

Research Article

Discrete Models of Disease and Competition

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Received 9 June 2017; Revised 25 July 2017; Accepted 10 August 2017; Published 20 September 2017

Academic Editor: Vicenç Mendez

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The aim of this work is to analyze the influence of the fast development of a disease on competition dynamics. To this end we present two discrete time ecoepidemic models. The first one corresponds to the case of one parasite affecting demography and intraspecific competition in a single host, whereas the second one contemplates the more complex case of competition between two different species, one of which is infected by the parasite. We carry out a complete mathematical analysis of the asymptotic behavior of the solutions of the corresponding systems of difference equations and derive interesting ecological information about the influence of a disease in competition dynamics. This includes an assessment of the impact of the disease on the equilibrium population of both species as well as some counterintuitive behaviors in which although we would expect the outbreak of the disease to negatively affect the infected species, the contrary happens.

1. Introduction

Species interactions are a central issue in ecology in order to explain community structure and its dynamics. Among the frameworks that try to explain ecosystems dynamics, ecoepidemiology has become a proper discipline in its own right [1, 2]. Loosely speaking, its main objective is to understand the effect of diseases or parasites on species interactions and vice versa. The empirical evidence of such an influence has motivated a great deal of mathematical modeling in order to reveal the underlying mechanisms.

In this work we focus on ecoepidemic competition models that have attracted great attention [3]. Empirical observations have revealed that disease/parasites can affect the outcome of species competition in several different ways. For instance, a shared parasite between the competing species can change the fate of competition, allowing the otherwise inferior competitor to rule out the superior competitor, as observed by Park [4] with *Tribolium castaneum* (the stronger competitor), *Tribolium confusum*, and the parasitoid *Adelina tribolii* that lowers the fecundity of *T. castaneum*. There is also evidence of species coexistence induced by specialist (i.e., affecting only one competitor) parasite. This is the

case of *Anolis* lizards in the Caribbean area [5]. *Anolis gingivinus* is a stronger competitor than *A. watsi* and coexisting populations are found only in the presence of the parasite *Plasmodium azurophilum*. This parasite that reduces the competitive abilities of the host rarely affects *A. watsi* but can be common in *A. gingivinus*.

It is usually assumed that disease/parasites reduce either the growth or competitive abilities of infected individuals or both of them. Nevertheless, this is not always the case. Regarding their effect on growth, some parasites spread through host offspring, so that the strategy consists on enhancing host fecundity to improve their spreading. For instance, this is the case of *Wolbachia* and its host *Bemisia tabaci* [6, 7]. *Bemisia t.* competes with *Trialeurodes vaporariorum* (Westwood) [8] and it has been reported that there are no evidences of Westwood infected by *Wolbachia* [9]. As far as competitive abilities are concerned, a laboratory study [10] dealt with the effect of the tapeworm *Hymenolepis diminuta* on competition between *T. castaneum* and *T. confusum*. *T. castaneum* (as we have said, the superior competitor) has higher infection and fitness costs from parasitism, so that a parasite-mediated reversal of the outcome of competition was expected. Nevertheless, *T. confusum* was excluded even faster

than what had happened in the absence of infection; it was suggested that intraguild predation is behind this outcome [10].

From the mathematical point of view, complete ecoepidemic competition models are known for the difficulty of their analytical study (see [3] box 2.3 and references therein). There exist a number of studies in continuous time [3] but, to our knowledge, our work is the first attempt in discrete time.

An ecoepidemic competition model combines three different processes: each species demography, intra- and/or interspecies competition, and epidemics. To our knowledge, all the ecoepidemic competition models in the literature consider that the three processes evolve within the same time scale. In other words, it is assumed that the effects of demography, competition, and infection are accounted for at the population level. This assumption is fairly true for certain long-term infections (as AIDS, tuberculosis, etc.) but is not for some others. Indeed, there are diseases such that a number of infection/recovery episodes take place within each demography period. This assumption is equivalent to that of the existence of different time scales associated with each process. An example of this scenario is the *human trachoma*, an infectious disease that is the leading cause of blindness worldwide and is caused by a microorganism called *Chlamydia trachomatis*. The immune system of human beings can recover but reinfections happen often [11]. After repeated episodes, successive scars cause the edge of the eyelid to fold inward, causing pain and even permanent damage to the cornea which may lead to irreversible blindness. In fact, the assumption of an endemic background scenario can be understood as the effect on the population dynamics of the disease having attained one of its equilibrium states.

In this work we consider two models of competition and disease. The first one is a single species model in which there is intraspecific competition and the population is affected by a disease-parasite. Demography-competition is assumed to be governed by an adaptation of the Beverton-Holt model and the disease corresponds to a discrete SIS epidemic model with frequency-dependent transmission [12]. The second model corresponds to two competing species, one of them affected by specialist disease/parasite. Competition is built up from the classical discrete time competition Leslie-Gower model [13], and the disease is again introduced by means of a discrete SIS epidemic model. This ecoepidemic model happens to be rich enough to exhibit the complex behaviors described above but simple enough to be analytically tractable.

One of the differences between continuous and discrete models is that in the former all processes involved in the model (demography, competition, and infection/recovery) occur instantaneously at the same time, whereas in the later it is usual to consider that processes take place sequentially [14]. Keeping this idea as well as the separation of time scales in mind, in the construction of both models we assume that a large number k of epidemic-related events take place within each demographic/competition step. Concentrating our attention on the case of the two competing populations, the resulting model is a three-dimensional system. Making use of the existence of different time scales, it is possible to build up a reduced two-dimensional system that approximates

the behavior of the original system and that simplifies the analysis of the model (see [15] for a recent review in the field of discrete approximate reduction techniques). The resulting reduced system can be regarded as a generalization of the Leslie-Gower competition model and retains some of its properties, as the fact that the unique attractor states are equilibrium points. However, the reduced model allows for disease-induced multistability scenarios.

This work is organized as follows: in Section 2 we consider the interplay between intraspecific competition and a disease on a single species model. In Section 3 we introduce a competing species and carry on with the mathematical analysis of the resulting system and its ecological interpretation. Subsequently, we analyze separately the scenarios of a disease affecting either species growth or species competitive abilities. The conclusions in Section 4 and Appendix with the proof of the mathematical results complete the manuscript.

2. Disease and Intraspecific Competition

We consider a host population with density dependent regulation that is affected by a disease which acts on a shorter time scale than the demographic dynamics. The time unit of the discrete model is the one associated with its demographic part. We consider that, in this time unit, called slow, a single episode of demographic change following a number k of disease infection-recovery cycles happens. In this way we intend to describe the fact that pathogens exhibit outbreaks on short time scales, on the order of days or weeks, whereas demographic changes might be considered annual.

Time in the slow time unit is denoted by t . $N_S(t)$ and $N_I(t)$ represent the susceptible and the infected individuals, 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.

The disease dynamics is defined by means of the discrete time SIS epidemic model studied in [12] whose associated map is

$$F(N_S, N_I) = (F_S(N_S, N_I), F_I(N_S, N_I)) \\ = \left(N_S - \frac{\beta N_S N_I}{N_S + N_I} + \gamma N_I, N_I + \frac{\beta N_S N_I}{N_S + N_I} - \gamma N_I \right), \quad (1)$$

so that the total effect of disease during one slow time unit is represented by its k th iterate that we denote as $F^{(k)}$.

We assume 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 dynamics keeps constant the population size

$$F_S(N_S, N_I) + F_I(N_S, N_I) = N_S + N_I. \quad (2)$$

Henceforth we assume that the following inequalities hold:

$$\beta < (1 + \sqrt{\gamma})^2, \quad \gamma \leq 1, \quad (3)$$

so that if we denote $\mathbf{R}_+^2 = [0, \infty) \times [0, \infty)$ and $\mathring{\mathbf{R}}_+^2 = (0, \infty) \times (0, \infty)$, one has $F(\mathbf{R}_+^2) \subset \mathring{\mathbf{R}}_+^2$ [12]. Note that condition $\gamma \leq 1$

amounts to requiring that the number of recovered individuals in one time step can not be greater than the number of infected individuals, which clearly is necessary for the number of infected individuals to remain positive. The second condition in (3) says that the transmission coefficient can not be too high with respect to the recovery coefficient because, if this were not so, the number of susceptibles might become negative.

