta}, \quad D = \frac{b_2}{\beta}, \quad E = \frac{\alpha}{\gamma}, \quad F = \frac{\eta}{\beta}$$

the system becomes

$$\begin{aligned}
\dot{p} &= \frac{r_1 p}{m} - ABp^2 - Bpu - pu - pv - pq - Cpv \\
\dot{u} &= \frac{r_2 u}{m} - ABpu - Bu^2 + Apu - \frac{Duq}{C} - Duv + mApv \\
\dot{q} &= -q - Euq - Fqv + pq + \frac{ADuq}{C} \\
\dot{v} &= -v + \frac{Fqv}{C} + pv + \frac{ADuv}{C} + \frac{Euq}{C}.
\end{aligned} \tag{2}$$

There are several equilibria, whose local stability can be analysed by considering the eigenvalues of the system's Jacobian,

$$\begin{pmatrix}
J_{11} & -p(B+1) & -p & -p(1+C) \\
u(-AB+A)+mAv & J_{22} & -\frac{D}{C}u & -Du+mAp \\
q & q(AD-CE)\frac{1}{C} & J_{33} & -Fq \\
v & \frac{1}{C}(ADv+Eq) & \frac{1}{C}(Fv+Eu) & J_{44}
\end{pmatrix} \tag{3}$$

where

$$\begin{aligned}
J_{11} &= \frac{r_1}{m} - 2ABp - Bu - u - v - q - Cv, \quad J_{22} = \frac{r_2}{m} - ABp - 2Bu + Ap - \frac{Dq}{C} - Dv \\
J_{33} &= -1 - Eu - Fv + p + \frac{ADu}{C}, \quad J_{44} = \frac{Fq}{C} - 1 + p + \frac{ADu}{C}
\end{aligned}$$

Some of the equilibria are easily found and analyzed.

The origin has the eigenvalues $\frac{r_1}{m}$, $\frac{r_2}{m}$, -1, -1 showing its instability. Next the point $Q_2 = (0, \frac{r_2}{Bm}, 0, 0)$ has the eigenvalues

$$\frac{-Br_2 + Br_1 - r_2}{Bm}, \quad -\frac{r_2}{m}, \quad -\frac{BmC + Er_2C - r_2AD}{BmC}, \quad \frac{r_2AD - BmC}{BmC}$$

showing stability if the following conditions are satisfied,

$$B(r_1 - r_2) < r_2, \quad r_2AD < BmC.$$

The remaining equilibria are the following ones:

$$Q_3 = (\frac{r_1}{ABm}, 0, 0, 0), \quad Q_4 = \Big(\frac{-Br_2 + Br_1 - r_2}{mA}, \frac{Br_2 - Br_1 + r_1}{m}, 0, 0\Big),$$
$$Q_5 = \Big(0, \frac{C}{AD}, 0, \frac{r_2AD - BmC}{D^2mA}\Big), \quad Q_6 = \Big(1, 0, \frac{r_1 - ABm}{m}, 0\Big), \quad Q_7 = (p_7^{\pm}, u_7^{\pm}, 0, v_7^{\pm})$$

the last one being actually a double equilibrium point, with components specified later.

Note that Q_3 is always feasible. Its eigenvalues are

$$-\frac{r_1}{m}, -\frac{-Br_2 + Br_1 - r_1}{Bm}, -\frac{-r_1 + ABm}{ABm}, -\frac{-r_1 + ABm}{ABm}$$

so that it is conditionally stable, namely if

$$r_1 - ABm < 0, \qquad r_1 + B(r_2 - r_1) < 0. \tag{4}$$

Q_4 is feasible when both the following conditions hold

$$K_1 = Br_2 - Br_1 + r_1 \geq 0, \quad K_2 = Br_1 - Br_2 - r_2 \geq 0,$$

which imply $r_1 \geq r_2$. Its eigenvalues are

$$\frac{Z}{mAC}, \quad \frac{Z - EACK_1}{mAC}, \quad \frac{B(r_2 - r_1) \pm \sqrt{5B^2(r_2 - r_1)^2 - 4X}}{2m}$$

where

$$Z = -mAC + A^2DK_1 + CK_2, \quad X = Br_1^2 - Br_2^2 - r_1r_2.$$

Now, $Z < 0$ implies the negativity of the first two eigenvalues. Also, if $K_1, K_2 > 0$ we can show that $X > 0$. Indeed when Q_4 is feasible from $K_2 > 0$ it follows

$$0 < r_1(Br_1 - Br_2 - r_2) + r_2(Br_1 - Br_2 - r_2) \equiv K_2(r_1 + r_2) = X - r_2^2$$

It then follows that $X > 0$.

Moreover, when Q_4 is feasible also $r_2 - r_1 < 0$ must hold. In fact, summing K_1 and K_2 we find $0 < Br_2 - Br_1 + r_1 + Br_1 - Br_2 - r_2 = r_1 - r_2$. As a consequence the fourth eigenvalue, with minus sign, clearly has negative real part. To have the same result also for the third one, we must have

$$|B(r_2 - r_1)| > \sqrt{5B^2(r_2 - r_1)^2 - 4X}$$

from which

$$B^2(r_2 - r_1)^2 > 5B^2(r_2 - r_1)^2 - 4X$$

and further

$$K_1K_2 \equiv X - B^2(r_2 - r_1)^2 \leq 0.$$

The strict inequality holds as long as the nonzero components of Q_4 do not degenerate. In conclusion the only stability condition for Q_4 reduces to

$$Z < 0. \tag{5}$$

If $K_1 = 0$ or $K_2 = 0$ or both, we need to require also $r_1 < r_2$.

