An incubating diseased-predator ecoepidemic model

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Abstract We present a model for transmissible diseases spreading among predators in a predator–prey system. Upon successful contact, a susceptible individual becomes infected but is not yet able to spread the disease further. After an incubation period, the diseased individual becomes infectious. We investigate the system’s equilibria by analytical and numerical means. For a suitable set of parameter values, the system shows persistent oscillations. The model also exhibits bistability of the coexistence equilibrium with the prey-only equilibrium.

Keywords Incubating diseases · Latency · Exposed · Ecoepidemiology · Ecosystems

AMS Mathematics Subject Classifications Primary 92D30; Secondary 92D25 · 92D40

1 Introduction

This paper describes and analyzes an ecoepidemic system, i.e., an environment where diseased interacting populations live. Ecoepidemiology is a fairly recent extension and merging of epidemiology and demographic studies (see [1] for an account of the first fifteen years of this topic). Epidemic models in the presence of an underlying demographic evolution had already been studied in the late 1980s and early 1990s [2, 3]. From these models, the study was extended to interacting populations [4–8]. Research in ecoepidemics has mostly dealt with systems in which prey are affected by a disease [9–13]. However, epidemics among predators have also been considered [6, 14].

Here we consider a system of the latter kind, namely predators affected by a disease and hunting a population of prey. The disease spreads by interaction between susceptible

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and infectious individuals. Different stages characterize the illness. At first, a “successful” contact between susceptible and infectious individuals leads to a new case of infection: the sound individual contracts the disease but cannot spread it further. This new recruit therefore belongs to a special class, namely the exposed individuals. The disease affects them mildly, impairing somewhat their hunting and reproductive capacities. Secondly, after an incubation period, the exposed predators become infectious, i.e., they migrate to the class of infectious individuals and the disease fully displays its debilitating effects. Recovery is allowed but it does not grant future immunity. Individuals who do not recover eventually die.

The subclass of predators is represented by an SEIS model, where as usual S stands for the susceptible class, E for those who can become exposed and I for the infectious population, who could finally recover and return to a susceptible state. There is no vertical transmission of the disease, i.e., newborns from sick parents are born sound.

This study represents a kind of extension and continuation of the investigation undertaken in [15]. In a former study, a predator–prey model with a disease spreading among the prey was considered. Former ecoepidemic models were limited to the case of SI, i.e., unrecoverable, or at most SIS, i.e., recoverable diseases where no incubation was taken into account. The main contribution of [15] consisted in considering for the first time ecoepidemic models with an incubation period. This paper is a further advancement in ecoepidemic research, where in a second step the SEI epidemic is assumed to spread among the predators and include also the possibility of recovery. In the context of epidemiology; see [16] for a review, the topic of incubation of a disease has classically been analyzed in [17–19]. The model introduced here also represents a generalization of the mass action model presented in [20], with the addition of an exposed class and allowing different functional responses for infected and susceptible predators.

The paper is organized as follows. After presenting the model in Section 2, we show boundedness for the system’s trajectories in Section 3. We study the boundary equilibria in Section 4 and the coexistence equilibria in Section 5, providing numerical simulations in all cases. In Section 6 we discuss the model in the absence of an exposed class. The final Section 7 contains a discussion of the results.

2 The model

Let $P = P(t)$ denote the healthy prey population and $S = S(t)$, $E = E(t)$ and $I = I(t)$ the healthy, exposed, i.e., infected but not yet infectious, and infectious predators, respectively. The model is

$$\begin{aligned}\frac{dP}{dt} &= rP \left(1 - \frac{P}{K}\right) - aSP - bEP - cIP, \\ \frac{dS}{dt} &= -mS + aePS + be(1 - \delta)EP + ce(1 - \theta)IP - \gamma SI + \rho I, \\ \frac{dE}{dt} &= \gamma SI - (m + \alpha)E, \\ \frac{dI}{dt} &= \alpha E - \mu I - mI - \rho I.\end{aligned}\tag{1}$$

The first equation models a logistic growth of the prey, with net reproduction rate r and carrying capacity K (first term); prey are also hunted by three kinds of predators at different rates, namely a , b , c for the classes S , E , I respectively (last three terms). Here we assume

that the disease affects, depending on the stage, the individuals differently according to its virulence.

The second equation contains the evolution of the sound predators. They are subject to natural mortality at rate m and they turn captured prey into newborns with conversion factor e . Since the disease is not vertically transmitted, all newborns from parents belonging to the two stages of the disease are born sound. We assume that the disease affects at least a bit of the reproductive capacity of the individuals, for which $\delta < 1$ and $\theta < 1$ represent the relative loss in fertility of the incubating and infected predators, respectively. Individuals leave the susceptible class upon becoming infected at rate γ and join it again upon disease recovery at rate ρ (last two terms).

