Simple metaecoepidemic models

Ezio Venturino Simple metaecoepidemic models. *Bulletin of Mathematical Biology*, Volume 73, Number 5, 917-950, 2011,
DOI:10.1007/s11538-010-9542-3.

Simple metaecoepidemic models

Ezio Venturino
Dipartimento di Matematica "Giuseppe Peano",
Universita' di Torino,
via Carlo Alberto 10, 10123 Torino, Italy
E-mail: ezio.venturino@unito.it

Abstract

We consider a simple predator-prey system with two possible habitats and where an epidemic spreads by contact among the prey, but it cannot affect the predators. Only the prey population can freely move from one environment to another. Several models are studied, for different assumptions on the structure of the demographic interactions and on the predators' feeding. Some counterintuitive results are derived. The role the safety refuge may in some cases entail negative consequences for the whole ecosystem. Also, depending on the system formulation, coexistence of all the populations may not always be supported.

Keywords: Eco-epidemiology; Local Stability; Global Stability; Holling Type-II; Hopf-bifurcation; Metapopulation; Habitat.

1 Introduction

In recent years classical population theory has evolved from the study of interacting populations and food chains to more complex situations encompassing communities living in separate environments, joined by possible migrations. Heterogeneous environments and landscape fragmentation due to natural causes or human activities threaten persistence of wild populations. The conservation issue in these habitats has become a major concern of environmentalists [47]. A tool for the understanding of population dynamics in these circumstances is provided by the metapopulation theory, [46]. Local population dynamics and interpatch migrations are responsible for metapopulation

dynamics, with the possible result that the population persists globally, although in some cases the local populations become extinct [12, 17, 20, 46, 49]. Gathering migrations data between patches is however problematic. In fact these activities are in general not undertaken [12, 17, 27]. From this the role of models becomes relevant to predict possible outcomes of specific situations [27]. For instance metapopulation dynamics has been applied to model the spotted owl (*Strix occidentalis*), or the mountain sheep (*Ovis canadensis*), [18], where only the most favorable habitats are populated and the remaining ground in between is used for interpatch migrations. Furthermore human activity also causes loss of habitat, since it tends to break the territorial distribution via human artifacts as buildings, roads, or clearing wild areas for creating new fields for agricultural purposes. The original population living in the unperturbed environment becomes separated into subpopulations, which continue to live independently, but may become now more sensible to adverse conditions. This situation may ultimately cause species extinction.

Ecologists have been looking for ways of assessing population dynamics in patched environments [40] and metapopulations represent a current answer, [21]. In the classical Levins model [31], colonization depends on just the portion of the environment that is actually inhabited. More recent models used to study the butterfly *Melitaea cinxia* in Finland [22] and also other species [34, 35], do not make this assumption. They rather use the concept of incidence function. In the case of the butterfly, however, it has been remarked that variations in local populations may depend on the interaction with a specialist braconid parasitoid, *Cotesia melitaearum* [30], suggesting the need for a metapopulation approach explicitly modeling a host-parasitoid metapopulation dynamics [23]. In the light of these remarks, the need for accounting for diseases in the above type of models is evident.

Ecoepidemiology is a rather new branch of population theory, dealing with the study of systems in which diseases spread among interacting populations. An introduction can be found in [32]. From the first papers on the subject, [19, 15, 5, 41, 42, 44], various situations more or less complicated have been considered to date in the literature, in a time span which is reaching the two decades. From the first researches dealing mostly with the quadratic predator-prey case, more complex models have also been introduced, [9, 2]. But other demographics have been considered, namely competition models and interactions of symbiotic nature, [43, 45].

In this investigation we do not aim at an approach providing a general solution, but rather content ourselves with a first step in the direction, allowing a simple interacting populations model incorporating a diseased population, with two possible living environments, in which one of them might constitute a refuge.

In this paper we thus consider a predator-prey system where two possible habitats are assumed to exist. Furthermore, an epidemic is propagating by contact among the prey, but it cannot affect the predators. The sound prey population can freely move from one environment to another. Several models are built on these basic assumptions, differing on the structure of the underlying demographic models, and on the type of predators' hunting.

In all models throughout the paper, the notation is consistent, namely S denote the sound prey, I the infected prey and P the predators. Indices serve to distinguish the populations in the two patches.

The paper is organized as follows. We consider four different situations distinguished by the populations living in the separate patches and by the way predators feed. In Section 2 we provide a few realistic biological situations. At first, we consider Holling type II hunting: in Section 3, the predator-prey system occupies the first patch, and the epidemic model patch 2; Section 4 instead deals with an ecoepidemic system in patch 1, while patch 2 serves only as a possible safety refuge for the sound prey. The following Section 5 relates the numerical experiments. The next two sections describe systems in which hunting is modeled via a quadratic mass action term. Section 6 contains again predator-prey and epidemics patches and Section 7 the ecoepidemic model in the first patch and the safety refuge in patch 2. Some further numerical simulations on this last situation are reported in the final section. At the referees' request we move most of the relevant mathematics to an Appendix.

2 Biological background

As discussed in the Introduction, the bottom line of the ecoepidemic research shows that since diseases are a fact in nature, their influence on the dynamics of populations cannot be ignored. It makes therefore sense to investigate their consequences on metapopulation models as well. To give specific biological situations, we provide here a few examples of ecosystems that fit in our description.

Lepidoptera have several predators: birds, bats, parasitoids, small mammals, reptiles and insects such as ants and dragonflies are the most important ones. In particular larvae constitute a major portion of the diet of some species, *Parus caerulens, P. major* [11]. Some species of bats can eat Lepidoptera up to half their weight per night, [6, 8]. Parasitoids, in particular Hymenoptera and Diptera attack Lepidoptera, either by killing or immobilizing their hosts immediately, or by implanting in them an egg, which develops and will later on kill the host, [16, 37]. Lepidoptera are also af-

fected by viruses, such as nuclear polyhedrosis virus (NPV), cytoplasmatic polyhedrosis virus (CPV), granulosis virus (GV), entomopox virus (EPV), small RNA viruses [36]. The outcomes are sensible effects, mainly both reproduction rates and pupae weight reduction. *Bacillus thuringiensis* var. kurstaki is a bacteria (Bacillaceae) quite common in nature that ingested by caterpillars kills them, poisoning the insect's digestive system. The Btk release a crystalline protein called "endotoxin" that acts by killing cells and dissolving holes in the lining of the insect's gut. Butterflies escape from their predators by several means: mimetism (expecially moth), assumption from some plants of some compounds that make them unpalatable for the predators (in particular in diurnal butterfly), for instance in the case of *Melitaea cinxia* feeding on *Cotesia melitaearum*, the latter getting alkaloids from the plant on which it feeds, [30]. In other cases they have shapes, or produce colors or sounds which discourage the predators, [37]. An important defense mechanism is myrmecophily, i.e. a kind of association with ants. In the weakest forms, the caterpillars seek refuge nearby the ant nests, along their feeding routes. The latter constitute an enemy-free space, i.e. they are free from predators and parasitoids, [3].

The second case is represented by the predator-prey system with red fox *Vulpes vulpes* (L.) and rabbits *Oryctolagus cuniculus* (L.), affected by the *Myxoma* virus. This is a classical case, since myxomatosis was artificially introduced among the wild rabbits in Australia in order to try to control their population size, [29, 28, 33, 48].

A third example concerns once again *Ovis canadensis*. Its predators are mainly the wolf (*Canis lupus*), coyote (*Canis latrans*), bear (*Ursus*), Canada lynx (*Lynx canadensis*), mountain lion (*Puma concolor*), golden eagle (*Aquila chrysaetos*), [13]. Bighorn sheep are hosts of a number of parasites. Nematode lungworms, *Protostrongylus stilesi* and *P. rushi*, infect all bighorn sheep individuals and probably coevolved with these sheep in North America. Most sheep do not experience any significant deleterious effects of lungworms, [14]. Other ones are *Cysticercus tenuicollis*, *Wyominia tetoni*, *Marshallagia marshalli*, *Ostertagia circumcincta*, *O. lyrata*, *O. occidentalis*, *O. ostertagi*; *Cooperia oncophora*, *C. surnabada*; *Nematodirus archari*, *N. davtiani*, *N. helvetianus*, *N. lanceolatus*, *N. spathiger*; *Trichostrongylus sp.*, *Protostrongylus rushi*; *Dermacentor albipictus* and *D. venustus*, [4].

Another further situation is represented by *Strix occidentalis*, which has as main parasites the helmints. Among its main predators there is the great horned owl, *Bubo virginianus*. At times the great horned owl hunts the spotted owl in order both directly to feed on it as well as to eliminate a possible competitor for resources, since the latter shares several prey with the great horned owl, [24]. Note that the prey of *Strix occidentalis*, mainly

small rodents, play an important role, as they appear to be the vector by which the spotted owl gets the parasites, i.e. the infection, [24].

With this background, we now turn to the construction of a few mathematical systems to model metaecoepidemic situations of the nature described here above. We consider a simple ecosystem in which only two patches are present, in order to be able to perform a mathematical analysis. The basic assumptions common to all of them, is that only prey are allowed to migrate from one patch to the other one. Further, the infected prey are too weak to contribute to intraspecific demographic pressure.

3 Holling type II SP-SI model

Let the patch common to both species be denoted by the index 1 while the index 2 is reserved for the refuge unreachable by the predators. Only in this patch where prey thrive, the epidemics occurs. We take the disease to strongly affect the infected individuals, so that they cannot compete with the sound ones, i.e. the susceptible prey do not feel any intraspecific demographic pressure from the infected ones, nor do the latter reproduce. Migration occurs back and forth from this refuge to the territory in which prey is hunted, but only for sound individuals. This assumption, common to this and all the subsequent models, is plausible, since the disease weakens the infected individuals and if some effort must be exerted to reach the other patch, the weaker animals may well not be able to make it. Figure 1 graphically depicts the situation.

Let S denote the sound prey, I the infected ones and P the predators. The model reads then

$$\begin{aligned}
\frac{dS_1}{dt} &= r_1 S_1 \left(1 - \frac{S_1}{K_1}\right) - a\frac{S_1 P}{H + S_1} - m_{21} S_1 + m_{12} S_2, \qquad (1)\\
\frac{dS_2}{dt} &= r_2 S_2 \left(1 - \frac{S_2}{K_2}\right) - \gamma S_2 I + \nu I + m_{21} S_1 - m_{12} S_2,\\
\frac{dI}{dt} &= I[\gamma S_2 - \mu - \nu],\\
\frac{dP}{dt} &= P\left(\frac{eS_1}{H + S_1} - b\right).
\end{aligned}$$

The first equation describes the logistic evolution of the prey population which is hunted. We assume that its net reproduction rate r_1 and the carrying capacity K_1 depend on this environment. The predation rate is a, migration toward patch 2 occurs at rate m_{21}, m_{12} is the migration rate in the opposite direction. The second equation describes the prey in the safe

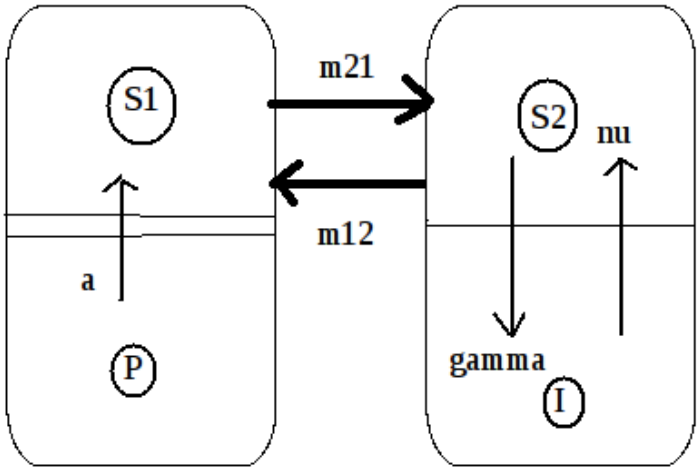

Figure 1: Graphical illustration of model (1).

refuge, with migration rates having opposite signs, net reproduction rate r_2 and carrying capacity K_2 now related to this habitat. In view of the remarks on the strength of the disease, in the logistic correction term in the sound prey evolution equation, no contribution from the infected is present. Thus infectives do not contribute to intraspecific competition, so that sound individuals do not feel their presence. The new feature here is the fact that sound prey can contract the disease, at rate γ. This process is simply described by a mass-action law. We assume that the disease is recoverable. The third equation states that new infectives are generated via the contact rate term appearing as a loss in the former equation, and leave this class via either a natural disease-related mortality μ or a recovery rate ν. No reproduction of infectives is allowed, nor do they feel the interspecific population pressure. The last equation describes the predators dynamics, accounting for natural mortality b and a Michaelis-Menten or Holling type II term for the feeding

behavior, with half saturation constant H. The parameter e represents the rate of food conversion into newborns.

3.1 Preliminaries

The system (1) is constructed by assuming that migration occurs among two territories. In each one of them, populations obey different type of dynamics. For later reference and comparison purposes, we investigate briefly the results of each of these classical models for each territory separately, in terms of our notation.