The asymptotic behavior of the solutions of the discrete system defined by map F (1) is studied in [12]. 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 infected individuals monotonically decreases to 0; that is, positive solutions converge to the disease-free equilibrium $N_0^* = (N, 0)$, where N is the constant population size. On the other hand, if $R_0 > 1$ the disease becomes endemic. Assuming, in addition to inequalities (3), that

$$\gamma < \beta \leq 2 + \gamma, \quad (4)$$

positive solutions converge to an asymptotically stable endemic equilibrium

$$N_e^* = (N_S^*, N_I^*) = \left(\left(\frac{\gamma}{\beta} \right) N, \left(1 - \frac{\gamma}{\beta} \right) N \right). \quad (5)$$

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

In the following we will restrict to the case where (4) holds and therefore the endemic equilibrium is stable. The fact that the fast disease process attains an equilibrium point in the long term allows one to reduce the dimension of the system that we are proposing. This equilibrium is expressed as the limit, \bar{F} , of the iterates of map F

$$\bar{F}(N_S, N_I) := \lim_{k \rightarrow \infty} F^{(k)}(N_S, N_I) = (\nu N, (1 - \nu)N), \quad (6)$$

where $N := N_S + N_I$ and ν is defined by

$$\nu := \begin{cases} 1, & \text{if } \beta \leq \gamma, \\ \frac{\gamma}{\beta}, & \text{if } \gamma < \beta \leq 2 + \gamma. \end{cases} \quad (7)$$

Note that $\nu = 1$ implies that the disease is eradicated.

To build up our demographic model where we have to take into account susceptible and infected individuals, we start from the Beverton-Holt model [16]

$$N(t+1) = \frac{bN(t)}{1 + cN(t)}, \quad (8)$$

where the positive parameters b and c are the intrinsic growth rate and the intraspecific competition coefficient. There are two possible asymptotic behaviors of the solutions of equation (8) with positive initial condition:

- (1) If $b \leq 1$ then $\lim_{t \rightarrow \infty} N(t) = 0$.
- (2) If $b > 1$ then $\lim_{t \rightarrow \infty} N(t) = (b - 1)/c$.

Note that this corresponds to a logistic behavior with carrying capacity $(b - 1)/c$.

Now we adapt the Beverton-Holt model to consider individuals classified into susceptible and infected. The trait-mediated indirect effects of parasites on hosts are considered both in the growth and in the intraspecific competition. We assume different intrinsic growth rates for susceptible, b_S , and infected, b_I , individuals. We also distinguish four intraspecific competition coefficients: c_{SS} , c_{SI} , c_{IS} , and c_{II} . The resulting model is

$$\begin{aligned} N_S(t+1) &= \frac{b_S N_S(t)}{1 + c_{SS} N_S(t) + c_{SI} N_I(t)}, \\ N_I(t+1) &= \frac{b_I N_I(t)}{1 + c_{IS} N_S(t) + c_{II} N_I(t)}. \end{aligned} \quad (9)$$

To build up the complete model combining the demographic and the disease processes we compose the k th iterate $F^{(k)}$ of map F (1) and the map defining system (9). Using the notation $F^{(k)}(N_S, N_I) = (F_S^{(k)}(N_S, N_I), F_I^{(k)}(N_S, N_I))$ the complete model has the form

$$\begin{aligned} N_S(t+1) &= \frac{b_S F_S^{(k)}(N_S(t), N_I(t))}{1 + c_{SS} F_S^{(k)}(N_S(t), N_I(t)) + c_{SI} F_I^{(k)}(N_S(t), N_I(t))}, \\ N_I(t+1) &= \frac{b_I F_I^{(k)}(N_S(t), N_I(t))}{1 + c_{IS} F_S^{(k)}(N_S(t), N_I(t)) + c_{II} F_I^{(k)}(N_S(t), N_I(t))}. \end{aligned} \quad (10)$$

System (10) can be reduced to a scalar equation from which we can approximate the asymptotic behavior of its solutions. To do so we make use of discrete approximate reduction techniques and follow the reduction procedure presented in [15, 17]. Assuming that the disease process has attained its equilibrium (6), the dynamics of the total number of individuals in the population, $N = N_S + N_I$, can be approximated by the following equation:

$$\begin{aligned} N(t+1) &= \frac{b_S \nu N(t)}{1 + c_{SS} \nu N(t) + c_{SI} (1 - \nu) N(t)} \\ &+ \frac{b_I (1 - \nu) N(t)}{1 + c_{IS} \nu N(t) + c_{II} (1 - \nu) N(t)}, \end{aligned} \quad (11)$$

obtained by adding up the two equations in (10) and using the following approximations: $N_S \approx \nu N$ and $N_I \approx (1 - \nu)N$ [18].

We can write (11) in the next simplified form

$$N(t+1) = \frac{b_1 N(t)}{1 + c_1 N(t)} + \frac{b_2 N(t)}{1 + c_2 N(t)}, \quad (12)$$

with four parameters defined as $b_1 = b_S \nu$, $b_2 = b_I (1 - \nu)$, $c_1 = c_{SS} \nu + c_{SI} (1 - \nu)$, and $c_2 = c_{IS} \nu + c_{II} (1 - \nu)$. The asymptotic behavior of its solutions is stated in the next theorem.

Theorem 1. *One considers equation (12) with b_1 , c_1 , and c_2 being positive and b_2 nonnegative. Let $N(t)$ be any solution of (12) with positive initial condition:*

- (1) If $b_1 + b_2 \leq 1$ then $\lim_{t \rightarrow \infty} N(t) = 0$.
 (2) If $b_1 + b_2 > 1$ then $\lim_{t \rightarrow \infty} N(t) = \bar{N}$, where

$$\bar{N} = \frac{1}{2c_1c_2} \left((b_2 - 1)c_1 + (b_1 - 1)c_2 + \sqrt{((1 - b_2)c_1 + (1 - b_1)c_2)^2 + 4(b_1 + b_2 - 1)c_1c_2} \right). \quad (13)$$

Proof. See Th. A.4 in [18]. \square

Making use of the last theorem and the results on approximate aggregation in [18], we can decide the asymptotic behavior of solutions of system (10). We assume that the disease becomes endemic ($R_0 > 1$, i.e., $\nu < 1$) because otherwise system (10) behaves as the scalar Beverton-Holt model. Therefore we have that, for any positive initial condition, the solution $(N_S(t), N_I(t))$ of system (10) verifies the following:

- (1) If $b_S\nu + b_I(1 - \nu) \leq 1$ then it approximately tends to $(0, 0)$.
 (2) If $b_S\nu + b_I(1 - \nu) > 1$ then it approximately tends to $W(\bar{N}/R_0, (1 - 1/R_0)\bar{N})$ where W is the transformation associated with the competition process (9); that is,

$$W \left(\frac{\bar{N}}{R_0}, \left(1 - \frac{1}{R_0}\right)\bar{N} \right) = \left(\frac{b_S^1 \bar{N}}{R_0(1 + c_1 \bar{N})}, \frac{b_I^1 (1 - 1/R_0) \bar{N}}{1 + c_2 \bar{N}} \right). \quad (14)$$

Note that if the disease is established, either the population tends to extinction or it tends to an endemic equilibrium. To analyze the influence of the parasite on the population dynamics we compare the asymptotic behaviors of the solutions of (8), representing the dynamics of the disease-free population, and (11), which yields the approximate long-term behavior of system (10). For the comparison we assume that parameters affecting susceptible individuals in system (10) coincide with the corresponding ones in (8), that is, $b = b_S$ and $c = c_{SS}$.

It is straightforward to check that $\max\{b_S, b_I\} < 1$ implies extinction and $\min\{b_S, b_I\} > 1$ endemic stabilization. On the other hand, if $b_S > 1$, that is, if the population without disease tends to $(b_S - 1)/c_{SS}$, then the disease leads the population to extinction for b_I small enough, specifically when $b_I < (1 - b_S\nu)/(1 - \nu)$. Conversely, if the population is not viable without disease, $b_S < 1$, a large enough infected growth rate, $b_I > (1 - b_S\nu)/(1 - \nu)$, entails the population attaining equilibrium $(\bar{N}/R_0, (1 - 1/R_0)\bar{N})$.

If the population attains a positive equilibrium in both cases, with and without disease, we can explore the influence of the disease by comparing the two final population sizes. Without disease it is $(b_S - 1)/c_{SS}$ and with disease \bar{N} . We do the comparison in the relevant particular case in which the disease does not affect the competitive abilities of individuals, that is, $c_{SS} = c_{SI} = c_{II} = c$. In this case we obtain the ratio

$$\frac{\bar{N}}{(b_S - 1)/c} = \frac{b_S\nu + b_I(1 - \nu) - 1}{b_S - 1}. \quad (15)$$

As a function of the growth rate of infected individuals, b_I , this ratio is increasing and equals 1 for $b_I = b_S$. More interesting is its dependence on $\nu = 1/R_0$

$$\frac{\bar{N}}{(b_S - 1)/c} = \frac{b_S - b_I}{b_S - 1}\nu + \frac{b_I - 1}{b_S - 1}. \quad (16)$$

If $b_S > b_I$ then it increases with ν ; that is, the larger R_0 , the smaller the ratio. Conversely, if $b_S < b_I$ the larger R_0 , the larger the ratio.

