A Discrete Predator-Prey Ecoepidemic Model.

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Abstract. In this work we present a discrete predator-prey ecoepidemic model. The predator-prey interactions are represented by a discrete Leslie-Gower model with prey intra-specific competition. The disease dynamics follows a discrete SIS epidemic model with frequency-dependent transmission. We focus on the case of disease only affecting prey though the case of a parasite of the predators is also presented. We assume that parasites provoke density- and trait-mediated indirect interactions in the predator-prey community that occur on a shorter time scale. This is included in the model considering that in each time unit t here exist a number k of episodes of epidemic changes followed by a single episode of demographic change, all of them occurring separately. The aim of this work is examining the effects of parasites on the long-term prey-predators interactions. These interactions in the absence of disease are governed by the Leslie-Gower model. In the case of endemic disease they can be analyzed through a reduced predator-prey model which summarizes the disease dynamics in its parameters. Conditions for the disease to drive extinct the whole community are obtained. When the community keeps stabilized different cases of the influence of disease on populations sizes are presented.

Keywords and phrases: Predator-prey system; SIS epidemic model, discrete-time system; time scales

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1. Introduction.

Parasites play a role in many processes within host populations and communities that influence species coexistence and ecosystem function [14]. Parasites account for a substantial proportion of the biomass of certain ecosystems and can alter food web structure. The study of these broadly extended systems requires the combination of community ecology and parasitology elements, approach that is sometimes called ecosystem parasitology [14]. The mathematical models representing these biological systems are studied in mathematical ecology and mathematical epidemiology [7], respectively, whereas the mathematical counterpart of ecosystem parasitology could be called mathematical eco-epidemiology. The papers by Anderson and May [4] and Hadeler and Freedman [13] in the mid eighties are the two key eco-epidemiological papers, that appeared when this denomination was still not used. During the nineties some other papers on eco-epidemiology started to be published, most of them following one of those two papers. A suggestion of the late O. Arino to J. Chattopadhyay [9] is at the origin of the name of the

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field: *ecoepidemics*. Since then the field of mathematical eco-epidemiology has grown enormously, see the recent review [21] by E. Venturino, one of its early pioneers.

The studies of communities focus on how species interactions affect the populations densities. These interactions can be direct, as the case of predation or interference competition, where individuals of both species have direct effects on each other. At the same level of importance are the indirects interactions, those arising when the impact of one species on another is mediated by the action of a third. Indirect interactions are classified into trait- and density-mediated. Parasites are a source of both classes of indirect interactions in a community. Killing their hosts provokes a density-mediated indirect interaction while changing host physiology could yield a trait-mediated one [14]. Trait-mediated effects can occur on shorter timescales than direct effects being thus crucial in structuring communities [6].

In this work we examine the effects of parasites on interactions between species at different trophic levels: predator-prey interactions. There are different ways in which parasites can enter into a predator-prey module and their influence on population dynamics will certainly depend on their position in the module. We focus on parasitism on the prey species, though we also briefly present the case of the predators being the hosts of the parasites. On the other hand, we do not consider the case of both species hosting the parasites. A relevant feature of these modules is the degree of predator specialisation. We assume a specialist predator, therefore more sensitive to the impact of parasitism on the prey. Concerning the indirect effects of parasitism, we reflect the density-mediated ones considering different intrinsic growth rates for susceptible a infected preys, whereas the trait-mediated ones are included by assuming different intra-species competitive abilities and selective predation for or against infected prey.

The theory of predator-prey interactions with infected prey is well developed for continuous time. Apart from the aforementioned seminal works [4, 9], Venturino already in 1994, [22], considered a Lotka-Volterra prey-predator model and studied the effects on the community of a disease affecting either the prey or the predator population. Since then an important number of works have been devoted to the subject, see the references in [21].

In discrete time we only found a work studying a predator-prey ecoepidemic model, [15]. In it a discrete predator-prey model with disease in prey is obtained by discretization of a continuous model and studied with the aim of finding more complex dynamical behaviour than in its continuous counterpart. In this work we present a discrete predator-prey ecoepidemic model that is built up from a simple discrete predator-prey model, the Leslie-Gower model [18], and the disease is introduced by means of a discrete SIS epidemic model with frequency-dependent transmission [1]. One of the main drawbacks of continuous models is that all the processes involved in population dynamics such as births, growth, interactions and infection must operate together at all times [3]. In discrete models it is natural to break time steps up into distinct stages so that each process occurs separately. To take into account that the disease mediated effects on the predator-prey community occur on a shorter timescale we assume that each time unit contains a single episode of demographic change and a number k of epidemic changes, all of them occurring separately [16].

The proposed model takes the form of a three dimensional system of difference equations with two time scales. The two time scales are introduced in the system by letting, in each time unit, the fast process, the disease dynamics, act a number k of times followed by the slow process, the demographic dynamics, acting a single time. The construction of this kind of systems, together with a reduction method that simplifies their analysis, is reviewed by the authors in [8]. The application of the reduction method to the proposed system yields a two dimensional one representing a predator-prey model including the effects of the disease in its parameters. This latter system turns out to be more general than the initial Leslie-Gower one and, since we have not found it in the literature, it is studied in the appendix.

The long term behaviour of the solutions of the predator-prey model without disease is characterized by means of equilibrium points. It exhibits, depending on parameter values, three different steady states: extinction of the whole community, prey at their carrying capacity together with predator disappearance, and coexistence of both populations. The reduced system allows to perform an analogous analysis of the long term behaviour of the model with disease, obtaining the corresponding three different cases. The

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changes that the disease introduces in the community are thus observable in two different ways. The first one has to do with changing the kind of equilibrium point defining the asymptotic behaviour: conditions are found for the disease to drive extinct a community otherwise viable and the other way around, making viable an inviable community, in some cases of symbiotic parasites. If there is no change through disease in the kind of equilibrium point defining the stationary state of the community, the second way of detecting the effects of the disease is by comparing populations sizes and community structures: neat results of this kind are obtained in some particular cases.

The rest of this paper is organized as follows: Section 2 presents the discrete SIS epidemic model and its asymptotic behaviour in 2.1, introduces the discrete Leslie-Gower predator-prey model with prey intra-specific competition in 2.2, couples both models together to obtain the two time-scales discrete eco-epidemic model in 2.3, and builds up the reduced system by means of which the complete model is analyzed in 2.4; Section 3 focuses on the analysis of the influence of parasites on the dynamics of the predator-prey community; Section 4 briefly develops an analogous model with the disease affecting the predators; In Section 5, we collect some observations and conclusions; In the appendix the reduction of the complete system is justified (A.1) and the reduced systems are studied (A.2).

2. Presentation of the model.

The model describes in discrete time a predator-prey community with the prey affected by a disease which acts on a shorter time scale than the predator-prey dynamics. The time unit of the model is considered to be the one associated to its demographic part, typically one year. In this time unit, referred to as slow, there is a single episode of demographic change. On the other hand, the fact that pathogens exhibit outbreaks on short time scales, on the order of days or weeks, lead us to explicitly include a second time scale in the model allowing a number k of disease infection-recovery cycles in one slow time unit.

Time in the slow time unit is denoted t. The time step on which the outbreak is modelled is $\triangle$. $P(t)$, $N_S(t)$ and $N_I(t)$ represents the predators, the susceptible prey and the infected prey, respectively, at time t. Between time t and time $t+1$ we consider the disease dynamics acting sequentially k times followed by one demographic episode. To distinguish the different outbreaks episodes we use the notation $N_S(t+m\triangle)$ and $N_I(t+m\triangle)$ with $m = 1, \ldots, k$.

We now proceed to present the disease dynamics and the prey-predator model. This is followed by the construction of the complete model together with an associated reduced system that we use for its analysis.

2.1. Prey infection: SIS model.

Following [1] we propose the following discrete-time SIS epidemic model for the prey:

$$
\begin{aligned}
N_S(t+(m+1)\triangle) &= N_S(t+m\triangle)\left(1 - \frac{\beta N_I(t+m\triangle)}{N_S(t+m\triangle)+N_I(t+m\triangle)}\right) + \gamma N_I(t+m\triangle) \\
N_I(t+(m+1)\triangle) &= N_I(t+m\triangle)\left(1 + \frac{\beta N_S(t+m\triangle)}{N_S(t+m\triangle)+N_I(t+m\triangle)} - \gamma\right)
\end{aligned}
\tag{2.1}
$$

As a consequence of assuming a fast development of the disease, the number of contacts is supposed to be constant irrespective of the density of the population. This implies [5] that transmission is frequency-dependent, with β being the transmission coefficient. The recovery coefficient γ represents the fraction of infected individuals that recover in a unit of time.