Q_5 is feasible for

$$G = r_2AD - BmC \geq 0. \tag{6}$$

The eigenvalues are

$$-\frac{G(1+C)+D(BCm+mC-r_1AD)}{D^2mA}, \quad -\frac{FG+EDmC}{D^2mA}, \quad -\frac{BmC\pm\sqrt{(BmC)^2-4DmAG}}{2Dm}.$$

If $G>0$ it is easy to show that the last two eigenvalues have always negative real part, while for $G=0$ the equilibrium is always unstable. Moreover the second one is always negative, in view of (6). The only stability condition comes then from the negativity of the first eigenvalue, a sufficient condition for which is given namely by

$$BCm+mC-r_1AD>0. \tag{7}$$

Q_6 is feasible if

$$B_1=r_1-ABm\geq 0.$$

Its eigenvalues are

$$-\frac{ABm\pm\sqrt{(ABm)^2-4mB_1}}{2m}, \quad \frac{W\pm\sqrt{W^2+4B_1D_1}}{2mC}$$

with $C_1=r_2+Am-ABm$, $D_1=-CFC_1+DFB_1+EACm^2$, $W=B_1(F-D)+C_1C$. If $B_1=0$ the equilibrium is always unstable. Let us take then $B_1>0$. It is easy to see that the first two eigenvalues have always nonpositive real part. To have stability, the remaining eigenvalues must have negative real parts. This is ensured by the following conditions: for the root with a minus sign, if $C_1>0$ we need to require $W<0$, $D_1<0$, while for $C_1<0$ the root is always negative; for the other root with the plus sign, in case $C_1>0$ the conditions are $W<0$, $D_1<0$, while for $C_1<0$ the eigenvalue is positive and therefore the equilibrium is unstable. In summary, this equilibrium would be stable for $C_1>0$, $W<0$, $D_1<0$. However, if $W<0$ we find $B_1(F-D)+C_1C<0$ implying $C_1C-DB_1<0$. Since $D_1=-F(CC_1-DB_1)+EACm^2$ it follows then $D_1>0$. Therefore Q_6 is inconditionally unstable.

Finally the last double equilibrium is quite complicated, as the nonvanishing components are explicitly determined by solving three quadratic equations. To study feasibility, let

$$\begin{aligned}
R&=C(C+1)(r_2+Am-AmB)+D(mA^2+C)(mAB-r_1)+m^2AB(A^2D-C)-m^2AC,\\
K&=A^2m^2C(C+1)(-AD(AmB-r_1)+B^2m(A^2D-C))+m^2ACD(A^2m+C)(mAB-r_1)\\
&\quad +m^2AB(A^2D-C)(-D(mAB-r_1)(mA^2-C)-Cr_1(C+1)),\\
J&=(A^2m+C)D(B(A^2D-C)-C)-(C+1)C(B(A^2D-C)-A^2D).
\end{aligned}$$

From now on we take $J>0$, as conclusions similar to the ones we are about to derive can be drawn also in the opposite case. We then have

$$u_7^{\pm}=\frac{R\pm\sqrt{R^2+4K}}{2mJ}C$$

with feasibility conditions for $K<0$ given by $R>0$ together with $R^2+4K\geq 0$ to have real roots; for $K>0$, u_7^- is always negative and u_7^+ is always positive.

For

$$p_7^{\pm}=1-AD\left(\frac{R\pm\sqrt{R^2+4K}}{2mJ}\right)$$

positivity holds unconditionally if $\left|AD\left(\frac{R\pm\sqrt{R^2+4K}}{2mJ}\right)\right|<1$. Conversely, there are two cases: if $K<0$ we need $R<0$ together with $R^2+4K\geq 0$ to ensure real roots, while for $K>0$, p_7^- is always positive and p_7^+ is always negative. Further we have

$$v_7^{\pm}=\frac{(r_1-AmB)+\frac{R\pm\sqrt{R^2+4K}}{2mJ}Wm}{m(C+1)}$$

with $W=A^2BD-BC-C$. To have positive values, we need real roots, ensured by $R^2+4K\geq 0$ and in case

$$\left|\frac{R\pm\sqrt{R^2+4K}}{2mJ}Wm\right|>|r_1-AmB|,$$

if $K>0$ positivity for v_7^+ is ensured by $W>0$ while for v_7^- we need $RW>0$; conversely for $K<0$ we need $RW>0$ for v_7^+ and for v_7^- we need $W<0$.

Alternatively positivity is also ensured by

$$\left|\frac{R\pm\sqrt{R^2+4K}}{2mJ}Wm\right|<|r_1-AmB|\,.$$

In summary, we state here below the only acceptable equilibria under the assumptions $J>0$ and $R^2+4K\geq 0$.