3. Disease and Interspecific Competition

3.1. Model Construction and Reduction. In this section we generalize the setting of the previous one and introduce a second species, which we consider disease free, which competes, according to the well-known Leslie-Gower model [13], with the species which is affected by the disease. Specifically, let us consider two species that we denote by 1 and 2, in competition and a disease defined by system (1) and which affects only species 1. Let N_S^1 and N_I^1 represent the number of susceptible and infected individuals of species 1 and let N^2 be the amount of individuals of species 2. Then, the slow dynamics is described by

$$\begin{aligned} N_S^1(t+1) &= \frac{b_S^1 N_S^1(t)}{1 + c_{SS} N_S^1(t) + c_{SI} N_I^1(t) + c_{S2} N^2(t)}, \\ N_I^1(t+1) &= \frac{b_I^1 N_I^1(t)}{1 + c_{IS} N_S^1(t) + c_{II} N_I^1(t) + c_{I2} N^2(t)}, \\ N^2(t+1) &= \frac{b^2 N^2(t)}{1 + c_{2S} N_S^1(t) + c_{2I} N_I^1(t) + c_{22} N^2(t)}, \end{aligned} \quad (17)$$

where all growth and competition coefficients are assumed to be positive.

As in Section 2, we consider that in the time unit of the model a single episode of demographic change happens, defined by (17), followed by a number k of disease infection-recovery cycles corresponding to the SIS model (1) introduced in Section 2. Then, the complete system that takes into account the joint effect of the slow and fast processes takes the form

$$N_S^1(t+1) = \frac{b_S^1 F_S^{(k)}(N_S^1(t), N_I^1(t))}{1 + c_{SS} F_S^{(k)}(N_S^1(t), N_I^1(t)) + c_{SI} F_I^{(k)}(N_S^1(t), N_I^1(t)) + c_{S2} N^2(t)},$$

$$\begin{aligned}
 N_I^1(t+1) &= \frac{b_I^1 F_I^{(k)}(N_S^1(t), N_I^1(t))}{1 + c_{IS} F_S^{(k)}(N_S^1(t), N_I^1(t)) + c_{II} F_I^{(k)}(N_S^1(t), N_I^1(t)) + c_{I2} N^2(t)}, \\
 N^2(t+1) &= \frac{b^2 N^2(t)}{1 + c_{2S} F_S^{(k)}(N_S^1(t), N_I^1(t)) + c_{2I} F_I^{(k)}(N_S^1(t), N_I^1(t)) + c_{22} N^2(t)}.
 \end{aligned}
 \tag{18}$$

If we follow the reduction procedure presented in [15, 17, 18], which essentially consists in adding up the first two equations in (18) and using the approximations $N_S^1 \approx \nu(N_S^1 + N_I^1)$ and $N_I^1 \approx (1 - \nu)(N_S^1 + N_I^1)$, we obtain the reduced system

$$\begin{aligned}
 N^1(t+1) &= \frac{b_S^1 \nu N^1(t)}{1 + c_{S1} N^1(t) + c_{S2} N^2(t)} \\
 &+ \frac{b_I^1 (1 - \nu) N^1(t)}{1 + c_{I1} N^1(t) + c_{I2} N^2(t)}, \tag{19} \\
 N^2(t+1) &= \frac{b^2 N^2(t)}{1 + c_{21} N^1(t) + c_{22} N^2(t)},
 \end{aligned}$$

where $N^1 := N_S^1 + N_I^1$, $c_{S1} := c_{SS}\nu + c_{SI}(1 - \nu)$, $c_{I1} := c_{IS}\nu + c_{II}(1 - \nu)$, and $c_{21} := c_{2S}\nu + c_{2I}(1 - \nu)$.

Note that system (19) generalizes the classical Leslie-Gower competition model [13] in the sense that at $\nu = 1$ (i.e., the disease-free scenario) system (19) becomes the usual Leslie-Gower competition model.

3.2. Model Analysis. In order to carry out the mathematical treatment of system (19) we express it in the form

$$(N^1(t+1), N^2(t+1)) = H(N^1(t), N^2(t)), \tag{20}$$

where H is the map defined by

$$\begin{aligned}
 H(x_1, x_2) &= (H_1(x_1, x_2), H_2(x_1, x_2)) \\
 &= (\phi_1(x_1, x_2) x_1, \phi_2(x_1, x_2) x_2), \\
 \phi_1(x_1, x_2) &:= \frac{r_S}{1 + c_{S1} x_1 + c_{S2} x_2} + \frac{r_I}{1 + c_{I1} x_1 + c_{I2} x_2}, \tag{21} \\
 \phi_2(x_1, x_2) &:= \frac{r_2}{1 + c_{21} x_1 + c_{22} x_2}, \\
 r_S &:= b_S^1 \nu, \quad r_I := b_I^1 (1 - \nu), \quad r_2 = b^2.
 \end{aligned}$$

Note that all the parameters of the model are positive except possibly r_I that is nonnegative. As we will see, the numbers

$$\begin{aligned}
 \phi_1(0, 0) &= r_S + r_I = b_S^1 \nu + b_I^1 (1 - \nu), \\
 \phi_2(0, 0) &:= r_2 = b^2
 \end{aligned}
 \tag{22}$$

will play an important role in the dynamics of the system.

We note that the functional form of system (20) is a particular case of a model obtained in [19] which studies

the dynamics of two spatially distributed populations in competition in which there is fast migration of individuals between the different patches. In that work an analytical study of the system was carried out in the case in which $\phi_i(0, 0) \leq 1$ for at least one value of $i = 1, 2$, which essentially corresponds to our Proposition 3 and Theorem 4. In Theorem 6 we provide a full analytical study of the behavior of the orbits of the system in the case $\phi_i(0, 0) > 1$ for $i = 1, 2$, which was only addressed numerically in [19].

Let $A_1 = \{(x_1, 0) : x_1 > 0\}$ and $A_2 = \{(0, x_2) : x_2 > 0\}$ be the positive axes. It is immediate to realize that the sets \mathbf{R}_+^2 , \mathbf{R}_+^2 , and A_i , $i = 1, 2$ are forward invariant by H . In the sequel, unless otherwise stated we will always assume that we are working on \mathbf{R}_+^2 .

Let us study the isoclines of the system, that is, the sets defined by $x_1 = H_1(x_1, x_2)$ and $x_2 = H_2(x_1, x_2)$. Clearly, besides the positive axes A_1 (resp., A_2) in which the variable x_2 (resp., x_1) is constant, the isoclines are the curves Γ_i defined by $\phi_i(x_1, x_2) = 1$, for $i = 1, 2$.

Lemma 2. *The isoclines Γ_1 and Γ_2 are, respectively, a hyperbola and a straight line. For each $i = 1, 2$, Γ_i intersects \mathbf{R}_+^2 if and only if $\phi_i(0, 0) > 1$ and in that case Γ_i intersects both the positive axes A_1 and A_2 . Moreover $\Gamma_1 \cap \mathbf{R}_+^2$ can be written in the form $x_2 = \Phi(x_1)$, $x_1 \in [0, R]$ for a certain $R > 0$, where Φ is a strictly decreasing convex function.*

Except in the degenerate case in which Γ_1 and Γ_2 are in fact the same straight line, there can exist at most two positive equilibriums.

Proof. See Appendix. □

Let us now consider the existence of equilibriums for system (20). Note that $E_0^* := (0, 0)$ is an equilibrium point for all values of the parameters. Using Lemma 2 we conclude the following:

- (i) For each $i = 1, 2$, there exists a semitrivial equilibrium point E_i^* on the positive axis A_i if and only if $\phi_i(0, 0) > 1$. In that case the semitrivial equilibrium is unique.
- (ii) A necessary condition for the existence of a positive equilibrium is that $\phi_i(0, 0) > 1$ for both $i = 1, 2$, and in that case there can exist at most two such equilibriums.

In what follows we will write $(x_1, x_2) \leq (x'_1, x'_2)$ (resp., $(x_1, x_2) < (x'_1, x'_2)$) to denote that $x_1 \leq x'_1$ and $x_2 \leq x'_2$ (resp., $x_1 < x'_1$ and $x_2 < x'_2$). Similarly, we will write

$(x_1, x_2) \leq_K (x'_1, x'_2)$ (resp., $(x_1, x_2) <_K (x'_1, x'_2)$) to denote that $x_1 \leq x'_1$ and $x_2 \geq x'_2$ (resp., $x_1 < x'_1$ and $x_2 > x'_2$).

Proposition 3. *Let us consider system (20).*

(a) *All solutions in \mathbf{R}_+^2 are forward bounded. More specifically,*

$$H(\mathbf{R}_+^2) \subset S := \left[0, \frac{r_S}{c_{S1}} + \frac{r_I}{c_{I1}}\right) \times \left[0, \frac{r_2}{c_{22}}\right). \quad (23)$$

(b) *H is strongly competitive in \mathbf{R}_+^2 ; that is, if $x, x' \in \mathbf{R}_+^2$ are distinct points with $x \leq_K x'$ it follows that $H(x) <_K H(x')$ [20].*

(c) *All orbits in \mathbf{R}_+^2 are eventually componentwise monotone; that is, for each $(N^1(0), N^2(0)) \in \mathbf{R}_+^2$, the corresponding solution $(N^1(t), N^2(t))$ verifies that $N^i(t)$ is eventually monotone for each $i = 1, 2$. Moreover, all orbits tend to an equilibrium as $t \rightarrow \infty$.*

Proof. See Appendix. \square

The next result analyzes the behavior of solutions of system (20) except in the case in which $\phi_i(0, 0) > 1$, $i = 1, 2$.