The disease process is represented by the map

$$
\mathbf{F}(N_S, N_I) = \left(N_S - \frac{\beta N_S N_I}{N_S + N_I} + \gamma N_I, N_I + \frac{\beta N_S N_I}{N_S + N_I} - \gamma N_I\right)
\tag{2.2}
$$

so that the total effect of the k outbreaks episodes during one slow time unit is defined by its k-th iterate $\mathbf{F}^k$.

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The prey population size $N(t) := N_S(t) + N_I(t)$ at the beginning of a slow time unit remains constant, $N_S(t + m\triangle) + N_I(t + m\triangle) = N(t)$ $(m = 1, \ldots, k)$, till the next demographic episode at the end of the time unit.

Henceforth we assume that the following inequalities hold:

$$\gamma \leq 1 \text{ and } \beta < \left(1 + \sqrt{\gamma}\right)^2, \tag{2.3}$$

so that solutions of system (2.1) are positive for all positive initial conditions [1].

The asymptotic behaviour of the solutions of system (2.1) is studied in [1]. The basic reproduction number for this model is $R_0 = \beta/\gamma$. If $R_0 \leq 1$ then for any positive initial condition the number of infecteds monotonically decreases to 0, i.e., the positive solutions of (2.1) converge to the disease-free equilibrium $\mathbf{N}_0^* = (N, 0)$, where N is the (constant) prey population size. On the other hand, if $R_0 > 1$ the disease becomes endemic. Assuming, in addition to inequalities (2.3), that

$$\gamma < \beta \leq 2 + \gamma, \tag{2.4}$$

the positive solutions of (2.1) converge to an asymptotically stable endemic equilibrium:

$$\mathbf{N}_e^* = (N_S^*, N_I^*) = \left((\gamma/\beta)N, (1 - \gamma/\beta) N\right). \tag{2.5}$$

If $2 + \gamma < \beta < \left(1 + \sqrt{\gamma}\right)^2$ then the monotonic convergence to an endemic equilibrium disappears giving rise to period-doubling and chaotic behaviour.

As we will see in Section 2.4, in order to reduce the complete model we will need to make use of the equilibrium of the fast disease process. This equilibrium is expressed as the limit, $\bar{F}$, of the iterates of map F. According to the previous reasonings

$$\bar{\mathbf{F}}(N_S, N_I) := \lim_{k \to \infty} \mathbf{F}^k(N_S, N_I) = (\nu^* N, (1 - \nu^*)N), \tag{2.6}$$

with $N = N_S + N_I$, $\nu^* = 1$ if $\beta \leq \gamma$ and $\nu^* = \gamma/\beta = 1/R_0$ if $\gamma < \beta \leq 2 + \gamma$. Note that $\nu^* = 1$ implies that the disease is eradicated.

2.2. Demography: Predator-prey interaction.

The basic model we use to represent the interactions between the populations of preys, $N(t)$, and predators, $P(t)$, is the discrete predator-prey Leslie-Gower model with prey intra-specific competition:

$$\begin{aligned} N(t+1) &= \frac{a_1 N(t)}{1 + b_1 N(t) + c_1 P(t)} \\ P(t+1) &= \frac{a_2 P(t)}{1 + c_2 P(t)/N(t)} \end{aligned} \tag{2.7}$$

Parameters a_1 and b_1 are the growth rate and the intra-specific competition coefficient of the prey in the absence of predators, i.e, Beverton-Holt model with carrying capacity $(a_1 - 1)/b_1$ [2]. Parameter c_1 weights the effect of predation on prey growth rate and parameter a_2 is the predators growth rate in the case of infinitely abundant prey. Finally, parameter c_2 weights the ratio $P(t)/N(t)$, number of predators per prey, so that the larger c_2 is the greater the reduction of the predators growth rate [18].

As we show in Theorem (A.3) in the Appendix there are three possible asymptotic behaviours of the solutions of system (2.7) with positive initial conditions:

1. If $a_1 \leq 1$ then $\lim_{t \to \infty} (N(t), P(t)) = (0, 0)$.
2. If $a_1 > 1$ and $a_2 \leq 1$ then $\lim_{t \to \infty} (N(t), P(t)) = (\bar{n}, 0) = ((a_1 - 1)/b_1, 0)$.
3. If $a_1 > 1$ and $a_2 > 1$ then system (2.7) possesses a unique positive equilibrium point:

$$(n^*, p^*) = \left(\frac{(a_1 - 1)c_2}{b_1 c_2 + (a_2 - 1)c_1}, \frac{(a_1 - 1)(a_2 - 1)}{b_1 c_2 + (a_2 - 1)c_1}\right), \tag{2.8}$$

which is asymptotically stable.

2.3. The complete model.

The previous predator-prey model can be adapted to contemplate prey individuals classified into susceptible and infected. The trait-mediated indirect effects of parasites are considered in the growth and intra-specific competition of prey as well as in the predation related parameters. More specifically, we assume different intrinsic growth rates for susceptible, a_1^S, and infected, a_1^I, preys. We also distinguish four prey intra-specific competition coefficients: b_1^{SS}, b_1^{SI}, b_1^{IS} and b_1^{II}. Parameters c_1^S and c_1^I refer to the effect of predation on susceptible and infected prey. Finally, parameter d^I allows one to differentiate between susceptible and infected prey as resource for predators growth. $d^I = 1$ means that both types of prey contribute equally to predators growth, whereas $0 < d^I < 1$ (resp. $d^I > 1$) imply that infected prey have a lesser (resp. larger) influence than susceptible prey.

Now, the proposed generalization of the discrete predator-prey Leslie-Gower model with prey intra-specific competition reads as follows:

$$N_S(t+1) = \frac{a_1^S N_S(t)}{1 + b_1^{SS} N_S(t) + b_1^{SI} N_I(t) + c_1^S P(t)}$$
$$N_I(t+1) = \frac{a_1^I N_I(t)}{1 + b_1^{IS} N_S(t) + b_1^{II} N_I(t) + c_1^I P(t)} \tag{2.9}$$
$$P(t+1) = \frac{a_2 P(t)}{1 + c_2 P(t)/(N_S(t) + d^I N_I(t))}$$

We build up the complete model by combining the demographic and the disease processes, i.e., the disease process represented in time interval $[t, t+1]$ by the k-th iterate $\mathbf{F}^k$ of map $\mathbf{F}$ (2.2), and the demography described through system (2.9) (see system (A.1) in the Appendix).

Using the notation $\mathbf{F}^k(N_S, N_I) = \left(\mathbf{F}_S^k(N_S, N_I), \mathbf{F}_I^k(N_S, N_I)\right)$ the complete model has the form

$$N_S(t+1) = \frac{a_1^S \mathbf{F}_S^k(N_S, N_I)}{1 + b_1^{SS} \mathbf{F}_S^k(N_S, N_I) + b_1^{SI} \mathbf{F}_I^k(N_S, N_I) + c_1^S P(t)}$$
$$N_I(t+1) = \frac{a_1^I \left(\mathbf{F}^k\right)_I (N_S, N_I)}{1 + b_1^{IS} \mathbf{F}_S^k(N_S, N_I) + b_1^{II} \mathbf{F}_I^k(N_S, N_I) + c_1^I P(t)} \tag{2.10}$$
$$P(t+1) = \frac{a_2 P(t)}{1 + c_2 P(t)/(\mathbf{F}_S^k(N_S, N_I) + d^I \mathbf{F}_I^k(N_S, N_I))}$$