Assume first that there in no patch 2, i.e. no safety refuge. The SI-SP model, in this case becomes a simple SP, predator-prey model, with equilibria

$$Z_0 = O, \quad Z_1 = (K_1, 0), \quad Z_2 = \left(\frac{bH}{e-b}, \frac{r_1}{aK_1} \left(H + \frac{bH}{e-b} \right) \left(K_1 - \frac{bH}{e-b} \right) \right).$$

The latter is feasible if $(e-b)K_1 > bH$, i.e. introducing a kind of reproduction number σ, for

$$\sigma \equiv \frac{(e-b)K_1}{bH} > 1, \quad e > b. \tag{2}$$

Then Z_0 is unstable, Z_1 is stable for $(e-b)K_1 < bH$, i.e. if the basic reproduction number σ is smaller than 1,

$$\sigma < 1, \tag{3}$$

but no Hopf bifurcation can arise here. The equilibrium Z_2 is stable for

$$H^2 r_1 > \left(1 - \frac{b}{e} \right)^2 aPK_1 \tag{4}$$

and imposing equality in (4), a Hopf bifurcation occurs.

The model without patch 1 is an SIS model, with equilibria

$$W_0 = O, \quad W_1 = (K_2, 0), \quad W_2 = \left(\frac{\mu+\nu}{\gamma}, \frac{r_2}{\mu} \frac{\mu+\nu}{\gamma} \left(1 - \frac{\mu+\nu}{\gamma K_2} \right) \right).$$

W_2 is feasible for the basic reproduction number, [26]

$$\rho \equiv \frac{\mu+\nu}{\gamma} \leq K_2. \tag{5}$$

The origin is unstable; W_1 is stable for

$$K_2 < \rho, \tag{6}$$

and W_2 is stable if

$$\mu^2 + \gamma\nu K_2 > \nu^2. \tag{7}$$

Thus stability of W_1 occurs if and only if W_2 is infeasible. Note that instability of W_2 implies that $\mu\nu^{-1}(\mu+\nu) + \gamma K_2 < \mu + \nu$, which violates (5), and thus entails infeasibility of W_2 and therefore feasibility of W_1, see (5). Thus this system can only have either the disease-free or the endemic equilibrium. No other dynamics is possible. The final outcome of the system is determined only by the basic reproductive ratio $\gamma K_2(\mu+\nu)^{-1}$. Note that for the SI model, $\nu = 0$, (7) is always true.

3.2 Equilibria

We consider now (1). Its boundedness can easily be established by introducing the total environment population $\Pi = S_1 + S_2 + I + P$.

The only possible equilibria of (1) are also easily found, to be the origin $E_0 \equiv O$ together with the boundary points $E_1 \equiv (S_1^{(1)}, S_2^{(1)}, 0, 0)$, $E_2 \equiv (S_1^{(2)}, S_2^{(2)}, 0, P^{(2)})$, $E_3 \equiv (S_1^{(3)}, S_2^{(3)}, I^{(3)}, 0)$, and the coexistence one $E_4 \equiv (S_1^{(4)}, S_2^{(4)}, I^{(4)}, P^{(4)})$.

Equilibrium E_1 does not have an explicit representation, but its feasibility is ensured by

$$m_{21} < r_1, \quad m_{12} < r_2, \quad r_1 r_2 \geq r_1 m_{12} + r_2 m_{21}. \tag{8}$$

For the other equilibria, we have

$$S_1^{(2)} = \frac{bH}{e-b} = \frac{K_1}{\sigma}, \quad P^{(2)} = \frac{eH}{(e-b)a}\left[m_{12}S_2^{(2)}\frac{e-b}{bH} + r_1 - m_{21} - \frac{r_1}{\sigma}\right],$$
$$S_2^{(2)} = \frac{K_2}{2r_2}\left[r_2 - m_{12} + \sqrt{(r_2-m_{12})^2 + 4r_2 m_{21}\frac{bH}{(e-b)K_2}}\right]$$

so that feasibility for E_2 is given by

$$e > b, \quad S_2^{(2)} > \left[\frac{m_{21}-r_1}{m_{12}} + \frac{r_1 bH}{m_{12}K_1(e-b)}\right]\frac{bH}{e-b}\ . \tag{9}$$

Then

$$S_1^{(3)} = \frac{K_1}{2r_1}\left[r_1 - m_{21} + \sqrt{(r_1-m_{21})^2 + 4\frac{\rho}{K_1}r_1 m_{12}}\right], \quad S_2^{(3)} = \rho,$$
$$I^{(3)} = \frac{1}{\mu}\left[r_2\rho\left(1 - \frac{\rho}{K_2}\right) + m_{21}S_1^{(3)} - m_{12}\rho\right]\ ,$$

so that E_3 is feasible for

$$S_1^{(3)} \geq \frac{\rho}{m_{21}}\left[m_{12} - r_2 + r_2\frac{\rho}{K_2}\right]. \tag{10}$$

E_4 has the following components

$$S_1^{(4)} = \frac{bH}{e-b}, \quad P^{(4)} = \frac{eH}{a(e-b)}\left[r_1 - m_{21} - \frac{r_1}{\sigma} + m_{12}\frac{\sigma\rho}{K_1}\right],$$
$$S_2^{(4)} = \rho, \quad I^{(4)} = \rho\left[r_2 - m_{12} - \frac{r_2\rho}{K_2} + m_{21}\frac{bH}{(e-b)\rho}\right]$$

and it is feasible first of all if $e \geq b$ and furthermore if

$$\frac{m_{21}K_2}{(m_{12}-r_2)K_2 + r_2\rho} \geq \frac{\sigma\rho}{K_1} \geq \frac{m_{21}-r_1}{m_{12}} + \frac{r_1}{m_{12}\sigma}. \tag{11}$$

3.3 Stability

The eigenvalues of the Jacobian at the origin are $-\nu - \mu$ and $-b$ and the roots of the quadratic $\lambda^2 + \lambda(m_{21} + m_{12} - r_1 - r_2) + r_1r_2 - r_1m_{12} - r_2m_{21} = 0$. The Routh-Hurwitz conditions ensure stability for

$$r_1r_2 > r_1m_{12} + r_2m_{21}, \quad r_1 + r_2 < m_{12} + m_{21}. \tag{12}$$

At E_1 stability occurs if and only if

$$S_1^{(1)} < \frac{bH}{e-b}, \quad S_2^{(1)} < \rho \tag{13}$$

and

$$r_1 + r_2 < 2\left(r_1\frac{S_1^{(1)}}{K_1} + r_2\frac{S_2^{(1)}}{K_2}\right) + m_{12} + m_{21}, \tag{14}$$
$$r_1r_2\left(1 - 2\frac{S_1^{(1)}}{K_1}\right)\left(1 - 2\frac{S_2^{(1)}}{K_2}\right) > r_1m_{12}\left(1 - 2\frac{S_1^{(1)}}{K_1}\right) + r_2m_{21}\left(1 - 2\frac{S_2^{(1)}}{K_2}\right).$$

E_2 is unconditionally unstable.

At E_3, one eigenvalue is $(H + S_1^{(3)})^{-1}[(e-b)S_1^{(3)} - bH]$, giving the first stability condition $S_1^{(3)}(e-b) < bH$ or

$$\rho\sigma < K_1. \tag{15}$$

The Routh-Hurwitz conditions for the remaining eigenvalues would then be implied by

$$S_1^{(3)} > \frac{1}{2}K_1, \quad S_2^{(3)} > \frac{1}{2}K_2. \tag{16}$$

The Routh-Hurwitz conditions ensure stability at E_4 if

$$\left(J_{11}^{(4)} + J_{22}^{(4)}\right)\left[J_{11}^{(4)}J_{22}^{(4)} - m_{12}m_{21}\right] + J_{11}^{(4)}\frac{aeHP^{(4)}S_1^{(4)}}{(H+S_1^{(4)})^3} + J_{22}^{(4)}\gamma\mu I^{(4)} < 0, \tag{17}$$

$$J_{22}^{(4)}\frac{aeHP^{(4)}S_1^{(4)}}{(H+S_1^{(4)})^3} + \gamma\mu I^{(4)}J_{11}^{(4)} + \gamma\mu I^{(4)}\frac{\left(J_{11}^{(4)} + J_{22}^{(4)}\right)^2}{(b_2b_3 - b_0)}\frac{aeHP^{(4)}S_1^{(4)}}{(H+S_1^{(4)})^3} < 0.$$

3.4 Hopf bifurcations

At the origin if we impose the quadratic to have purely imaginary roots, via

$$r_1 + r_2 = m_{12} + m_{21}, \quad r_1r_2 < r_1m_{12} + r_2m_{21}, \tag{18}$$

we see that limit cycles can be obtained, as in the $m_{12} - m_{21}$ parameter plane the conditions (18) are seen to have solutions.

At E_1 again we can obtain a Hopf bifurcation by acting on the quadratic characteristic equation, while requiring the remaining eigenvalues, one of which differs in the two cases, to be negative. We are led to

$$r_1 + r_2 = m_{12} + m_{21} + 2\left(\frac{r_1}{K_1}S_1^{(1)} + \frac{r_2}{K_2}S_2^{(1)}\right), \tag{19}$$

$$r_1\left[1 - \frac{2S_1^{(1)}}{K_1}\right]r_2\left[1 - \frac{2S_2^{(1)}}{K_2}\right] < r_1\left[1 - \frac{2S_1^{(1)}}{K_1}\right]m_{12} + r_2\left[1 - \frac{2S_2^{(1)}}{K_2}m_{21}\right], \tag{20}$$

which again are seen to have a solution in the $m_{12} - m_{21}$ parameter plane, taking into account that (19) requires

$$r_1 - 2\frac{r_1}{K_1}S_1^{(1)} + r_2 - 2\frac{r_2}{K_2}S_2^{(1)} > 0$$

and thus the case of both terms on the left of (20) being simultaneously negative cannot occur.

At E_3 the characteristic equation is a cubic. It can be factored and a sufficient condition to ensure one feasible value for the bifurcation parameter $r_2^{\dagger}$, can analytically be found, (50).

Also at E_4 a feasible value for the Hopf bifurcation to occur r_2^* is obtained by imposing condition (53).

3.5 Results interpretations

At first, we observe that the origin E_0 can be stabilized, i.e. that the whole ecosystem may be wiped out, under suitably unfavorable conditions, (12), in contrast to what happens to the two separate models corresponding to each single patch, namely equilibria Z_0 and W_0. Thus, surprisingly, the "refuge" could be an endangerment for the whole environment, threatening its long term sustainability. On a deeper analysis, however, it can easily be established that the purely demographic model obtained by removing the infection, and the infected class, from (1), has exactly the same feature, the origin can be stabilized under the very same conditions (12). This appears to be reasonable, as in conditions (12) no parameters related to disease appear, but only the migration and the reproduction rates. On the other hand, field experiments show that without disease, the refuge is still beneficial, see [25]. However, these two results are not in contrast, as in the field experiments, both predator and prey are allowed to migrate, while for us the predators are prevented to reach the prey refuge. For this counter-intuitive effect of the refuge, infection is therefore by no means crucial, as the phenomenon can be ascribed to the assumptions on migration.

In addition the only other possible stable equilibria are the predator-free one E_3, the predator- and disease-free point E_1 and the coexistence of the whole ecosystem E_4. Thus if the origin is unstable, the prey can never be wiped out of the system. In this sense the existence of the refuge protects them. Note however that this happens also for the SP submodel, since the equilibria Z_1 and Z_2 contain the prey. The instability of E_2 coupled with the one of the origin and of E_1 renders impossible the disease eradication, thus in this case the refuge establishes the disease in the ecosystem. When E_3 is stable, the predators are wiped out.

Note that for the SP subsystem for

$$\sigma > 1 \tag{21}$$

the predators invade the environment, since the predator-free equilibrium Z_1 becomes unstable. For the basic reproduction number $\rho < 1$ in the SI subsystem the disease gets eradicated, while it remains endemic conversely.

In the combined model, the disease together with the predators can be wiped out for low enough prey levels, see (13). The latter can be restated as $S_1^{(1)}\sigma < K_1$, $S_2^{(1)} < \rho$, or new modified reproduction numbers can be introduced, namely

$$\sigma_m \equiv \frac{S_1^{(1)}\sigma}{K_1} < 1, \quad \rho_m \equiv \frac{S_2^{(1)}}{\rho} < 1, \tag{22}$$

to ensure stability of E_1. Therefore the possibility of eradicating the disease in the SI subsystem, for small ρ, entails possibly a large ρ_m, thereby making the eradication of the disease and of the predators together in the metaecoepidemic model more unlikely. Note that the effect of σ is instead directly proportional to σ_m, so that predators' disappearance in the pure SP submodel favors also the instability of E_1.

A similar condition for the prey in patch 1 must be ensured for stability of E_3, still given by the first condition (13), but here stability is instead ensured if the prey in both patches are also above certain levels, see (16). Thus the wiping out of predators in the metaecoepidemic model requires, in addition to (21), also that the prey at equilibrium are above both patches' carrying capacities. The disease for the predator-free equilibrium stability needs only not to be too much virulent, its basic reproduction number must be bounded above, (15).