The third equation describes the evolution of the exposed individuals. The first term is a source, accounting for the new infection arising by a successful contact between sound and infectious individuals. Exposed individuals are affected merely by natural mortality at rate m (second term), since at this stage we assume that the disease is not yet deadly. The same term also states that they migrate into the class of infectious individuals at rate α . Note that the larger the α , the shorter the incubation period.

The last equation refers to the dynamics of infectious predators. The only positive contribution to this class comes from the end of the incubation stage of exposed individuals, at rate α (first term). The last three terms contain the departures from this class. Infectious individuals may die, either because of the disease at rate μ , or for natural reasons at rate m , or else recover at rate ρ . Recovered predators migrate back to the class S of susceptibles, thereby not acquiring immunity from the disease.

All the model parameters are assumed to be positive.

For later use in the study of stability properties, we report here the Jacobian J of system (1),

$$J = \begin{bmatrix} J_{11} & -aP & -bP & -cP \\ J_{21} & -m + aeP - \gamma I & be(1 - \delta)P & ce(1 - \theta)P - \gamma S + \rho \\ 0 & \gamma I & -(m + \alpha) & \gamma S \\ 0 & 0 & \alpha & -(\mu + m + p) \end{bmatrix} \quad (2)$$

where

$$J_{11} = r \left(1 - \frac{2P}{K} \right) - aS - bE - cI,$$

$$J_{21} = aeS + be(1 - \delta)E + ce(1 - \theta)I.$$

3 Boundedness

Defining the total ecosystem population $\psi(t) := P(t) + S(t) + E(t) + I(t)$ and summing the equations in (1), we have

$$\begin{aligned} \frac{d\varphi}{dt} &= P \left[r \left(1 - \frac{P}{K} \right) - aS - bE - cI + aeS + be(1 - \delta)E + ce(1 - \theta)I \right] \\ &\quad - m(S + E + I) - \mu I \\ &= -m\varphi - \mu I \\ &\quad + P \left[r \left(1 - \frac{P}{K} \right) - aS(1 - e) - bE(1 - e + e\delta) - cI(1 - e + e\theta) + m \right]. \end{aligned}$$

Thus we obtain the following estimates

$$\begin{aligned} \frac{d\varphi}{dt} + m\varphi &\leq P \left[r \left(1 - \frac{P}{K} \right) + m - aS(1-e) - bE(1-e) - cI(1-e) \right] \\ &\leq P \left[r \left(1 - \frac{P}{K} \right) + m \right] \leq \frac{K}{4} (r+m)^2 \equiv \overline{M}, \end{aligned}$$

since by definition the conversion factor is a fraction, $e < 1$. The last estimate follows on taking the maximum of the parabola in P . Finally, from the theory of differential inequalities we have

$$\varphi(t) \leq e^{-mt} + \frac{\overline{M}}{m} (1 - e^{-mt}) \leq \frac{\overline{M}}{m} + 1 = M.$$

Since the total population is bounded, each subpopulation P, S, E, I is bounded as well for all future times.

Remark 1 Furthermore, since the first quadrant is an invariant domain, in view of the fact that (1) is a homogeneous system, so that the coordinate hyperplanes in the phase hyperspace cannot be crossed by the existence and uniqueness theorem, the solutions are always positive. This is a good result, since, together with the instability result of the origin below, it shows that the ecosystem is preserved.

4 Boundary equilibria

As a first equilibrium, we find the origin $E_1 = (0, 0, 0, 0)$, with Jacobian eigenvalues $r, -m, -(m + \alpha), -(\mu + m + \rho)$. E_1 is therefore unconditionally unstable.

Next, we find the equilibrium $E_2 = (K, 0, 0, 0)$, which is always feasible. The eigenvalues of $J(E_2)$ are $-r, -m + aeK, -(m + \alpha), -(m + \mu + \rho)$. Since they are all real, no Hopf bifurcations arise in this case. Three of the eigenvalues are always negative and the remaining one must be negative to ensure stability. This condition is then

$$R_0^P \equiv \frac{aeK}{m} < 1, \quad (3)$$

where we have used the same notation introduced in [21] to denote the predator's disease-free reproduction number. It expresses the same concept as in [21] since it gives the number of offspring aeK of a predator, while the prey are at carrying capacity K , during the mean predator's lifetime m^{-1} , in absence of an infected predator.

A numerical example of the system's evolution towards the equilibrium E_2 is obtained through the parameter values: $r = 6.3, K = 800, a = 0.01, b = 0.005, c = 0.001, e = 0.02, m = 0.2, \mu = 0.9, \alpha = 0.2, \delta = 0.5, \theta = 0.7, \gamma = 0.15, \rho = 0.1$.