2.4. The reduced model.

In order to become analytically tractable, system (2.10) must be reduced to a two-dimensional system. To do so we make use of discrete approximate reduction techniques and follow the reduction procedure presented in Appendix A.1. Assuming that the disease process has attained its equilibrium (2.6), the dynamics of variables $N = N_S + N_I$, total number of preys, and P, number of predators, can be approximated by the following system, built as (A.2) in the Appendix:

$$N(t+1) = \frac{\nu^* a_1^S N(t)}{1 + \left(\nu^* b_1^{SS} + (1-\nu^*) b_1^{SI}\right) N(t) + c_1^S P(t)} + \frac{(1-\nu^*) a_1^I N(t)}{1 + \left(\nu^* b_1^{IS} + (1-\nu^*) b_1^{II}\right) N(t) + c_1^I P(t)}$$
$$P(t+1) = \frac{a_2 P(t)}{1 + \dfrac{c_2}{\nu^* + (1-\nu^*) d^I} \cdot \dfrac{P(t)}{N(t)}}$$

$$\tag{2.11}$$

that we write in the next simplified form

$$N(t+1) = \frac{\alpha_1 N(t)}{1 + \beta_1 N(t) + \gamma_1 P(t)} + \frac{\alpha_2 N(t)}{1 + \beta_2 N(t) + \gamma_2 P(t)}$$
$$P(t+1) = \frac{\alpha_3 P(t)}{1 + \gamma_3 P(t)/N(t)} \tag{2.12}$$

where we have defined

$$\alpha_1 = \nu^* a_1^S, \ \alpha_2 = (1 - \nu^*)a_1^I, \ \alpha_3 = a_2, \ \beta_1 = \nu^* b_1^{SS} + (1 - \nu^*)b_1^{SI} \qquad (2.13)$$
$$\beta_2 = \nu^* b_1^{IS} + (1 - \nu^*)b_1^{II}, \ \gamma_1 = c_1^S, \ \gamma_2 = c_1^I, \ \gamma_3 = c_2 / \left(\nu^* + (1 - \nu^*)d^I \right)$$

Notice that system (2.7) has the same form of system (2.12) when me make $\alpha_2 = 0$, what allows one to study the dynamics of the Leslie-Gower predator-prey model with intraspecific competition as a particular case of (2.12).

The asymptotic behaviour of solutions of system (2.11) is established in Theorem (A.3). Let us summarize here its results:

1. If $R_0 = \beta/\gamma < 1$, then $\nu^* = 1$ so that the disease is eradicated at the fast time scale and the system acts as the predator-prey model (2.7).
2. If $R_0 = \beta/\gamma > 1$, then $\nu^* = \gamma/\beta = 1/R_0$ and the disease becomes endemic. Depending on parameters values, either both preys and predators go extinct, or predators disappear and preys stabilize, or the community attains a positive equilibrium state. In this case:

   (a) If $a_1^S/R_0 + (1 - 1/R_0)a_1^I \leq 1$ then $\lim_{t \to \infty} (N(t), P(t)) = (0, 0)$.
   (b) If $a_1^S/R_0 + (1 - 1/R_0)a_1^I > 1$ and $a_2 \leq 1$ then $\lim_{t \to \infty} (N(t), P(t)) = (\bar{N}, 0)$, where $\bar{N}$ is (A.5).
   (c) If $a_1^S/R_0 + (1 - 1/R_0)a_1^I > 1$ and $a_2 > 1$ then system (2.11) possesses a unique positive equilibrium point (N^*, P^*) given by (A.6) which is asymptotically stable.

Theorem A.2 in the Appendix guarantees that these three behaviours are inherited by system (2.10), where the number of susceptible and infected preys is given by N/R_0 and $(1 - 1/R_0)N$ respectively. Indeed, the positive solutions $(N_S(t), N_I(t), P(t))$ of system (2.10) approximately tend to $(0, 0, 0)$ in case (a) and to $\left(\bar{N}/R_0, (1 - 1/R_0)\bar{N}, 0 \right)$ in case (b) whereas in case (c) the system has an equilibrium point which is asymptotically stable and can be approximated by $(N^*/R_0, (1 - 1/R_0)N^*, P^*)$.

3. Effects of the disease on predator-prey interactions.

In order to frame the discussion, we notice that the disease modifies the vital features (growth rate) as well as species interaction abilities (intra specific competition and predation effects on prey a predator) of the infected individuals.

To analyze the influence of parasite on the dynamics of the predator-prey community we compare the asymptotic behaviours of the solutions of systems (2.7), representing the disease free community state, and (2.11), which reflects the long term behaviour of system (2.10) where the disease acting in a shorter time scale is taken into account. For the comparison we assume that (a) parameters affecting susceptible prey in system (2.10) coincide with the corresponding ones affecting prey in system (2.7), i.e., $a_1^S = a_1$, $b_1^{SS} = b_1$ y $c_1^S = c_1$, and (b) $R_0 > 1$, i.e., the disease is endemic, since otherwise we can not measure its effect.

The intensity of the disease is characterized by its basic reproduction number R_0. As it was previously mentioned there are four ways through which the introduction of the disease affects the coefficients of the predator-prey model without disease:

(i) Effect on the net prey growth rate in the absence of density dependence, i.e., $a_1^I \neq a_1^S$.

(ii) Effect on predation, i.e., $c_1^I \neq c_1^S$.

(iii) Effect on the capacity of infected prey to contribute to predator growth, i.e., $d^I \neq 1$.

(iv) Effect on the intracompetitive abilities of prey, i.e., at least one of the coefficients $b_1^{SI}, b_1^{SS}, b_1^{II}$ and b_1^{IS} is different from the others.

3.1. Effects on the survival of prey and predator population.

In this section we consider that the only way the disease affects the survival of prey population is through item (i) above, i.e., through the value of a_1^I compared with that of a_1^S.

In many cases the disease has a negative impact on infected individuals, so that we first assume that $a_1^I < a_1^S$. Obviously, the occurrence of a disease outbreak that becomes endemic does not affect the survival of prey whenever $1 < a_1^I < a_1^S$ or $a_1^I < a_1^S < 1$. Thus, we assume now that

$$a_1^I < 1 < a_1^S, \tag{3.1}$$

that is, the susceptible prey subpopulation can survive on its own whereas the infective prey subpopulation can not. The fate of the whole prey population is driven by the value of $\alpha_1 + \alpha_2 = a_1^S/R_0 + a_1^I(1 - 1/R_0)$ from where it follows that a sufficient large value of R_0 can lead the prey population to extinction. In particular, $a_1^S/R_0 + a_1^I(1 - 1/R_0) < 1$ if, and only if

$$\frac{a_1^S - a_1^I}{1 - a_1^I} < R_0, \tag{3.2}$$

so that the left hand side of expression (3.2) is a lower threshold for the reproductive number R_0 to eradicate prey population. On the other hand, if the coefficients are such that inequality (3.2) is fully reversed, then the susceptible part of the prey population is strong enough to compensate for the otherwise non-viable infected prey population.

Now we turn our attention to the case in which the disease has a positive impact on the fitness of infected individuals. For example, the interaction between hosts and parasites does not always lead to a reduction in the number of hosts since sometimes parasites enhance the host fitness as a way of spreading out, i.e, it is a symbiotic relationship. This is the case, for instance, of Wolbachia and its host Bemisia tabachi [11,12], and its natural predators (among them, different species of coleoptera) [10,20]. Therefore, condition $a_1^S < a_1^I$ is plausible in some situations. As before, we focus on the case

$$a_1^S < 1 < a_1^I. \tag{3.3}$$

Analogue calculations to those leading to (3.2) can be carried out to obtain threshold values that decide whether or not the prey population goes extinct for a certain value of a_1^S, a_1^I and R_0. We stress the fact that the presence of parasites in a symbiotic relationship with prey might avoid the extinction of the latter even in the case in which $a_1^S < 1$.

In addition, note that given the prey population can establish, the disease has no effect whatsoever in the extinction or non-extinction of predators, for this is controlled exclusively by parameter a_2.

3.2. Effect on population fitness

To this end, we compare the size of each species population at equilibrium in systems (2.7) without disease, and (2.11). We assume that $a_1 = a_1^S > 1$, $a_1^S/R_0 + a_1^I(1 - 1/R_0) > 1$ so that in both systems prey tend to a positive equilibrium. Moreover, in order to reduce the complexity of the different expressions and proceed analytically we assume throughout this section that intraspecific prey interactions (item (iv) above) are not altered by the disease, so that we set $b_1^{SS} = b_1^{SI} = b_1^{IS} = b_1^{II} = b_1$. We recall the parameter definitions (2.13) (where $v^* = 1/R_0$) and that the notation used for the equilibrium points of submodel (2.7) is equivalent to that of the aggregated system but with lower case letters.