4 Holling type II SIP-S model

In this second model the ecoepidemics occurs in the first patch, the second one is a safe refuge only for the sound prey, which are the only ones able to migrate there. Once again, the diseased individuals are assumed not to be able to make the effort to reach the safe environment. Predators do not feed on infected prey, though. Again we illustrate the model in Figure 2.

$$\begin{aligned}
\frac{dS_1}{dt} &= r_1 S_1 \left(1 - \frac{S_1}{K_1}\right) - a\frac{S_1 P}{H + S_1} - m_{21} S_1 + m_{12} S_2 - \gamma S_1 I + \nu I, \\
\frac{dS_2}{dt} &= r_2 S_2 \left(1 - \frac{S_2}{K_2}\right) + m_{21} S_1 - m_{12} S_2, \\
\frac{dI}{dt} &= I[\gamma S_1 - \mu - \nu], \\
\frac{dP}{dt} &= P\left(\frac{eS_1}{H + S_1} - b\right).
\end{aligned} \tag{23}$$

Boundedness for (23) is established as for the model (1).

The equilibria are $Q_0 = E_0 = O$, $Q_1 = E_1$, with the same feasibility conditions (8), $Q_2 = E_2$, again with same feasibility conditions (9) and $Q_3 = (\widetilde{S_1^{(3)}}, \widetilde{S_2^{(3)}}, \widetilde{I^{(3)}}, 0)$, with components given by

$$\widetilde{S_1^{(3)}} = \rho, \quad \widetilde{S_2^{(3)}} = \frac{K_2}{2r_2}\left[r_2 - m_{12} + \sqrt{(r_2 - m_{12})^2 + 4m_{21}\frac{r_2}{K_2}\rho}\right],$$

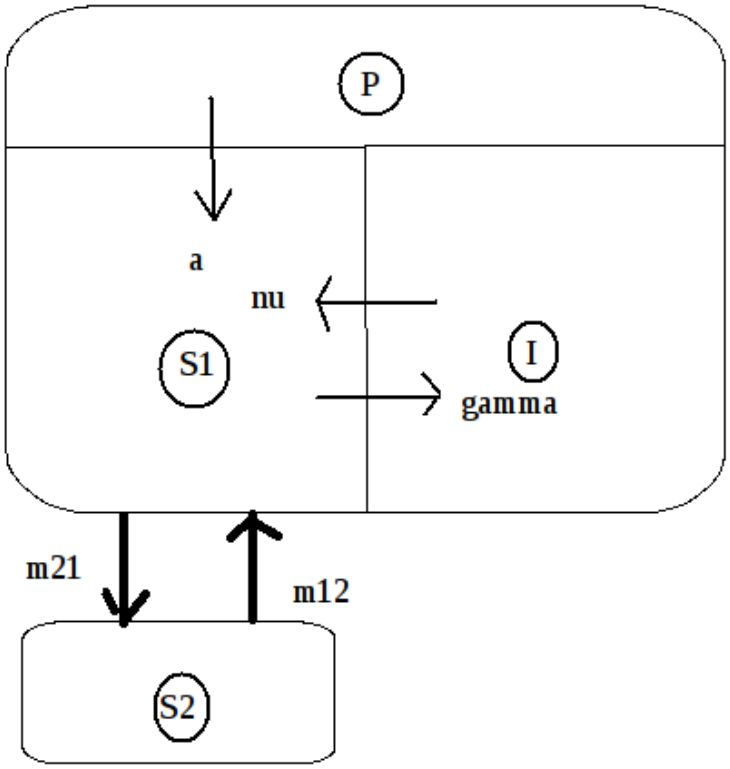

Figure 2: Graphical illustration of model (23).

$$\widetilde{I^{(3)}} = \frac{1}{\mu}\left[r_1\rho\left(1-\frac{\rho}{K_1}\right) - m_{21}\rho + m_{12}\widetilde{S_2^{(3)}}\right].$$

Feasibility amounts to requiring $\widetilde{S_2^{(3)}}$ to be real, and both $\widetilde{S_2^{(3)}}$ and $\widetilde{I^{(3)}}$ to be nonnegative. Note that in this case the interior equilibrium does not exist, i.e. coexistence in this metaecoepidemic environment is not possible.

4.1 Stability

The eigenvalues of J at the origin are exactly the same as for E_0 in (1), thus the stability conditions coincide with (12).

At Q_1 stability occurs if and only if conditions (14) hold, together with

$$S_1^{(1)} < \min\left\{\frac{\mu+\nu}{\gamma}, \frac{bH}{e-b}\right\} \equiv \min\left\{\rho, \frac{K_1}{\sigma}\right\}. \tag{24}$$

Q_2 is instead inconditionally unstable.

At Q_3 one eigenvalue is $(H+\widetilde{S_1^{(3)}})^{-1}[(e-b)\widetilde{S_1^{(3)}}-bH]$, which of course needs to be negative for stability, giving again the same stability condition (15), the others are the roots of a cubic (43) with coefficients

$$\widetilde{a_0}=-\gamma\mu\widetilde{I^{(3)}}\widetilde{J_{22}^{(3)}},\quad \widetilde{a_1}=\widetilde{J_{11}^{(3)}}\widetilde{J_{22}^{(3)}}+\gamma\mu\widetilde{I^{(3)}}-m_{12}m_{21},\quad \widetilde{a_2}=-(\widetilde{J_{11}^{(3)}}+\widetilde{J_{22}^{(3)}}).$$

Considering the equilibrium definition it follows that

$$\widetilde{J_{11}^{(3)}}=-\frac{\nu\widetilde{I^{(3)}}+m_{12}\widetilde{S_2^{(3)}}}{\widetilde{S_1^{(3)}}}-\frac{r_1\widetilde{S_1^{(3)}}}{K_1}<0,\quad \widetilde{J_{22}^{(3)}}=-\frac{m_{21}\widetilde{S_1^{(3)}}}{\widetilde{S_2^{(3)}}}-\frac{r_2\widetilde{S_2^{(3)}}}{K_2}<0.$$

Thus $\widetilde{a_0}>0$, $\widetilde{a_2}>0$ and the remaining condition for stability becomes

$$-(\widetilde{J_{11}^{(3)}}+\widetilde{J_{22}^{(3)}})(\widetilde{J_{11}^{(3)}}\widetilde{J_{22}^{(3)}}-m_{12}m_{21})-\gamma\mu\widetilde{I^{(3)}}\widetilde{J_{11}^{(3)}}>0 \tag{25}$$

and the latter is implied by (16) evaluated at $\widetilde{S_1^{(3)}}$ and $\widetilde{S_2^{(3)}}$.

4.2 Hopf bifurcations

The analysis for the points Q_0 and Q_1 is exactly the same as for E_0 and E_1.

At Q_3 to have a feasible value for the Hopf bifurcation parameter $r_1^\dagger$ it is enough to require (56).

4.3 Discussion

Note that for the alternative model, we can delete only patch 2, to make a reasonable comparison. We get an ecoepidemic model in patch 1 which has been analyzed and is known to produce bifurcations, [10] for the case of no external removal. A situation common for this case and to (1) is that the parameter a plays no role whatsoever in the bifurcations. Thus the predators' hunting rate in the metaecoepidemic model cannot be the cause of stable cyclic populations oscillations. This is in contrast to the result for the simple ecoepidemic model, see Theorem 4.4 of [10], where the predation rate appears to possess a role for the Hopf bifurcation to occur.

Again in the metaecoepidemic system the origin can be stabilized, with the same conditions as for the former model, namely (12). Once again this indicates the possibility of extinction, caused both by the existence of the survival refuge and by the assumption that only prey can migrate.

Also here as well as for (1), the instability of $Q_2\equiv E_2$ means that the disease alone cannot be eradicated, while preserving the other populations.

Recalling the discussion following (43), the instability is to be ascribed to the fact that $J_{11}^{(2)} J_{22}^{(2)} < 0$. But note that the latter quantities, being evaluated at Q_2, are independent of the disease. Therefore the refuge acts as a tool for keeping the disease endemic in the system and this is due only to demographic effects.

Equilibrium Q_1 has a more stringent condition on the prey size in patch 1 (compare (24) and (13)) and a more relaxed one for the prey in the safety refuge, since the latter have only to obey conditions (14) as in (1).

But the main result in this context is that the metaecoepidemic model (23) does not sustain all the populations. Therefore the existence of a safety refuge for the prey surprisingly becomes a negative factor from the biodiversity point of view.

5 Simulations

To further investigate the metaecoepidemic model with Holling type II dynamics, we have performed numerical experiments on (1). Some results are reported here. In all the figures the left column contains the graphs of the populations in the two patches as separate entities, without any communication between them. With the same parameters, this time including nonzero migration rates, we run the simulations again and report their results on the right column.

Remark. Here and in the rest of the paper, populations are counted by individuals, i.e. pure numbers. The left-hand side of (1), (23) and of the models that will be introduced later, (26) and (38), are therefore frequencies. This makes all the terms on the right hand sides of the equations also frequencies. Thus since K_1, K_2 and H must be pure numbers, all other parameters r_1, r_2, a, γ, ν, μ, e, b, m_{12}, m_{21}, are also frequencies, with time measured in years, unless otherwise stated.

We use the reference values of parameters from the real situations described in Section 2. Note that data gathered from the literature allow us to set the value of the reproduction parameters. In case of *Ovis canadensis*, there are an average 2.5 offsprings per year, and an average life span of 10.5 years, [13]. Finally, *Strix occidentalis* has about 2 to 3 offsprings per year, [39].

This means that we can fix the reproduction values. In particular we consider the case of *Ovis canadensis* or of *Strix occidentalis*. We can then take $r_1 = 2.5$ for the fertility of sound individuals. The value of $r_2 = 1$ has been chosen assuming that the disease affects heavily the infected. For instance, experimental results on insects show a decrease in net reproduction

rate of about 50% in disease-affected individuals, see Fig. 2 of [36]. The remaining parameter values are arbitrarily chosen to simulate a hypothetical environment.

Figure 3 shows that the limit cycles present in the underlying demographic model in patch 1 get transferred via the migration also to the second patch. In this particular case we observe also that the disease is eradicated in patch 2, but the oscillations amplitudes become larger and larger and ultimately the system collapses. The figure is obtained from the following parameter values $K_1 = 100$, $a = 0.5$, $H = 1$, $m_{21} = 3$, $m_{12} = 2$, $K_2 = 150$, $\gamma = 0.5$, $\nu = 0.3$, $\mu = 0.3$, $e = 1.3$, $b = 0.1$. In Figure 4 we show instead that stable coexistence

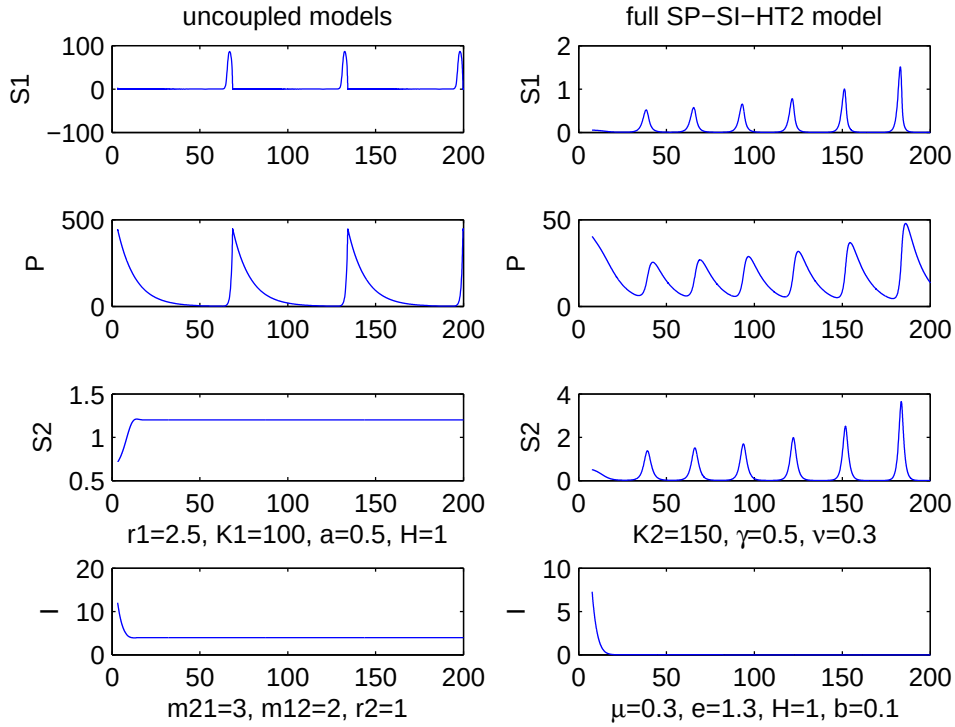

Figure 3: Demographic oscillations are carried through to the epidemics patch. Here and in all subsequent figures, the left column contains the graphs of the populations in two patches as separate entities, without any communication between them, while the right one shows the metaecosystem populations.

