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by

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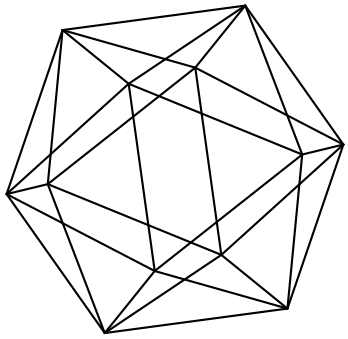

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A minimal model for ecoepidemics with group defense. *

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Abstract

The new idea of group defense as recently introduced by the author in the context of two interacting populations is in this paper applied to communities subject also to a disease. The system is formulated with the bare minimum of interactions among all the populations involved in order to highlight the effects of the nonlinearity describing the defense mechanism. A key parameter identified in the purely demographic model, which completely describes its outcomes, is seen here to have an important role also, in that its dropping below a threshold prevents the disease from invading the environment and causes the healthy prey and predators to coexist via persistent oscillations.

1 Introduction

Recently the author has proposed new models for population interactions, in the joint papers [2, 3]. In spite of the fact that only two-dimensional dynamical systems are considered, there is a novelty in the basic idea that has led to the formulation of such models. Since the early works of Lotka and Volterra [15, 10] the basic assumption in population theory considers always one to one interactions between prey and predators, assumed to be free to wander about in the environment and therefore subject to random encounters among isolated individuals of each population. Instead in [2, 3]

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a new idea of group defense has been introduced, quite different from earlier ones, see for instance [7]. The key point here is that prey, identified for instance in the large herbivores populating the savannas, gather together in huge herds, with generally the strongest individuals on the border and the weakest being concentrated in the middle of the bunch. This has the consequence that the capture of a prey by a successful predator's attack occurs mainly on the boundary, involving therefore mostly the individuals that occupy the outermost positions in the herd.

This situation is mathematically achieved in the model by observing that if a population spreads in a two-dimensional domain, the number of individuals on its boundary will be proportional to the length of the perimeter of the area occupied, and therefore proportional to the square root of the population density. The consequences of this assumption are that in a such a kind of predator-prey model a key parameter can be identified which describes completely the ensuing system dynamics.

In this paper we extend these ideas to ecoepidemic systems. The latter describe demographic interactions among populations in which also a disease spreads by contact. They originate from the research in epidemiology which started to consider varying size populations, [5, 8, 12]. Since the constraint of a fixed population implicit in the classical epidemic models was then removed, the natural next step consisted in allowing a disease also among interacting populations, that therefore do not have constant values, see for instance [14]. Note that predator-prey interactions are not the only possible models, as also symbiosis and competition can be considered, [16, 4, 1]. For a more comprehensive account of ecoepidemic models in these contexts, see [11]. More specifically, here we consider the prey population, which grazes together in herds, similarly as to what done in [2, 3], but in which an epidemic occurs. In the environment are also present predators, who maintain an individualistic behavior. We differentiate the way prey wander about, assuming as stated that they stick together, when sound. However, the diseased animals are assumed to be left behind by the herd, or to abandon it voluntarily. This habit is common for instance among elephants. This difference in behavior entails also a different description of interactions with predators. In fact for the sick prey, the latter are on a one to one basis as in the classical models. Therefore, they are modeled via the standard mass action term.

The paper is organized as follows. We present the model in the next Section, then we analyse its equilibria. Their stability is studied in Section 4. We then present the results of numerical simulations, and a final discussion concludes the paper.

2 The model

We consider a "minimal" model for species interactions, subject also to a disease spreading in this case among the prey. This means that in the dynamical system describing the structure of the ecosystem, we account only for the bare fundamental relationships existing among the populations, in order to better elucidate the role of the "group defense" assumption we are making. In this way it will better be possible to study and understand its consequences on the ecosystem behavior.

Specifically, we ignore prey subpopulations intraspecific competition, assuming namely that only sound individuals would compete for resources, and we take the disease to be of type SI, i.e. there is no recovery from it, namely individuals, once infected, keep the disease for their life. We do not consider either more realistic descriptions of the feeding, like the Holling type II usually proposed nowadays in ecoepidemic literature. This because the Holling type II interaction is in general the key nonlinear element in a dynamical system that makes it oscillate. Its presence would then obscure the influence of, or intermingle with, the other square root nonlinearity we introduce.

Consider therefore the following system

$$\begin{aligned} \frac{d\widehat{R}}{d\tau} &= r\widehat{R}\left(1-\frac{\widehat{R}}{K}\right) - \widehat{\lambda}\widehat{R}\widehat{I} - \widehat{a}\sqrt{\widehat{R}}\widehat{F}, \\ \frac{d\widehat{I}}{d\tau} &= \widehat{I}\left(\widehat{\lambda}\widehat{R} - \widehat{b}\widehat{F} - \widehat{\mu}\right), \\ \frac{d\widehat{F}}{d\tau} &= \widehat{F}\left(\widehat{a}\widehat{e}\sqrt{\widehat{R}} + \widehat{b}\widehat{e}\widehat{I} - \widehat{m}\right). \end{aligned} \tag{1}$$

The fundamental feature that distinguishes it from classical predator-prey interactions, and also from earlier models describing population interactions subject to an epidemics spreading in the environment, is the last term of the first equation, which has a counterpart in the third equation as well. It corresponds to the fact that among populations that gather together to graze, it is the outermost individuals who bear the burden of suffering most from the attacks of possible predators from the outside. Hunting therefore occurs mostly on the perimeter of sound herd and it is modelled via this square root term. The remaining terms in the first equation describe logistic growth of the prey, in which as stated above only sound individuals contribute, and the infection mechanism, described via a simple mass action law. The second equation describes the infected evolution, who have an individualistic behavior, being isolated from the rest of their consimilar. Therefore, they are hunted as in the classical predator-prey models via a mass action term.

Infected prey are recruited into this class through successful contact between a susceptible and an infected, and they are subject to hunting by predators, as mentioned. Here $\widehat{\mu}$ represents natural plus disease-related mortality. The third equation contains the dynamics of the predators, who in absence of prey starve, with natural mortality $\widehat{m}$. The first two terms instead account for the reward obtained by hunting sound and infected prey respectively.