For $\left|AD\left(\frac{R\pm\sqrt{R^2+4K}}{2mJ}\right)\right|>1$, and $K>0$, there are two possibilities. If $\left|\frac{R\pm\sqrt{R^2+4K}}{2mJ}Wm\right|>|r_1-AmB|$ the feasible equilibrium is $(p^-,u^+,0,v^+)$ for $W>0$, while conversely it is $(p^-,u^+,0,v^-)$ for $W<0$. Instead in case of $\left|\frac{R\pm\sqrt{R^2+4K}}{2mJ}Wm\right|<|r_1-AmB|$ and $r_1-AmB>0$ both the points $(p^-,u^+,0,v^\pm)$ are feasible.

For $\left|AD\left(\frac{R\pm\sqrt{R^2+4K}}{2mJ}\right)\right|<1$ instead, again two subcases arise. If $\left|\frac{R\pm\sqrt{R^2+4K}}{2mJ}Wm\right|>|r_1-AmB|$: for $K<0$, and $R>0$, $W>0$ the equilibria $(p^\pm,u^\pm,0,v^\pm)$ are both feasible; for $K>0$ and $W>0$ the feasible equilibria are $(p^\pm,u^+,0,v^+)$ and for $K>0$ and $W<0$ they are $(p^\pm,u^+,0,v^-)$. If instead $\left|\frac{R\pm\sqrt{R^2+4K}}{2mJ}Wm\right|<|r_1-AmB|$, and $r_1-AmB>0$, for $K>0$ we have the feasible points $(p^\pm,u^+,0,v^\pm)$ and for $K<0$ and $R>0$ the feasible equilibria are $(p^\pm,u^\pm,0,v^\pm)$.

For this equilibrium the stability investigation is quite complicated and is done numerically.

Figures 1-5 contain the simulations results. We respectively show sound and infected prey and total prey populations, top row, sound and infected predators and total predator populations, bottom row. From the latter it is evident that a feasible Q_7 can indeed be attained, and moreover that a bifurcation leading to limit cycles arises around it. Figures 1 and 2 show such limit cycles with different amplitudes respectively. Taking as reference the values of Figure 1, namely $r_1=2.8$, $r_2=.7$, $m=0.1$, $A=0.9$, $B=0.3$, $C=0.21$, $D=0.3$, $E=0.4$, $F=0.52$, and $r_1=12.8$, $r_2=0.7$, $m=0.1$, $A=0.7$, $B=0.3$, $C=0.021$, $D=0.83$, $E=0.004$, $F=0.0052$, we have then investigated the influence of each parameter as bifurcation parameter. It seems that the bifurcation is insensitive only to E. Figures 3-5 contain the stable equilibrium with some components at nonzero very low level, obtained for the first value of each bifurcation parameter. The bifurcation values are summarized here: a stable behavior is ensured below the following values: $r_1=2.45$, $A=0.71$, $D=0.258$, and above the following ones: $r_2=0.825$, $B=0.475$, $C=0.269$, $F=1.52$, $m=0.35$. The stable behavior however leads to equilibrium Q_5 for all the parameters but B, in which case the trajectories tend to the point $Q_7=(0.0083,0.7713,0.0000,22.197)$, showing thus that for this bifurcation parameter the Hopf bifurcation at Q_7 is obtained. The same occurs for the larger value of the parameter $D=1.49$, the oscillations seem to damp down toward Q_7.

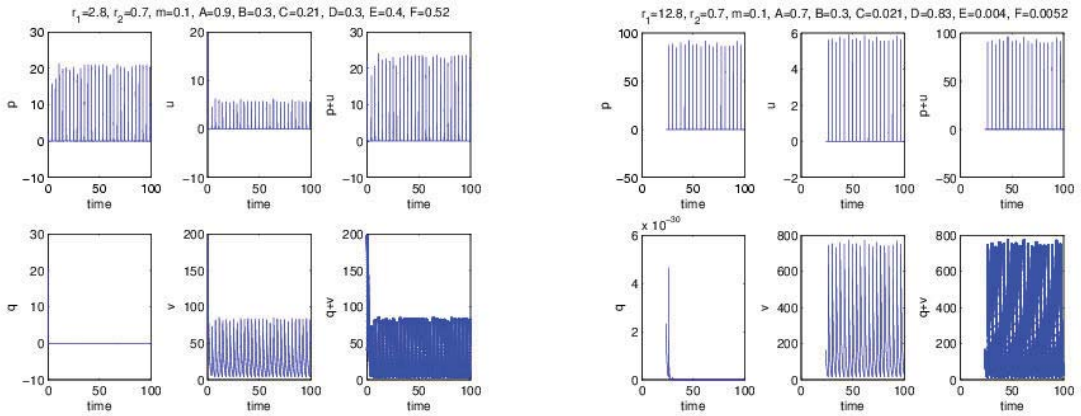

Figure 1: Limit cycles for the parameter values: $r_1=2.8$, $r_2=.7$, $m=0.1$, $A=0.9$, $B=0.3$, $C=0.21$, $D=0.3$, $E=0.4$, $F=0.52$, (left) and $r_1=12.8$, $r_2=0.7$, $m=0.1$, $A=0.7$, $B=0.3$, $C=0.021$, $D=0.83$, $E=0.004$, $F=0.0052$ (right)