Equilibria in the two patches can be reached in spite of the fact that in the purely demographic model oscillations are present. The parameters in this case are $K_1 = 100$, $a = 1.5$, $H = 1$, $m_{21} = 3$, $m_{12} = 2$, $K_2 = 150$, $\gamma = 0.75$,

$\nu = 0.03$, $\mu = 0.03$, $e = 1.3$, $b = 0.1$. Figure 5 instead shows that the

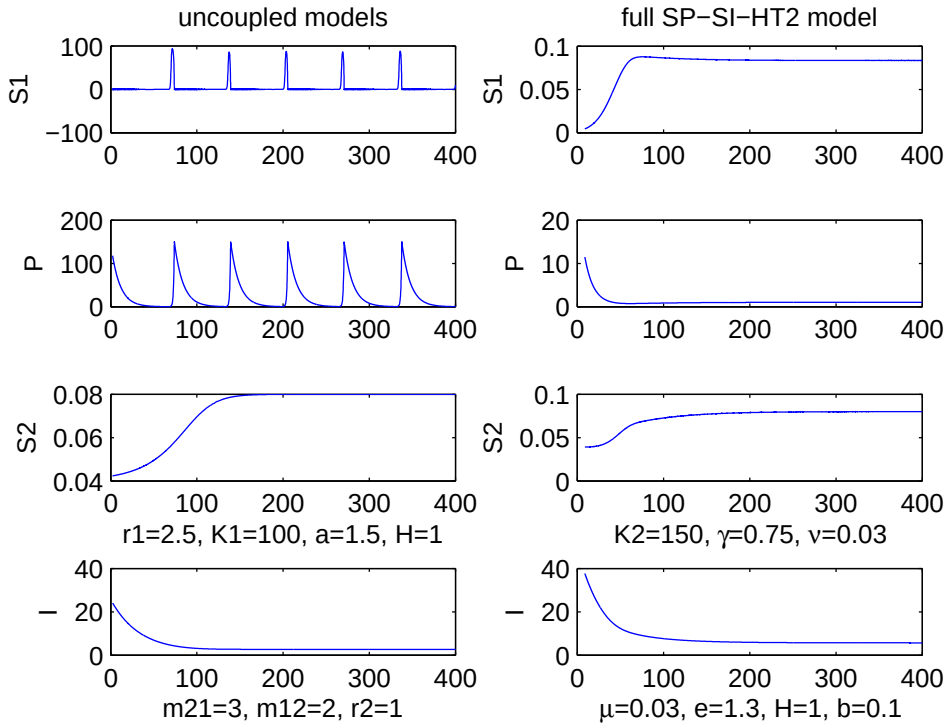

Figure 4: Demographic oscillations are damped in the metaecoepidemic model.

limit cycles inherited by the underlying demographic model are present in both patches, but in this case the disease, endemic in the isolated patch 2, is wiped out in the model with migrations, for the parameter values $K_1 = 100$, $a = 0.7$, $H = 1$, $m_{21} = 3$, $m_{12} = 2$, $K_2 = 150$, $\gamma = 0.75$, $\nu = 0.3$, $\mu = 0.03$, $e = 1.3$, $b = 0.1$; Figure 6, obtained for the parameter values $K_1 = 100$, $a = 1.5$, $H = 1$, $m_{21} = 3$, $m_{12} = 2$, $K_2 = 150$, $\gamma = .5$, $\nu = 0.3$, $\mu = 0.3$, $e = 0.8$, $b = 0.9$, instead shows that the same behavior of the two separate patches can be shown also by the combined metaecoepidemic model, which settles to the predator-free equilibrium E_3.

6 Holling type I SI-SP case

We turn now to the setting up and to the analysis of metaecoepidemic models with mass action predation terms. Recalling Figure 1, at first we formulate

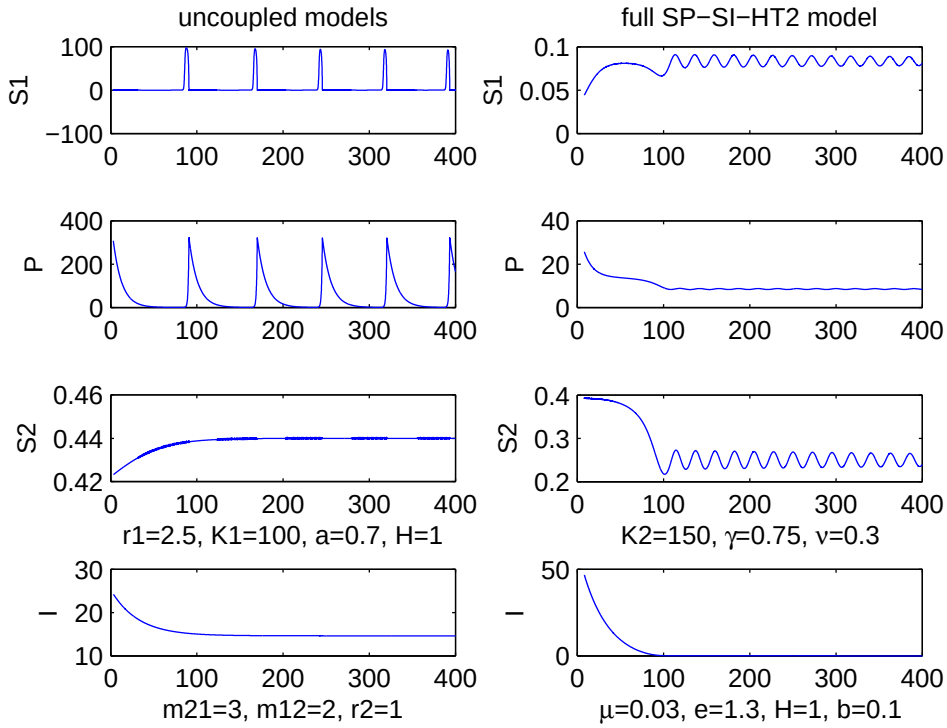

Figure 5: The disease is wiped out in the metaecoepidemic model.

the analogous model of system (1), namely

$$\begin{aligned}
\frac{dS_1}{dt} &= r_1 S_1 \left(1 - \frac{S_1}{K_1}\right) - a S_1 P - m_{21} S_1 + m_{12} S_2, \qquad (26)\\
\frac{dS_2}{dt} &= r_2 S_2 \left(1 - \frac{S_2}{K_2}\right) - \gamma S_2 I + \nu I + m_{21} S_1 - m_{12} S_2,\\
\frac{dI}{dt} &= I[\gamma S_2 - \mu - \nu],\\
\frac{dP}{dt} &= P\left(e S_1 - b\right).
\end{aligned}$$

Note that of the two independent subsystems, SI coincides with the former one analyzed in Subsection 3.1, while the SP model here admits as equilibria the origin Y_0, unstable, the predator-free point $Y_1 = (K_1, 0)$, which is stable for

$$K_1 < \frac{b}{e}, \qquad (27)$$

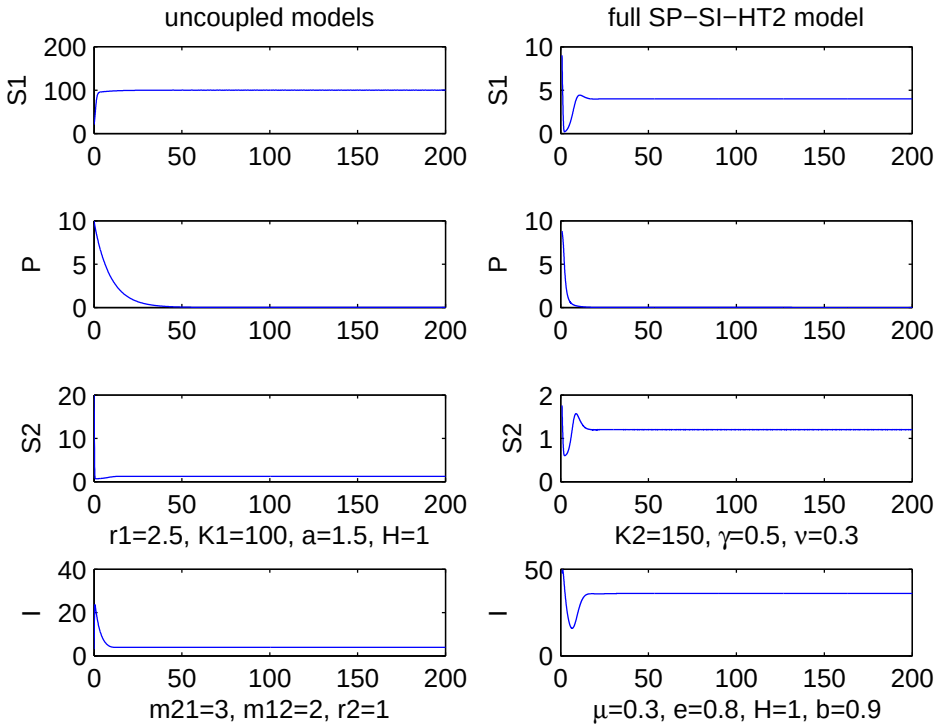

Figure 6: The metaecoepidemic model shows the same behavior of the two separate models, settling to equilibrium E_3.

and the unconditionally stable coexistence one

$$Y_2 = \left(\frac{b}{e}, \frac{r_1}{a}\left[1 - \frac{b}{eK_1}\right]\right),$$

feasible for

$$K_1 > \frac{b}{e} \tag{28}$$

i.e. the opposite of condition (27). Hence only one of the two equilibria is at the same time feasible and stable, making it globally asymptotically stable, Y_1 when (27) holds, and Y_2 in the opposite case.

For model (26), equilibria are the points $U_0 = O$, $U_1 = E_1$, $U_2 = (\frac{b}{e}, \widehat{S_2^{(2)}}, 0, \widehat{P^{(2)}})$ here given by

$$\widehat{S_2^{(2)}} = \left[r_2 - m_{12} + \sqrt{(r_2 - m_{12})^2 + 4m_{21}\frac{br_2}{eK_2}}\right]\frac{K_2}{2r_2}, \tag{29}$$

$$\widehat{P^{(2)}} = \left[r_1 \frac{b}{e} \left(1 - \frac{b}{eK_1} \right) + m_{12} \widehat{S_2^{(2)}} - m_{21} \frac{b}{e} \right] \frac{e}{ab} \tag{30}$$

feasible for

$$r_1 \frac{b}{e} \left(1 - \frac{b}{eK_1} \right) + m_{12} \widehat{S_2^{(2)}} \geq m_{21} \frac{b}{e}. \tag{31}$$

Also, $U_3 \equiv E_3$ with the very same feasibility conditions (10). For U_4, the coexistence equilibrium, we have

$$\widehat{S_1^{(4)}} = \frac{b}{e}, \quad \widehat{I^{(4)}} = \frac{1}{\mu} \left[r_2 \rho \left(1 - \frac{\rho}{K_2} \right) + m_{21} \frac{b}{e} - m_{12} \rho \right] \tag{32}$$

$$\widehat{S_2^{(4)}} = \rho, \quad \widehat{P^{(4)}} = \left[r_1 \frac{b}{e} \left(1 - \frac{b}{eK_1} \right) + m_{12} \rho - m_{21} \frac{b}{e} \right] \frac{e}{ab}, \tag{33}$$

with feasibility conditions

$$r_2 \rho \left(1 - \frac{\rho}{K_2} \right) \geq m_{12} \rho - m_{21} \frac{b}{e} \geq r_1 \frac{b}{e} \left(\frac{b}{eK_1} - 1 \right). \tag{34}$$

The boundedness of the solution trajectories is established with the same steps as for (1), recalling that $e < a$.

The origin has the same stability properties as for (1), namely it is stable when (12) hold. The point U_1 coincides in part with E_1 also for stability purposes, i.e. it has two different eigenvalues, $\gamma S_2^{(1)} - \mu - \nu$, $eS_1^{(1)} - b$, while the remaining ones originate from the same quadratic as for E_1, so that its stability is ensured by (14) and

$$S_1^{(1)} < \frac{b}{e}, \quad S_2^{(1)} < \rho. \tag{35}$$

For U_2 the analysis follows the same steps as for E_2 and it is again unconditionally unstable. At U_3 one eigenvalue is $eS_1^{(3)} - b$ which is negative if

$$e\rho < b \tag{36}$$

and once again the remaining stability analysis coincides with the one of E_3, thus stability is ensured by (16).

For stability at U_4 we need (57). But $J_{11}, J_{22} < 0$ leads to some simplifications, since the first two conditions (57) are satisfied.

In this case the search for Hopf bifurcations for Q_3 coincides with the one for E_3, while in the case of Q_4, we are led once again to a cubic

$$\sum_{k=0}^{3} \pi_k r_2^k = 0$$

in the bifurcation parameter r_2. Then a sufficient condition for having a feasible value of this parameter would again be

$$\pi_3 \pi_0 < 0. \tag{37}$$

The particular case SI-SP model with no patch 2, gives the equilibria $L_0 \equiv O$, $L_1 = (\frac{b}{e}, \frac{r_1}{a}[1 - \frac{b}{3K_1}])$. The former is unstable, the latter always stable. When patch 1 is absent, we get the same equilibria W_0, W_1, W_2 found earlier.