3 Some preliminary analysis

The system (1) has a singularity in the Jacobian, stemming from the square root term. In order to better analyze it, we use a singularity removal device. Namely, let $\widehat{P}^2 = \widehat{R}$, then assuming $\widehat{P} \neq 0$, we have

$$\begin{aligned} \frac{d\widehat{P}}{d\tau} &= \frac{r}{2}\widehat{P}\left(1 - \frac{\widehat{P}^2}{K}\right) - \widehat{a}\widehat{F} - \widehat{\lambda}\widehat{P}\widehat{I} \\ \frac{d\widehat{I}}{d\tau} &= \widehat{I}\left(\widehat{\lambda}\widehat{P}^2 - \widehat{b}\widehat{F} - \widehat{\mu}\right) \\ \frac{d\widehat{F}}{d\tau} &= \widehat{F}\left(\widehat{a}\widehat{e}\widehat{P} + \widehat{b}\widehat{e}\widehat{I} - \widehat{m}\right) \end{aligned} \tag{2}$$

We now proceed to the adimensionalization step. Using $P(t) = \alpha\widehat{P}(\tau)$, $I(t) = \beta\widehat{I}(\tau)$, $F(t) = \gamma\widehat{F}(\tau)$, $t = \delta\tau$ and then setting

$$\alpha^2 K = 1, \quad \frac{r}{2\delta} = 1, \quad \frac{\widehat{\lambda}}{\alpha^2\delta} = \frac{\widehat{\lambda}}{2\beta\delta}, \quad \frac{\widehat{b}}{\gamma\delta} = \frac{\widehat{b}}{\beta\delta} \tag{3}$$

we find

$$\alpha = \frac{1}{\sqrt{K}}, \quad \delta = \frac{r}{2}, \quad \beta = \gamma = \frac{1}{2K}.$$

We then get the rescaled system

$$\begin{aligned} \frac{dP}{dt} &= P\left(1 - P^2\right) - aF - \lambda PI \\ \frac{dI}{dt} &= I\left(\lambda P^2 - bF - \mu\right) \\ \frac{dF}{dt} &= F\left[e(aP + bI) - m\right] \end{aligned} \tag{4}$$

with

$$a = 2\frac{\widehat{a}}{r}\sqrt{K}, \quad \lambda = 2\widehat{\lambda}\frac{K}{r}, \quad b = 4\frac{\widehat{b}}{r}K, \quad \mu = \frac{2}{r}\widehat{\mu}, \quad m = \frac{2}{r}\widehat{m}.$$

At this point we need to look back at the adimensionalization used in [3]. Over there, it was possible to choose $a = 1$, a choice that we do not follow here. In order to be able to use the former results in our context for comparison purposes, we discuss briefly how the results of [3] change if we use a similar adimensionalization as above. In fact, the predator-prey model of [3] is obtained from (2) by setting $\widehat{\lambda} = 0$, $\widehat{b} = 0$, $\widehat{\mu} = 0$. In this way, using again the first two of (3) supplemented by

$$\frac{a\alpha}{2\gamma\delta} = \frac{a}{\alpha\delta}$$

we obtain once more the simplified model (4) in which $\lambda = 0$, $b = 0$, $\mu = 0$. At this point, however, in the former rescaling [3] a new fundamental parameter is introduced, $\rho = m/e$. In this context, however, its definition must be changed to

$$\rho = \frac{m}{ae}. \tag{5}$$

This change however, luckily, does not imply relevant changes in the subsequent analysis. Namely the results of [3] still hold for the simplified two-dimensional version of (4) with this modified definition of ρ. In summary, they are as follows: for $\rho > 1$ the system settles to the predator-free equilibrium, for $3^{-1/2} < \rho < 1$ the system shows coexistence of prey and predators at a stable equilibrium, for $0 < \rho < 3^{-1/2}$ predators and prey coexist, but through persistent oscillations. These results will be referred to in what follows.

3.1 Boundedness

The following argument is close to the one found in the literature, see for instance [9, 13]. Define then the quantity $T = \frac{1}{2}P^2 + I + F$. Differentiating and using (4), we have for an arbitrary $0 < \eta < \min\{m, \mu\}$

$$\frac{dT}{dt} + \eta T \leq \Phi(P), \quad \Phi(P) = P^2[1 + \eta - P^2].$$

The quartic attains the maximum value $M = \frac{1}{4}(1+\eta)^2$ at $P = \frac{1}{2}\sqrt{2(1+\eta)}$, so that from the differential inequality $\dot{T} + \eta T \leq M$ it follows $T(t) \leq M\eta^{-1} + \epsilon$ for any $t \geq 0$, with an arbitrary $\epsilon > 0$. This result establishes boundedness for each population of the system.

3.2 Equilibria

The equilibria are the origin E_0, the sound prey-only point $E_1 \equiv (1, 0, 0)$, the disease-free one $E_2 \equiv (P_2, 0, F_2)$, the predator-free one $E_3 \equiv (P_3, I_3, 0)$

and the coexistence one $E_4 \equiv (P_4, I_4, F_4)$. Explicitly, their components are

$$P_2 = \frac{m}{ae} = \rho, \quad F_2 = \frac{m}{a^2 e}\left(1 - \frac{m^2}{a^2 e^2}\right) = \frac{\rho}{a}\left(1 - \rho^2\right), \tag{6}$$

$$P_3 = \sqrt{\frac{\mu}{\lambda}}, \quad I_3 = \frac{1}{\lambda}\left(1 - \frac{\mu}{\lambda}\right), \tag{7}$$

with feasibility conditions for E_2 and E_3 are respectively

$$\mathcal{R}_p = \frac{ae}{m} = \frac{1}{\rho} \geq 1, \tag{8}$$

and

$$\mathcal{R}_0 = \frac{\lambda}{\mu} \geq 1. \tag{9}$$

For the coexistence equilibrium we have

$$F_4 = \frac{1}{b}\left(\lambda P_4^2 - \mu\right), \quad I_4 = \frac{1}{be}\left(m - aeP_4\right) = \frac{a}{b}\left(\rho - P_4\right)$$

and P_4 is a root of the cubic

$$\Psi(P) \equiv P^3 + \left(\frac{\lambda m}{be} - 1\right) P^2 - \frac{a}{b}\mu = 0.$$

Clearly from Descartes' rule it has one positive root, giving a feasible equilibrium, provided that

$$\frac{m}{ae} = \rho \geq P_4 \geq \sqrt{\frac{\mu}{\lambda}}. \tag{10}$$

It is also easily seen that differentiating, $\Psi'(P) = 0$ for the two values

$$\widetilde{P}_{\pm} = \pm\frac{1}{\sqrt{3}}\sqrt{\frac{be - \lambda m}{be}}$$

which exist for $be \geq \lambda m$. But in such case we also find $\Psi''(\widetilde{P}_+) > 0$ so that $\widetilde{P}_+$ is a minimum. Combining this result with $\Psi(0) < 0$, it is therefore apparent that the case of three positive roots cannot occur. $\Psi(P)$ admits always only one positive root, i.e. there is always one possible coexistence equilibrium E_4, feasible when the conditions (10) are satisfied. In term of the model parameters, (10) becomes explicitly

$$\rho^2\left(\frac{a\mu}{b\rho^3} - 1\right) = \frac{m^2}{a^2 e^2}\left(\frac{a^4 e^3 \mu}{bm^3} - 1\right) \leq \frac{\lambda m}{be} - 1 \leq \mu\left(\frac{a}{b}\sqrt{\frac{\lambda}{\mu}} - \frac{1}{\lambda}\right).$$