We focus first on prey population when predators can not establish, i.e., $a_2 = \alpha_3 \leq 1$. The effect of an endemic disease on the prey population is captured by ratio $\bar{N}/\bar{n}$ and it is straightforward to show that $\bar{N}/\bar{n} = \delta$ where

$$\delta := \frac{\frac{1}{R_0}a_1^S + \left(1 - \frac{1}{R_0}\right)a_1^I - 1}{a_1^S - 1}. \tag{3.4}$$

Note that δ is independent on items (ii) and (iii) above, and depends only on item (i). Clearly δ is an increasing function of a_1^I and equals one if $a_1^I = a_1^S$, so the ratio $\bar{N}/\bar{n}$ is smaller (resp. larger) that 1, and consequently the disease reduces (resp. increases) the equilibrium value of prey, if and only if $a_1^I < a_1^S$ (resp. $a_1^I > a_1^S$). For $a_1^I > a_1^S$ it admits the following bound:

$$\frac{\bar{N}}{\bar{n}} < \frac{a_1^I - 1}{a_1^S - 1} \tag{3.5}$$

Let us now concentrate in the case in which predators are able to establish, i.e., $a_2 > 1$. The ratios N^*/n^* and P^*/p^* that characterize the effect of the disease on prey and predators, now depend on items (i), (ii) and (iii) and in the general case their analytical study is untractable, so later on we will introduce some simplifying assumptions.

Before that, let us notice that

$$\frac{P^*}{p^*} = \left(\frac{1}{R_0} + (1 - \frac{1}{R_0})d^I \right) \frac{N^*}{n^*}. \tag{3.6}$$

In words, the effect of the disease on predators depends on both its effect on prey population, represented by N^*/n^* and the parameter d^I modeling the relative contribution of infected prey to predator growth. Note that when $d^I = 1$ (both susceptible and infected prey contribute equally to predators growth) then $P^*/p^* = N^*/n^*$ and so the effect on the disease on predators is exactly the same as its effect on prey. Moreover, when $d^I < 1$ we have $P^*/p^* < N^*/n^*$ and when $d^I > 1$ we have $P^*/p^* > N^*/n^*$, i.e., when infected prey have a lower (resp. larger) impact on predators growth than non-infected ones, the effect of the disease on the number of predators is stronger (resp. milder) than its effect on the number of prey.

In any case, (3.6) shows that P^*/p^* can be easily studied once we know N^*/n^*, and so in the sequel we will concentrate our attention mainly on the latter.

3.2.1. Disease affecting only prey growth rate and prey contribution to predator growth

Let us suppose that the predation coefficients (item (ii) above) are not altered by the disease, so that we set $c_1^S = c_1^I = c_1$. Then it follows that

$$\frac{N^*}{n^*} = \delta q, \tag{3.7}$$

where δ is given by (3.4) and

$$q := \frac{b_1 c_2 + (a_2 - 1)\, c_1}{b_1 c_2 + (a_2 - 1)\, c_1 \left(\frac{1}{R_0} + (1 - \frac{1}{R_0})d^I \right)}$$

Note that when $d^I = 1$ then $q = 1$ and so N^*/n^* is independent on b_1, c_2 and c_1 and depends only on a_1, a_1^I and R_0. Moreover, $q < 1$ (resp. $q > 1$) if and only if $d^I > 1$ (resp. $d^I < 1$). Therefore, from (3.7) it follows that when $a_1^I < a_1^S$ and $d^I \geq 1$, then $N^*/n^* < 1$ and so the introduction of the disease decreases the equilibrium population of prey. The contrary happens when the inequalities are fully reversed.

3.2.2. Disease affecting only predation

Another interesting problem is the analysis of the effect on population fitness of item (ii) above, that is, selective predation for or against infected prey represented by the fact that $c_1^I \neq c_1^S = c_1$. Indeed, there is empiric evidence of both selective predation for uninfected prey and for infected prey, where in the case of parasites the latter is also known as parasite-induced vulnerability to predation [17].

In order to simplify the study we suppose that the disease does not affect items (i) and (iii) above so that we set $a_1^I = a_1^S = a_1 > 1$ and $d^I = 1$. Clearly, in the case $c_1^I = c_1^S$ we have that system (2.11) with the disease degenerates in disease-free system (2.7), and so it must be $N^*/n^* = 1$. In addition, from (A.7) it is immediate to see that, for any fixed values of the rest of the parameters, N^* is a strictly decreasing function of c_1^I and, therefore, this is also the case for the ratio N^*/n^*. As a consequence, N^*/n^* is less

than one if and only if $c_1^I > c_1^S$, that is, selective predation for infected prey reduces the fitness of the prey population. The contrary happens when $c_1^I < c_1^S$ so that selective predation for uninfected prey increases the fitness of the prey population.

4. Disease in the predator population.

In this section we present a model analogous to the one developed in Section 2 but with the disease affecting only the predator population. Calling P_S and P_I the susceptible and the infected predators, respectively, the disease process is represented by the map

$$\mathbf{F}(P_S, P_I) = \left(P_S - \frac{\beta P_S P_I}{P_S + P_I} + \gamma P_I, P_I + \frac{\beta P_S P_I}{P_S + P_I} - \gamma P_I \right) \tag{4.1}$$

where, as in the case of Section 2 we are assuming conditions (2.3) and (2.4). Its equilibrium has the form

$$\bar{\mathbf{F}}(P_S, P_I) := \lim_{k \to \infty} \mathbf{F}^k(P_S, P_I) = (\nu^* P, (1 - \nu^*)P), \tag{4.2}$$

with $P = P_S + P_I$, $\nu^* = 1$ if $\beta \leq \gamma$ and $\nu^* = \gamma/\beta = 1/R_0$ if $\gamma < \beta \leq 2 + \gamma$.

Assuming different intrinsic growth rates, a_2^S and a_2^I, and predation parameters, c_1^S and c_1^I, c_2^S and c_2^I, for susceptible and infected predators, the Leslie-Gower predator-prey model (2.7) can be adapted to the case of predator affected by the disease:

$$
\begin{aligned}
N(t+1) &= \frac{a_1 N_S(t)}{1 + b_1 N(t) + c_1^S P_S(t) + c_1^I P_I(t)} \\
P_S(t+1) &= \frac{a_2^S P_S(t)}{1 + c_2^S P(t)/N(t)} \\
P_I(t+1) &= \frac{a_2^I P_I(t)}{1 + c_2^I P(t)/N(t)}
\end{aligned}
\tag{4.3}
$$

We can now build up the complete model combining the two processes as carried out in Section 2.3. To study the models, whose writing we omit, we follow the reduction procedure of Appendix A.1 and end up with the following reduced system, where N is the number of preys and $P = P_S + P_I$ the total number of predators.

$$
\begin{aligned}
N(t+1) &= \frac{a_1 N_S(t)}{1 + b_1 N(t) + \left(\nu^* c_1^S + (1-\nu^*)c_1^I\right) P(t)} \\
P(t+1) &= \frac{\nu^* a_2^S P(t)}{1 + c_2^S P(t)/N(t)} + \frac{(1-\nu^*)a_2^I P(t)}{1 + c_2^I P(t)/N(t)}
\end{aligned}
\tag{4.4}
$$

The asymptotic behaviour of the solutions of system (4.4) is analyzed in Theorem A.5, and with the help of Theorem A.2 we can extend the results to the solutions $(N(t), P_S(t), P_I(t))$ of the complete system. We state here the results for the case $R_0 > 1$ where the disease becomes endemic:

1. If $a_1 \leq 1$ all positive initial conditions approximately tend $(0, 0, 0)$.
2. If $a_1 > 1$ and $a_2^S/R_0 + (1 - 1/R_0)a_2^I \leq 1$ all positive initial conditions approximately tend to $((a_1 - 1)/b_1, 0, 0)$.
3. If $a_1 > 1$ and $a_2^S/R_0 + (1 - 1/R_0)a_2^I > 1$ the system has an equilibrium point which is asymptotically stable and can be approximated by $(N^*, P^*/R_0, (1 - 1/R_0)P^*)$, where (N^*, P^*) is given by (A.10).

Let us extract some brief conclusions. If the prey is not viable without the disease, this is still the case with it and so the community goes extinct. The disease can eliminate the predators from the community, and this happens independently of the parameters a_1, b_1, c_1^S and c_1^I characterizing the dynamics of the prey. Moreover, the disease can keep the community in an equilibrium in which the total populations of prey and predators are a function of disease and disease-mediated parameters.

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5. Conclusion.