In this case, the extinction of all classes of predators is due only to demographic reasons, as all parameters appearing in (3) are not disease-related. Indeed, similar results are obtained by also setting γ to very small values, showing that the cause of the predator's disappearance is really due to the demographic parameters only, i.e., the fact that the food income rate for sound predators is lower than their mortality rate. In this case, we have $aeK = 0.16 < m = 0.2$, thereby satisfying the stability condition (3) for E_2 . Figure 1 contains the simulation result with the initial conditions

$$P_0 = 500, S_0 = 70, E_0 = 50, I_0 = 30.$$

Fig. 1 Solution trajectories of (1) leading to E_2 for the parameter values $r = 6.3$, $K = 800$, $a = 0.01$, $b = 0.005$, $c = 0.001$, $e = 0.02$, $m = 0.2$, $\mu = 0.9$, $\alpha = 0.2$, $\delta = 0.5$, $\theta = 0.7$, $\gamma = 0.15$ and $\rho = 0.1$. Clockwise from the top left, we find the prey P , the healthy predators S , the diseased predators I , and the exposed predators E

We then have the equilibrium

$$E_3 = \left(\frac{m}{ae}, \frac{r}{a} \left(1 - \frac{m}{aeK} \right), 0, 0 \right) \quad (4)$$

feasible for

$$1 < R_0^P. \quad (5)$$

The meaning of the predator's disease-free reproduction number is here apparent, since only if the predators reproduce fast enough can they be present in the environment. There is a transcritical bifurcation in the system when $m = aeK$ since in such a case E_2 becomes unstable, while E_3 becomes feasible and the two equilibria coincide. At E_3 the Jacobian has a block structure

$$J|_{E_3} = \begin{bmatrix} A & B \\ 0 & D \end{bmatrix}$$

and the characteristic equation factors into the product of two quadratic equations. For the first one, condition (5) leads to

$$-\text{tr}(A) = \frac{rm}{aeK} > 0 \quad \det(A) = mr \left(1 - \frac{m}{aeK} \right) > 0,$$

for which the corresponding eigenvalues have a negative real part. For the second quadratic equation

$$\lambda^2 + (2m + \mu + \alpha + \rho)\lambda + (m + \alpha)(m + \mu + \rho) - \frac{\alpha\gamma r}{a} \left(1 - \frac{m}{aeK} \right) = 0 \quad (6)$$

we have

$$-\operatorname{tr}(D) = 2m + \mu + \alpha + \rho > 0 \quad \det(D) = (m + \alpha)(m + \mu + \rho) - \frac{\alpha\gamma r}{a} \left(1 - \frac{m}{aeK}\right).$$

The Routh–Hurwitz conditions for stability then reduce to

$$(m + \alpha)(m + \mu + \rho) > \frac{\alpha\gamma r}{a} \left(1 - \frac{m}{aeK}\right). \quad (7)$$

Solving quadratic equation (6) we obtain

$$\lambda_{1,2} = \frac{1}{2} \left\{ -(2m + \alpha + \mu + \rho) \pm \sqrt{\Delta_3} \right\},$$

where

$$\Delta_3 := (\mu + \rho - \alpha)^2 + 4 \frac{\alpha\gamma r}{a} \left(1 - \frac{m}{aeK}\right).$$

The discriminant Δ_3 is strictly positive due to the feasibility of E_3 , (5), and therefore both the eigenvalues associated with the matrix D are real. One is always negative and the sign of the other depends on the parameter values. Specifically, it is strictly negative if and only if (7) holds. The latter is thus necessary and sufficient to ensure the stability of E_3 . One can prove that such a condition is satisfied for $\alpha\mu > \gamma r$. Other suitable relations on the parameter values can be found that ensure (7) be satisfied. Here, we do not discuss this point further. No Hopf bifurcations arise at E_3 , since matrix A has a strictly negative trace, while the trace of D is strictly positive, i.e., the associated eigenvalues cannot be pure imaginary.

Note that following [21], introducing the epizootiological reproduction numbers for exposed and infected predators, respectively, given by

$$\mathcal{R}_0^E = \frac{\gamma}{m + \alpha}, \quad \mathcal{R}_0^I = \frac{\alpha}{m + \mu + \rho},$$

expressing the average number of recruitments into the classes E and I by a single exposed, respectively infected, individual during its residence times in these respective classes and letting $\mathcal{R}_0 = \mathcal{R}_0^E \mathcal{R}_0^I$, condition (7) can be restated as

$$\frac{r}{a} \mathcal{R}_0 \left(1 - \frac{1}{\mathcal{R}_0^p}\right) < 1.$$

It follows that the disease can certainly invade the predators if the above inequality is reversed. Interestingly, this condition can also be favored by a suitably high prey reproduction rate r or by their low removal rate a via healthy predator predation.

For the parameter values $r = 5.5$, $K = 800$, $a = 0.2$, $b = 0.08$, $c = 0.01$, $e = 0.02$, $m = 0.2$, $\mu = 0.9$, $\alpha = 0.2$, $\delta = 0.7$, $\theta = 0.95$, $\gamma = 0.03$, and $\rho = 0.1$, the system settles to the equilibrium E_3 . The disease is eradicated. We find specifically that $aeK = 3.2 > m = 0.2$ and $\mu a = 0.18 > \gamma r = 0.165$, so that $E_3 \approx (50, 26, 0, 0)$ is stable. Note that we take $b > c$, as we assume the incubating diseased predators to be less affected by the disease than the infectious individuals.