4 Some particular cases

4.1 The demographic model

The underlying disease-free population model is the following predator-prey model

$$\dot{p}=p\left(\frac{r_1}{m}-ABp-q\right), \tag{8}$$

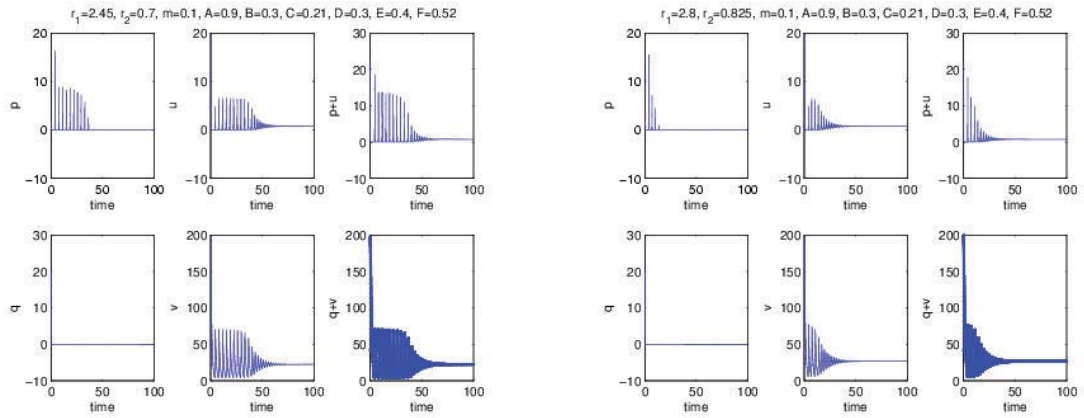

Figure 2: Oscillations damp toward Q_5 for $r_1 = 2.45$ (left) and $r_2 = 0.825$ (right)

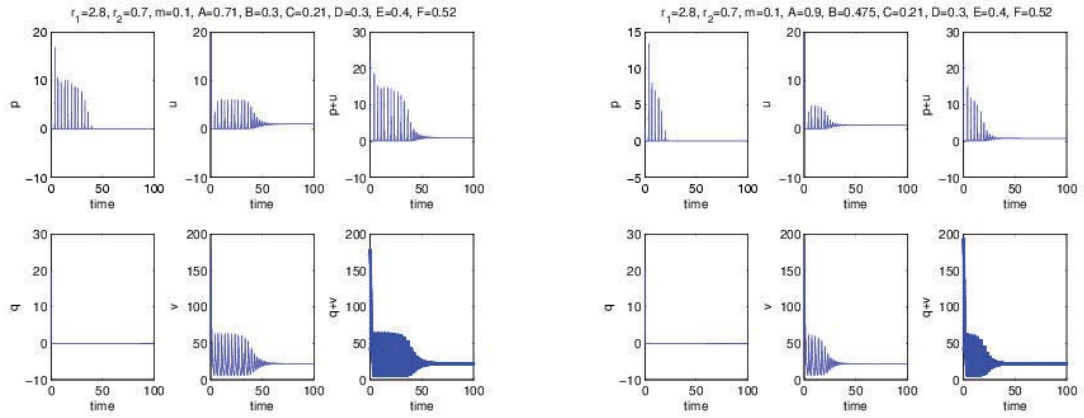

Figure 3: Oscillations for $A = 0.71$ damp toward Q_5 (left) and onset of bifurcation for $B = 0.475$ (right); in the latter case the equilibrium reached is $Q_7 = (0.0083, 0.7713, 0.0000, 22.197)$

$$\dot{q} = q(p-1),$$

whose equilibria are the origin, always unstable in view of the eigenvalues $\frac{r_1}{m}$, -1, the point $M_2 = (\frac{r_1}{ABm}, 0)$, stable if

$$r_1 - ABm < 0. \tag{9}$$

Also, the coexistence equilibrium $M_3 = (1, \frac{r_1 - ABm}{m})$ is feasible for

$$r_1 - ABm \geq 0. \tag{10}$$

Its eigenvalues are

$$\frac{-ABm \pm \sqrt{(ABm)^2 - 4m(r_1 - ABm)}}{2m}$$

so that in view of (10) for the strict inequality $r_1 - ABm > 0$ it is always stable.

If we now compare M_3 with Q_6, the former is unconditionally stable for $r_1 - ABm > 0$, while the latter is always unstable. Thus the disease introduction in the demographic system makes unstable the coexistence equilibrium, which is instead stable in the purely demographic model. In this case the disease acts then as a destabilizing factor. Figure 6 shows this effect.

4.2 Model without predators

The model with no predators is the following purely epidemics model

$$\begin{aligned} \dot{p} &= p\Big(\frac{r_1}{m} - ABp - Bu - u\Big), \\ \dot{u} &= u\Big(\frac{r_2}{m} - ABp - Bu + Ap\Big), \end{aligned} \tag{11}$$