Theorem 4. *Let us consider system (20).*

(a) *For each $i = 1, 2$, if $\phi_i(0, 0) \leq 1$ then for any initial value on \mathbf{R}_+^2 species i tends to extinction as $t \rightarrow \infty$.*

(b) *If $\phi_i(0, 0) \leq 1$ for $i = 1, 2$, all orbits in \mathbf{R}_+^2 tend to E_0^* , and if the inequalities are strict then E_0^* is hyperbolic. If $\phi_i(0, 0) > 1$ for $i = 1, 2$, then E_0^* is a repeller and therefore no orbit can converge to E_0^* .*

(c) *If $\phi_1(0, 0) > 1$ and $\phi_2(0, 0) \leq 1$ then (1) all orbits with $N^1(0) = 0$ tend to E_0^* and (2) all orbits with $N^1(0) > 0$ tend to E_1^* .*

(d) *If $\phi_2(0, 0) > 1$ and $\phi_1(0, 0) \leq 1$ then (1) all orbits with $N^2(0) = 0$ tend to E_0^* and (2) all orbits with $N^2(0) > 0$ tend to E_2^* .*

Proof. See Appendix. \square

Let us now consider the case in which $\phi_i(0, 0) > 1$ for $i = 1, 2$. In the first place we study the local stability of the semitrivial equilibria and the behavior of orbits on the positive axes.

Proposition 5. *Let us consider system (20) and let us assume that $\phi_i(0, 0) > 1$ for $i = 1, 2$.*

(a) *All orbits starting on the positive i -axis A_i converge to E_i^* , $i = 1, 2$.*

(b) *Let $i = 1, 2$ be fixed. If $\phi_j(E_i^*) < 1$, $j \neq i$, then E_i^* is hyperbolic and attracting. If $\phi_j(E_i^*) > 1$, $j \neq i$, then E_i^* is hyperbolic and unstable.*

Proof. (a) Let $i = 1, 2$ be fixed. We know that A_i is invariant for H and then any orbit corresponding to an initial condition

on A_i must converge to either E_0^* or E_i^* . From Theorem 4 part (b) E_0^* is a repeller and therefore the result follows.

(b) It follows from the usual analysis of the eigenvalues of the corresponding Jacobian matrix. Standard calculations lead to the results bearing in mind that $\phi_i(E_i^*) = 1$. \square

In order to study the behavior of solutions for positive initial conditions, we will consider different cases based on the relative position of the intercepts of the isoclines. To this end, we define R_{ij} as the j -intercept of Γ_i , $i, j = 1, 2$. It is immediate to check that R_{ij} is given by

$$R_{1j} = \frac{1}{2c_{Sj}c_{Ij}} \left(\alpha_j + \sqrt{\alpha_j^2 + 4c_{Sj}c_{Ij}(r_S + r_I - 1)} \right), \quad j = 1, 2, \quad (24)$$

$$R_{2j} = \frac{r_2 - 1}{c_{2j}}, \quad j = 1, 2,$$

where $\alpha_j := r_S(c_{Ij} - 1) + r_I(c_{Sj} - 1)$, $j = 1, 2$.

Now we distinguish the following scenarios:

(i) Case A: $R_{11} < R_{21}$ and $R_{12} > R_{22}$.

(ii) Case B: $R_{11} > R_{21}$ and $R_{12} < R_{22}$.

(iii) Case C_1 : $R_{11} > R_{21}$ and $R_{12} > R_{22}$.

(iv) Case C_2 : $R_{11} < R_{21}$ and $R_{12} < R_{22}$.

Taking into account Lemma 2, the isoclines divide \mathbf{R}_+^2 in a finite number of open connected and disjoint sets U_k , $k = 1, \dots, q$, in which x_i is either strictly increasing or strictly decreasing for each $i = 1, 2$. To describe the kind of monotonicity in each region we will use arrows, in such a way that, for example, the situation in which x_1 decreases and x_2 increases corresponding to a horizontal arrow pointing to the left and a vertical arrow pointing up. Figure 1 shows the different configurations. Note that in case C_1 we can distinguish two generic cases depending on whether the two isoclines do not intersect (case C_{1a}) or intersect at two points (case C_{1b}).

The following result deals with the number and location of the coexistence equilibria and with the global behavior of solutions for positive initial conditions.

Theorem 6. *Let us consider system (20) and let us assume that $\phi_i(0, 0) > 1$ for $i = 1, 2$.*

(1) *If there exists only one positive equilibrium that one denotes as $E_3^* = (e_1^*, e_2^*)$, then one has $0 < e_1^* < R_{11}$ and $0 < e_2^* < R_{22}$. In the case that there exist two positive equilibria $E_3^* = (e_{31}^*, e_{32}^*)$ and $E_4^* = (e_{41}^*, e_{42}^*)$, then they can be ordered in such a way that $0 < e_{31}^* < e_{41}^* < R_{11}$ and $0 < e_{42}^* < e_{32}^* < R_{22}$.*

(2) *Study of the different cases is as follows:*

(i) *Case A. E_1^* and E_2^* are hyperbolic and unstable. There exists exactly one positive equilibrium E_3^* and it is hyperbolic and attracting. All orbits starting on \mathbf{R}_+^2 converge to E_3^* .*

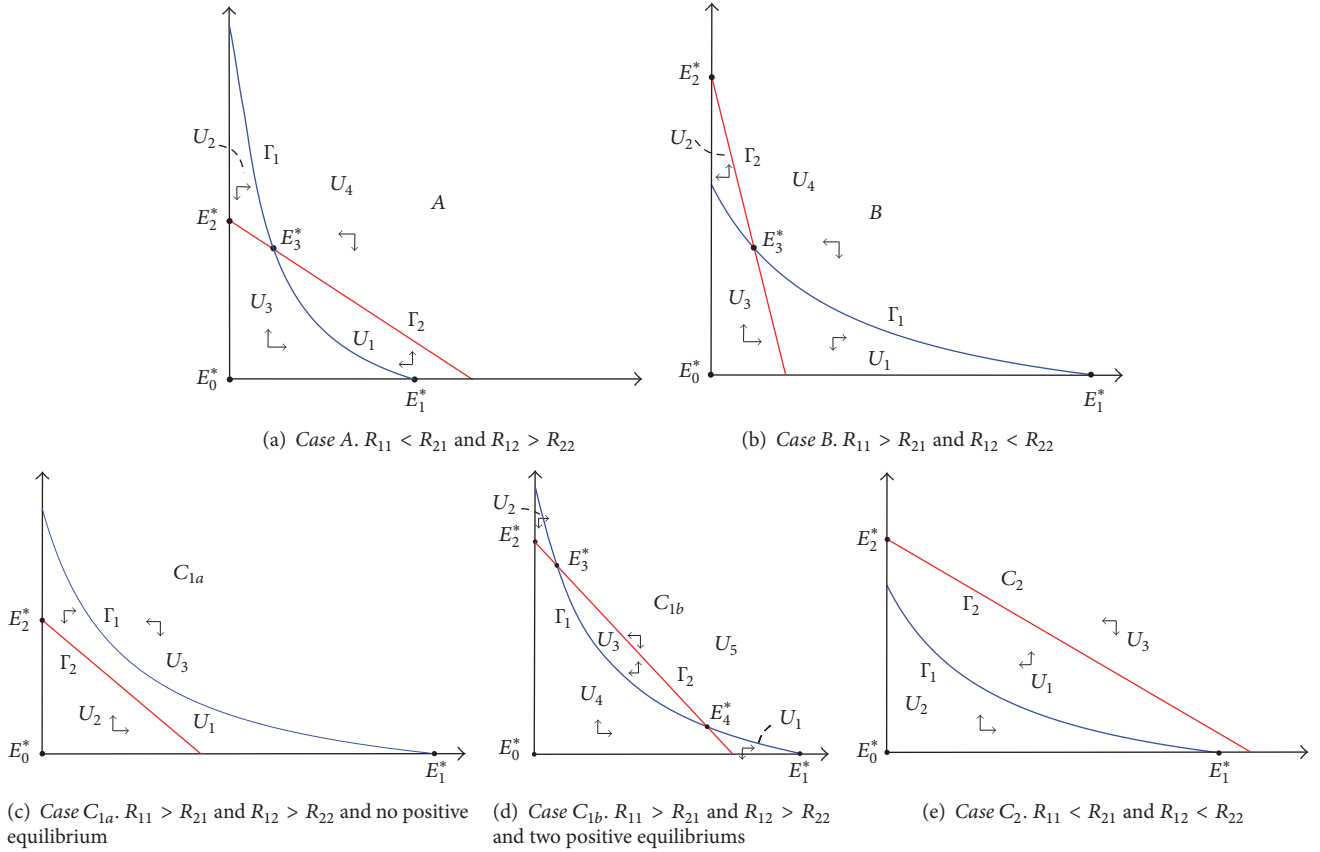


FIGURE 1: Different configurations of isoclines and equilibrium points of system (20) when $\phi_i(0, 0) > 1$ for $i = 1, 2$, in terms of the relative position of the intercepts of isoclines, R_{ij} (24).