We present in this work a model of a predator-prey community with parasites that infect one of the two species, focusing on the case of infected prey. The treatment of the subject differs from most of the existing literature in two aspects: the model is in discrete time and the parasites dynamics occurs on a shorter time scale than the predator-prey interactions. We keep this work simple enough to be able to find analytic results stressing the effects of the disease on the community. The Leslie-Gower model that we use to describe the community dynamics without disease has a simple long term behaviour that it is inherited in its simplicity by the complete system that encompasses predator-prey-parasite dynamics. Assuming that the disease is endemic, there exist three different possibilities of long term behaviour: extinction of the whole community, extinction of the predator species with stabilization of the prey species, and coexistence of the two species at stable population sizes. In the last two cases, in addition, there are constant proportions of susceptible and infected preys.

Parasites infecting prey species can be considered competing with predators for the resource: the prey/host species. Different from more classical models of exploitative competition for an explicit resource, the model presented in this work admits that both competing species coexist on the single resource. The endemicity of the disease depends on the transmission-recovery ratio, $R_0 > 1$, and does not change with populations densities, thus parasites persist if prey do. On the other hand, predators viability depends on their growth rate a_2 being greater than one. The coexistence of the module predator-prey-parasite depends then on the weighted mean of susceptible and infected prey growth rates, a_1^S and a_1^I:

$$a_1^S/R_0 + (1 - 1/R_0)a_1^I > 1,$$

where the weights are the constant proportions of susceptible and infected preys. Out of this inequality it is easy to find conditions for a disease to drive extinct an otherwise population in coexistence and vice-versa.

Parasites infecting only the predator species can be considered as their consumers so that the module predator-prey-parasite turns out to be a linear food chain with parasites as super-predators. Assuming $R_0 > 1$ and the prey growth rate $a_1 > 1$, the coexistence of the module depends now on:

$$a_2^S/R_0 + (1 - 1/R_0)a_2^I > 1,$$

the weighted mean of susceptible and infected predators growth rates, a_1^S and a_1^I. In this case the disease can just alter the fate of predators since prey is regulated in the absence of predator and parasite.

In the conditions for coexistence of the module predator-prey-parasite, the effects of the disease can be analyzed by comparing the stable sizes, with and without disease, of the populations of prey, N^* and n^*, and predator, P^* and p^*. The parameter d^I modeling the relative contribution of infected prey to predator growth has a direct influence on the stable structure of the predator-prey community; keeping all the other parameters constant, $d^I < 1$ entails a stronger effect of the disease on the number of predators than its effect on the number of prey $P^*/p^* < N^*/n^*$; the contrary happens if $d^I > 1$. If the disease affects negatively (resp. positively) the growth of infected prey, $a_1^I < a_1^S$ (resp. $a_1^I > a_1^S$), and the infected prey contribution to predator growth is larger (resp. less) than the susceptible one, $d^I > 1$ (resp. $d^I < 1$), then the size of both populations decreases (resp. increases), $N^* < n^*$ and $P^* < p^*$ (resp. $N^* > n^*$ and $P^* > p^*$). If predators attack infected prey more (resp. less) frequently than susceptible prey, $c_1^I > c_1^S$ (resp. $c_1^I < c_1^S$) then, assuming the rest of parameters unaltered by the disease, both populations decrease (resp. increase).

Appendices

A.1. Reduction procedure

The coupled dynamics of any three component (N_S, N_I and P) system in discrete time can be modeled as:

$$N_S(t+1) = f_S(N_S(t), N_I(t), P(t))$$
$$N_I(t+1) = f_I(N_S(t), N_I(t), P(t))$$
$$P(t+1) = g(N_S(t), N_I(t), P(t))$$

Supposing that the first two component of the community (N_S and N_I) correspond to the susceptible and infected individuals of a species affected by a disease, as described in section 2.1, a general complete model that includes species interactions on a slow time scale and disease action on a fast time scale would read as follows:

$$N_S(t+1) = f_S\left(\left(\mathbf{F}^k\right)_S(N_S(t), N_I(t)), \left(\mathbf{F}^k\right)_I(N_S(t), N_I(t)), P(t)\right)$$
$$N_I(t+1) = f_I\left(\left(\mathbf{F}^k\right)_S(N_S(t), N_I(t)), \left(\mathbf{F}^k\right)_I(N_S(t), N_I(t)), P(t)\right) \quad (A.1)$$
$$P(t+1) = g\left(\left(\mathbf{F}^k\right)_S(N_S(t), N_I(t)), \left(\mathbf{F}^k\right)_I(N_S(t), N_I(t)), P(t)\right)$$

where $(\mathbf{F}^k)_S(N_S(t), N_I(t))$ and $(\mathbf{F}^k)_I(N_S(t), N_I(t))$ are the first and the second components of the k-th iterate of the map $\mathbf{F}$ describing the disease process.

Assuming that the fast process has attained its equilibrium (2.6), the dynamics of the variables $N = N_S + N_I$ and P are governed by the following system

$$N(t+1) = f_S\left(\nu^*N(t), (1-\nu^*)N(t), P(t)\right) + f_I\left(\nu^*N(t), (1-\nu^*)N(t), P(t)\right)$$
$$P(t+1) = g\left(\nu^*N(t), (1-\nu^*)N(t), P(t)\right) \quad (A.2)$$

where $\nu^* = 1$ if $\beta \leq \gamma$ and $\nu^* = \gamma/\beta$ if $\gamma < \beta \leq 2 + \gamma$.

The asymptotic behaviour of the solutions of systems (A.1) and (A.2) can be related by making use of results in [19] regarding approximate reduction techniques. The condition for the results to hold is that $\mathbf{F}^{(k)}$ converges to $\bar{\mathbf{F}}$ uniformly on compact sets and the same happens with their differentials, i.e., $\lim_{k\to\infty} D\mathbf{F}^{(k)}(N_S, N_I) = D\bar{\mathbf{F}}(N_S, N_I)$ uniformly on compact sets. Now we proceed to prove these facts:

Lemma A.1. *Let $\mathbf{F}$ defined in* (2.2)

$$\mathbf{F}(N_S, N_I) = \left(N_S - \frac{\beta N_S N_I}{N_S + N_I} + \gamma N_I, N_I + \frac{\beta N_S N_I}{N_S + N_I} - \gamma N_I\right),$$

with the positive parameters β and γ verifying conditions (2.3), $\gamma \leq 1$ and $\beta < \left(1 + \sqrt{\gamma}\right)^2$, and (2.4), $\beta < 2 + \gamma$. Then the following two limits exist uniformly on compacts sets of $\Omega := [0, \infty) \times (0, \infty)$:

1. $\displaystyle\lim_{k\to\infty} \mathbf{F}^{(k)}(N_S, N_I) = \bar{\mathbf{F}}(N_S, N_I) = (\nu^*N, (1-\nu^*)N)$, *with $N = N_S + N_I$.*

2. $\displaystyle\lim_{k\to\infty} D\mathbf{F}^{(k)}(N_S, N_I) = D\bar{\mathbf{F}}(N_S, N_I) = \begin{pmatrix} \nu^* & \nu^* \\ 1-\nu^* & 1-\nu^* \end{pmatrix}$.