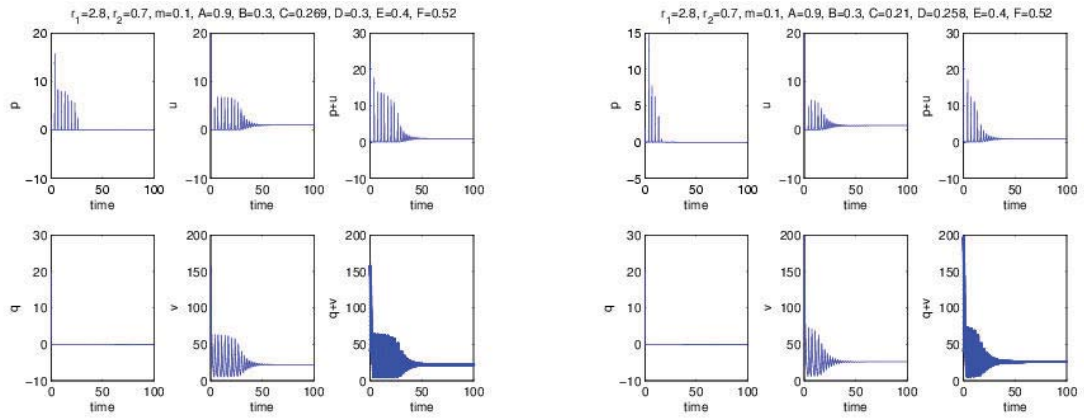

Figure 4: Oscillations damp toward Q_5 for $C = 0.269$(left) and $D = 0.258$ (right)

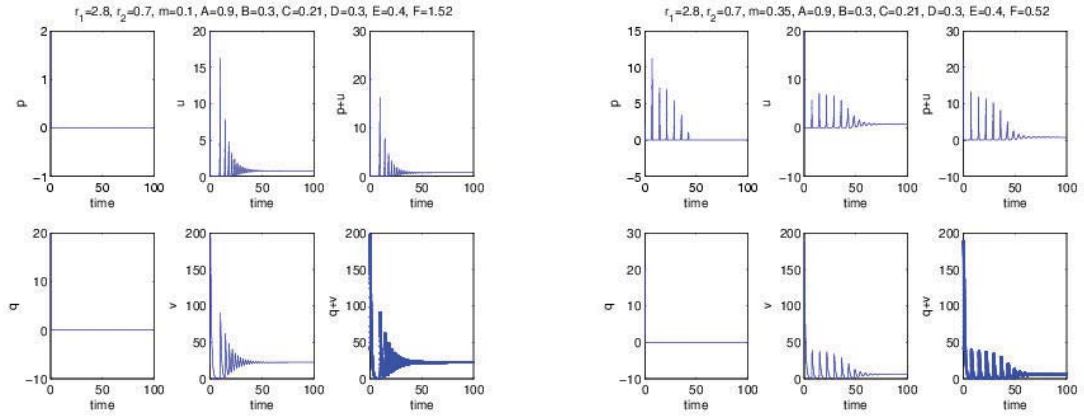

Figure 5: Oscillations damp toward Q_5 for $F = 1.52$ (left) and $m = 0.35$ (right)

which possesses the trivial equilibrium, unstable in view of the eigenvalues $\frac{r_1}{m}$, $\frac{r_2}{m}$. There are three more equilibria,

$$P_2 = \Big(0, \frac{r_2}{Bm}\Big), \quad P_3 = \Big(\frac{r_1}{ABm}, 0\Big), \quad P_4 = \Big(\frac{r_1B - r_2 - r_2B}{Am}, \frac{r_1 - r_1B + r_2B}{m}\Big).$$

P_2 is conditionally stable, namely for

$$r_1B - r_2B - r_2 < 0, \tag{12}$$

since the other eigenvalue is always negative, $-\frac{r_2}{m}$. Also P_3 has a negative eigenvalue, $-\frac{r_1}{m}$, and is also conditionally stable, if

$$r_1 + r_2B - r_1B < 0. \tag{13}$$

P_4 instead is feasible only if the following conditions hold

$$r_1B - r_2 - r_2B \geq 0, \quad r_1 - r_1B + r_2B \geq 0. \tag{14}$$

Note that these conditions imply $r_2 \leq r_1$. Its eigenvalues are

$$\frac{B(r_2 - r_1) \pm \sqrt{(B(r_2 - r_1))^2 - 4(r_1B - r_2 - r_2B)(r_1 - r_1B + r_2B)}}{2m}$$

so using (14) in case of strict inequalities it is inconditionally stable. Note that the feasibility of P_4 prevents the stability of both P_2 and P_3 and vice versa, the stability of at least one of the latter prevents the feasibility of P_4.

On comparing P_4 and Q_4 the former, if feasible with strict inequalities, is always stable, while the latter is stable only if $Z < 0$. Therefore introducing the predators in an environment in which the prey are subject to a disease, the equilibrium situation reached by the system in absence of predators is disturbed by the presence of the latter. To verify the above assertion we ran a simulation. Figure 7 contains the systems behavior when both species are present, top, and when the predators are missing, bottom. For the parameter values

$$A = 0.1, B = 0.5, C = 0.9, D = 0.2, E = 1.1, F = 1, m = 0.2, r_1 = 1.5, r_2 = 0.1$$