4 Stability

The Jacobian of the system 4 is

$$J = \begin{pmatrix} 1 - 3P^2 - \lambda I & -\lambda P & -a \\ 2\lambda IP & \lambda P^2 - bF - \mu & -bI \\ aeF & beF & -m + e(aP + bI) \end{pmatrix} \tag{11}$$

Evaluating it at E_0 we find the eigenvalues 1, $-m$, $-\mu$, so that the origin is unstable. At E_1 we find instead -2, $\lambda - \mu$, $ae - m$, for which we get conditional stability, namely for, recall the definitions (8) and (9),

$$\mathcal{R}_p < 1, \quad \mathcal{R}_0 < 1. \tag{12}$$

The former coupled with (8) shows that there is a transcritical bifurcation when E_2 becomes feasible collides with the stable equilibrium E_1, the latter losing then its stability. A similar result holds for E_3 and E_1, by using the second (12) together with (9). Let us now define the quantity

$$R^* = \max\left\{\mathcal{R}_p, \mathcal{R}_0\right\}. \tag{13}$$

It follows that if $R^* > 1$, in the system at least one of either the disease or the predators will establish itself. More specifically, if $\mathcal{R}_p > 1$ the predators could invade the environment, while for $\mathcal{R}_0$ it is the disease that can become endemic.

At E_2 one eigenvalue of (11) explicitly is $\lambda P_2^2 - bF_2 - \mu$ and the remaining two are the roots of a quadratic, coming from a 2 by 2 minor $\widetilde{J}$ of (11), for which the stability conditions, since $aeP_2 - m = 0$ here, require the positivity of the quantities

$$-\mathrm{tr}(\widetilde{J}) = 3P_2^2 - 1 > 0, \quad \det(\widetilde{J}) = a^2 eF_2 > 0.$$

Thus the latter is verified and the former gives

$$\rho > \frac{1}{\sqrt{3}}. \tag{14}$$

In addition to (14), stability of E_2 is thus regulated by the first eigenvalue. By performing some algebraic manipulations on the latter, we can now introduce the quantity

$$R_0^A = \max\left\{\frac{m^2(a^2e\lambda + bm)}{a^2e^2(a^2e\mu + bm)}, \frac{ae}{\sqrt{3}m}\right\} = \max\left\{\rho^2\frac{(a\lambda + b\rho)}{(a\mu + b\rho)}, \frac{1}{\sqrt{3}\rho}\right\}. \tag{15}$$

If $R_0^A > 1$, it follows that E_2 is unstable and provided also that $R^* > 1$, the disease becomes endemic in the ecosystem.

Note further that for the above condition on the trace (14) reducing to an equality, namely for

$$\rho = \frac{1}{\sqrt{3}}. \tag{16}$$

we have a supercritical Hopf bifurcation, since in such case two purely imaginary roots of the quadratic appear, giving purely imaginary eigenvalues. Therefore the equilibrium becomes unstable and a stable limit cycle appears around it.

At equilibrium E_3, one eigenvalue is $e(aP_3 + bI_3) - m$ and the other two again come from a suitable minor $\widehat{J}$ of (11) for which the Routh-Hurwitz conditions simplify again to give

$$-\mathrm{tr}(\widehat{J}) = 2\frac{\mu}{\lambda} > 0, \quad \det(\widehat{J}) = 2\lambda^2 P_3^2 I_3 > 0$$

and are therefore clearly satisfied. Thus stability is ensured by $e(aP_3+bI_3) < m$ or, explicitly,

$$\sqrt{\frac{\mu}{\lambda}} + \frac{b}{a\lambda}\left(1 - \frac{\mu}{\lambda}\right) < \rho. \tag{17}$$

Introducing the quantity

$$R_0^B = \frac{\lambda^2 m + be\mu}{\lambda e(a\sqrt{\lambda\mu} + b)} \tag{18}$$

stability can be recast in the form $R_0^B < 1$. If $R_0^B > 1$ and at the same time $R^* > 1$ it follows therefore that the predators invade the environment. Also, here no Hopf bifurcation can arise since $-\mathrm{tr}(\widehat{J}) > 0$ always.

We finally investigate the coexistence equilibrium E_4. In the Jacobian (11) the elements J_{22} and J_{33} now vanish. Letting $M_2(J(P_4))$ denote the sum of the principal minors of order 2 of (11), for which we have

$$M_2(J(P_4)) = b^2 e I_4 F_4 + a^2 e F_4 + 2\lambda^2 P_4^2 I_4,$$

the characteristic equation at E_4 can be written as the cubic

$$\sigma^3 - \mathrm{tr}(J(P_4))\sigma^2 + M_2(J(P_4))\sigma - \det(J(P_4)) = 0.$$

The Routh-Hurwitz conditions for stability give the three following inequalities

$$-\mathrm{tr}(J(P_4)) = 3P_4^2 + \lambda I_4 - 1 > 0 \tag{19}$$

$$-\det(J(P_4)) > 0 \tag{20}$$

$$\mathrm{tr}(J(P_4))M_2(J(P_4)) + \det(J(P_4)) > 0 \tag{21}$$

Studying these inequalities, for the equation associated with (19) we find the roots

$$P_4^{\pm} = \frac{\lambda a}{6b} \pm \frac{1}{6}\sqrt{\frac{\lambda^2 a^2}{b^2} - 12\left(\frac{\lambda m}{be} - 1\right)}$$

so that the solution interval for the inequality (19) for the two alternative cases $\lambda m > be$ and $\lambda m < be$ is respectively

$$P_4 \in (0, P_4^-) \cup (P_4^+, +\infty); \qquad P_4 \in (P_4^+, +\infty). \tag{22}$$

For (20) we are led to

$$0 > \det(J(P_4)) = beF_4 I_4 \left[b(1 - 3P_4^2 - \lambda I_4) - a\lambda P_4\right],$$

which leads to another quadratic inequality,

$$3beP_4^2 + \lambda m - be > 0.$$

This condition is automatically ensured if $be < \lambda m$. Else, the roots of the associated equation are

$$P_{4\pm} = \pm\sqrt{1 - \frac{\lambda m}{3be}}$$

and in such case (20) holds for $P > P_{4+}$.