(ii) Case B. E_1^* and E_2^* are hyperbolic and attracting. There exists exactly only one positive equilibrium E_3^* which is hyperbolic and a saddle. Moreover E_3^* can not attract any open set and therefore almost all orbits starting in $\overset{\circ}{\mathbf{R}}_+^2$ converge to either E_1^* or E_2^* .

(iii) Case C_1 . E_1^* is hyperbolic and attracting, E_2^* is hyperbolic and unstable, and there can exist zero (case C_{1a}), two (case C_{1b}), or one (nongeneric case) positive equilibria. Let us deal with the generic cases:

(a) Case C_{1a} . All orbits starting on $\overset{\circ}{\mathbf{R}}_+^2$ converge to E_1^* .

(b) Case C_{1b} . If one orders the two positive equilibria E_3^* and E_4^* as indicated in part (1), then no orbits starting in $\overset{\circ}{\mathbf{R}}_+^2$ can converge to E_2^* . Besides E_3^* is hyperbolic and attracting and E_4^* is hyperbolic and a saddle. Moreover E_4^* can not attract any open set and therefore almost all orbits starting in $\overset{\circ}{\mathbf{R}}_+^2$ converge to either E_1^* or E_3^* .

(iv) Case C_2 . E_2^* is hyperbolic and asymptotically stable, E_1^* is hyperbolic and unstable, and there

are no positive equilibria. All orbits starting on $\overset{\circ}{\mathbf{R}}_+^2$ converge to E_2^* .

Proof. See Appendix. □

The analytic expression of the positive equilibrium points is quite complex and we omit it, although we will specify it in a particular case considered in Section 3.4.

In order to get a qualitative idea of the effect of the disease in the competition dynamics, let us compare the different scenarios regarding the long-term behavior of the classical Leslie-Gower competition model (in the case where there can exist positive equilibria) with those resulting from Theorem 6 in which the effect of a disease in species 1 is taken into account. In the classical Leslie-Gower competition model, we can have (a) competitive exclusion of one of the two species for any initial condition (corresponding to our cases C_{1a} and C_2), (b) competitive exclusion of one species depending on the initial condition (corresponding to our case B), and (c) species coexistence for any initial condition (corresponding to our case A). Therefore, the introduction of the disease includes a new scenario which is exclusion of species 2 or species coexistence depending on initial conditions (our case C_{1b}).

As we did in Section 2, the results on approximate aggregation in [18] together with Theorems 4 and 6 allow

one to study the asymptotic behavior of solutions of system (18). Loosely speaking, the results in [18] guarantee that when a solution of the reduced system (20) with initial condition (a_1, a_2) tends to a hyperbolic equilibrium (x_1^*, x_2^*) then if k is large enough, the corresponding solution of system (18) (i.e., a solution of (18) with initial condition such that $N_S^1(0) + N_I^1(0) = a_1$, $N^2(0) = a_2$) tends to an equilibrium which is approximately $T(\nu x_1^*, (1 - \nu)x_1^*, x_2^*)$ where T is the mapping, defined by (17), that corresponds to the competition process; that is,

$$T(\nu x_1^*, (1 - \nu)x_1^*, x_2^*) = \left(\frac{b_S^1 \nu x_1^*}{1 + c_{S1} x_1^* + c_{S2} x_2^*}, \frac{b_I^1 (1 - \nu) x_1^*}{1 + c_{I1} x_1^* + c_{I2} x_2^*}, \frac{b^2 x_2^*}{1 + c_{21} x_1^* + c_{22} x_2^*} \right). \quad (25)$$

In the case in which the disease is endemic, $R_0 > 1$, the previous expression has the form $T(x_1^*/R_0, (1 - 1/R_0)x_1^*, x_2^*)$.

Figure 2 shows the basins of attraction of E_1^* and E_3^* for the reduced system together with some solutions of the original system for a choice of parameters corresponding to case C_{1b} . Note in particular that, as predicted by the theory, when the initial condition is such that the solution of the aggregated system tends to $E_1^* = (x_1^*, 0)$, the solution of the original system converges to $T(\nu x_1^*, (1 - \nu)x_1^*, 0) = (b_S^1 \nu x_1^*/(1 + c_{S1} x_1^*), b_I^1 (1 - \nu)x_1^*/(1 + c_{I1} x_1^*), 0)$.

In the next sections we analyze particular cases of the above setting in order to gain more insight into the effect of infection in the dynamics of competition.

3.3. Disease Modified Competitive Abilities. In [21] the consequences of parasite-modified competitive abilities of prey are studied in a predator-prey system. They find that they are crucial by showing that they may lead to coexistence of predator and prey, a situation that is impossible otherwise. These findings enhance the importance of the indirect effects of parasite-mediated modifications of host life-history traits beyond the direct effects as, that is, on reproduction.

We consider a first particular case in which disease affects only the competitive abilities of infected individuals. Thus we assume that the growth rate of species 1 is unaffected by the disease, that is, $b^1 := b_S^1 = b$. Moreover, we suppose that the competitive abilities of infected individuals in species 1 are worse than those corresponding to the susceptible individuals. Specifically we assume $c_{SS} > c_{SI}$, $c_{2S} > c_{2I}$; that is, the interference the susceptible individuals produce either on themselves or on species 2 individuals is larger than the corresponding interference exerted by infected individuals. At the same time we suppose that the rest of intraspecific competition coefficients of species 1 are equal, that is, $c_{II} = c_{IS} = c_{SS}$.

At first glance we could predict that our assumptions will yield a negative impact in the capacity of species 1 to compete against species 2 in the presence of infected individuals. However we will see that, for certain range of parameter values, the combined effect of competition and fast disease dynamics gives rise to scenarios in which the disease endemicity ($1/\nu = R_0 > 1$) improves the outcome of competition for species 1 with respect to the disease-free setting ($\nu = 1$).

To illustrate this last point we treat the particular example of system (17) with parameters values verifying the above relations. Let $b^1 := b_S^1 = b_I^1 > 1$, $b^2 = 5$, $c_{SS} = 3$, $c_{SI} = 2.8$, $c_{S2} = c_{IS} = c_{II} = c_{I2} = c_{22} = 1$, $c_{2S} = 2$, $c_{2I} = 1.8$, and $\nu \in (0, 1]$. Applying Theorem 6 to the corresponding reduced system (19) we show in Figure 3 the asymptotic behavior of solutions of system (17) for a range of values of parameters ν and b^1 .

In the first place, notice that all five scenarios shown in Theorem 6 can happen. Secondly, we highlight two unexpected behaviors that appear as the basic reproduction number of the disease $R_0 = 1/\nu$ increases. The first one corresponds to values of parameter b^1 in the approximate range 3.3 to 5. Solutions tend to species 1 exclusion, case C_2 , in the disease-free situation ($\nu = 1$) whereas increasing R_0 (decreasing ν) makes case B appear, that is, the exclusion of either species 1 or species 2 depending on the initial sizes of both populations. A second unexpected change of asymptotic behavior appears for $b^1 > 5$. The tendency to coexistence, case A , in the disease-free scenario is transformed, by increasing R_0 , into case C_{1a} , exclusion of species 2, sometimes going through C_{1b} , which also entails exclusion of species 2 depending on initial conditions.