6.1 Results interpretation

The results here indicate once again that the ecosystem will collapse under the very same conditions (12) of the corresponding model with Holling type II functional response and also the disease-free equilibrium U_2 is always unstable, i.e. it is not possible to eradicate the disease, keeping all populations alive. The disease- and predator-free equilibrium U_1 can be stabilized, i.e. is attainable under different conditions of the corresponding equilibrium E_1 for the Holling type II model, compare the first conditions of (13) and of (35). In particular, if $e < b$, (13) cannot be satisfied, while it may still be possible to render U_1 stable. The predator-free equilibrium exhibits instead the same existence and stability properties in both models. This is not surprising, in view of the fact that by setting $P = 0$, the two submodels of (1) and (26) coincide. Coexistence of all the populations in the ecosystem is possible both in (1) as well as in (26).

7 Holling type I SIP-S case

The final model we consider is the counterpart of (23), see for reference Figure 2. Again, no "satiation effect", modeled by a Michaelis-Menten term is here assumed:

$$\begin{aligned}
\frac{dS_1}{dt} &= r_1 S_1 \left(1 - \frac{S_1}{K_1}\right) - a S_1 P - m_{21} S_1 + m_{12} S_2 - \gamma S_1 I + \nu I, \quad (38)\\
\frac{dS_2}{dt} &= r_2 S_2 \left(1 - \frac{S_2}{K_2}\right) + m_{21} S_1 - m_{12} S_2,\\
\frac{dI}{dt} &= I[\gamma S_1 - \mu - \nu],\\
\frac{dP}{dt} &= P\,(e S_1 - b)\,.
\end{aligned}$$

The equilibria are found as follows: $R_0 = O$, $R_1 \equiv E_1$, $R_2 \equiv U_2$, $R_3 \equiv Q_3$, but the interior equilibrium does not exist in general, unless $b\gamma = e(\mu + \nu)$ in which case it is not unique, it becomes a line of equilibria and will not be analyzed any further.

The Jacobian in this case differs from (54) in the elements

$$J_{11} = r_1 - 2r_1\frac{S_1}{K_1} - aP - \gamma I - m_{21}, \quad J_{14} = -aS_1, \quad J_{41} = eP, \quad J_{44} = eS_1 - b.$$

At R_0 the stability analysis is the same as for E_0, at R_1 two eigenvalues are $\gamma S_1^{(1)} - \mu - \nu$, $eS_1^{(1)} - b$, and the remaining quadratic coincides with the one of E_1. Thus stability is implied by (14) and

$$S_1^{(1)} < \min\left\{\frac{\mu + \nu}{\gamma}, \frac{b}{e}\right\} \equiv \min\left\{\rho, \frac{b}{e}\right\}. \tag{39}$$

For R_2 only the first eigenvalue differs, namely $\gamma\frac{b}{e} - \mu - \nu$, the others are those of U_2 and therefore it is again unconditionally unstable. At R_3 the first eigenvalue is $eS_1^{(3)} - b$, the others are those of Q_3, so that stability is achieved for (36), i.e. implied by (16), and (25).

Note that the particular cases of the uncoupled SIP-S model reduce only to the standard quadratic ecoepidemic model, [42], the other one being a trivial logistic one population model.

8 Simulations

To illustrate the theoretical analysis, more experiments have been carried out. We report here some of the results obtained for those related to model (38).

Recalling the remark of Section 5, all parameters are frequencies, with time measured in years, except those related to populations, i.e. the carrying capacities K_1 and K_2. In this case, we chose the parameter values obtained from the populations of *Vulpes vulpes* (L.) and *Oryctolagus cuniculus* (L.). They are as follows: for the red fox there are 4.5 offsprings per year, [7], while for the rabbit the offsprings are between 4 to 14, and they reproduce from 5 to 7 times per year, [1]. Thus, for the sound prey reproduction rate we take the maximum from the above data, $r_1 = 100$, while for the infected fertility we assume the minimum, namely $r_2 = 20$. For the predators instead, we let $a = 4.5$.

Figure 7 shows that the predator-free equilibrium in the uncoupled model contains the endemic disease with a large prevalence. In presence of the

refuge, while the susceptible levels in both patches are essentially unaltered, the infected experience an increase of 50%. Evidently, if the goal is the disease fight, the safety refuge in this case is to be avoided. On the contrary, in a two-patch ecosystem as described here, the removal of the safety refuge contributes to significantly decreasing the disease prevalence. Note that in this case the predators are wiped out, both in the uncoupled systems and in the metaecoepidemic model.

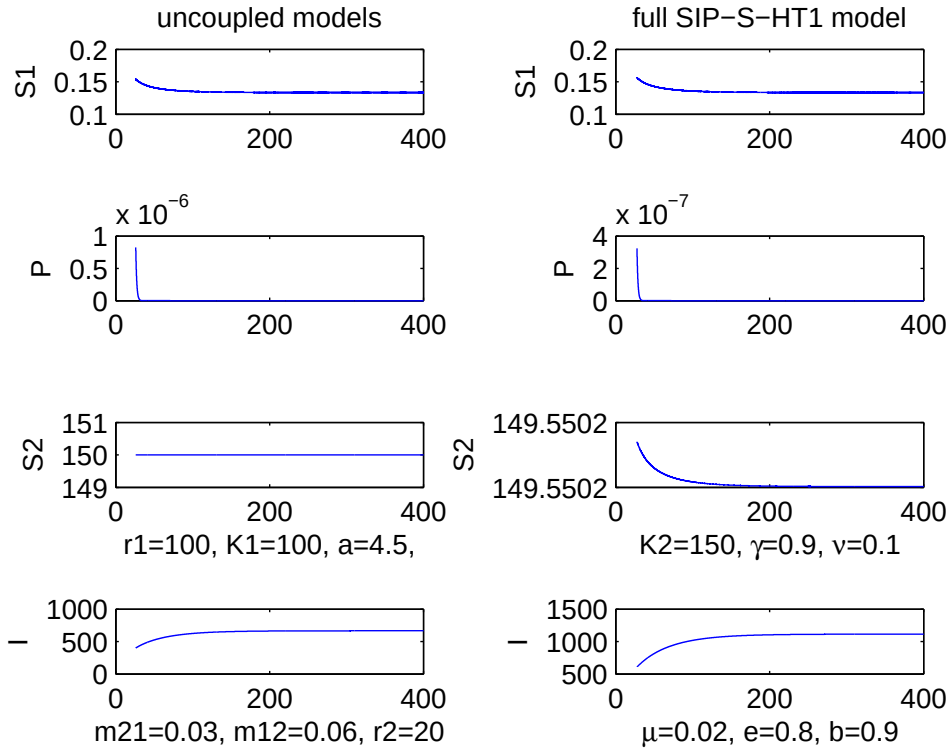

Figure 7: Equilibrium R_3, for the parameter values $r_1 = 100$, $K_1 = 100$, $a = 4.5$, $m_{21} = 0.03$, $m_{12} = 0.06$, $r_2 = 20$, $K_2 = 150$, $\gamma = 0.9$, $\nu = 0.1$, $\mu = 0.02$, $e = 0.8$, $b = 0.9$.

In Figure 8 we show instead that the disease-free stable equilibrium in the SIP phase space of the single patch model is also reached in the metaecoepidemic model, equilibrium R_2, for the parameter values $K_1 = 100$, $m_{21} = 3$, $m_{12} = 5$, $K_2 = 150$, $\gamma = 0.25$, $\nu = 0.1$, $\mu = 0.2$, $e = 0.8$, $b = 0.2$. Lower migration rates seem to significantly decrease the number of predators and to correspondingly markedly increase the sound prey in the refuge, leaving

the sound prey in patch 1 essentially at the same level.

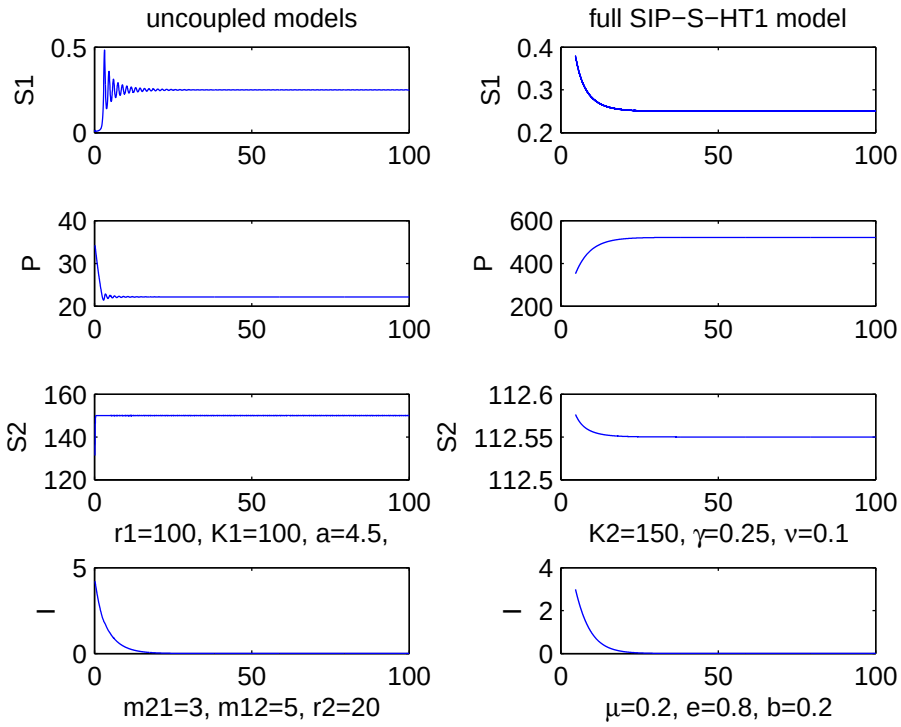

Figure 8: Equilibrium R_2, for the parameter values $r_1 = 100$, $K_1 = 100$, $a = 4.5$, $m_{21} = 3$, $m_{12} = 5$, $r_2 = 20$, $K_2 = 150$, $\gamma = 0.25$, $\nu = 0.1$, $\mu = 0.2$, $e = 0.8$, $b = 0.2$.

Figure 9 shows that the stable disease-free coexistence equilibrium is reached by the single patch system, while in the second patch the susceptible population thrives at carrying capacity. By allowing migration rates among the patches, the latter drops significantly, while the equilibrium in patch 1 remains essentially unaltered, but for the predators that experience a 7-fold increase.

For a relevant difference between the migration rates, $m_{21} = 100$, $m_{12} = 0.01$ and a highly virulent disease, $\gamma = 0.95$, coupled with the remaining parameters $K_1 = 10$, $K_2 = 150$, $\nu = 0.7$, $\mu = 0.2$, $e = 0.2$, $b = 0.2$, one can observe that the predator-free endemic equilibrium of patch 1 is converted into a disease-free equilibrium in the metaecoepidemic model, Figure 10. Large differences in migration rates between patches are indeed frequent in real situations, due for instance to different available nutrients in the two patches, with migration occurring naturally toward the richer one, with no

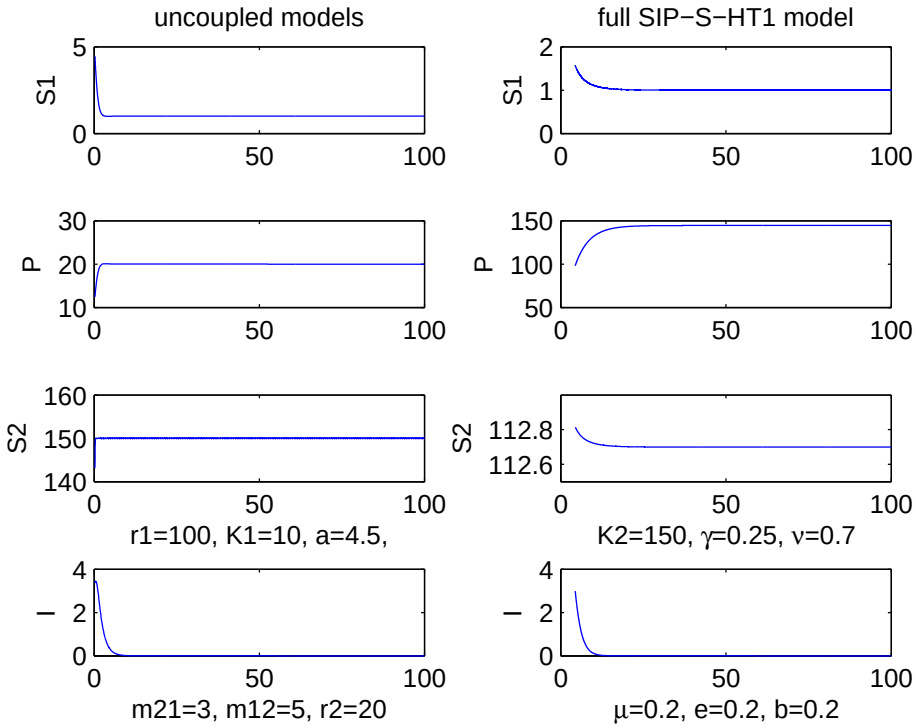

Figure 9: Equilibrium R_2, for the parameter values $r_1 = 100$, $K_1 = 10$, $a = 4.5$, $m_{21} = 3$, $m_{12} = 5$, $r_2 = 20$, $K_2 = 150$, $\gamma = 0.25$, $\nu = 0.7$, $\mu = 0.2$, $e = 0.2$, $b = 0.2$.

coming back. Also important is patch overcrowding, implying that emigration towards the less populated patch takes place especially for the younger individuals. Furthermore the available space in less crowded patches makes them sinks, attracting migrants and thus constituting ecological traps for them, i.e. sink habitats low in quality for reproduction and survival, yet preferred over other available, high quality habitats. For these remarks, see [38].