Proof. We can write $\mathbf{F}$ in terms of the polinomial function $\phi(x) = x\left(1 + \beta(1-x) - \gamma\right)$:

$$\mathbf{F}(N_S, N_I) = ((1 - \phi(N_I/N))N, \phi(N_I/N)N) \quad (A.3)$$

and, having in mind that $\mathbf{F}$ keeps N constant, we can also express its k-th iterate in terms of ϕ^k, the k-th iterate of ϕ:

$$\mathbf{F}^k(N_S, N_I) = \left((1 - \phi^k(N_I/N))N, \phi^k(N_I/N)N\right). \quad (A.4)$$

For any compact set $C \subset \Omega$ let us define $M_C = \max\limits_{(N_S,N_I)\in C} (N_S + N_I)$, $\alpha_C = \min\limits_{(N_S,N_I)\in C} N_I/N > 0$ and $\beta_C = \max\limits_{(N_S,N_I)\in C} N_I/N \leq 1$. We then have

$$\max_{(N_S,N_I)\in C} \|\mathbf{F}^k(N_S,N_I) - \bar{\mathbf{F}}(N_S,N_I)\| = \max_{(N_S,N_I)\in C} N \left\|\left(1 - \phi^k(N_I/N) - v^*, \phi^k(N_I/N) - 1 + v^*\right)\right\| \leq$$

$$\leq \sqrt{2} M_C \max_{(N_S,N_I)\in C} |\phi^k(N_I/N) - (1-\nu^*)| \leq \sqrt{2} M_C \max_{x\in[\alpha_C,\beta_C]} |\phi^k(x) - (1-\nu^*)|$$

Now it is straightforward to see that the scalar difference equation $x_{t+1} = \phi(x_t)$ with initial conditions $x_0 \in [\alpha_C, \beta_C] \subset (0,1]$ converges monotonically, and therefore uniformly in $[\alpha_C, \beta_C]$, to $1 - v^*$. Therefore the first limit converges uniformly in compact sets as we wanted to show. To prove the uniform convergence of the second limit we first express the differential of $\mathbf{F}^k$ in terms of the derivative of ϕ^k:

$$D\mathbf{F}^k(N_S,N_I) = \begin{pmatrix} 1 - \phi^k(\frac{N_I}{N}) + \frac{N_I}{N}(\phi^k)'(\frac{N_I}{N}) & 1 - \phi^k(\frac{N_I}{N}) + (\frac{N_I}{N} - 1)(\phi^k)'(\frac{N_I}{N}) \\ \phi^k(\frac{N_I}{N}) - \frac{N_I}{N}(\phi^k)'(\frac{N_I}{N}) & \phi^k(\frac{N_I}{N}) + (1 - \frac{N_I}{N})(\phi^k)'(\frac{N_I}{N}) \end{pmatrix}$$

which can be decomposed as

$$D\mathbf{F}^k(N_S,N_I) = \begin{pmatrix} 1 - \phi^k(\frac{N_I}{N}) & 1 - \phi^k(\frac{N_I}{N}) \\ \phi^k(\frac{N_I}{N}) & \phi^k(\frac{N_I}{N}) \end{pmatrix} + (\phi^k)'(N_I/N) \begin{pmatrix} \frac{N_I}{N} & \frac{N_I}{N} - 1 \\ -\frac{N_I}{N} & 1 - \frac{N_I}{N} \end{pmatrix}$$

As a consequence of the first limit we have that

$$\lim_{k\to\infty} \begin{pmatrix} 1 - \phi^k(\frac{N_I}{N}) & 1 - \phi^k(\frac{N_I}{N}) \\ \phi^k(\frac{N_I}{N}) & \phi^k(\frac{N_I}{N}) \end{pmatrix} = \begin{pmatrix} \nu^* & \nu^* \\ 1 - \nu^* & 1 - \nu^* \end{pmatrix} = D\bar{\mathbf{F}}(N_S,N_I)$$

uniformly on compacts sets of Ω. So, bearing in mind that $\begin{pmatrix} \frac{N_I}{N} & \frac{N_I}{N} - 1 \\ -\frac{N_I}{N} & 1 - \frac{N_I}{N} \end{pmatrix}$ is bounded, to finish the proof we only need to show that the following limit is uniform on compact sets of $(0,1]$

$$\lim_{k\to\infty} (\phi^k)'(x) = 0,$$

Since $|\phi'(1-v^*)| < 1$ there exist $\alpha < 1$ and a neighbourhood $I \subset (0,1]$ of $1 - \nu^*$ such that for every $x \in I$ we have $|\phi'(x)| < \alpha$. Now, the uniform convergence to $1 - \nu^*$ of the solutions of the scalar difference equation $x_{t+1} = \phi(x_t)$ with initial conditions $x_0 \in (0,1]$ and the chain rule to calculate $(\phi^k)'(x)$ complete the proof. □

The next theorem relates the asymptotic behavior of systems (A.1) and (A.2) for big enough values of parameter k.

Theorem A.2. *Let us assume that $f_S, f_I, g \in C^1(\mathbf{R}_+^3)$ and that the hypotheses in Lemma A.1 hold. Let (N^*, P^*) be a hyperbolic equilibrium point of (A.2). Then there exists $k_0 \in \mathbb{N}$ such that for each $k \geq k_0$ there exists a hyperbolic equilibrium point $(N_{S,k}^*, N_{I,k}^*, P_k^*)$ of (A.1) satisfying*

$$\lim_{k\to\infty} \left(N_{S,k}^*, N_{I,k}^*, P_k^*\right) = \left(\nu^* N^*, (1-\nu^*)N^*, P^*\right).$$

Moreover,

1. If (N^*, P^*) is asymptotically stable (resp. unstable) then $(N^*_{S,k}, N^*_{I,k}, P^*_k)$ is asymptotically stable (resp. unstable) for each $k \geq k_0$.

2. In the case of (N^*, P^*) being asymptotically stable, for each $k \geq k_0$, if $(N_S(0) + N_I(0), P(0))$ is in the basin of attraction of (N^*, P^*) then $(N_S(0), N_I(0), P(0))$ is in the basin of attraction of $(N^*_{S,k}, N^*_{I,k}, P^*_k)$.

Analogous results hold for periodic solutions.

Proof. It is a direct consequence of the results in [19] defining

$$F(N_S, N_I, P) = \left(N_S - \frac{\beta N_S N_I}{N_S + N_I} + \gamma N_I, N_I + \frac{\beta N_S N_I}{N_S + N_I} - \gamma N_I, P \right),$$

$$S(N_S, N_I, P) = (f_S(N_S, N_I, P), f_I(N_S, N_I, P), g(N_S, N_I, P))$$

and using Lemma A.1. $\square$

A.2. Asymptotic behaviour of systems (2.11) and (4.4)

Theorem A.3. *Let us consider system* (2.12) *with all its parameters being positive but α_2 that we assume to be nonnegative.*

1. *If $\alpha_1 + \alpha_2 \leq 1$ then any solution $(N(t), P(t))$ of system* (2.12) *with positive initial conditions verifies*

$$\lim_{t \to \infty} (N(t), P(t)) = (0, 0).$$

2. *If $\alpha_3 \leq 1$ and $\alpha_1 + \alpha_2 > 1$ then any solution $(N(t), P(t))$ of system* (2.12) *with positive initial conditions verifies*

$$\lim_{t \to \infty} N(t) = \bar{N} \ \text{and} \ \lim_{t \to \infty} P(t) = 0,$$

with

$$\bar{N} = \frac{(\alpha_2 - 1)\beta_1 + (\alpha_1 - 1)\beta_2 + \sqrt{((1 - \alpha_2)\beta_1 + (1 - \alpha_1)\beta_2)^2 + 4(\alpha_1 + \alpha_2 - 1)\beta_1\beta_2}}{2\beta_1\beta_2}. \tag{A.5}$$

3. *If $\alpha_1 + \alpha_2 > 1$ and $\alpha_3 > 1$ then system* (2.12) *possesses a unique positive equilibrium point (N^*, P^*) which is asymptotically stable:*

$$N^* = \frac{(\alpha_2 - 1)\bar{\beta}_1 + (\alpha_1 - 1)\bar{\beta}_2 + \sqrt{((1 - \alpha_2)\bar{\beta}_1 + (1 - \alpha_1)\bar{\beta}_2)^2 + 4(\alpha_1 + \alpha_2 - 1)\bar{\beta}_1\bar{\beta}_2}}{2\bar{\beta}_1\bar{\beta}_2}$$

$$P^* = \frac{(\alpha_3 - 1)N^*}{\gamma_3} \tag{A.6}$$

with $\bar{\beta}_1 = \beta_1 + (\alpha_3 - 1)\gamma_1/\gamma_3$ and $\bar{\beta}_2 = \beta_2 + (\alpha_3 - 1)\gamma_2/\gamma_3$.

Proof.