The study of (21) is much more involved. The inequality involves a quintic polynomial in P_4, but it can be recast into the following form, involving respectively two rational functions,

$$L(P_4) < Q(P_4),$$

with

$$L(P_4) = be + \frac{a^2 e}{bI_4} + 2\frac{\lambda^2 P_4^2}{bF_4}, \quad Q(P_4) = \frac{3beP_4^2 + \lambda m - be}{1 - 3P_4^2 + \lambda I_4}.$$

Now L has two vertical asymptotes at $P_4^a = \rho$ and at $P_4^b = \sqrt{\frac{\lambda}{\mu}}$. Depending on their respective positions, the function can have different behaviors. When $P_4^a < P_4^b$, it raises from the positive value $L(0) = \frac{a^2}{m} + \frac{b}{e}$ to infinity, then in between the asymptotes it raises from $-\infty$ up to a maximum, which may or may not lie above the P_4 axis, and goes down to $-\infty$ again; beyond this second vertical asymptote, it raises then again from $-\infty$ up to the horizontal asymptote $\frac{b}{e} + 2\lambda$. For $P_4^a < P_4^b$ instead, it moves from $L(0) > 0$ to $-\infty$, the vertical asymptote P_4^b, then on its right it goes down from $+\infty$ to a minimum and then it raises up to $+\infty$ at the other asymptote P_4^a; on the right of this it raises up from $-\infty$ to the same horizontal asymptote.

Either one of these curves must be intersected with Q, which is another rational function tending to the horizontal asymptote at height $-be$ as $P_4 \to \infty$. Further, $Q(0) = \lambda m - be$, for which it can have both signs, and it has the vertical asymptotes at

$$\widehat{P}_4^+ = \frac{1}{6be}\left\{-ae\lambda + \left[a^2e^2\lambda^2 + 12be(be+\lambda m)\right]\right\}.$$

From the point at height $\frac{\lambda m - be}{\lambda m + be}$ on the vertical axis Q raises up to $+\infty$ at $\widehat{P}_4^+$, then it lies below the horizontal asymptote $-be$.

Seeking the solutions of the inequality $L < Q$ gives four fundamental cases, depending on whether the inequalities $be > \lambda m$ and $\rho = \frac{m}{ae} < \sqrt{\frac{\lambda}{\mu}}$ are satisfied or not. One has to account in each also the relative positions of the vertical asymptotes of the two rational functions. In several such situations other various possibilities arise, giving many different possible cases. In all of these cases, intervals in which (21) is satisfied can be shown to exist, generally near the vertical asymptotes. These must be combined with the solutions of (19) and (20) to give ranges for the position of P_4 for which the Routh-Hurwitz conditions hold, ensuring stability for the coexistence equilibrium. This result motivates the simulations of the next Section, showing that indeed this equilibrium can be attained.

Furthermore, Hopf bifurcations would arise when the cubic has two purely imaginary roots, a condition which is equivalent to having the inequality (21) become an equality, therefore this holds at the intersections of the two rational functions L and Q. The nature of the bifurcation is established from the sign of the remaining root of the cubic, given by the coefficient of the square term, namely $-\mathrm{tr}(J(P_4))$.

5 Simulations

Assume to have a value of ρ that exceeds 1. Figure 1 contains the simulation result giving equilibrium E_1 for the disease-free system. From the sound prey-only equilibrium, any positive disease incidence shifts it to the predator-free one, in which the disease is present, Fig. 2. This occurs provided that the disease incidence is larger than the disease-related mortality. If the disease-related mortality exceeds λ, then equilibrium E_3 becomes unstable and the system sets back to equilibrium E_1, Fig 3.

In these transitions the value of the hunting rate b on infected has some role. It influences the outcome of the system, since for small values of it, a small amount of predators establishes itself in the system, see Figure 4, so

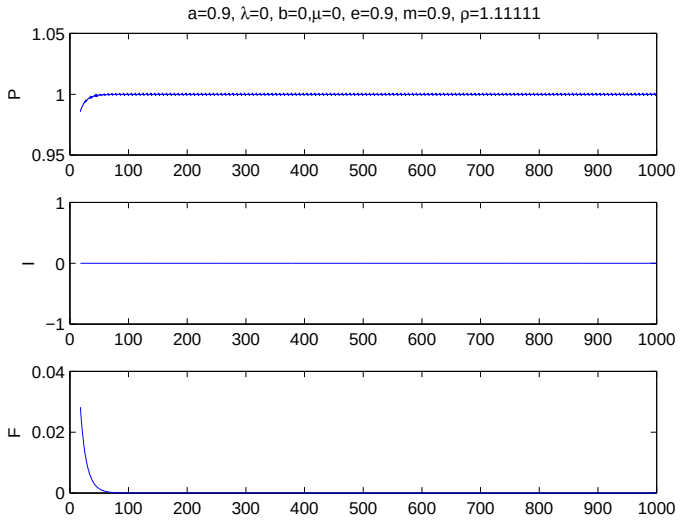

Figure 1: The prey-only equilibrium for the purely demographic model obtained for the parameter values $a = 0.9$, $\lambda = 0.0$, $b = 0.0$, $\mu = 0.0$, $e = 0.9$, $m = 0.9$ implying $\rho = 1.11$.

that the coexistence equilibrium of all subpopulations E_4 is achieved; in the same conditions, for a larger disease-related mortality instead, the system settles down to E_1 once more, even with a larger b, Figure 5. If we increase the hunting rate on infected further, the system once again settles to the prey-only equilibrium but this time achieving it with damped oscillations, Figure 6. An additional increase of b leads to sustained oscillations, Figure 7, which however in the long run dampen toward E_1, only more slowly. The larger the value of b, the slower the decay rate. In these situations there is an almost constant value for the sound prey, with the other populations almost at vanishing levels, followed by a sudden drop caused by an epidemic, which is in turn followed by an upsurge of predators.

All these dynamics are proper of the ecoepidemic model introduced here, as for the underlying demographic model with no epidemic, only equilibrium E_1 is possible in these circumstances, as $\lambda = \mu = b = 0$.