The simulation leading to this equilibrium is reported in Fig. 2, with the initial conditions

$$P_0 = 500, \quad S_0 = 70, \quad E_0 = 50, \quad I_0 = 30.$$

For the parameter set $r = 2$, $K = 800$, $a = 5$, $b = 4$, $c = 1$, $e = 0.02$, $m = 0.1$, $\mu = 0.5$, $\alpha = 0.2$, $\delta = 0.5$, $\theta = 0.7$, $\gamma = 0.1$ and $\rho = 0.1$ we have another simulation, in Fig. 3, leading to oscillations, which in spite of looking like limit cycles, in view of our theoretical

Fig. 2 Solution trajectories of (1) leading to E_3 with parameter values $r = 5.5$, $K = 800$, $a = 0.2$, $b = 0.08$, $c = 0.01$, $e = 0.02$, $m = 0.2$, $\mu = 0.9$, $\alpha = 0.2$, $\delta = 0.7$, $\theta = 0.95$, $\gamma = 0.03$ and $\rho = 0.1$. Clockwise from the top left, we find the prey P , the healthy predators S , the diseased predators I , and the exposed predators E

Fig. 3 Solution trajectories of (1) with parameter values $r = 2$, $K = 800$, $a = 5$, $b = 4$, $c = 1$, $e = 0.02$, $m = 0.1$, $\mu = 0.5$, $\alpha = 0.2$, $\delta = 0.5$, $\theta = 0.7$, $\gamma = 0.1$ and $\rho = 0.1$ showing, for large time, very slowly damped oscillations. This is due to the pair of conjugate eigenvalues $-0.0012 \pm 0.4469i$. Clockwise from the top left, we find the prey P , the healthy predators S , the diseased predators I , and the exposed predators E

analysis, are instead very slowly damped. In fact, for these parameter values, the Jacobian has the pair of conjugate eigenvalues $-0.0012 \pm 0.4469i$.

Remark 2 The prey-free point $\widehat{E}(0, \widehat{S}, \widehat{E}, \widehat{I})$ looks at first sight to be a possible equilibrium, but on deeper analysis, it turns out to be infeasible. This is to be expected because predators cannot survive in absence of the prey, the latter being indeed their only food source.

5 Coexistence equilibrium

The equilibrium configuration of system (1) can be explicitly solved, in the following order, to find the coexistence equilibrium $E_4 = (P^*, S^*, E^*, I^*)$.

Start from the fourth equation, solving for E , then substitute into the third equation to find S^* and finally use these expressions in the second equation to obtain an expression of I . Explicitly,

$$E^* = \frac{m + \mu + \rho}{\alpha} I^*, \quad S^* = \frac{(m + \mu + \rho)(m + \alpha)}{\gamma \alpha}, \quad I^* = \frac{r \left(1 - \frac{P^*}{K}\right) - a S^*}{b(m + \mu + \rho) + c \alpha} \alpha. \quad (8)$$

Note that the value of I^* and therefore also the value of E^* depends on P^* . The latter is found by back substitution of the above quantities into the first equilibrium equation. We obtain the following quadratic

$$AP^2 + BP + C = 0, \quad (9)$$

with

$$\begin{aligned} A &= \frac{-be(1 - \delta)(\mu + m + \rho)r - ce(1 - \theta)r\alpha}{(b(\mu + m + \rho) + c\alpha)K} < 0, \\ B &= aeS^* + \frac{Ke(r - aS^*)[b(1 - \delta)(\mu + m + \rho) + c\alpha(1 - \theta)] + (\gamma S^* - \rho)r\alpha}{K[b(\mu + m + \rho) + c\alpha]}, \\ C &= -mS^* + \frac{(\rho - \gamma S^*)(r - aS^*)\alpha}{b(\mu + m + \rho) + c\alpha}, \end{aligned}$$

whose roots determine the values of P^* . The values of the remaining components of E_4 , E^* and I^* can then be found via back substitution into (8). We can thus find up to two coexistence equilibria, corresponding to the roots P_1 and P_2 of (9). To have real roots, the feasibility condition

$$\Delta := B^2 - 4AC \geq 0 \quad (10)$$

must be satisfied. To ensure that both roots are positive, we have to impose the additional condition

$$\sqrt{B^2 - 4AC} - B < 0,$$

implying that B must be non-negative and also $C < 0$, in view of the sign of A . Again, the parameter space can be explored to find suitable relations ensuring that these conditions hold. We find for instance that if

$$\frac{\rho}{\gamma} < S^* < \frac{r}{\alpha}, \quad (11)$$

then $B > 0$ and $C < 0$. The following conditions must all hold to ensure that the infectious population I is non-negative for both values of P :

$$r - \alpha S^* - \frac{r - B - \sqrt{B^2 - 4AC}}{K} > 0, \quad B > 0, \quad C < 0 \quad (12)$$

Conversely, $C > 0$ implies that exactly one positive P^* root always exists.