3.4. Disease Modified Growth Capabilities. We consider now another particular case in which we can strengthen some of the analytical results of Section 3.2 and we can extract some interesting quantitative biological information. Let us assume that the disease does not affect the competitive abilities of individuals but it only modifies the reproductive capabilities of the infected species; that is,

$$\begin{aligned} c_{11} &:= c_{SS} = c_{SI} = c_{IS} = c_{II}, \\ c_{12} &:= c_{S2} = c_{I2}, \\ c_{21} &:= c_{2S} = c_{2I}. \end{aligned} \quad (26)$$

Then system (19) takes the form

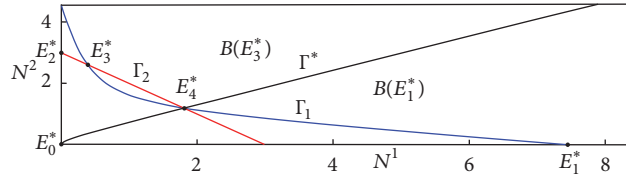
$$\begin{aligned} N^1(t+1) &= \frac{(r_S + r_I) N^1(t)}{1 + c_{11} N^1(t) + c_{12} N^2(t)}, \\ N^2(t+1) &= \frac{r_2 N^2(t)}{1 + c_{21} N^1(t) + c_{22} N^2(t)} \end{aligned} \quad (27)$$

which is a classical Leslie-Gower model [22] with disease modified growth rates. From the general setting of Section 3.1, the isocline Γ_1 now degenerates into a straight line. The isoclines intercepts (24) simplify to

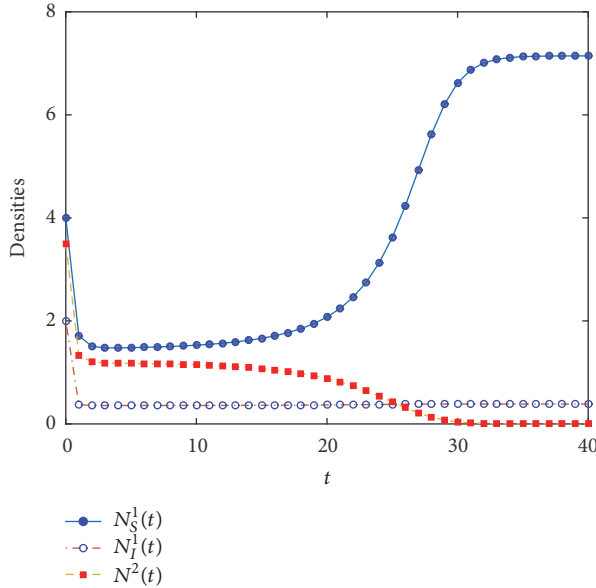
$$\begin{aligned} R_{1j} &= \frac{r_S + r_I - 1}{c_{1j}} = \frac{b_S^1 \nu + b_I^1 (1 - \nu) - 1}{c_{1j}}, \\ R_{2j} &= \frac{b^2 - 1}{c_{2j}}, \end{aligned} \quad (28)$$

$$j = 1, 2.$$

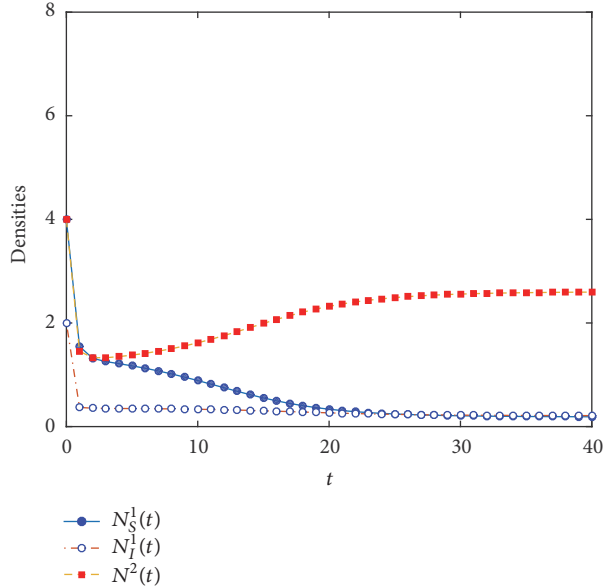
Then we can sharpen the results in Theorem 6. In the first place, since the isoclines are straight lines there can be at



(a) Basins of attraction \$B(E_1^*)\$ and \$B(E_3^*)\$ of \$E_1^*\$ and \$E_3^*\$ for system (19) and separatrix curve \$\Gamma^*\$



(b) Solution of (18) for initial conditions \$N_S^1(0) = 4\$, \$N_I^1(0) = 2\$, and \$N^2(0) = 4\$, belonging to \$B(E_1^*)\$



(c) Solution of (18) for initial conditions \$N_S^1(0) = 4\$, \$N_I^1(0) = 2\$, and \$N^2(0) = 3.5\$, belonging to \$B(E_3^*)\$

FIGURE 2: Basins of attraction \$B(E_1^*)\$ and \$B(E_3^*)\$ of \$E_1^*\$ and \$E_3^*\$ for the aggregated system (19) and solutions versus time for the original system (18). The parameter values are \$k = 20\$, \$\gamma = 0.9\$, \$\beta = 1.125\$, \$b_S^1 = 10\$, \$b_I^1 = 20\$, \$b^2 = 4\$, \$c_{SS} = 0.75\$, \$c_{SI} = 2\$, \$c_{IS} = 9\$, \$c_{II} = 14\$, \$c_{S2} = 6\$, \$c_{I2} = 1\$, \$c_{2S} = 0.5\$, \$c_{2I} = 3\$, and \$c_{22} = 1\$, which correspond to case \$C_{1b}\$.

most one positive equilibrium \$E_3^* = (e_1^*, e_2^*)\$ which can be computed explicitly as

$$e_1^* = \frac{c_{22}(r_S + r_I - 1) - c_{12}(r_2 - 1)}{c_{11}c_{22} - c_{12}c_{21}}, \quad (29)$$

$$e_2^* = \frac{c_{11}(r_2 - 1) - c_{21}(r_S + r_I - 1)}{c_{11}c_{22} - c_{12}c_{21}}$$

and, moreover, we have the following.

Corollary 7. Consider system (27). Then, in addition to the results in Theorem 6 one has that case \$C_{1b}\$ can not happen and therefore in case \$C_1\$ (\$R_{11} > R_{21}\$ and \$R_{12} > R_{22}\$) all orbits starting on \$\mathbf{R}_+^2\$ converge to \$E_1^*\$.

Proof. The result is immediate taking into account that \$\Gamma_1\$ and \$\Gamma_2\$ are now straight lines and therefore conditions \$R_{11} > R_{21}\$ and \$R_{12} > R_{22}\$ imply that they do not intersect. \$\square\$

Let us concentrate in case A (coexistence for any initial condition). In order to quantify the effect of the disease in the equilibrium population, we set \$b^1 = b_S^1\$, \$b_I^1 = \alpha b^1\$, where \$\alpha > 0\$

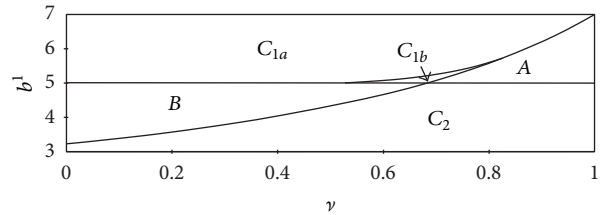


FIGURE 3: Asymptotic behavior cases (Theorem 6) of the solutions of system (17) for parameters values \$b^1 := b_S^1 = b_I^1 > 1\$, \$b^2 = 5\$, \$c_{SS} = 3\$, \$c_{SI} = 2.8\$, \$c_{S2} = c_{IS} = c_{II} = c_{I2} = c_{22} = 1\$, \$c_{2S} = 2\$, \$c_{2I} = 1.8\$, and \$\nu \in (0, 1]\$.

is a measure of the effect of the disease on the reproductive capability of infected individuals in species 1. In this way, using (29) we have that the quotient between the equilibrium population of species 1 with (\$0 < \nu < 1\$) and without disease (\$\nu = 1\$) is given by

$$\frac{c_{22}(b^1[\alpha + (1 - \alpha)\nu] - 1) - c_{12}(b^2 - 1)}{c_{22}(b^1 - 1) - c_{12}(b^2 - 1)}, \quad (30)$$

while the corresponding quotient for species 2 is

$$\frac{c_{21} (b^1 [\alpha + (1 - \alpha) \nu] - 1) - c_{11} (b^2 - 1)}{c_{21} (b^1 - 1) - c_{11} (b^2 - 1)}, \quad (31)$$

so that in particular if $\alpha < 1$ (resp., $\alpha > 1$) the long-term population size of species 1 is reduced (resp., increased) and the opposite happens to species 2.

4. Conclusion

In this work we have built up an ecoepidemic model in which disease dynamics is fast with respect to competition-demography and have carried out its mathematical analysis, which reveals a number of possible long-term scenarios for the populations.

From an applied point of view, biological controls as predators or competitors, along with disease-induced population size reduction, are often used to control harmful species. Some key questions are whether a given control strategy will be effective or whether it hides unexpected effects (as the well-known hydra effect [23]). In this direction we have obtained threshold values for species persistence/extinction (as in Theorems 1 and 4 or Proposition 5) and expressions (like (16), (30), and (31)) that provide an estimation of the impact on the community of a disease outbreak. Our analysis also reveals that, for certain values of the parameters, unexpected outcomes as those described in Section 3.3 may appear. There we have shown that there are situations in which as the basic reproductive number R_0 increases, the infected species improves as a competitor even though the interspecific competitive pressure exercised by infected individuals is lower than that exercised by susceptible individuals.

The theoretical results obtained in this work can encourage laboratory experiments to validate some of the unexpected results predicted by the model as well as modeling work in order to find plausible explanations for these scenarios. Our findings also suggest possible future lines of work, for example, the study of competition models in which both species are infected by either a shared disease or, alternatively, different diseases.