We now take $3^{-1/2} < \rho < 1$. In this case the equilibrium reached by the underlying purely demographic model is the predator-prey coexistence, corresponding to the disease-free one, E_2, in the three-dimensional model, for the parameter values $a = 1.2$, $\lambda = 0.08$, $b = 160.6$, $\mu = 0.3$, $e = 0.9$, $m = 0.9$ implying $\rho = 0.8333$, Figure 8. From this equilibrium, making $m = 1.9$ i.e. getting $\rho > 1$ we go back to E_1, as the theory prescribes, since (14), or what is the same, the second (15) is violated. This equilibrium is obtained

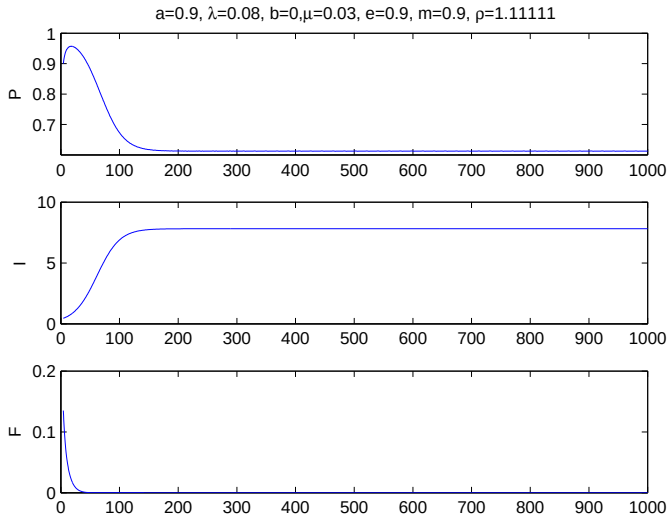

Figure 2: The predator-free equilibrium for the ecoepidemic model obtained for the parameter values $a = 0.9$, $\lambda = 0.08$, $b = 0.0$, $\mu = 0.03$, $e = 0.9$, $m = 0.9$ implying $\rho = 1.11$.

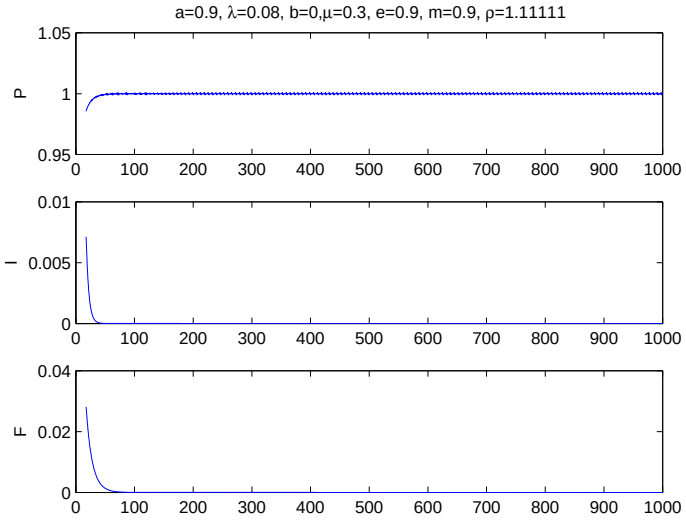

Figure 3: Again the prey-only equilibrium for the ecoepidemic model obtained for the parameter values $a = 0.9$, $\lambda = 0.08$, $b = 0.0$, $\mu = 0.3$, $e = 0.9$, $m = 0.9$ implying $\rho = 1.11$.

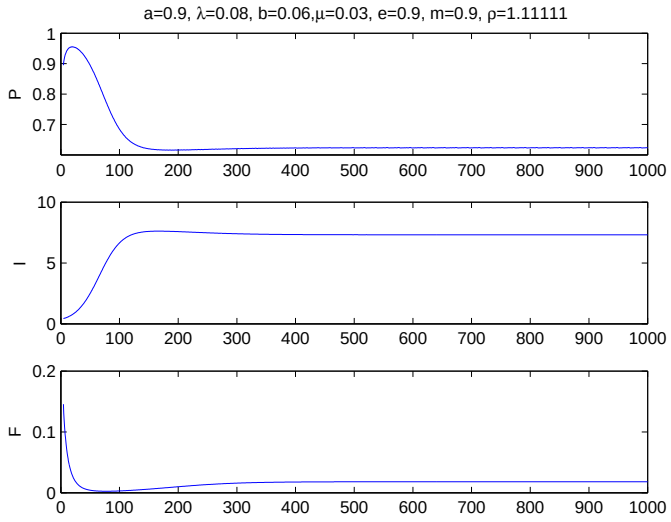

Figure 4: Coexistence equilibrium for the ecoepidemic model obtained for the parameter values $a = 0.9$, $\lambda = 0.08$, $b = 0.06$, $\mu = 0.03$, $e = 0.9$, $m = 0.9$ implying $\rho = 1.11$.

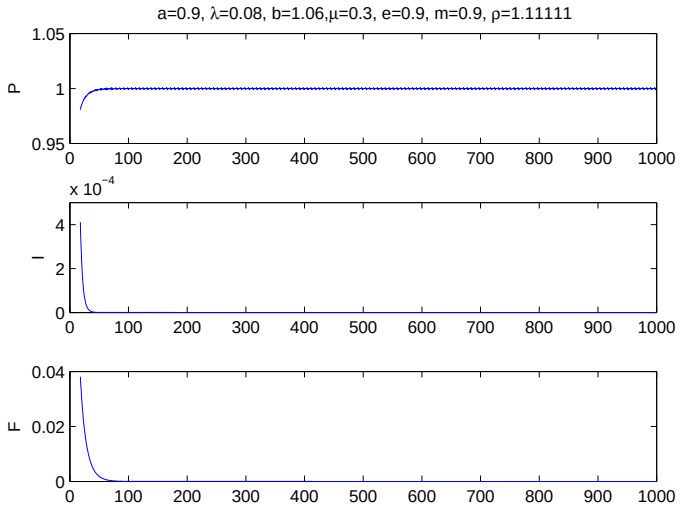

Figure 5: Once more the prey-only equilibrium for the ecoepidemic model obtained for the parameter values $a = 0.9$, $\lambda = 0.08$, $b = 1.06$, $\mu = 0.3$, $e = 0.9$, $m = 0.9$ implying $\rho = 1.11$.