Solving the system analytically is impossible. Investigating these conditions numerically, we do not find a parameter set such that the two equilibria are feasible and stable at the same time.

For the parameter values $r = 10$, $K = 800$, $a = 0.1$, $b = 0.08$, $c = 0.01$, $e = 0.02$, $m = 0.1$, $\mu = 0.5$, $\alpha = 0.2$, $\delta = 0.5$, $\theta = 0.7$, $\gamma = 0.1$ and $\rho = 0.1$ and the initial conditions

$$P_0 = 500, \quad S_0 = 70, \quad E_0 = 50, \quad I_0 = 30,$$

system (1) evolves toward a coexistence equilibrium, as shown in Fig. 4.

Condition (10) is satisfied in this case, since $\Delta \approx 7 \cdot 10^{-3}$. The two coexistence equilibria are

$$E_4 \approx (256, 9, 61, 20), \quad E_5 \approx (963, 9, -30, -10),$$

the second one being infeasible. Note that the condition $10 = r > aS^* = 0.9$ holds in this case: there are two coinciding roots $P = 9$ of the quadratic (9). However, the ultimate feasibility of this equilibrium also depends on the non-negativity of its remaining components, which in this case for E_5 does not hold, since conditions (12) are not all satisfied. E_4 is

Fig. 4 The model (1) evolves toward coexistence of all populations with parameter values $r = 10$, $K = 800$, $a = 0.1$, $b = 0.08$, $c = 0.01$, $e = 0.02$, $m = 0.1$, $\mu = 0.5$, $\alpha = 0.2$, $\delta = 0.5$, $\theta = 0.7$, $\gamma = 0.1$ and $\rho = 0.1$. Clockwise from the top left, we find the prey P , the healthy predators S , the diseased predators I , and the exposed predators E

Fig. 5 Persistent oscillations of all the populations obtained for the parameter values $r = 1$, $K = 800$, $a = 0.1$, $b = 3.8$, $c = 12.91$, $e = 0.2$, $m = 0.1$, $\mu = 0.8$, $\alpha = 0.2$, $\delta = 0.99$, $\theta = 0.99$, $\gamma = 1.5$ and $\rho = 5.001$. Top to bottom P , S , E , I

stable, with two negative eigenvalues and two complex eigenvalues having a negative real part, thus giving populations exhibiting damped oscillations.

Further, for the following parameter values $r = 1$, $K = 800$, $a = 0.1$, $b = 3.8$, $c = 12.91$, $e = 0.2$, $m = 0.1$, $\mu = 0.8$, $\alpha = 0.2$, $\delta = 0.99$, $\theta = 0.99$, $\gamma = 1.5$ and $\rho = 5.001$, we find what appear to be persistent oscillations of all the populations. We have followed them for very long times and they do not appear to decay; see Fig. 5 for a plot of a section of the time span reaching time 8×10^5 .

6 The system without the exposed class

The basic ecoepidemic system presented here, apart from the introduction of the exposed class, differs from an earlier model in the literature in that here the prey are taken to be the only food source for the predators, while for the latter other resources were assumed to be available in [14]. Therefore, a direct comparison is not possible. We however consider here briefly the special case of the ecoepidemic model (1) without the exposed class. Keeping the same earlier notation, the system becomes

$$\begin{aligned} \frac{dP}{dt} &= rP \left(1 - \frac{P}{K} \right) - aSP - cIP, \\ \frac{dS}{dt} &= -mS + aePS + ce(1 - \theta)IP - \gamma SI + \rho I, \\ \frac{dI}{dt} &= \gamma SI - \mu I - mI - I, \end{aligned} \quad (13)$$

with Jacobian

$$\hat{J} = \begin{bmatrix} r \left(1 - \frac{2P}{K}\right) - aS - cI & -aP & -cP \\ aeS + ce(1-\theta)I & -m + aeP - \gamma I & ce(1-\theta)P - \gamma S + \rho \\ 0 & \gamma I & \gamma S - (\mu + m + \rho) \end{bmatrix}. \quad (14)$$

Retaining the same numbering as for the original system (1), the equilibria here are again the origin, unstable in view of the eigenvalue $r > 0$, the prey-only equilibrium $\hat{E}_2 = (K, 0, 0)$, the disease-free equilibrium $\hat{E}_3 = (\hat{P}_3, \hat{S}_3, 0)$ and coexistence $\hat{E}_4 = (\hat{P}_4, \hat{S}_4, \hat{I}_4)$. Once again, note that the prey-free equilibrium is infeasible, because the prey are the only food for predators, as is the sound predator-free point, which gives a negative infected population. Now, the disease-free equilibrium $\hat{E}_3$ coincides with the one formerly found, E_3 . This is also not a big surprise, since even in case of model (1) this is a demographic equilibrium and therefore it must be the same both in (1) as well as in (13), because the demographic underlying structure is the same in both systems. Feasibility is thus given once again by (5). Stability in this case is slightly different from (7). This is evident as (7) contains the parameter α that is not present in (13). The stability condition for $\hat{E}_3$ hinges on one eigenvalue only, since the other two always have a negative real part in view of the Routh–Hurwitz conditions for a quadratic, and becomes

$$\gamma \frac{r}{a} \left(1 - \frac{m}{aeK}\right) < \mu + m + \rho. \quad (15)$$

However, it is easily seen that as $\alpha \rightarrow +\infty$, condition (7) tends to (15). On the other hand, if $\alpha \rightarrow 0^+$, the stability condition (7) is always verified.