1. Let $(N(t), P(t))$ be the solution of system (2.12) for initial conditions $N(0) > 0$ and $P(0) > 0$. For any $t \geq 0$, $0 < N(t+1) < \dfrac{(\alpha_1 + \alpha_2)N(t)}{1 + \bar{\beta}N(t)}$, with $\bar{\beta} = \min\{\beta_1, \beta_2\}$. If we define $g(x) = (\alpha_1 + \alpha_2)x/(1 + \bar{\beta}x)$ we see that it is an increasing function, what yields $0 < N(t) < g^{(t)}(N(0))$ for all $t \geq 0$. As $g(x)$ is a continuous function from $[0, \infty)$ to $[0, \infty)$ verifying that $0 < g(x) < x$ for all $x > 0$ Theorem 2.5 in [2] applies and we have that $\lim_{t \to \infty} g^{(t)}(x_0) = 0$ for any $x_0 > 0$. This implies that $0 \leq \lim_{t \to \infty} N(t) \leq \lim_{t \to \infty} g^{(t)}(N(0)) = 0$, that is, $\lim_{t \to \infty} N(t) = 0$. On the other hand, $P(t+1) \leq \dfrac{\alpha_3 P(t)}{\gamma_3 P(t)/N(t)} = \dfrac{\alpha_3 N(t)}{\gamma_3}$, for $t \geq 0$. So, $\lim_{t \to \infty} N(t) = 0$ yields that $\lim_{t \to \infty} P(t) = 0$.

2. Let $(N(t), P(t))$ be the solution of system (2.12) for initial conditions $N(0) > 0$ and $P(0) > 0$. We are using the family of functions f_δ for $\delta \geq 0$, defined as:

$$f_\delta(x) = \frac{\alpha_1 x}{1 + \beta_1 x + \delta} + \frac{\alpha_2 x}{1 + \beta_2 x + \delta}.$$

We notice that $f_0(x) < \alpha_1/\beta_1 + \alpha_2/\beta_2$ for all $x > 0$. As $N(t+1) < f_0(N(t))$ we also have that $N(t) < \alpha_1/\beta_1 + \alpha_2/\beta_2$ for all $t > 0$. From the last inequality we deduce that $P(t+1) < \dfrac{\alpha_3 P(t)}{1 + \bar{\gamma} P(t)}$, where $\bar{\gamma} = \dfrac{\gamma_3}{\alpha_1/\beta_1 + \alpha_2/\beta_2}$. Thus $\alpha_3 \leq 1$ implies, reasoning as in the previous item, that $\lim_{t\to\infty} P(t) = 0$. To prove the convergence of $N(t)$ we first characterize the asymptotic behaviour of the positive solutions of the difference equation $x(t+1) = f_\delta(x(t))$. We have that $f_\delta(0) = 0$, $f_\delta(x) > 0$ for $x > 0$, and $\lim_{x\to\infty} f_\delta(x) = \alpha_1/\beta_1 + \alpha_2/\beta_2$. We also have $f_\delta'(x) = \dfrac{\alpha_1(1+\delta)}{(1+\beta_1 x+\delta)^2} + \dfrac{\alpha_2(1+\delta)}{(1+\beta_2 x+\delta)^2} > 0$ so that f_δ is strictly increasing and f_δ' strictly decreasing for $x > 0$. If $\alpha_1 + \alpha_2 > 1$ then $f_\delta'(0) = \dfrac{\alpha_1 + \alpha_2}{1+\delta} > 1$ for $\delta < \alpha_1 + \alpha_2 - 1$. Now it is straightforward to prove that $x(t+1) = f_\delta(x(t))$ possesses a unique positive fixed point

$$x_\delta^* = \frac{(\alpha_2 - 1 - \delta)\beta_1 + (\alpha_1 - 1 - \delta)\beta_2 + \sqrt{\left((1 - \alpha_2 + \delta)\beta_1 + (1 - \alpha_1 + \delta)\beta_2\right)^2 + 4(1+\delta)(\alpha_1 + \alpha_2 - 1 - \delta)\beta_1\beta_2}}{2\beta_1\beta_2}.$$

Moreover, function f_δ verifies that $x < f_\delta(x) < x_\delta^*$ for $x \in (0, x_\delta^*)$ and $x_\delta^* < f_\delta(x) < x$ for $x \in (x_\delta^*, \infty)$ what implies (Theorem 2.8 in [2]) that $\lim_{t\to\infty} f_\delta^{(t)}(x_0) = 0$ for any $x_0 > 0$. We now use the fact that $\lim_{t\to\infty} P(t) = 0$, what implies that for any $\delta > 0$ there exists $t_\delta \geq 0$ such that for any $t \geq t_\delta$ we have that $\max\{\gamma_1 P(t), \gamma_2 P(t)\} \leq \delta$, and also, calling $F(N, P) = \dfrac{\alpha_1 N}{1 + \beta_1 N + \gamma_1 P} + \dfrac{\alpha_2 N}{1 + \beta_2 N + \gamma_2 P}$, that

$$f_\delta(N(t)) \leq F(N(t), P(t)) \leq f_0(N(t)).$$

As f_δ is an increasing function we obtain the following inequalities:

$$F(N(t+1), P(t+1)) \leq f_0(N(t+1)) = f_0\left(F(N(t), P(t))\right) \leq f_0\left(f_0(N(t))\right) = f_0^{(2)}(N(t)),$$

$$f_\delta^{(2)}(N(t)) = f_\delta\left(f_\delta(N(t))\right) \leq f_\delta\left(F(N(t), P(t))\right) = f_\delta(N(t+1)) \leq F(N(t+1), P(t+1))$$

and by induction, for any $k > 0$, that

$$f_\delta^{(k)}(N(t)) \leq F(N(t+k), P(t+k)) \leq f_0^{(k)}(N(t))$$

Letting δ tend to 0 and using the continuity of f_δ respect to δ, we obtain

$$f_0^{(k)}(N(t)) \leq F(N(t+k), P(t+k)) \leq f_0^{(k)}(N(t))$$

and now, making k tend to ∞ it follows that

$$\lim_{t\to\infty} N(t) = x_0^* = \bar{N}.$$

3. The equilibrium points of system (2.12) are the solutions (N^*, P^*) of the next system

$$\begin{cases} 1 = \dfrac{\alpha_1}{1 + \beta_1 N^* + \gamma_1 P^*} + \dfrac{\alpha_2}{1 + \beta_2 N^* + \gamma_2 P^*} \\[2ex] 1 = \dfrac{\alpha_3}{1 + \gamma_3 P^*/N^*} \end{cases}$$

Solving the second equation we obtain $P^* = \dfrac{\alpha_3 - 1}{\gamma_3} N^*$, where we see that $\alpha_3 > 1$ is a necessary condition for a positive equilibrium point to exist. Substituting into the first equation we get

$$1 = \frac{\alpha_1}{1 + \bar{\beta}_1 N^*} + \frac{\alpha_2}{1 + \bar{\beta}_2 N^*} \tag{A.7}$$

where $\bar{\beta}_1 = \beta_1 + (\alpha_3 - 1)\gamma_1/\gamma_3$ and $\bar{\beta}_2 = \beta_2 + (\alpha_3 - 1)\gamma_2/\gamma_3$. This equation becomes

$$\bar{\beta}_1 \bar{\beta}_2 (N^*)^2 + \big((1 - \alpha_2)\bar{\beta}_1 + (1 - \alpha_1)\bar{\beta}_2\big) N^* + 1 - (\alpha_1 + \alpha_2) = 0$$

from where we see that $\alpha_1 + \alpha_2 > 1$ is the necessary and sufficient condition for the existence of a positive solution, which moreover is unique:

$$N^* = \frac{(\alpha_2 - 1)\bar{\beta}_1 + (\alpha_1 - 1)\bar{\beta}_2 + \sqrt{\big((1 - \alpha_2)\bar{\beta}_1 + (1 - \alpha_1)\bar{\beta}_2\big)^2 + 4(\alpha_1 + \alpha_2 - 1)\bar{\beta}_1 \bar{\beta}_2}}{2\bar{\beta}_1 \bar{\beta}_2}$$