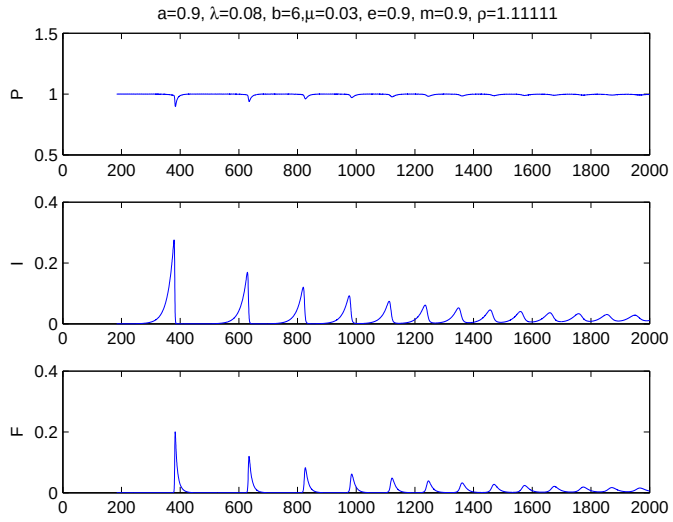

Figure 6: Once more the prey-only equilibrium for the ecoepidemic model obtained for the parameter values $a = 0.9$, $\lambda = 0.08$, $b = 6.0$, $\mu = 0.03$, $e = 0.9$, $m = 0.9$ implying $\rho = 1.11$.

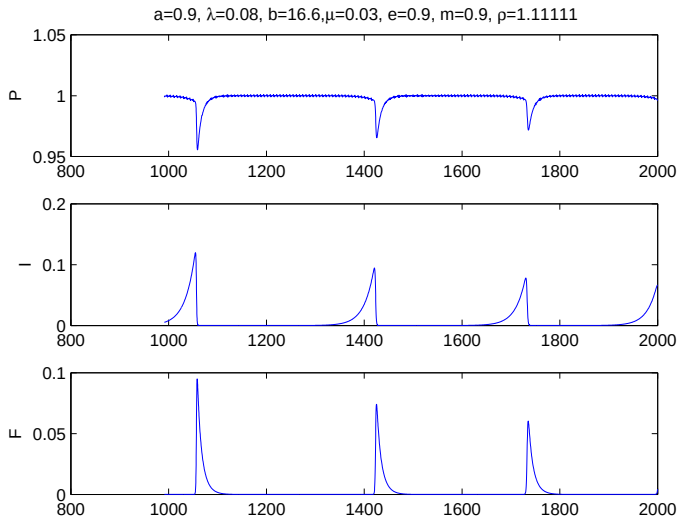

Figure 7: Dampened oscillations toward E_1 for the ecoepidemic model obtained for the parameter values $a = 0.9$, $\lambda = 0.08$, $b = 16.6$, $\mu = 0.03$, $e = 0.9$, $m = 0.9$ implying $\rho = 1.11$.

also for a one order of magnitude smaller value of $b = 16.6$. From it if we increase the disease incidence to $\lambda = 6.8$, so that the value of $\rho < 1$ remains unaltered, then E_2 is destabilized, but this time the system moves toward the coexistence equilibrium E_4. This occurs however with a very low value of infected, Figure 9. Here $P_4 = 0.7862$, $I_4 = 0.0031$, $F_4 = 0.2351$. From these values, we have performed a check on the system's behavior subject to changes in the parameters. Variations in λ both above and below the reference value correspond to slight changes in the equilibrium values, until $\lambda = 5$ or nearby values, at which point the infected drop to very low values and the system moves to the disease-free equilibrium E_2. At $\lambda = 10.9$ we have obtained very narrow limit cycles in the three dimensional space, Figure 10. They seem also to exist in a very narrow interval around this value of λ. Changes for higher values of b do not affect much the equilibrium. Values lower than the reference one, say around $b = 14$ lead to the invasion of the disease; in fact equilibrium E_4 is obtained, although with initially a low number of infected. Around $b = 6$ limit cycles involving all the populations appear, Figure 11. For much smaller values, around $b = 3.5$ for instance, the equilibrium once again moves back to the predator-free equilibrium E_3. Larger values of the disease-related mortality lead to lower values of infected, until about $\mu \approx 0.9$, after which essentially the disease gets eradicated. Lower values of μ still yield the coexistence equilibrium.

The parameters appearing in ρ are a, e and m. We found that a affects the outcome, in fact larger values, up to $a = 1.5$, lead to E_2, still with $\rho < 1$; at $a = 1.8$ we found limit cycles of the two dimensional healthy prey-predators demographic subsystem, Figure 12, but in this case the parameter ρ has dropped below the threshold, namely $\rho = 0.5556 < 0.5774$. Lower values give still E_4 but with higher infected levels, up to $a = 1$ at which point we have $\rho = 1$, and this trend continues past these values. Changes in e: higher values than the reference value lead to the disease-free equilibrium E_2, for lower values we have $\rho < 1$ only if $e \geq 0.75$. But in decreasing the conversion factor, the infected grow slightly, the sound prey and predators decrease, instead. Past $e = 0.75$, we are outside the range of ρ that we are exploring, but E_4 is still achieved, until around $e = 0.3$ at which point the system shifts to the predator-free equilibrium E_3. Although outside the range interest here, for $e = 0.39$ we have discovered very small limit cycles of the whole system, Figure 13. To show that these are not dampened oscillations, we ran the same simulation over a much larger time horizon, obtaining the results shown in Figure 14, with the same oscillation amplitudes. We now consider changes in m. Larger values lead to an increase in infected and a decrease in both sound prey and predators, up to around $m = 1.06$ at which point the value of ρ becomes larger than 1. A decrease of m leads to opposite

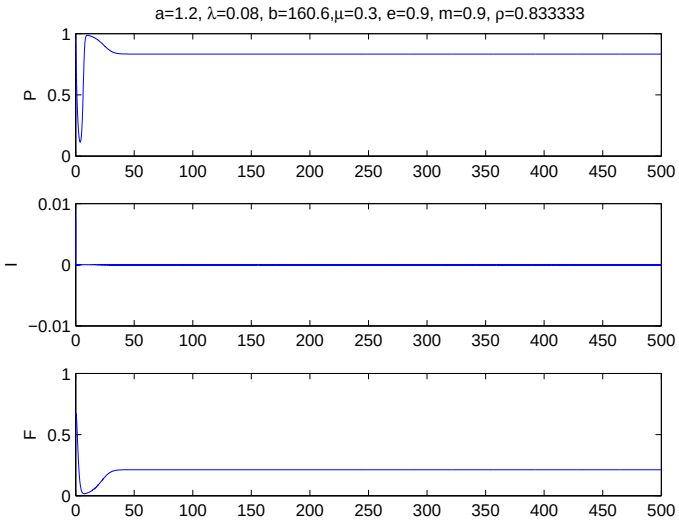

Figure 8: Disease-free equilibrium E_2 for the ecoepidemic model obtained for the parameter values $a = 1.2$, $\lambda = 0.08$, $b = 160.6$, $\mu = 0.3$, $e = 0.9$, $m = 0.9$ implying $\rho = 0.8333$.

changes and quickly to the disease-free equilibrium E_2. These results are to be expected, except perhaps the one for the reduction of healthy prey when m increases, but it can be explained by the fact that a reduction of predators puts less pressure also on infected and therefore the effects of the disease might become more relevant.