Appendix

Proofs of Results

Proof of Lemma 2. Clearly Γ_2 is a straight line. We can write Γ_1 as a conic $Ax_1^2 + 2Bx_2 + Cx_2^2 + Dx_1 + Ex_2 + F = 0$, where $A = c_{S1}c_{I1}$, $C = c_{S2}c_{I2}$, and $B = (c_{S1}c_{I2} + c_{S2}c_{I1})/2$ and D, E , and F are certain coefficients. The discriminant is $D = AC - B^2 = -(c_{S1}c_{I2} + c_{S2}c_{I1})^2/4$ which is negative, so the conic is a hyperbola. For each $x_1 \geq 0$, function $\phi_i(x_1, *)$ is strictly decreasing and tends to zero when $x_2 \rightarrow \infty$ (*) and the same happens if we interchange the variables; that is, for each $x_2 \geq 0$ function $\phi_i(*, x_2)$ is strictly decreasing and tends to zero when $x_2 \rightarrow \infty$ (**). As a consequence, the equation $\phi_i(x_1, x_2) = 1$ has a solution in $\mathring{\mathbf{R}}_+^2$ if and only if $\phi_i(0, 0) > 1$.

Clearly, if $\phi_2(0, 0) > 1$, then Γ_2 intersects both the positive axes. Let us now assume that $\phi_1(0, 0) > 1$. Using (*) we have

that for each value of $x_1 \geq 0$ there exists a unique value of x_2 such that $\phi_1(x_1, x_2) = 1$ and so we can write Γ_1 in the form $x_2 = \Phi(x_1)$, $x_1 \geq 0$ for a certain Φ . Using (*) and (**) it follows that the equation $\phi_1(0, x_2) = 1$ (resp., $\phi_1(x_1, 0) = 1$) has a unique positive solution and so Γ_1 intersects each positive axes in one point. Differentiating twice in $\phi_1(x_1, \Phi(x_1)) = 1$ we obtain after straightforward computations that $\Phi'(x_1) < 0$ and $\Phi''(x_1) \geq 0$ for $x_1 \geq 0$ and so the convexity of Φ follows.

The last part of the result follows taking into account the fact that a branch of a hyperbola can intersect a straight line in at most 2 points. \square

Proof of Proposition 3. The proof of (a) is straightforward. Regarding (b), it is immediate to check that, for $i, j \in \{1, 2\}$, $i \neq j$, the map $H_i(x_1, x_2)$ is strictly increasing as a function of x_i and strictly decreasing as a function of x_j . Therefore it follows trivially that if $x, x' \in \mathbf{R}_+^2$ are distinct points with $x \leq_K x'$ then $H(x) <_K H(x')$.

(c) In the first place we prove that H verifies property (O+) in [20, p. 343]. To do so we apply [20, Lemma 4.3] to $H : \bar{S} \rightarrow \bar{S}$ where S is defined in (23). We need to prove the following four conditions:

- (i) It is immediate that \bar{S} contains order intervals [20, p. 345] and is \leq_K -convex [20, p. 339].
- (ii) To prove that $\det DH(x_1, x_2) > 0$ for $(x_1, x_2) \in \bar{S}$ we used the CAS Maxima. The resulting expression contains a number of summands all of which are positive.
- (iii) Direct calculations prove that $DH(x_1, x_2)$ is K -positive in \bar{S} [20, p. 338]; that is, $\partial H_1/\partial x_1 > 0$, $\partial H_2/\partial x_2 > 0$, $\partial H_1/\partial x_2 \leq 0$, and $\partial H_2/\partial x_1 \leq 0$ in \bar{S} .
- (iv) The last hypothesis to prove is that H is injective. For that, following [20, Remark 4.2], it suffices to confirm that \bar{S} is compact and connected and that $H^{-1}(0, 0)$ is a single point, which is the case.

Now, using (a) and (b) and property (O+) we can apply Theorem 4.2 in [20] so that all orbits in \mathbf{R}_+^2 are eventually componentwise monotone and converge to an equilibrium. \square

Proof of Theorem 4. (a) Let $i \in \{1, 2\}$ such that $\phi_i(0, 0) \leq 1$. Then the only equilibria in \mathbf{R}_+^2 are E_0^* and, possibly, E_j^* with $j \neq i$. As any positive solution must tend to one of these equilibrium points we have that $\lim_{t \rightarrow \infty} N^i(t) = 0$.

(b) The first part is trivial from (a) and the rest of the result is easy to check using the linearization of H in E_0^* .

(c) If $N^1(0) = 0$ then $N^1(t) = 0$ for all t and from $\phi_2(0, 0) \leq 1$ we have that $N^2(t) \rightarrow 0$ so (1) follows. In order to prove (2), let us take $N^1(0) > 0$ so that in particular $N^1(t) > 0$ for all $t \geq 0$. The solution $(N^1(t), N^2(t))$ must converge to either E_0^* or E_1^* . As the convergence of $N^1(t)$ must be eventually monotone and $\phi_1(0, 0) > 1$, the only possibility is that the solution converges to E_1^* .

(d) The proof of the result is analogous to that of (c). \square

Lemma A.1. (a) For any initial condition $x_0 \in \overset{\circ}{\mathbf{R}}_+^2$ there exists $n_0 \in \mathbb{N}$ and a region U_k such that $H^{(n)}(x_0) \in \bar{U}_k$ for all $n \geq n_0$.
 (b) Let

$$\begin{aligned} W &= \{x \in \mathbf{R}_+^2 : H(x) \leq_K x\}, \\ W' &= \{x \in \mathbf{R}_+^2 : H(x) \geq_K x\}. \end{aligned} \quad (\text{A.1})$$

Then W and W' are forward invariant for H .
 (c) Let

$$\begin{aligned} T &= \{x \in \mathbf{R}_+^2 : H(x) < x\}, \\ T' &= \{x \in \mathbf{R}_+^2 : H(x) > x\}. \end{aligned} \quad (\text{A.2})$$

Then $H(T) \subset (T \cup W \cup W')$ and $H(T') \subset (T' \cup W \cup W')$.

Proof. (a) The result is a direct consequence of the fact that orbits are eventually componentwise monotone.

(b) The result is a direct consequence of the fact that H is competitive. Indeed, let $x \in W$ so that $H(x) \leq_K x$. Using the fact that H is competitive it follows that $H(H(x)) \leq_K H(x)$ which implies that $H(x) \in W$ and W is forward invariant as we wanted to prove. An analogous reasoning leads to the conclusion that W' is forward invariant.

(c) The result is a direct consequence of the fact that property (O+) (part (c) of Proposition 3) holds. \square

Lemma A.2. Let $x^* \in \overset{\circ}{\mathbf{R}}_+^2$ be a positive fixed point of H . Then x^* is nonhyperbolic if and only if $\nabla\phi_1(x^*)$ and $\nabla\phi_2(x^*)$ are linearly dependent.

Proof. Let $x^* = (x_1^*, x_2^*) \in \overset{\circ}{\mathbf{R}}_+^2$ be a positive fixed point of H and let

$$\begin{aligned} J := DH(x^*) &= \begin{pmatrix} \frac{\partial H_1}{\partial x_1}(x^*) & \frac{\partial H_1}{\partial x_2}(x^*) \\ \frac{\partial H_2}{\partial x_1}(x^*) & \frac{\partial H_2}{\partial x_2}(x^*) \end{pmatrix} \\ &= \begin{pmatrix} 1 + x_1^* \frac{\partial \phi_1}{\partial x_1}(x^*) & x_1^* \frac{\partial \phi_1}{\partial x_2}(x^*) \\ x_2^* \frac{\partial \phi_2}{\partial x_1}(x^*) & 1 + x_2^* \frac{\partial \phi_2}{\partial x_2}(x^*) \end{pmatrix} \end{aligned} \quad (\text{A.3})$$

be the Jacobian matrix at x^* , where we have used the fact that $\phi_i(x^*) = 1$, $i = 1, 2$. It is immediate to check that $\partial H_1 / \partial x_2 < 0$ and $\partial H_2 / \partial x_1 < 0$ in $\overset{\circ}{\mathbf{R}}_+^2$ and so $(\partial H_1 / \partial x_2)(\partial H_2 / \partial x_1) > 0$. J has complex eigenvalues if and only if the discriminant $\Delta := (\text{tr } J)^2 - 4 \det J$ is negative. Δ can be expressed as $\Delta = ((\partial H_1 / \partial x_1)(x^*) - (\partial H_2 / \partial x_2)(x^*))^2 + 4(\partial H_1 / \partial x_2)(x^*)(\partial H_2 / \partial x_1)(x^*) > 0$ and therefore J cannot have complex eigenvalues.