Note that introducing the epizootiological reproduction number [21] for this model as

$$\mathcal{R}_0^* = \frac{\gamma}{m + \mu + \rho},$$

the condition (15) can be restated as

$$\frac{r}{a} \mathcal{R}_0^* \left(1 - \frac{1}{R_0^p}\right) < 1.$$

Here too, as for the model with latency, a high reproducing prey may favor the endemicity of the disease in the predator population.

Further, $\hat{E}_2$ is the very same point E_2 , apart from the dimension of the phase space, with the very same stability condition (3). Once again, this result is to be ascribed to demographic reasons only. Therefore, the prey-only equilibrium is not affected by the presence or absence of the incubating class among predators.

The coexistence equilibrium easily has two components as follows

$$\hat{S}_4 = \frac{\mu + m + \rho}{\gamma}, \quad \hat{I}_4 = \frac{r}{c} \left(1 - \frac{P}{K}\right) - a \frac{\mu + m + \rho}{\gamma c};$$

note that as $\alpha \rightarrow +\infty$, $S^* \rightarrow \hat{S}_4$. For feasibility, we need

$$\frac{r}{K} P_4 \leq r - a \frac{\mu + m + \rho}{\gamma}. \quad (16)$$

The prey are found as roots of the quadratic (9) with new coefficients

$$\begin{aligned}\widehat{A} &= -\frac{r}{K}e(1-\theta), \quad \widehat{C} = -m\frac{\mu+m+\rho}{\gamma} - \frac{1}{c}(m+\mu)\left(r - a\frac{\mu+m+\rho}{\gamma}\right), \\ \widehat{B} &= ae\frac{\mu+m+\rho}{\gamma} + (1-\theta)e\left(r - a\frac{\mu+m+\rho}{\gamma}\right) + \frac{r}{cK}(m+\mu).\end{aligned}$$

Note that as $\alpha \rightarrow +\infty$, $A \rightarrow \widehat{A}$, $B \rightarrow \widehat{B}$ and $C \rightarrow \widehat{C}$. Further, $\widehat{A} < 0$ and $\widehat{C} < 0$ in view of (16). Hence, the same discussion for the existence of the roots of this quadratic as in Section 5 follows unchanged.

Finally, the Routh–Hurwitz stability conditions

$$-\operatorname{tr}(\widehat{J}_4) > 0, \quad -\det(\widehat{J}_4) > 0, \quad -\operatorname{tr}(\widehat{J}_4)M_4 > -\det(\widehat{J}_4) \quad (17)$$

for the Jacobian $\widehat{J}_4 \equiv \widehat{J}(\widehat{E}_4)$ evaluated at $\widehat{E}_4$ can be written down explicitly, observing that

$$\begin{aligned}-\operatorname{tr}(\widehat{J}_4) &= \frac{r}{K}\widehat{P}_4 + m - ae\widehat{P}_4 + \gamma\widehat{I}_4, \\ M_4 &= a\widehat{P}_4[ae\widehat{S}_4 + ce(1-\theta)\widehat{I}_4] - \frac{r}{K}\widehat{P}_4[ae\widehat{P}_4 - \gamma\widehat{I}_4 - m] \\ &\quad - \gamma\widehat{I}_4[ce(1-\theta)\widehat{P}_4 - \gamma\widehat{S}_4 + \rho], \\ \det(\widehat{J}_4) &= \gamma\widehat{I}_4\widehat{P}_4\left[ce(a\widehat{S}_4 + c(1-\theta)\widehat{I}_4) - \frac{r}{K}(ce(1-\theta)\widehat{P}_4 - \gamma\widehat{S}_4 + \rho)\right].\end{aligned}$$

7 Discussion

In this paper we have described a model for the evolution of a predator–prey system in which a disease spreads among the predators. The model includes a class of exposed predators, which are mildly affected by the disease and momentarily unable to spread it further. After an incubation period, an exposed individual migrates to the class of infectious predators; its hunting and reproductive abilities are severely impaired. In this condition, it contributes to spreading the disease by further contacts with sound predators. The model includes recovery from the disease. Since recovery does not imply immunity, the system is classified as P-S-E-I-S.

The system can evolve only toward the predator-free equilibrium E_2 , the disease-free equilibrium E_3 , given by (4), the coexistence equilibrium E_4 given by (8) and the solutions of (9). We studied the stability of these equilibria by analytical and numerical means. In particular, we provided conditions for E_2 and E_3 to be stable, (3) and (7), respectively.