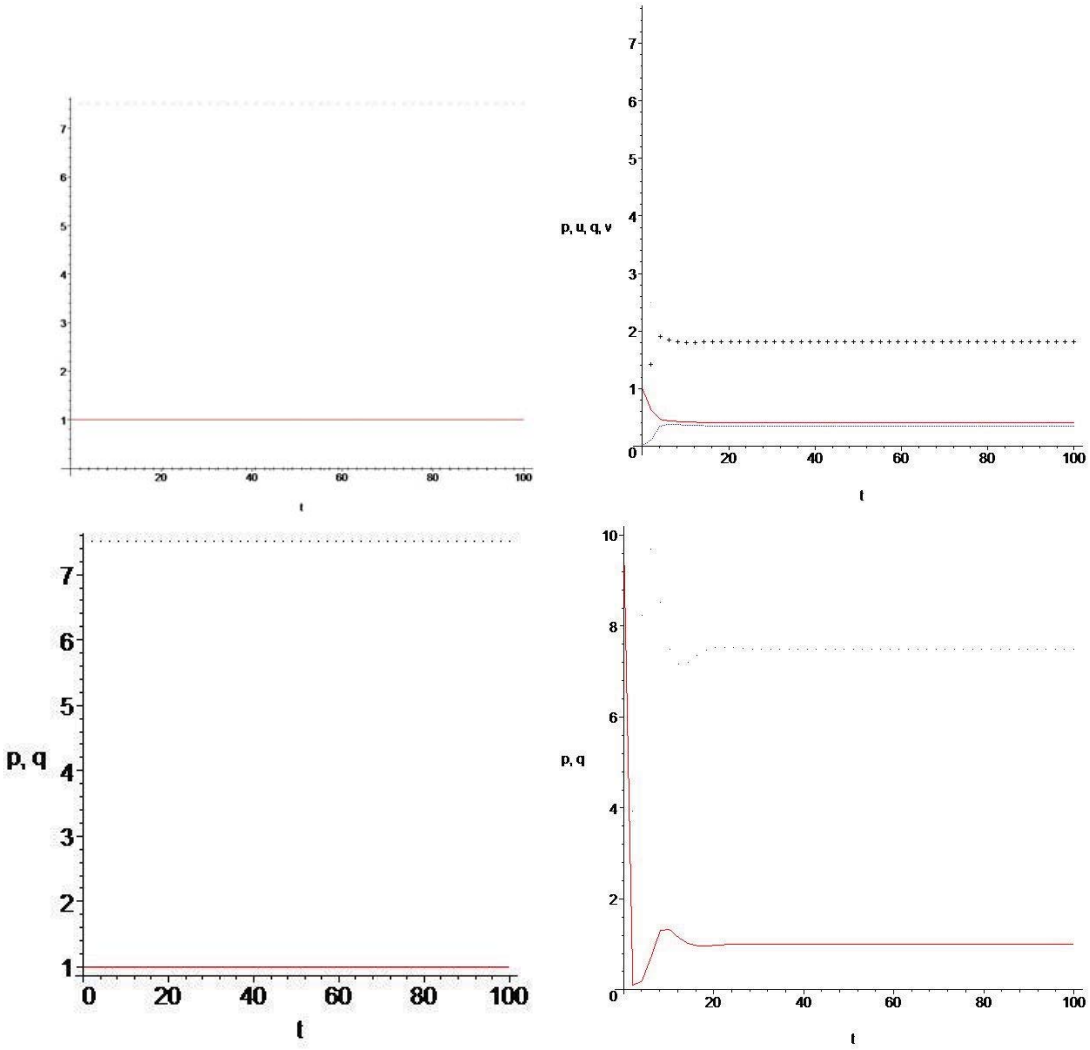

Figure 6: Destabilizing effect of the disease; top behavior near the unstable equilibrium $Q_6 = \{1, 0, 7.5, 0\}$; left: taking as initial conditions the point itself $(1, 0, 7.5, 0)$; right: taking slightly perturbed initial conditions: $(1, 0.01, 7.5, 0)$ the system moves away from Q_6; bottom behavior near the stable demographic equilibrium $M_3 = \{1, 7.5\}$; left: initial conditions on the equilibrium $(1, 7.5)$; right: the demographic system behavior with arbitrary initial conditions: $(10, 2)$ returns to the equilibrium value M_3.

$Q_4 \equiv (30, 4, 0, 0)$ is feasible but not stable. If we give initial conditions on the equilibrium, we find that it is preserved, but if we slightly perturb the initial conditions $(30, 4, 0.001, 0)$ we find that the trajectories drift away from Q_4. On the other hand, for the same parameter values, the equilibrium $P_4 = (30, 4)$, is found both starting with P_4 itself as well as from $(10, 4)$.

4.3 Model with no disease in the prey

Another simplified situation occurs if we disregard the disease among the prey to get the model:

$$\begin{aligned} \dot{p} &= p\Big(\frac{r_1}{m} - ABp - q - (C+1)v\Big) \\ \dot{q} &= q(-1 - Fv + p) \\ \dot{v} &= v\Big(-1 + \frac{Fq}{C} + p\Big). \end{aligned} \tag{15}$$

In this case there are at most four possibly feasible equilibria: the origin which is unstable, since it has the eigenvalues $\frac{r_1}{m}$, -1, double; then the points

$$M_{p2} = \Big(\frac{r_1}{ABm}, 0, 0\Big), \quad M_{p3} = \Big(1, \frac{r_1 - ABm}{m}, 0\Big), \quad M_{p4} = \Big(1, 0, \frac{r_1 - ABm}{mC}\Big).$$

M_{p2} is conditionally stable, in view of the negative eigenvalue $-\frac{r_1}{m}$ and another double one, leading to the stability