We have also run some test cases for the case of *Ovis canadensis* or *Strix occidentalis*. Setting $r_1 = 2.5$, $r_2 = 1$, for the remaining set of parameter values given by $K_1 = 100$, $a = 1.5$, $m_{21} = 0.03$, $m_{12} = 0.06$, $K_2 = 150$, $\gamma = 0.9$, $\nu = 0.1$, $\mu = 0.2$, $e = 0.8$, $b = 0.9$, equilibrium R_3 is recovered, Figure 11. We note that the system with two patches experiences an 9-fold increase in the number of infectives, while the number of sound individuals does not sensibly change, comparing with the equilibrium of the classical SIP model. If the goal is to fight the disease, the safety refuge should be

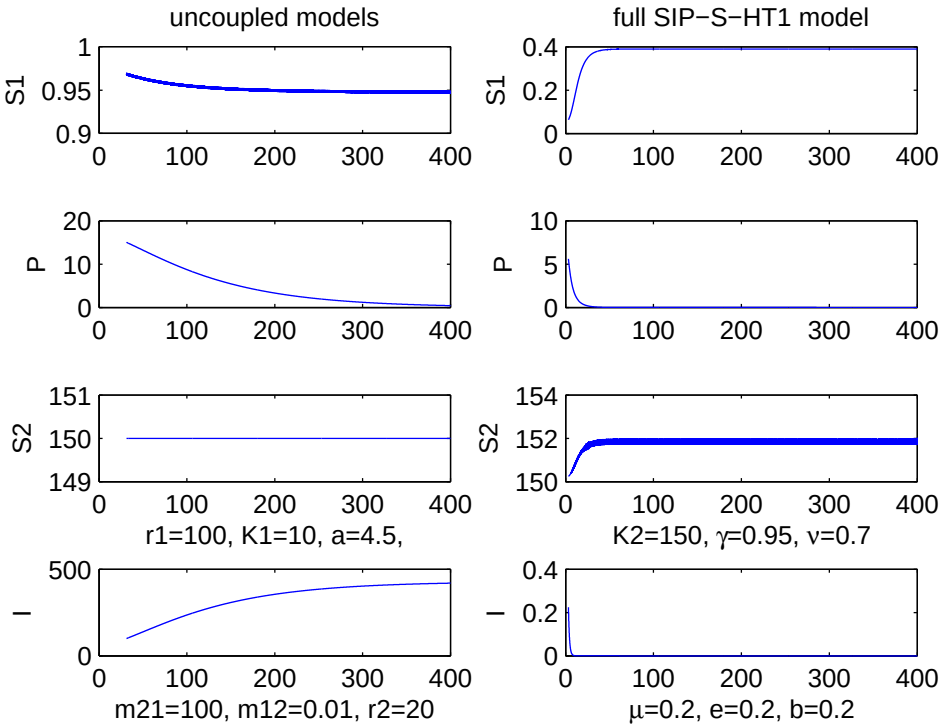

Figure 10: The predator-free equilibrium of patch 1 is converted into a disease-free equilibrium in the metaecoepidemic system, for the parameter values $r_1 = 100$, $K_1 = 10$, $a = 4.5$, $m_{21} = 100$, $m_{12} = 0.01$, $r_2 = 20$, $K_2 = 150$, $\gamma = 0.95$, $\nu = 0.7$, $\mu = 0.2$, $e = 0.2$, $b = 0.2$.

avoided. On the contrary, in a two-patch ecosystem as described here, the removal of the safety refuge contributes to significantly decreasing the disease prevalence. Also, for migration rates that are 10 times higher, $m_{21} = 0.3$, $m_{12} = 0.6$, the the sound population in patch 2 is about half, while the infected in patch 1 increase almost 45 times. Also, in these conditions a decrease of the disease incidence to $\gamma = 0.5$ does not seem to affect the final infectives' equilibrium values, remaining at about 190. In decreasing the incidence value we found essentially little change in the endemic value of the disease equilibrium, until about $\gamma = 0.265$ where the equilibrium shifts to R_2.

Keeping the same reproduction and hunting rates, if we choose instead the set of parameter values $K_1 = 100$, $a = 3.5$, $m_{21} = 3$, $m_{12} = 5$, $K_2 = 150$, $\gamma = 0.25$, $\nu = 0.1$, $\mu = 0.2$, $e = 0.8$, $b = 0.2$ the disease-free equilibrium is obtained. If we change the migration rates, making them ten times smaller,

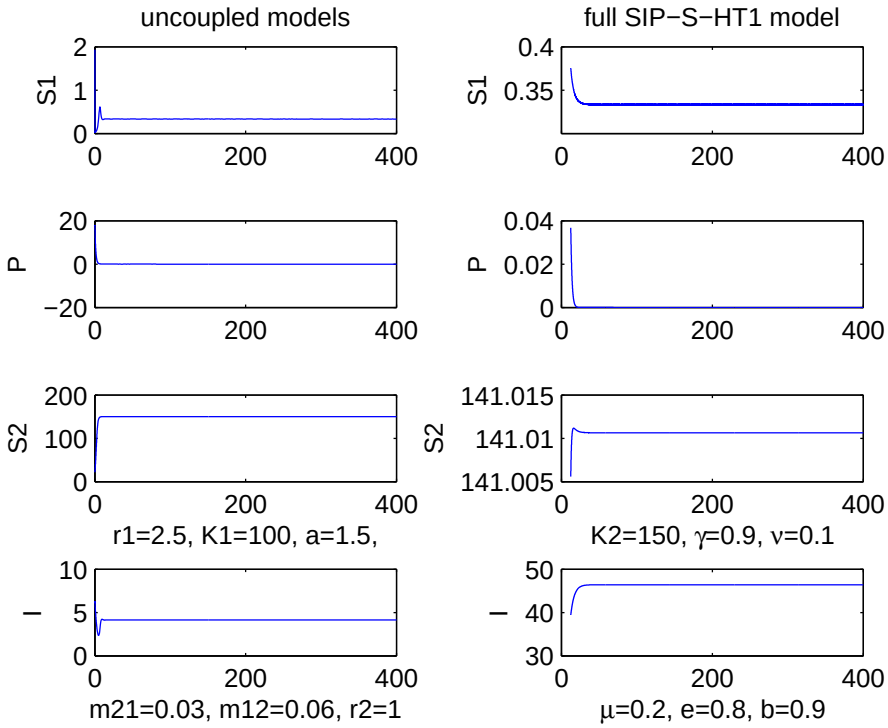

Figure 11: The predator-free equilibrium obtained for the case of *Ovis canadensis* or *Strix occidentalis* for the parameter values $r_1 = 2.5$, $K_1 = 100$, $a = 1.5$, $m_{21} = 0.03$, $m_{12} = 0.06$, $r_2 = 1$, $K_2 = 150$, $\gamma = 0.9$, $\nu = 0.1$, $\mu = 0.2$, $e = 0.8$, $b = 0.9$.

$m_{21} = 0.3$, $m_{12} = 0.5$, we notice that the predators get an increase of 47%, while the sound prey in the refuge experience a 415-fold increase. Note that in these last conditions, an increase of the disease incidence to $\gamma = 0.9$ does not affect the final equilibrium values.

Finally, for the set of parameter values $K_1 = 10$, $a = 4.5$, $m_{21} = 3$, $m_{12} = 5$, $K_2 = 150$, $\gamma = 0.25$, $\nu = 0.7$, $\mu = 0.2$, $e = 0.2$, $b = 0.2$ we obtain the disease-free equilibrium. Lowering the emigration rate from patch 2 to $m_{12} = 0.01$, the metaecoepidemic model does exhibit the same behavior as the single patch 1 system. Allowing instead a much larger m_{12} than m_{21} produces only much lower levels of susceptibles in patch 2; they are about half.

8.1 Results interpretation

As for the SIP-S Holling type II case, coexistence of the ecosystem cannot be ensured in general. Again, all the populations in the model could collapse. In the same way of the SI-SP models, Q_1 becomes unstable if $b > e$, but R_1 could still be stabilized. The disease cannot be eradicated from the ecosystem, keeping both predators and prey thriving in both patches, as it happens also in the corresponding Holling type II model. The stability of the predator-free equilibrium can still be ensured even if $b > e$, with similar sufficient conditions as in the nonlinear model (23).

9 Conclusion

Tables 1 and 2 contain respectively the summary of our findings. In the first one, we provide an interpretation of the equilibria, in the second one we collect all the feasibility and stability conditions of the various models.

In all these variants of the basic metaecoepidemic model with migration allowed only for sound prey, it is possible that the system collapses, under particularly unfavorable conditions, while this is excluded for each single patch subsystem, either consisting of a pure predator-prey system or of a pure epidemic model. This has to be ascribed to the combined effects of the existence of the refuge and of the migration limited only to prey. Infection plays therefore no role for this result.

If the origin is made unstable, i.e. if the system is prevented from collapse, the prey can never be wiped out of the system, as it happens for the classical SP submodel. In this sense the existence of the refuge protects them.

For all these metaecoepidemic models, it is not possible to eradicate the disease, keeping all populations alive. If the origin and E_1 are unstable, the disease cannot be eradicated, in this case the refuge establishes the epidemics in the ecosystem and the predators may or may not survive in this environment. The possible stable equilibria for the systems are the predator-free one, the predator- and disease-free point and the coexistence of the whole ecosystem. Thus the predators can be wiped out: for this to occur a sufficient condition is that the susceptible prey at equilibrium are above both patches' half carrying capacities for the SI-SP model and the sound prey in the SP patch be bounded above. The disease does seem to play only a marginal role for the predator-free equilibrium stability, in the sense that its basic reproduction number must be bounded above, (15).

Note that in the Holling type I models the disease- and predator-free equilibria can be stabilized, under somewhat more relaxed conditions than

the one of the corresponding equilibria for the Holling type II models.

There is a substantial difference between the two types of dynamics. The possibility of eradicating the disease in the SI subsystem with the Holling type II model, i.e. small ρ, entails possibly a large ρ_m, thereby making the eradication of the disease and the predators together in the metaecoepidemic model more unlikely. In the Holling type I system instead, a smaller ρ makes the corresponding equilibrium more prone to be unstable. So the effect is similar to what happens in the SI-only-patch model. Predators' disappearance in the pure SP submodel, due to a small σ, favors also the stability of E_1 in the HT-II model. The corresponding situation in the HT-I systems can be described as follows: in the SP single patch model, predators disappear if the prey carrying capacity is below a critical value, see (27), and a similar condition involving rather the prey population at the equilibrium must hold for the corresponding equilibria U_1 and R_1 in the metaecoepidemic model.

Comparing the two SIP-S models, we immediately see that both do not sustain coexistence of all subpopulations. Therefore the existence of a safety refuge for the prey surprisingly becomes a negative factor from the biodiversity point of view. Here too the disease alone cannot be eradicated, while preserving the other populations. Therefore the refuge acts as a tool for keeping the disease endemic in the system and this is due only to demographic effects.

Equilibrium Q_1, i.e. the disease- and predator-free one, in HT-II has a more stringent condition on the prey size in the SIP patch (compare (24) and (13)) and a more relaxed one for the prey in the safety refuge. If $b > e$ then Q_1 becomes unstable, but R_1 could still be stabilized in HT-I and the larger the gap between these parameters, the better is for stability of R_1. In fact the stability of the predator-free equilibrium in HT-I can still be ensured even if $b > e$, with similar sufficient conditions as in the HT-II model (23).

Finally, we remark here a major difference between simple ecoepidemic HT-II models and metaecoepidemic HT-II systems of both SI-SP and SIP-S kinds: the parameter a plays no role whatsoever in the analytical conditions leading to Hopf bifurcations, as instead it has been observed in the purely ecoepidemic system, [10]. Thus the predators' hunting rate in the metaecoepidemic model cannot be the cause of stable cyclic populations oscillations.

In our sample simulations we have further shown that demographic oscillations can either be carried through to the epidemics patch, or alternatively they can be damped in the metaecoepidemic model. In other cases the two simpler decoupled models show the same behavior as the metaecoepidemic one. The two decoupled models can lead to a predator-free equilibrium; but in presence of the refuge, with the same parameter values, a disease-free

equilibrium can instead be obtained.

Table 1: Summarizing Table of Equilibria: Interpretation

Model	Equilibrium	Interpretation
SP HT-II	Z_0	system collapse
1 patch	Z_1	prey-only
	Z_2	predator-prey coexistence
SIS	W_0	system collapse
1 patch	W_1	susceptible-only
	W_2	endemic disease
SP-SI HT-II	E_0	system collapse
	E_1	prey-only in 2 patches
	E_2	disease-free
	E_3	predator-free
	E_4	coexistence
SIP-S HT-II	Q_0	system collapse
	Q_1	prey-only in 2 patches
	Q_2	disease-free
	Q_3	predator-free
SP HT-I	Y_0	system collapse
1 patch	Y_1	predator-free
	Y_2	coexistence
SP-SI HT-I	U_0	system collapse
	U_1	prey-only in 2 patches
	U_2	disease-free
	U_3	predator-free
	U_4	coexistence
SIP-S HT-I	R_0	system collapse
	R_1	prey-only in 2 patches
	R_2	disease-free
	R_3	predator-free

Appendix

Proof of boundedness for model (1).