It does not seem possible for the disease alone to eradicate the predators. In fact, the predator-free equilibrium E_2 is stable when condition (3) holds, but in this condition no disease-related parameters appear. Hence, the disappearance of predators may be due only to demographic reasons.

Coexistence has been shown to be possible in the case of disease affecting the prey [15], as well as here. In [15], this equilibrium can bifurcate and lead to persistent limit cycles. We have discovered a kind of similar behavior here as well. Hopf bifurcations were also found in the simpler model [20]. Thus the sustained oscillations in the current manuscript are not due to the exposed class, the addition of latency being not responsible for this kind of dynamics. Rather, they perhaps could be ascribed to the four-dimensional structure of the model.

The comparison with a similar model where the disease affects the prey [15] reveals that similar types of equilibria exist in the two cases. Under the stated assumptions, both systems cannot disappear, since the origin is always unstable. In both cases, the boundary equilibria corresponding to the prey-only and the disease-free model are also present. However, in the simplest case of [15], persistent oscillations may appear. They can be attributed to the underlying demographic structure, which is a pure Lotka–Volterra system and thereby shows center oscillations. In fact, limit cycles have been shown to arise in various situations of the several models introduced in [15]. Here, on the contrary, we have an underlying demographic structure given by a predator–prey model with a logistic evolution of the prey. This prevents center oscillations from appearing and it is further known that the quadratic interaction terms do not lead to any type of persistent oscillations. In fact, no such cycles

Fig. 6 E_2 top and E_4 bottom, for the parameter values $r = 10$, $K = 40$, $a = 0.1$, $b = 0.8$, $c = 1.01$, $e = 0.02$, $m = 0.1$, $\mu = 0.05$, $\alpha = 0.2$, $\delta = 0.5$, $\theta = 0.7$, $\gamma = 0.1$ and $\rho = 0.1$ and initial conditions $P(0) = 40.2$, $S(0) = 0.7$, $E(0) = 0.5$, $I(0) = 0.3$ for E_2 and $P(0) = 500$, $S(0) = 70$, $E(0) = 50$, $I(0) = 30$ for E_4 . The latter are not completely evident from the graphs, since their initial portions have been cut out to have a picture with a vertical scale that better shows the equilibrium values reached by the simulations

are found near the boundary equilibria in this model as well, but, as mentioned, all the populations can show persistent oscillations.

Comparing instead the model with incubation to the ecoepidemic system without it, while the equilibria of the former tend to those of the latter when the rate out of the incubation class α becomes very large and therefore also the feasibility and stability conditions tend in the limit to coincide, we also found out that the prey-only equilibrium is not influenced by the presence of the incubating class among the predators. This is in a certain sense to be expected, because in the absence of all predator types at equilibrium, whether the model includes this class or not becomes an immaterial issue. The limit $\alpha \rightarrow +\infty$ corresponds to a very large speed out of class E . If we count populations by the number of their individuals, the rate becomes a frequency and its reciprocal then denotes the residence time in the class. Thus the result has an easy interpretation. In the limit individuals tend not to stay in class E but just to transit it on their way to class I , i.e., in the limit the class E can be deleted from the model without any harm.

Note also that in the limit $\alpha \rightarrow 0^+$ the third and fourth system's equations (1) imply $I = 0$ and $E = 0$. Therefore, only equilibria E_2 and E_3 are possible. Further, (7) becomes trivially satisfied, implying that E_3 whenever feasible is always stable. The transcritical bifurcation involving E_2 and E_3 is still present. This situation can be interpreted as saying that when the residence time in the exposed class becomes infinite, the exposed individuals will no longer migrate into the infected class. Thus, no infected individuals will eventually survive and so ultimately also no exposed individuals will be there, since the source of contagion will become exhausted. Hence, the only possible equilibria in the ecosystem are the demographic ones, involving prey only and coexistence among prey and healthy predators. The two coalesce when (3) or (5) become equalities.

Bistability of E_2 and E_4 is shown in Fig. 6 for the parameter values $r = 10$, $K = 40$, $a = 0.1$, $b = 0.8$, $c = 1.01$, $e = 0.02$, $m = 0.1$, $\mu = 0.05$, $\alpha = 0.2$, $\delta = 0.5$, $\theta = 0.7$, $\gamma = 0.1$ and $\rho = 0.1$ with the respective initial conditions $P(0) = 40.2$, $S(0) = 0.7$, $E(0) = 0.5$, $I(0) = 0.3$ and $P(0) = 500$, $S(0) = 70$, $E(0) = 50$, $I(0) = 30$. Multistability has recently been investigated in another similar context in [22].