The initial values used in all the above simulations are $P = 0.10$, $I = 0.04$, $F = 0.03$ unless otherwise specified.

We consider now the case $0 < \rho < 3^{-1/2}$. At first we investigate the parameters that do not change ρ. Starting from the reference values $a = 1.8$, $\lambda = 6.8$, $b = 16.6$, $\mu = 0.3$, $e = 0.9$, $m = 0.9$ implying $\rho = 0.5556 < 3^{-1/2} \equiv 0.5774$ as noted above, we change again the parameter values. Changing λ does not sensibly change the dynamics of the system, it is not possible via a larger disease incidence that the disease invades the environment. Limit cycles remain essentially the same in the healthy prey-predator subspace. Values of $b < 3.3$ lead to the predator-free equilibrium E_3. Higher values of b as it should be expected do not lead to changes in the limit cycles, as the infected are hunted at higher rate and therefore are unable to invade the enviroment. In a similar way the system does not change under variations in μ.

The parameter values that influence ρ do not affect much the system's outcome, when preserving ρ below the critical threshold value $3^{-1/2} = 0.5774$.

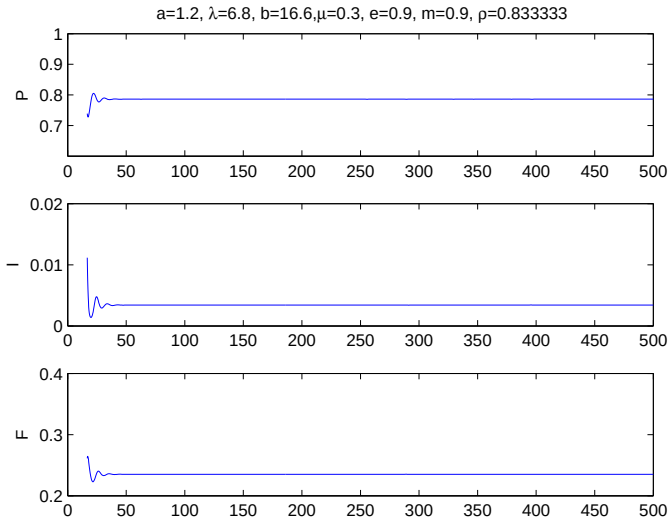

Figure 9: Coesistence equilibrium E_4 for the ecoepidemic model obtained from E_2 by a larger disease incidence with the parameter values $a = 1.2$, $\lambda = 6.8$, $b = 16.6$, $\mu = 0.3$, $e = 0.9$, $m = 0.9$ implying $\rho = 0.8333$.

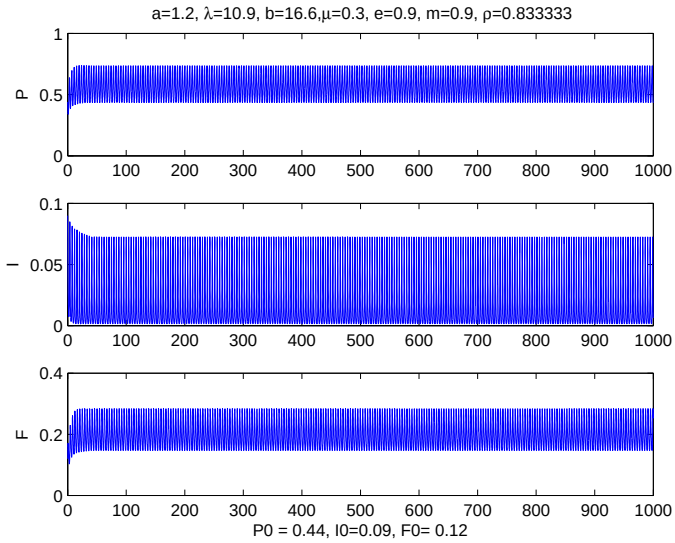

Figure 10: Limit cycles in the three dimensional space for the ecoepidemic model obtained for the parameter values $a = 1.2$, $\lambda = 10.9$, $b = 16.6$, $\mu = 0.3$, $e = 0.9$, $m = 0.9$ implying $\rho = 0.8333$.

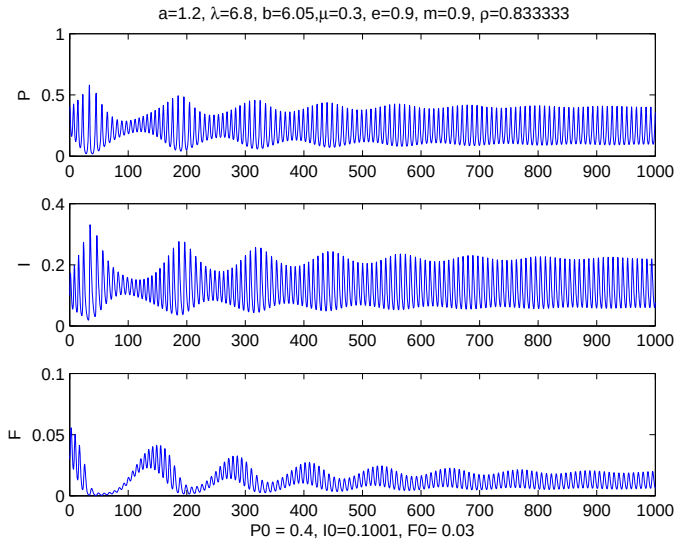

Figure 11: Limit cycles in the three dimensional space for the ecoepidemic model obtained for the parameter values $a = 1.2$, $\lambda = 6.8$, $b = 6.05$, $\mu = 0.3$, $e = 0.9$, $m = 0.9$ implying $\rho = 0.8333$.

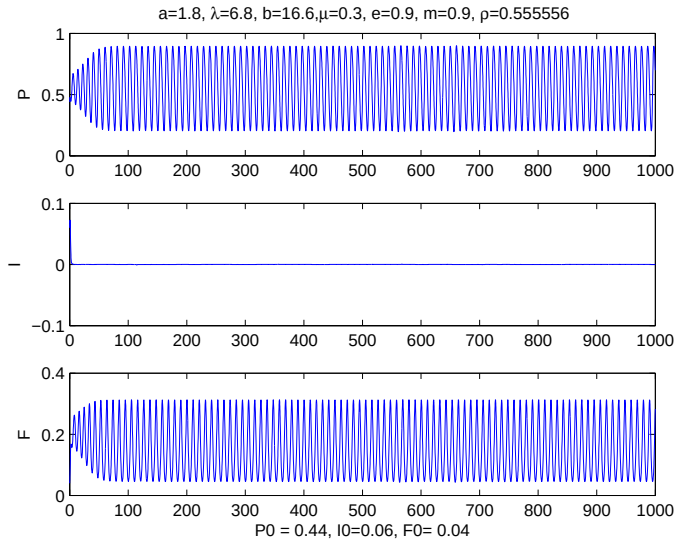

Figure 12: Limit cycles in the two dimensional space for the ecoepidemic model obtained for the parameter values $a = 1.8$, $\lambda = 6.8$, $b = 16.6$, $\mu = 0.3$, $e = 0.9$, $m = 0.9$ implying $\rho = 0.5556$.