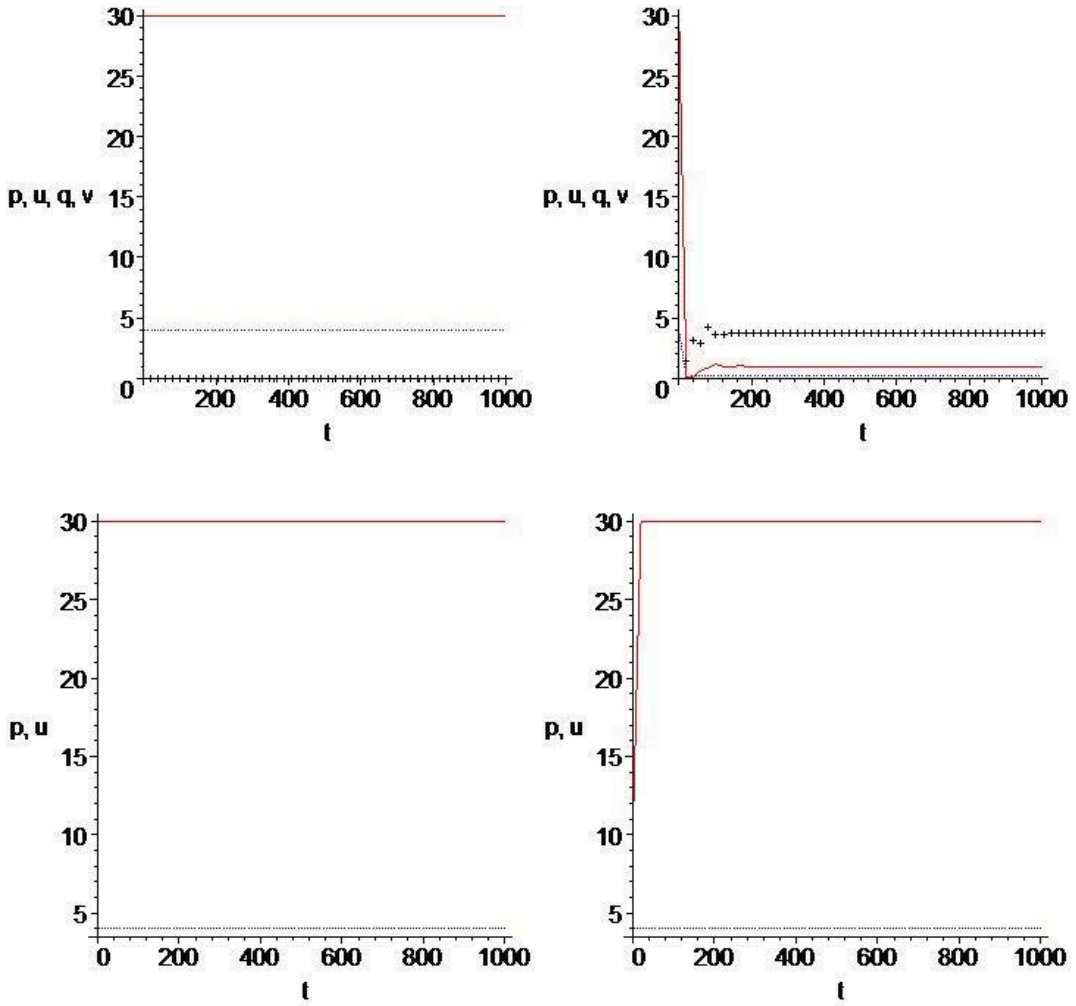

Figure 7: Full system behavior, top; left: trajectories starting at the equilibrium $Q_4 = \{30, 4, 0, 0\}$; right: trajectories with initial conditions $(30, 4, 0.001, 0)$; model with no predators, bottom; left: trajectories originating in $P_4 = \{30, 4\}$; right: system's behavior with initial conditions $(10, 4)$

condition

$$r_1 - ABm < 0. \tag{16}$$

The equilibrium M_{p3} is feasible for

$$r_1 - ABm \geq 0 \tag{17}$$

but has the eigenvalues

$$\frac{F(r_1 - ABm)}{mC}, \frac{-ABm \pm \sqrt{(ABm)^2 - 4m(r_1 - ABm)}}{2m},$$

so that taking the strict inequalities in (17) it is always unstable.

Finally M_{p4} is feasible again if (17) holds. Its eigenvalues are

$$\frac{-ABm \pm \sqrt{(ABm)^2 - 4m(r_1 - ABm)}}{2m}, -\frac{F(r_1 - ABm)}{mC}$$

and therefore the strict inequality in (17) makes it always stable.

At the equilibrium M_{p4} the sound prey and diseased predators coexist. A corresponding equilibrium in the full model, with disease also in the prey, does not exist. Thus the disease raging among the prey as well renders the disease invasion of predators impossible, while at the same time preserving the sound prey.