On summing the equations (1), for an arbitrary $0 < \eta < \min\{b, \mu\}$, we have

$$\frac{d\Pi}{dt}+\eta\Pi \leq (r_1+\eta)S_1-\frac{r_1S_1^2}{K_1}+(r_2+\eta)S_2-\frac{r_2S_2^2}{K_2} \leq (r_1+\eta)^2\frac{K_1}{4r_1}+(r_2+\eta)^2\frac{K_2}{4r_2} \equiv M$$

so that $\dot{\Pi} \leq -\eta\Pi + M$ and the solutions are ultimately bounded, $\Pi(t) \leq M\eta^{-1} \equiv M^*$ for every $t \geq 0$.

Equilibria of (1), their stability and bifurcations.

The equilibrium E_1 is obtained by intersecting the two parabolae one gets from the first two equations of (1), namely

$$S_2 = \varphi(S_1) \equiv \frac{S_1}{m_{12}}\left[m_{21} - r_1\left(1-\frac{S_1}{K_1}\right)\right],$$
$$S_1 = \psi(S_2) \equiv \frac{S_2}{m_{21}}\left[m_{12} - r_2\left(1-\frac{S_2}{K_2}\right)\right].$$

They meet at the origin, and have another intersection in the first quadrant if their other roots are positive and a suitable condition on their slopes at the origin is satisfied; these in turn yield the feasibility conditions of E_1, (8).

Stability

The Jacobian of the system (1) is

$$J = \begin{pmatrix} J_{11} & m_{12} & 0 & -\frac{aS_1}{H+S_1} \\ m_{21} & J_{22} & \nu-\gamma S_2 & 0 \\ 0 & \gamma I & \gamma S_2-\mu-\nu & 0 \\ \frac{ePH}{(H+S_1)^2} & 0 & 0 & \frac{eS_1}{H+S_1}-b \end{pmatrix} \tag{40}$$

with

$$J_{11} = r_1 - 2\frac{r_1}{K_1}S_1 - m_{21} - \frac{aPH}{(H+S_1)^2}, \quad J_{22} = r_2 - 2\frac{r_2}{K_2}S_2 - m_{12} - \gamma I.$$

At E_1 we find the eigenvalues

$$\gamma S_2^{(1)} - \mu - \nu, \quad \frac{eS_1^{(1)} - bH - bS_1^{(1)}}{H + S_1^{(1)}} \tag{41}$$

and those of the reduced matrix $J_{[1,2;1,2]}$, where the notation emphasizes the rows and columns of the original matrix J that are taken, thus

$$J_{[1,2;1,2]} = \begin{pmatrix} r_1 - 2\frac{r_1}{K_1}S_1^{(1)} - m_{21} & m_{12} \\ m_{21} & r_2 - 2\frac{r_2}{K_2}S_2^{(1)} - m_{12} \end{pmatrix}. \tag{42}$$

Using the Routh-Hurwitz conditions, combining with the earlier eigenvalues, stability occurs if and only if (13) and (14) hold.

For E_2 one eigenvalue is $\gamma S_2^{(2)} - \mu - \nu$. The other ones are roots of the following cubic, where by $J_{ik}^{(2)}$ we denote the ik element of the Jacobian J evaluated at E_2,

$$\sum_{i=0}^{3} a_i \lambda^i = 0, \tag{43}$$

with

$$a_2 = -(J_{11}^{(2)} + J_{22}^{(2)}), \quad a_1 = J_{11}^{(2)} J_{22}^{(2)} - m_{12}m_{21} - \frac{abPH}{(H + S_1^{(2)})^2}, \quad a_0 = \frac{abPHJ_{22}^{(2)}}{(H + S_1^{(2)})^2}. \tag{44}$$

Now the Routh-Hurwitz criterion for stability requires that $a_0 > 0$, $a_2 > 0$, $a_1 a_2 > a_0 a_3 = a_0$. From the definition of a_0 it follows then that we must have $J_{22}^{(2)} > 0$, and from the one of a_2, also $J_{11}^{(2)} < -J_{22}^{(2)} < 0$. Hence $J_{11}^{(2)} J_{22}^{(2)} < 0$ implying $a_1 < 0$, so that the remaining condition $a_1 a_2 > a_0$ is impossible to verify. Thus E_2 is unconditionally unstable.

The remaining eigenvalues of J at E_3 are the roots of the characteristic equation (43) which has coefficients

$$a_0 = -\mu\gamma I J_{11}^{(3)}, \quad a_1 = J_{11}^{(3)} + J_{22}^{(3)} + \mu\gamma I - m_{12}m_{21}, \quad a_2 = -(J_{11}^{(3)} + J_{22}^{(3)}),$$

and to satisfy the Routh-Hurwitz conditions, we need to require

$$J_{11}^{(3)} < 0, \quad J_{22}^{(3)} < -J_{11}^{(3)}$$

for $a_0 > 0$ and $a_2 > 0$ respectively, while the remaining condition becomes

$$J_{11}^{(3)}[J_{11}^{(3)} J_{22}^{(3)} - m_{12}m_{21}] + J_{22}^{(3)}[J_{11}^{(3)} J_{22}^{(3)} + \mu\gamma I - m_{12}m_{21}] < 0, \tag{45}$$

i.e.

$$J_{22}^{(3)} \mu\gamma I < (J_{11}^{(3)} + J_{22}^{(3)})[J_{11}^{(3)} J_{22}^{(3)} - m_{12}m_{21}]. \tag{46}$$

It is easily seen that (46) is impossible if $J_{22}^{(3)} > 0$, hence letting $J_{11}^{(3)} = A - m_{21}$, $J_{22}^{(3)} = B - m_{12}$, the conditions (45) and (46) are further implied by

$$AB - Am_{12} - Bm_{21} > 0$$

which is ensured if we take $A, B < 0$ i.e. by (16).

At E_4 the characteristic equation is a quartic,

$$\sum_{i=0}^{4} b_i \lambda^i = 0, \tag{47}$$

with

$$b_0 = \gamma\mu I^{(4)} \frac{aeHP^{(4)}S_1^{(4)}}{(H+S_1^{(4)})^3}, \quad b_1 = -\left[J_{22}^{(4)} \frac{aeHP^{(4)}S_1^{(4)}}{(H+S_1^{(4)})^3} + \gamma\mu I^{(4)} J_{11}^{(4)} \right],$$

$$b_2 = J_{11}^{(4)} J_{22}^{(4)} - m_{12}m_{21} + \frac{aeHP^{(4)}S_1^{(4)}}{(H+S_1^{(4)})^3} + \gamma\mu I^{(4)}, \quad b_3 = -\left(J_{11}^{(4)} + J_{22}^{(4)} \right).$$

The Routh-Hurwitz conditions ensure stability if

$$b_3 > 0, \quad b_0 > 0, \quad b_2 b_3 > b_1, \quad b_1(b_2 b_3 - b_1) > b_0 b_3^2.$$

Thus the first condition holds always and the last condition requires instead $J_{11}^{(4)} + J_{22}^{(4)} < 0$. The remaining ones give (17).

Bifurcations

The characteristic equation evaluated at E_3 is a cubic. In order to have a Hopf bifurcation it needs to be split as follows

$$\sum_{i=0}^{3} a_i \lambda^i = (\lambda^2 + \widehat{B}^2)(\lambda^2 + \widehat{C}) = 0$$

so that by expanding and equating coefficients of like powers, we find the relation

$$a_0 = a_1 a_2 \tag{48}$$

that must be satisfied, and $\widehat{B} = \sqrt{b_1}$, $\widehat{C} = a_2$. Writing then $J_{11} = A - m_{12}$, $J_{22} = B - m_{21}$ one can observe that $S_1^{(3)}$ and A are both independent of the parameters r_2 and K_2. Choosing as a bifurcation parameter the former, (48) can be expressed as a quadratic equation in r_2,

$$\sum_{k=0}^{2} r_2^k \eta_k = 0, \quad R = -\frac{\gamma}{\mu}\left(m_{21}S_1^{(3)} - m_{12}\frac{\mu+\nu}{\mu} \right) \tag{49}$$

$$\eta_2 = (A - m_{21}) \left(\frac{\nu^2 - \mu^2 - \nu\gamma K_2}{\gamma\mu K_2} \right)^2$$

$$+\left(1-\frac{\nu+\mu}{\gamma K_2}\right)(\nu+\mu)\left[\left(1-2\frac{S_1^{(3)}}{K_1}\right)-\frac{\nu+\mu}{\mu}\left(1-\frac{\nu+\mu}{\gamma K_2}\right)\right],$$

$$\eta_1=\left[2(A-m_{21})R-(A-m_{21})^2-m_{12}m_{21}\right]\frac{\nu^2-\mu^2-\nu\gamma K_2}{\gamma\mu K_2}$$

$$+R\left[\left(1-\frac{\nu+\mu}{\gamma K_2}\right)(\nu+\mu)-\left(1-2\frac{S_1^{(3)}}{K_1}\right)+\frac{\nu+\mu}{\mu}\left(1-\frac{\nu+\mu}{\gamma K_2}\right)\right],$$

$$\eta_0=(A-m_{21})R^2-(A-m_{12}-m_{21})Am_{12}-R^2+\left[(A-m_{21})^2-m_{12}m_{21}\right]R.$$

Thus a sufficient condition to ensure the existence of one positive root, i.e. one feasible value for the bifurcation parameter $r_2^\dagger$, is to impose

$$\eta_2\eta_0<0. \tag{50}$$

At E_4 we can proceed in a similar fashion, expanding (47)

$$\sum_{i=0}^{4}b_i\lambda^i=(\lambda^2+\omega^2)(\lambda+\rho)(\lambda+\sigma)=0$$

and equating coefficients to get the condition

$$b_1^2+b_3^2b_0=b_1b_2b_3. \tag{51}$$

An appropriate bifurcation parameter can once more be found to be again r_2. Expansion of the condition in terms of the system parameters leads this time to a cubic equation,

$$\sum_{k=0}^{3}r_2^k\phi_k=0. \tag{52}$$

The parameter expressions are very involved and therefore omitted. Once again a feasible value r_2^* for the Hopf bifurcation to occur is obtained by imposing

$$\phi_3\phi_0<0. \tag{53}$$

Stability and bifurcations for the system (23)

The Jacobian of the system differs slightly in the structure from (40),

$$J=\begin{pmatrix} J_{11} & m_{12} & \nu-\gamma S_1 & -\frac{aS_1}{H+S_1} \\ m_{21} & J_{22} & 0 & 0 \\ \gamma I & 0 & \gamma S_1-\mu-\nu & 0 \\ \frac{ePH}{(H+S_1)^2} & 0 & 0 & \frac{eS_1}{H+S_1}-b \end{pmatrix} \tag{54}$$

with

$$J_{11} = r_1 - 2\frac{r_1}{K_1}S_1 - m_{21} - \frac{aPH}{(H+S_1)^2} - \gamma I, \quad J_{22} = r_2 - 2\frac{r_2}{K_2}S_2 - m_{12}.$$

At Q_1 we find the eigenvalues

$$\gamma S_1^{(1)} - \mu - \nu, \quad \frac{(e-b)S_1^{(1)} - bH}{H + S_1^{(1)}}$$

the second of which coincides with the second eigenvalue of E_1 (41), and those remaining are also the other ones of E_1 coming from the quadratic of the same reduced matrix (42). Thus using the Routh-Hurwitz conditions, combining with the earlier eigenvalues, stability occurs if and only if conditions (14) hold, together with (24).

For Q_2 one eigenvalue differs from the corresponding one of E_2, namely it is $\gamma S_1^{(2)} - \mu - \nu$. The other ones are the roots of the cubic (43), with the same coefficients (44) as for the equilibrium E_2 of (1). Hence unconditional instability follows.