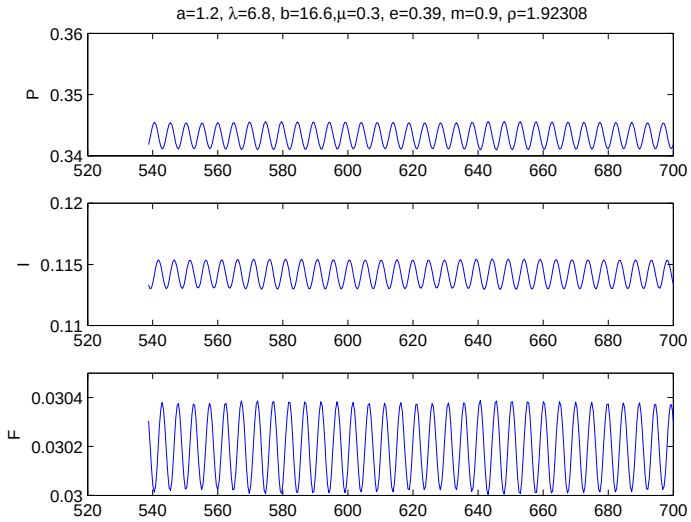

Figure 13: Small limit cycles around E_4 for the ecoepidemic model obtained with the parameter values $a = 1.2$, $\lambda = 6.8$, $b = 16.6$, $\mu = 0.3$, $e = 0.39$, $m = 0.9$ implying $\rho = 1.9231$.

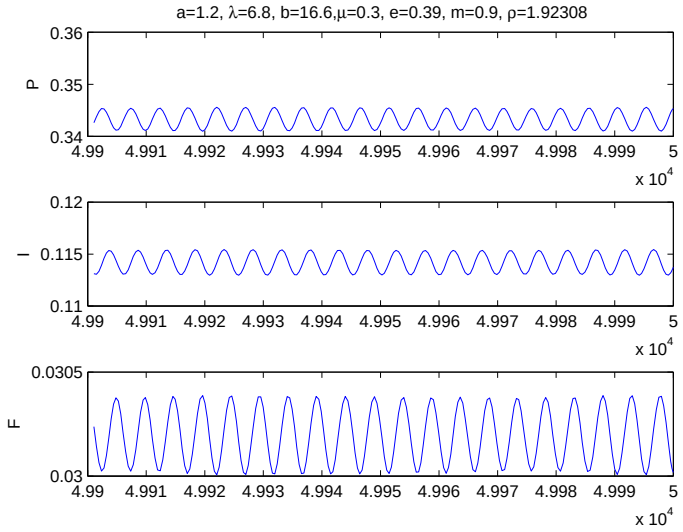

Figure 14: For the same parameter values of Figure 13, over a much wider timespan, the oscillation amplitudes are not changed.

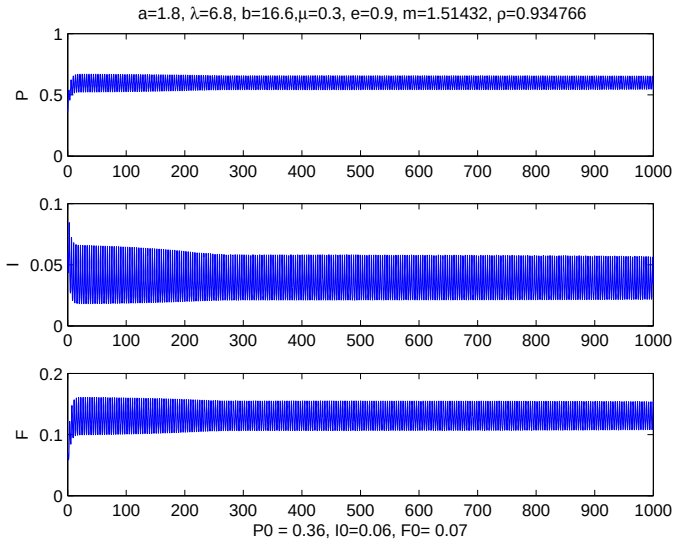

Figure 15: For the parameter values $a = 1.8$, $\lambda = 6.8$, $b = 16.6$, $\mu = 0.3$, $e = 0.9$, $m = 1.5143$ implying $\rho = 0.9348 > 0.5774$ limit cycles for all the system populations are found.

For higher values of m, namely $m = 1.5143$ leading to $\rho = 0.9348$, outside the range for ρ that we are considering here, we find limit cycles of the three populations, see Figure 15. Therefore the general conclusion in this case seems to be that for $\rho < 3^{-1/2}$ the two dimensional limit cycle of the demographic submodel attracts the system's trajectories.

6 Conclusion

The model presented here contains the new feature of prey group defense, introduced in [2] and [3]. It is here extended in the realm of ecoepidemic systems, to encompass also an infection spreading among the prey. The model has the following equilibria: the healthy prey-only equilibrium, stable if (12) holds; the disease-free point feasible for (8) and stable for (14); the predator-free equilibrium feasible for (9) and stable for (17); and the coexistence equilibrium. For the latter we have stated feasibility conditions, (19), (20), (21), tried to discuss them analytically for what is possible, but finally shown in the simulations that it can be attained. We have also identified quantities that determine when respectively the disease and the predators permanently remain in the system. The role of the key parameter ρ introduced in the two-dimensional purely demographic model, [2, 3] seems to bear

here a less important role. Only, for its values below the critical threshold $3^{-1/2}$, simulations show that the limit cycle in the healthy prey-predator domain is attractive also in the larger phase space in which infected prey are present.

With extensive simulations it is found that the disease effect correspond to a dampening of the system dynamics. This result bears some resemblance with the one of [6]. This occurs here in the assumption that infected individuals remain isolated and are therefore captured by predators according to the classical mass action interactions. It remains to be ascertained whether infected that remain in the heard instead of being left behind, voluntarily or expelled by the group, are able to provide a richer system behavior. This will constitute the next step in the investigation.

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