4.4 Model with no disease in predators

The model without disease in the predators is

$$\begin{aligned} \dot{p} &= p\left(\frac{r_1}{m} - ABp - (B+1)u - q\right), \\ \dot{u} &= u\left(\frac{r_2}{m} - A(B-1)p - Bu - \frac{Dq}{C}\right), \\ \dot{q} &= q\left(p - 1 + \frac{ADu}{C}\right) \end{aligned} \tag{18}$$

with equilibria given by the origin, unstable in view of the eigenvalues $\frac{r_1}{m}, \frac{r_2}{m}$, -1, by the coexistence equilibrium $S_{p7} = (p_7, u_7, q_7)$ and by the points

$$S_{p2} = \left(0, \frac{r_2}{Bm}, 0\right), \quad S_{p3} = \left(0, \frac{C}{AD}, \frac{C(-BCm + ADr_2)}{AD^2 m}\right), \quad S_{p4} = \left(\frac{r_1}{ABm}, 0, 0\right),$$
$$S_{p5} = \left(\frac{r_1 B - r_2 B - r_2}{mA}, \frac{r_1 + r_2 B - r_1 B}{m}, 0\right), \quad S_{p6} = \left(1, 0, \frac{r_1 - ABm}{m}\right).$$

Now, S_{p2} has eigenvalues

$$\frac{r_1 B - r_2 B - r_2}{Bm}, \quad -\frac{r_2}{m}, \quad \frac{r_2 AD - BmC}{BmC}$$

so that it is stable for

$$r_1 B - r_2 B - r_2 < 0, \qquad r_2 AD - BmC < 0. \tag{19}$$

Similar considerations hold for the all the other equilibria. S_{p3} is feasible for $H = -BCm + ADr_2 > 0$ with eigenvalues

$$-\frac{-BmC^2 + CDBm + Cr_2 AD + CDm - D^2 r_1 A}{AD^2 m}, \quad -\frac{BmC \pm \sqrt{(BmC)^2 - 4ADmH)}}{2DAm}$$

so that it is stable for

$$-BmC^2 + CDBm + Cr_2 AD + CDm - D^2 r_1 A > 0. \tag{20}$$

For S_{p4} the eigenvalues are

$$-\frac{r_1}{m}, \quad \frac{r_1 + Br_2 - Br_1}{Bm}, \quad \frac{r_1 - ABm}{ABm}$$

so that stability follows for

$$r_1 + Br_2 - Br_1 < 0, \qquad r_1 - ABm < 0. \tag{21}$$

The point S_{p5} instead is feasible for

$$H_1 = r_1 B - r_2 B - r_2 > 0, \qquad H_2 = r_1 + r_2 B - r_1 B > 0,$$

from which we find $r_2 - r_1 < 0$. Its eigenvalues are

$$\frac{B(r_2 - r_1) \pm \sqrt{(B(r_2 - r_1))^2 - 4H_1 H_2}}{2m}, \quad -\frac{H_3}{AmC}$$

where $H_3 = -BCr_1 + r_1 BA^2 D - r_1 A^2 D - Br_2 A^2 D + BCr_2 + AmC + r_2 C$. Stability follows for $H_3 > 0$.

The equilibrium S_{p6} is feasible for $r_1 - ABm > 0$ and has eigenvalues

$$-\frac{ABmC - AmC - DABm - r_2 C + Dr_1}{mC}, \quad \frac{-ABm \pm \sqrt{(ABm)^2 - 4m(r_1 - ABm)}}{2m}$$

so that it is stable for $C(ABm - Am - r_2) - D(ABm - r_1) > 0$.

Finally S_{p7} is difficult to study. Note however that this equilibrium is not present in the original model (1). Thus if the disease affects both populations, the predators alone cannot overcome it.

5 Interpretation

The model (1) could be made more general by allowing disease-related mortality for both species. Without going into details we just mention that the analysis of the latter would show that the equilibria are the same as for the original model (1), with more complicated feasibility and stability conditions. We have shown by simulations that system (1) exhibits limit cycles around the equilibrium with no sound predators.

The model without disease (8) has an always stable coexistence equilibrium but the corresponding point Q_6 of (1) is always unstable. Thus in the ecoepidemic model the disease cannot be removed while preserving both populations.

In the model without predators (11), i.e. the purely epidemics model obtained from (1) setting $q = v = 0$, the coexistence equilibrium when feasible is always stable. On the contrary in (1) stability for Q_4, the endemic prey equilibrium, is only conditional. Thus the predators' presence in the model has a system influence, to destabilize the prey endemic equilibrium population. It is however not easy to determine the outcome of a possible predator introduction into the environment, other than saying it will make the system (1) tend to one of its equilibria, where the disease for the prey may not or may be present, but perhaps in such case even affect them more. Thus the ecological measure of introducing a natural predator to fight the disease may not always be a sound measure, unless conditions for the stability of the sought equilibria are also met.

The model with no disease in the prey (15) has the unconditionally stable equilibrium M_{p4} with no sound predators, which is absent from (1). Thus the introduction of the disease in the prey may save the sound predators from extinction; this fact could instead happen should the prey be disease-free.

In the model with no disease in the predators (18), i.e. $v = 0$, the equilibrium with no infected prey S_{p6} can be stabilized while the disease-free equilibrium Q_6 in (1) is always unstable. The coexistence equilibrium S_{p7} has no counterpart $(p,u,q,0)$ in (1). Thus in the latter the disease cannot disappear just from the predators. Its only equilibria with $v = 0$ are Q_3 and Q_4 with all predators vanishing or the unstable disease-free Q_6.

Finally, the model without sound predators, i.e. for $q = 0$, shows the same characteristics as (1). Combining with earlier findings, we observe that the disease can vanish from the prey leaving only the sound prey as the sole subpopulation thriving in the system. Otherwise, the disease remains endemic in the ecosystem.

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