We prove the asymptotic stability of the equilibrium point (N^*, P^*) of system (2.12) by linearization. The jacobian of the transformation at (N^*, P^*) is

$$J(N^*, P^*) = \begin{pmatrix} 1 - \dfrac{\alpha_1 \beta_1 N^*}{\big(1 + \bar{\beta}_1 N^*\big)^2} - \dfrac{\alpha_2 \beta_2 N^*}{\big(1 + \bar{\beta}_2 N^*\big)^2} & -\dfrac{\alpha_1 \gamma_1 N^*}{\big(1 + \bar{\beta}_1 N^*\big)^2} - \dfrac{\alpha_2 \gamma_2 N^*}{\big(1 + \bar{\beta}_2 N^*\big)^2} \\ \dfrac{(\alpha_3 - 1)^2}{\alpha_3 \gamma_3} & \dfrac{1}{\alpha_3} \end{pmatrix}$$

where we have used (A.7) in order to obtain position $(1,1)$. We complete the proof by proving that $J(N^*, P^*)$ verifies the Jury conditions, i.e., $|\mathrm{Tr}(J(N^*, P^*))| < 1 + \det(J(N^*, P^*)) < 2$. We begin by proving that the trace of $J(N^*, P^*)$ is positive. Having in mind equality (A.7),

$$\mathrm{Tr}(J(N^*, P^*)) = 1 - \frac{\alpha_1 \beta_1 N^*}{\big(1 + \bar{\beta}_1 N^*\big)^2} - \frac{\alpha_2 \beta_2 N^*}{\big(1 + \bar{\beta}_2 N^*\big)^2} + \frac{1}{\alpha_3}$$

$$= \frac{1}{\alpha_3} + 1 - \left(\frac{\alpha_1}{1 + \bar{\beta}_1 N^*} \frac{\beta_1 N^*}{1 + \bar{\beta}_1 N^*} + \frac{\alpha_2}{1 + \bar{\beta}_2 N^*} \frac{\beta_2 N^*}{1 + \bar{\beta}_2 N^*} \right)$$

$$\geq \frac{1}{\alpha_3} + 1 - \max\left\{ \frac{\beta_1 N^*}{1 + \bar{\beta}_1 N^*}, \frac{\beta_2 N^*}{1 + \bar{\beta}_2 N^*} \right\} > \frac{1}{\alpha_3} > 0.$$

where in the second to last inequality we have used that $\bar{\beta}_1 \geq \beta_1$, $\bar{\beta}_2 \geq \beta_2$. Next we prove that $\det(J(N^*, P^*)) < 1$. Still using (A.7),

$$\det(J(N^*, P^*)) = \frac{1}{\alpha_3}\left(1 - \frac{\alpha_1 \beta_1 N^*}{\big(1 + \bar{\beta}_1 N^*\big)^2} - \frac{\alpha_2 \beta_2 N^*}{\big(1 + \bar{\beta}_2 N^*\big)^2}\right) + \frac{(\alpha_3 - 1)^2}{\alpha_3 \gamma_3}\left(\frac{\alpha_1 \gamma_1 N^*}{\big(1 + \bar{\beta}_1 N^*\big)^2} + \frac{\alpha_2 \gamma_2 N^*}{\big(1 + \bar{\beta}_2 N^*\big)^2}\right)$$

$$< \frac{1}{\alpha_3} + \frac{(\alpha_3 - 1)^2}{\alpha_3 \gamma_3}\left(\frac{\alpha_1}{1 + \bar{\beta}_1 N^*} \frac{\gamma_1 N^*}{1 + \bar{\beta}_1 N^*} + \frac{\alpha_2}{1 + \bar{\beta}_2 N^*} \frac{\gamma_2 N^*}{1 + \bar{\beta}_2 N^*}\right)$$

$$< \frac{1}{\alpha_3} + \frac{(\alpha_3 - 1)^2}{\alpha_3 \gamma_3} \max\left\{\frac{\gamma_1 N^*}{1 + \bar{\beta}_1 N^*}, \frac{\gamma_2 N^*}{1 + \bar{\beta}_2 N^*}\right\} < \frac{1}{\alpha_3} + \frac{(\alpha_3 - 1)^2}{\alpha_3 \gamma_3} \frac{\gamma_3}{\alpha_3 - 1} = 1.$$

where in the last inequality we have used that $1 + \bar{\beta}_i N^* > (\alpha_3 - 1)\gamma_i/\gamma_3$ for $i = 1, 2$. Finally we prove that $|\mathrm{Tr}(J(N^*, P^*))| < 1 + \det(J(N^*, P^*))$, that is, $\det(J(N^*, P^*)) + 1 - \mathrm{Tr}(J(N^*, P^*)) > 0$:

$$\det(J(N^*, P^*)) + 1 - \mathrm{Tr}(J(N^*, P^*)) = \frac{1}{\alpha_3}\left(1 - \frac{\alpha_1 \beta_1 N^*}{\big(1 + \bar{\beta}_1 N^*\big)^2} - \frac{\alpha_2 \beta_2 N^*}{\big(1 + \bar{\beta}_2 N^*\big)^2}\right)$$

$$+ \frac{(\alpha_3 - 1)^2}{\alpha_3 \gamma_3}\left(\frac{\alpha_1 \gamma_1 N^*}{\big(1 + \bar{\beta}_1 N^*\big)^2} + \frac{\alpha_2 \gamma_2 N^*}{\big(1 + \bar{\beta}_2 N^*\big)^2}\right) + \frac{\alpha_1 \beta_1 N^*}{\big(1 + \bar{\beta}_1 N^*\big)^2} + \frac{\alpha_2 \beta_2 N^*}{\big(1 + \bar{\beta}_2 N^*\big)^2} - \frac{1}{\alpha_3}$$

$$= \frac{\alpha_3 - 1}{\alpha_3}\left(\frac{\alpha_1 \beta_1 N^*}{\big(1 + \bar{\beta}_1 N^*\big)^2} + \frac{\alpha_2 \beta_2 N^*}{\big(1 + \bar{\beta}_2 N^*\big)^2}\right) + \frac{(\alpha_3 - 1)^2}{\alpha_3 \gamma_3}\left(\frac{\alpha_1 \gamma_1 N^*}{\big(1 + \bar{\beta}_1 N^*\big)^2} + \frac{\alpha_2 \gamma_2 N^*}{\big(1 + \bar{\beta}_2 N^*\big)^2}\right) > 0.$$

□

Theorem A.4. *System* (2.12) *without predators is the following difference equation:*

$$N(t+1) = \frac{\alpha_1 N(t)}{1 + \beta_1 N(t)} + \frac{\alpha_2 N(t)}{1 + \beta_2 N(t)}. \tag{A.8}$$

in which we consider that all its parameters are positive but α_2, that we assume to be nonnegative.

1. If $\alpha_1 + \alpha_2 \leq 1$ then any solution $N(t)$ of equation (A.8) *with positive initial condition verifies*

$$\lim_{t \to \infty} N(t) = 0.$$

2. If $\alpha_1 + \alpha_2 > 1$ then any solution $N(t)$ of equation (A.8) *with positive initial condition verifies*

$$\lim_{t \to \infty} N(t) = \bar{N},$$

where $\bar{N}$ is given by (A.5).

Proof. Analogous to the two first items of the previous theorem. □

Theorem A.5. *Let us consider the reduced system* (4.4), *corresponding to the case in which the disease affects the predator, in the following simplified form*

$$N(t+1) = \frac{\alpha_1 N(t)}{1 + \beta N(t) + \gamma_1 P(t)}$$
$$P(t+1) = \frac{\alpha_2 P(t)}{1 + \gamma_2 P(t)/N(t)} + \frac{\alpha_3 P(t)}{1 + \gamma_3 P(t)/N(t)} \tag{A.9}$$

and let us assume that all the parameters are positive but α_3, that we assume to be nonnegative.

1. If $\alpha_1 \leq 1$ then any solution $(N(t), P(t))$ of system (A.9) *with positive initial conditions verifies*

$$\lim_{t \to \infty} (N(t), P(t)) = (0, 0).$$

2. If $\alpha_1 > 1$ and $\alpha_2 + \alpha_3 \leq 1$ then any solution $(N(t), P(t))$ of system (2.12) *with positive initial conditions verifies*

$$\lim_{t \to \infty} (N(t), P(t)) = ((\alpha_1 - 1)/\beta, 0).$$

3. If $\alpha_1 > 1$ and $\alpha_2 + \alpha_3 > 1$ then system (A.9) *possesses a unique positive equilibrium point (N^*, P^*) which is asymptotically stable:*

$$N^* = \frac{2(\alpha_1 - 1)\gamma_2 \gamma_3}{2\beta\gamma_2\gamma_3 + \gamma_1\left((\alpha_2 - 1)\gamma_3 + (\alpha_3 - 1)\gamma_2 + \sqrt{((\alpha_2 - 1)\gamma_3 + (\alpha_3 - 1)\gamma_2)^2 + 4(\alpha_2 + \alpha_3 - 1)\gamma_2\gamma_3}\right)}$$

$$P^* = \frac{\alpha_1 - 1 - \beta N^*}{\gamma_1} \tag{A.10}$$

Proof. Analogous to the proof of Theorem A.3. □

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