Fig. 7 Bifurcation diagram showing only non-trivial states: *dashed* - unstable; *solid* - stable. The number of infectious predators are plotted as a function of the transmission parameter γ , for the parameter values $r = 10$, $K = 40$, $a = 0.1$, $b = 0.8$, $c = 1.01$, $e = 0.02$, $m = 0.1$, $\mu = 0.05$, $\alpha = 0.2$, $\delta = 0.5$, $\theta = 0.7$ and $\rho = 0.1$

On the other hand, we have tried to follow the transcritical bifurcation of E_2 leading to the two-species disease-free equilibrium E_4 , at the same time monitoring what happened to the coexistence equilibrium E_4 and found that as soon as E_3 becomes feasible and stable, E_4 coalesces with it. In other words, we do not have examples of bistability involving both E_3 and E_4 .

Running a numerical continuation analysis, Fig. 7 shows a saddle-node bifurcation actually giving rise to two non-trivial stationary states. The number of infectious predators are plotted versus the disease transmission parameter γ .

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References

1. Malchow, H., Petrovskii, S., Venturino, E.: *Spatiotemporal Patterns in Ecology and Epidemiology*. CRC, Boca Raton, Florida (2008)
2. Gao, L.Q., Hethcote, H.W.: Disease transmission models with density-dependent demographics. *J. Math. Biol.* **30**, 717–731 (1992)
3. Mena-Lorca, J., Hethcote, H.W.: Dynamic models of infectious diseases as regulator of population sizes. *J. Math. Biol.* **30**, 693–716 (1992)
4. Beltrami, E., Carroll, T.O.: Modelling the role of viral disease in recurrent phytoplankton blooms. *J. Math. Biol.* **32**, 857–863 (1994)
5. Hader, K.P., Freedman, H.I.: Predator–prey populations with parasitic infection. *J. Math. Biol.* **27**, 609–631 (1989)
6. Venturino, E.: The influence of diseases on Lotka–Volterra systems. IMA Preprint Series #913, IMA, Minneapolis, USA (1992)
7. Venturino, E.: The influence of diseases on Lotka–Volterra systems. *Rocky Mt. J. Math.* **24**, 381–402 (1994)
8. Venturino, E.: Epidemics in predator–prey models: disease among the prey. In: Arino, O., Axelrod, D., Kimmel, M., Langlais, M. (eds.) *Mathematical Population Dynamics: Analysis of Heterogeneity*, vol. 1: *Theory of Epidemics*, pp. 381–393. Wurtz Publishing Ltd, Winnipeg, Canada (1995)
9. Chattopadhyay, J., Arino, O.: A predator–prey model with disease in the prey. *Nonlinear Anal.* **36**, 747–766 (1999)
10. Arino, O., El Abdllaoui, A., Mikram, J., Chattopadhyay, J.: Infection on prey population may act as a biological control in ratio-dependent predator–prey model. *Nonlinearity* **17**, 1101–1116 (2004)
11. Delgado, M., Molina-Becerra, M., Suarez, A.: Relating disease and predation: equilibria of an epidemic model. *Math. Methods Appl. Sci.* **28**, 349–362 (2005)
12. Han, L., Ma, Z., Hethcote, H.W.: Four predator–prey models with infectious diseases. *Math. Comput. Model.* **30**, 849–858 (2001)
13. Haque, M., Venturino, E.: The role of transmissible diseases in Holling–Tanner predator–prey model. *Theor. Popul. Biol.* **70**, 273–288 (2006)
14. Venturino, E.: Epidemics in predator–prey models: disease in the predators. *IMA J. Math. Appl. Med. Biol.* **19**, 185–205 (2002)
15. Venturino, E.: Ecoepidemic models with disease incubation and selective hunting. *J. Comput. Appl. Math.* **234**, 2883–2901 (2010)
16. Hethcote, H.W.: The mathematics of infectious diseases. *SIAM Rev.* **42**, 599–653 (2000)
17. Pugliese, A.: An $S \rightarrow E \rightarrow I$ epidemic model with varying population size. In: Busenberg, S., Martelli, M. (eds.) *Differential Equations Models in Biology, Epidemiology and Ecology*, pp. 121–138. Springer, Berlin (1991)
18. Gao, L.Q., Mena-Lorca, J., Hethcote, H.W.: Four SEI endemic models with periodicity and separatrices. *Math. Biosci.* **128**, 157–184 (1995)
19. Gao, L.Q., Mena-Lorca, J., Hethcote, H.W.: Variations on a theme of SEI endemic models. In: Martelli, M., Cooke, C.L., Cumberbatch, E., Tang, B., Thieme, H.R. (eds.) *Differential Equations and Applications to Biology and Industry*, pp. 191–207. World Scientific, Singapore (1996)

20. Oliveira, N., Hilker, F.: Modelling disease introduction as biological control of invasive predators to preserve endangered prey. *Bull. Math. Biol.* **72**, 444–468 (2010)
21. Hilker, F., Schmitz, K.: Disease-induced stabilization of predator–prey oscillations. *J. Theor. Biol.* **255**, 299–306 (2008)
22. Hilker, F., Langlais, M., Malchow, H.: The Allee effect and infectious diseases: extinction, multistability, and the (dis-)appearance of oscillations. *Am. Nat.* **173**, 72–88 (2009)