In the proof of Proposition 3 we have shown that $\det DH(x) > 0$ for all $x \in \bar{S}$. As $x^* \in \bar{S}$, it follows that $\det J > 0$ and together with $\text{tr } J > 0$ yields the fact that the eigenvalues

of J are positive. In this case x^* is nonhyperbolic if and only if $\lambda = 1$ is an eigenvalue of J . Now, using (A.3) we obtain

$$\begin{aligned} \det(J - I) &= x_1^* x_2^* \left(\frac{\partial \phi_1}{\partial x_1}(x^*) \frac{\partial \phi_2}{\partial x_2}(x^*) \right. \\ &\quad \left. - \frac{\partial \phi_1}{\partial x_2}(x^*) \frac{\partial \phi_2}{\partial x_1}(x^*) \right) \end{aligned} \quad (\text{A.4})$$

which, since $x_1^*, x_2^* \neq 0$, is zero if and only if $\nabla\phi_1(x^*)$ and $\nabla\phi_2(x^*)$ are linearly dependent. \square

Proof of Theorem 6. (1) The bounds for the components of the positive equilibrium or equilibria are a trivial consequence of the fact that $E_1^* = (R_{11}, 0)$ and $E_2^* = (0, R_{22})$ and of the isoclines being curves given by strictly decreasing functions of the x_1 variable.

(2) Throughout the proof we will use implicitly the fact that $\overset{\circ}{\mathbf{R}}_+^2$ is forward invariant for H and that when $\phi_i(0, 0) > 1$ for $i = 1, 2$, E^* is a repeller and so no orbit can converge to E^* . In each one of the different cases that we study below, the hyperbolicity and the local stability properties (attracting or unstable) of the semitrivial equilibria follow directly from Proposition 5 and the relative positions of the isoclines intercepts.

Case A. The existence of a unique positive equilibrium is a direct consequence of the fact that $R_{11} < R_{21}$ and $R_{12} > R_{22}$ and of the properties of the isoclines (Lemma 2). Since x_1 is increasing in $\bar{U}_2 \cup \bar{U}_3$ (see Figure 1(a)), no orbit in $\overset{\circ}{\mathbf{R}}_+^2$ can converge to E_2^* . Similarly since x_2 is increasing in the regions $\bar{U}_1 \cup \bar{U}_3$, no orbit in them can converge to E_1^* . Since E_0^* (repeller), E_1^* , E_2^* , and E_3^* are the only equilibria and all orbits converge to an equilibrium, then necessarily all orbits must converge to E_3^* . Using Lemma A.2 and the fact that the isoclines are not tangent at E_3^* we have that E_3^* is hyperbolic.

Case B. The existence of a unique positive equilibrium E_3^* is a direct consequence of the fact that $R_{11} > R_{21}$ and $R_{12} < R_{22}$ and of the properties of the isoclines (Lemma 2).

Clearly $\bar{U}_1 \subset W$ and $\bar{U}_2 \subset W'$, where W, W' are defined in (A.1). Using Lemma A.1 part (b), we have that \bar{U}_1 and \bar{U}_2 are forward invariant for H , and from the monotonicity of H in these regions it follows that orbits starting in them can not converge to E_3^* . Since all orbits must necessarily converge to an equilibrium, those starting in \bar{U}_2 (resp., \bar{U}_1) must necessarily converge to E_2^* (resp., E_1^*). In particular E_3^* is unstable. Let B_i be the basin of attraction of E_i^* , $i = 1, 2$ in $\overset{\circ}{\mathbf{R}}_+^2$ which is open since E_i^* is attracting [24]. Clearly $X := \overset{\circ}{\mathbf{R}}_+^2 \setminus \{E_3^*\}$ is an open connected set and therefore, since B_1 and B_2 are disjoint, $X \setminus (B_1 \cup B_2)$ can not be empty. So we conclude that there are points in X such that their corresponding orbits do not converge to either E_1^* or E_2^* , and so they must converge to E_3^* .

Using Lemma A.2 and the fact that the isoclines are not tangent at E_3^* we have that E_3^* is hyperbolic, and so it must necessarily be a saddle, for it attracts some points and repels

others. In order to prove that E_3^* can not attract an open set we will argue by contradiction. Let U be a neighborhood of E_3^* and let $W_{\text{loc}}^s(E_3^*)$ be its corresponding local stable manifold that, as it is well known, is one-dimensional. Let us assume that there is an open set V that is attracted to E_3^* . Then we can choose two points $x, y \in U$ such that $x <_K y$, and, using the fact that H is strongly competitive and denoting $H^{(n)} = (H_1^{(n)}, H_2^{(n)})$, it follows that, for all $n \geq 1$, $H^{(n)}(x) <_K H^{(n)}(y)$ and so the rectangle $R_n := [H_1^{(n)}(x), H_1^{(n)}(y)] \times [H_2^{(n)}(x), H_2^{(n)}(y)]$ has nonempty interior for all n . Since $H^{(n)}(x) \rightarrow E_3^*$ and $H^{(n)}(y) \rightarrow E_3^*$, using a standard comparison argument it follows that R_n converges to E_3^* and therefore for large enough n it must be $R_n \subset U$, and so $R_n \subset W_{\text{loc}}^s(E_3^*)$ which contradicts the fact of $W_{\text{loc}}^s(E_3^*)$ being one-dimensional.

Case C_{1a}. H_1 is increasing in $U_1 \cup \Gamma_2 \cup U_2$ what precludes convergence of any orbit starting in $\overset{\circ}{R}_+$ to E_2^* , and so they must all converge to E_1^* .

Case C_{1b}. Using Lemma A.2 and the fact that the isoclines are not tangent at either E_3^* or E_4^* we have that both equilibria are hyperbolic.

Clearly $\bar{U}_1 \cup \bar{U}_2 \subset W$ and $\bar{U}_3 \subset W'$ (W and W' defined in (A.1)), and so using Lemma A.1 part (b) we have that $\bar{U}_1 \cup \bar{U}_2$ and \bar{U}_3 are forward invariant for H . Since H is continuous $H(\bar{U}_1)$ must be connected and since $E_1^* \in \bar{U}_1$ is a fixed point, $H(\bar{U}_1)$ can not intersect \bar{U}_2 and so we conclude that \bar{U}_1 is forward invariant. A similar argument shows that \bar{U}_2 is forward invariant too. The monotonicity of H in U_3 shows that orbits starting in it can not converge to E_4^* and therefore they must converge to E_3^* . Similarly, the monotonicity in U_1 (resp., U_2) shows that orbits starting in U_1 (resp., U_2) can not converge to E_4^* (resp., E_2^*) and so they must converge to E_1^* (resp., E_3^*). In particular E_4^* is unstable.

Let us now show that E_3^* is attracting. Let $\varepsilon > 0$ be small enough, let $B(E_3^*, \varepsilon)$ be the open ball with center E_3^* and radius ε , and let $x \in B(E_3^*, \varepsilon)$, so that x belongs to $\bar{U}_2 \cup \bar{U}_3 \cup U_4 \cup U_5$. Let us consider in turn the three following possibilities: (i) $x \in \bar{U}_2 \cup \bar{U}_3$, (ii) $x \in U_4$, and (iii) $x \in U_5$.

In scenario (i) we have already shown that the corresponding orbit converges to E_3^* . (ii) Clearly $U_4 \subset T'$ where T' is defined in (A.2). Then Lemma A.1 part (b) implies that if $x \in U_4$ then $H^{(n)}(x)$ can not enter U_5 for any value of n . Therefore either $H^{(n)}(x)$ enters $\bar{U}_2 \cup \bar{U}_3$ for a certain n and then we already know that the orbit must converge to E_3^* or, on the contrary, $H^{(n)}(x)$ remains in U_4 for all $n \geq 0$. In this latter case x is ε -close to E_3^* and the monotonicity in this region precludes convergence of the orbit to E_2^* or to E_4^* , and so it must necessarily converge to E_3^* . (iii) In this case we can carry out a reasoning completely analogous to that of (ii) replacing U_4 by U_5 , T' by T , and E_2^* by E_1^* . In conclusion we have that E_3^* attracts the open ball $B(E_3^*, \varepsilon)$ and so it is stable.

In order to prove that there are points different from E_4^* that are attracted to it we proceed as in case B: let B_3 and B_1 be the basins of attraction of E_3^* and E_1^* , respectively, which are

disjoint nonempty open sets since E_3^* and E_1^* are attracting.

$X := \overset{\circ}{R}_+^2 \setminus \{E_4^*\}$ is an open connected set and therefore $X \setminus (B_3 \cup B_1)$ can not be empty. Therefore, there are points in X such that their corresponding orbits do not converge to either E_3^* or E_1^* , and so they must converge to E_4^* .

The proof that E_4^* is hyperbolic and it can not attract any open set is completely analogous as that carried out for E_3^* in case B.

Case C₂. Since $R_{11} < R_{21}$, $R_{12} < R_{22}$, and Γ_1 is convex there can be no positive fixed points. This case is the symmetric of C_{1a} when we interchange x_1 with x_2 , E_1^* with E_2^* , and E_3^* with E_4^* , and so the proof for this case follows from the one carried out there. \square

Conflicts of Interest

The authors declare that they have no conflicts of interest.

Acknowledgments

The authors are supported by Ministerio de Economía y Competitividad (Spain), Project MTM2014-56022-C2-1-P.

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