For the bifurcation at Q_3, we proceed once again as for E_3 remarking that (48) in this case leads also to a quadratic equation, this time in the bifurcation parameter r_1,

$$\sum_{i=0}^{2} \theta_i r_1^i = 0.$$

In this case, we find

$$\theta_2 = -J_{22}\left[\left(1 - 2\frac{\widetilde{S_2^{(3)}}}{K_2}\right) - \frac{\gamma}{\mu}\left(1 - \frac{\widetilde{S_1^{(3)}}}{K_1}\right)\right]^2$$
$$-\gamma\left(1 - 2\frac{\widetilde{S_2^{(3)}}}{K_2}\right)\left(1 - \frac{\widetilde{S_1^{(3)}}}{K_1}\right)\widetilde{S_1^{(3)}} + \frac{\gamma^2}{\mu}\left(1 - \frac{\widetilde{S_1^{(3)}}}{K_1}\right)^2\left(\widetilde{S_1^{(3)}}\right)^2$$

and in view of (16) and the fact that $J_{22} < 0$, requiring then

$$\frac{1}{2}K_1 < \widetilde{S_1^{(3)}} < K_1 \tag{55}$$

ensures that $\theta_2 > 0$, so that to have a feasible value for the Hopf bifurcation parameter $r_1^\dagger$ it is enough to require

$$\theta_0 < 0. \tag{56}$$

Stability of model (26)

The Jacobian differs slightly from (40), namely some of its entries are different,

$$J_{11} = r_1 - 2\frac{r_1}{K_1}S_1 - m_{21} - aP, \quad J_{14} = -aS_1, \quad J_{41} = eP.$$

At U_4 we get a quartic, with coefficients

$$\widehat{a_0} = ae\gamma\widehat{P^{(4)}}\widehat{S_1^{(4)}}\widehat{I^{(4)}} > 0, \quad \widehat{a_1} = -(\gamma\mu\widehat{I^{(4)}}\widehat{J_{11}^{(4)}} + ae\widehat{P^{(4)}}\widehat{S_1^{(4)}}\widehat{J_{22}^{(4)}}),$$
$$\widehat{a_2} = \widehat{J_{11}^{(4)}} + \widehat{J_{22}^{(4)}} + \gamma\mu\widehat{I^{(4)}} + m_{12}m_{21} + ae\widehat{P^{(4)}}\widehat{S_1^{(4)}}, \quad \widehat{a_3} = -(\widehat{J_{11}^{(4)}} + \widehat{J_{22}^{(4)}}).$$

For stability we need

$$-(J_{11} + J_{22}) > 0, \quad \widehat{a_2}\widehat{a_3} > \widehat{a_1}, \quad \widehat{a_1}(\widehat{a_2}\widehat{a_3} - \widehat{a_1}) > \widehat{a_0}\widehat{a_3}^2. \tag{57}$$

If $J_{11}, J_{22} < 0$ the first two conditions (57) are satisfied.

Acknowledgments. The author expresses his deep gratitude to his colleagues Professor Guido Badino, Dr. Francesca Bona, Dr. Simona Bonelli and Dr. Luca Casacci for very useful discussions and for providing the biological reference material mentioned in the paper. The author also thanks the referees for their constructive criticism.

References

[1] G. Amori, L. Contoli, A. Nappi, (2008) Fauna d'Italia, (Italian Fauna) Mammalia III Erinaceomorpha, Soricomorpha, Lagomorpha, Rodentia, Calderini Bologna, Italy.

[2] O. Arino, A. El Abdllaoui, J. Mikram, J. Chattopadhyay (2004) Infection on prey population may act as a biological control in ratio-dependent predator-prey model, Nonlinearity, 17, 1101–1116.

[3] P. R. Atsatt (1981) Lycaenid butterflies and ants: selection for enemy-free space, The American Naturalist 118(5) 638–654.

[4] W. W. Becklund, C. M. Senger (1967) Parasites of *ovis canadiensis* in Montana, with a checklist of the internal and external parasites of the Rocky Mountain bighorn sheep in North America, The J. of Parasitology 53(1) 157-165.

[5] E. Beltrami, T. O. Carroll (1994) Modelling the role of viral disease in recurrent phytoplankton blooms, J. Math. Biol. 32, 857–863.

[6] H. L. Black (1974) A North Temperate Bat Community: Structure and Prey Populations, Journal of Mammalogy 55, 138–157.

[7] L. Boitani, S. Lovari S., A. Vigna Taglianti (2003) Fauna d'Italia, (Italian Fauna) Mammalia III Carnivora - Artiodactyla, Calderini, Bologna, Italy.

[8] V. Brack, Jr., R. K. LaVal (1985) Food Habits of the Indiana bat in Missouri, Journal of Mammalogy 66, 308–315.

[9] J. Chattopadhyay, O. Arino (1999) A predator-prey model with disease in the prey, Nonlinear Analysis, 36, 747–766.

[10] J. Chattopadhyay, R. R. Sarkar, G. Ghosal (2002) Removal of infected prey prevent limit cycle oscillations in an infected prey–predator system — a mathematical study, Ecological Modelling 156, 113–121.

[11] R. J. Cowie, S. A. Hinsley (1988) Feeding Ecology of Great Tits (*Parus major*) and Blue Tits (*Parus caeruleus*), breeding in suburban gardens, Journal of Animal Ecology 57, 611–626.

[12] J. T. Cronin (2003) Movement and spatial population structure of a prairie planthopper, Ecology, 84, 1179–1188.

[13] T. Dewey, L. Ballenger (1999) "*Ovis canadensis*" (Online), Animal Diversity Web, Accessed December 04, 2009 at http://animaldiversity.ummz.umich.edu/site/accounts/information/Ovis_canadensis.html.

[14] M. Festa-Bianchet (1999) Bighorn sheep, in D.E. Wilson, S. Ruff (Editors) The Smithsonian book of North American mammals, Washington: Smithsonian Institution Press, 348-350.

[15] H. I. Freedman (1990) A model of predator-prey dynamics as modified by the action of parasite, Math. Biosci. 99, 143–155.

[16] I. D. Gauld (1988) Evolutionary patterns of host utilization by ichneumonoid parasitoids (Hymenoptera: Ichneumonidae and Braconidae), Biological Journal of the Linnean Society 35, 351–377.

[17] E. J. Gustafson, R. H. Gardner (1996) The effect of landscape heterogeneity on the probability of patch colonization, Ecology 77, 94–107.

[18] R. J. Gutiérrez, S. Harrison (1996) Applying metapopulation theory to spotted owl management: a history and critique, in D. R. McCollough (Ed.) Metapopulations and wildlife conservation, Washington: Island Press, 167–185.

[19] K. P. Hadeler, H. I. Freedman, (1989), Predator-prey populations with parasitic infection, J. Math. Biology 27, 609–631.

[20] I. Hanski (1985) Single-species spatial dynamics may contribute to long-term rarity and commonness, Ecology 66, 335–343.

[21] I. Hanski, M. Gilpin (Ed.s) (1997) Metapopulation biology: ecology, genetics and evolution, London: Academic Press.

[22] I. Hanski, A. Moilanen, T. Pakkala, M. Kuussaari (1996) Metapopulation persistence of an endangered butterfly: a test of the quantitative incidence function model, Conservation Biology 10, 578–590.

[23] S. Harrison, A. Taylor (1997) Empirical evidence for metapopulation dynamics, in I. Hanski, M. Gilpin (Ed.s) Metapopulation biology: ecology, genetics and evolution. London: Academic Press, 27–42.

[24] E. P. Hoberg, G. S. Miller, E. Wallner-Pendleton, O. R. Hedstrom (1989) Helminth parasites of northern spotted owls (*Strix occidentalis caurina*) from Oregon, Journal of Wildlife Diseases, 25(2), 246-251

[25] M. Holyoak (2000) Effects of nutrient enrichment on predator-prey metapopulation dynamics, J. Anim. Ecol. 69, 985-997.

[26] H. Inaba, H. Nishiura (2008) The basic reproduction number of an infectious disease in a stable population: the impact of population growth rate on the eradication threshold, Math. Model. Nat. Phenom. 3 (7), 194–228.

[27] P. Kareiva (1990) Population Dynamics in Spatially Complex Environments: theory and data, Philosophical Transactions of the Royal Society of London B 330, 175–90.

[28] P. Kerr, G. McFadden (2002) Immune responses to Myxoma Virus, Viral Immunology 15(2), 229–246.

[29] P. J. Kerr, J. C. Merchant, L. Silvers, G. M. Hood, A. J. Robinson (2003) Monitoring the spread of myxoma virus in rabbit *Oryctolagus cuniculus* populations on the southern tablelands of New South Wales, Australia. II. Selection of a strain of virus for release, Epidemiol. Infect. 130, 123–133.

[30] G. Lei, I. Hanski (1997) Metapopulation structure of *Cotesia melitaearum*, a parasitoid of the butterfly *Melitaea cinxia*, Oikos 78, 91–100.

[31] R. Levins (1969) Some demographic and genetic consequences of environmental heterogeneity for biological control, Bulletin of the Entomological Society America 15, 237–240.

[32] H. Malchow, S. Petrovskii, E. Venturino (2008) Spatiotemporal patterns in Ecology and Epidemiology, Boca Raton: CRC.

[33] J. C. Merchant, P. J. Kerr, N. G. Simms, G. M. Hood, R. P. Pech, A. J. Robinson (2003) Monitoring the spread of myxoma virus in rabbit it Oryctolagus cuniculus populations on the southern tablelands of New South Wales, Australia. III. Release, persistence and rate of spread of an identifiable strain of myxoma virus, Epidemiology and Infection 130(1), 135–147.

[34] A. Moilanen, I. Hanski (1995) Habitat destruction and competitive coexistence in a spatially realistic metapopulation model, Journal of Animal Ecology 64, 141-144.

[35] A. Moilanen, A. Smith, I. Hanski (1998) Long-term dynamics in a metapopulation of the American pika, American Naturalist 152, 530–542.

[36] L. D. Rothman, J. H. Myers (1996) Debilitating effects of viral diseases on host lepidoptera J. of Invertebrate Pathology 67, 1–10.

[37] M. J. Scoble (1992) The lepidoptera: form, function, and diversity, Oxford: Oxford University Press.

[38] R. L., Schooley, L. C. Branch (2007) Spatial heterogeneity in habitat quality and cross-scale interactions in metapopulations, Ecosystems 10, 846-853.

[39] Semeyn, E. (1999) "*Strix occidentalis*" (On-line), Animal Diversity Web. Accessed December 04, 2009 at http://animaldiversity.ummz.umich.edu/site/accounts/information/Strix_occidentalis.html.

[40] D. Tilman, P. Kareiva (Ed.s) (1997) Spatial ecology, Princeton: Princeton University Press.

[41] E. Venturino (1994) The influence of diseases on Lotka Volterra systems. *Rocky Mountain Journal of Mathematics* 24, 381–402.

[42] E. Venturino (1995) Epidemics in predator-prey models: disease in prey, in O. Arino, D. Axelrod, M. Kimmel, M. Langlais (Ed.s) Mathematical Population dynamics 1: Analysis of heterogeneity, Winnipeg: Wuertz, 381–393.

[43] E. Venturino (2001) The effects of diseases on competing species, Math. Biosc. 174, 111–131.

[44] E. Venturino (2002) Epidemics in predator-prey models: disease in the predators, IMA Journal of Mathematics Applied in Medicine and Biology 19, 185–205.

[45] E. Venturino (2007) How diseases affect symbiotic communities, Math. Biosc. 206, 11–30.

[46] J. A. Wiens (1996) Wildlife in patchy environments: metapopulations, mosaics, and management, in D. R. McCullough (Ed.) Metapopulations and Wildlife Conservation, Washington: Island Press, 53–84.

[47] J. A. Wiens (1997) Metapopulation dynamics and landscape ecology, in I. A. Hanski, M. E. Gilpin (Ed.s), San Diego: Academic Press, 43-62.

[48] R. T.Williams, J. D.Dunsmore,I.Parer (1972) Evidence for the Existence of Latent Myxoma Virus in Rabbits (*Oryctolagus cuniculus (L.)*) Nature 238, 99–101.

[49] J. Wu (1994) Modeling dynamics of patchy landscapes: linking metapopulation theory, landscape ecology and conservation biology, in Yearbook in Systems Ecology (English edition) Beijing: Chinese Academy of Sciences.

Table 2: Summarizing Table of Equilibria: existence and stability conditions.

Model	Equilibrium	feasibility	stability	sufficient for stability
SP HT-II	Z_0	always	unstable	
1 patch	Z_1	always	(3) i.e. $\sigma < 1$	
	Z_2	(2) i.e. $\sigma > 1$, $e > b$	(4)	
SIS	W_0	always	unstable	
1 patch	W_1	always	(6) i.e. $K_2 < \rho$	
	W_2	(5) i.e. $K_2 > \rho$	(7)	
SP-SI HT-II	E_0	always	(12)	
	E_1	(8)	(13), (14)	
	E_2	(9)	unstable	
	E_3	(10)	(15), (45), (46)	(15), (16)
	E_4	$e > b$, (11)	(17)	
SIP-S HT-II	Q_0	always	(12)	
	Q_1	(8)	(14), (24)	
	Q_2	(9)	unstable	
	Q_3	$\widetilde{S_2^{(3)}} \geq 0$, $\widetilde{I^{(3)}} \geq 0$	(15), (25)	(15), (16)
SP HT-I	Y_0	always	unstable	
1 patch	Y_1	always	(27) i.e. $K_1 < \frac{b}{e}$	
	Y_2	(28) i.e. $K_1 > \frac{b}{e}$	always	
SP-SI HT-I	U_0	always	(12)	
	U_1	(8)	(14), (35)	
	U_2	(31)	unstable	
	U_3	(10)	(36), (45), (46)	(36), (16)
	U_4	(34)	(57)	(37)
SIP-S HT-I	R_0	always	(12)	
	R_1	(8)	(14), (39)	
	R_2	(31)	unstable	
	R_3	$\widetilde{S_2^{(3)}} \geq 0$, $\widetilde{I^{(3)}} \geq 0$	(36), (25)	(16), (25)