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A model for the evolution of parasite–host interactions based on the *Maculinea–Myrmica* system: Numerical simulations and multiple host behavior

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ABSTRACT

We present a mathematical model of parasite–host interactions inspired by the *Maculinea–Myrmica* system. Numerical simulations of the model were conducted in order to access the possibility of stable multiple host behavior in the model. Results indicate that multiple host behavior can be observed under natural conditions, although a division of the original parasite population into separate subpopulations, each adapted to one distinct host, is expected. Transitions from single to multiple host behavior are expected to occur if the relative host species abundances change or host's tolerance increases. Further model development and analysis are required to extend these results.

1. Introduction

Host–parasite interactions provide an excellent example of coevolution in nature, serving both as tests for evolutionary predictions and as sources of new insights on evolutionary dynamics. Given the time scales in which natural selection can occur, mathematical models are useful tools to investigate the dynamics of such systems.

To model host–parasite interactions a variety of approaches have been used with distinct objectives. The focus of such models are diverse: aspects of populations dynamics [1–3], evolutionary dynamics of parameters such as virulence [4], effects of geographic distribution on adaptation [5], dynamics of quantitative traits [6] and others. A full review of the recent developments in the field of evolutionary dynamics is beyond the scope of this paper and we refer to [7] as an excellent source of key references and concepts.

In this paper we present a model for parasite host interaction based on an *aspect space* approach, inspired by the seminal works of Lin and Segel [8]. The creation of the model was based in the *Maculinea* butterflies-*Myrmica* ants parasite–host system, but our model is quite general and may be applied to discuss other host–parasite interactions. In this paper we will refer to the specific system which has originated the model, leaving a general discussion of host–parasite interactions for future work.

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Also, we should remark that we have two very specifics objectives in this paper: to propose a model to the dynamics of host–parasite interactions in which is possible to discuss the emergence of multiple or single host behaviors and to explore the parameter space of the model to access the possibility of the existence of stable multiple host behavior in the simulations of the biological system that inspired the construction of the model.

2. Biological background

In this section we provide some information on the biological system that originated the model.

2.1. Life cycle

The most specialized social parasites among Lycaenidae belong to *Maculinea* genus, where all species have an obligatory relationship with ants. There are six species, currently described, although probably there are some more species in Asia which have not yet been described [9–11]. Large Blue butterflies possess unique and highly specialized life cycles [12]. During the summer females lay eggs on a specific food plant and after about three weeks, young larvae hatch from eggs and feed on seeds or flowers. One month later, at the start of the fourth larval instar, larvae drop to the ground and wait for foraging ants of the *Myrmica* genus, which take caterpillars into their nests. *Maculinea* larvae live inside *Myrmica* nests for 10–22 months [13–15] where they obtain more than 98% of their ultimate biomass [16,17]. Depending on the species, larvae pupate in May or June and remain in this stage about three weeks. After this time young butterflies emerge [18].

2.2. Host specificity in the Maculinea–Myrmica system

Early studies on *Maculinea* host ant specificity indicated that each of the five European *Maculinea* species had one and separate *Myrmica* host species, whose nests butterfly larvae survival was much higher than in other host ant species [19]. More recent researches show that the situation is more complicated and host ant specificity of *Maculinea* butterflies should be considered at a local scale [20–23]. A first well demonstrated example was found for *M. alcon* for which a host switch occurs across Europe [20]. In other studies, [24–26], the existence of multiple host populations in different *Maculinea* species, *i.e.*, a single population of *Maculinea* able to exploit more than one host at the same site, was demonstrated.

In this paper, our objective is to develop a mathematical model to discuss the evolution of the different host specificity patterns displayed by the *Maculinea* butterfly species in distinct field studies [27,24,19,28]. In that sense, it is important to alert that the focus of the model is on the emergence of *host specificity* patterns and not on the complex details of population dynamics. It is also worth noting that the whole complexity of the population dynamics of the species *can* influence the evolution of the host specificity behavior. However, for the sake of clarity and as a first approximation, we have chosen to simplify the aspects of population dynamics and focus on the dynamics of phenotypic changes in populations of *Maculinea* species and its hosts.

2.3. Chemical profiles and host specificity

In the case of *Maculinea* butterflies, similarly to other social parasites, one of the methods employed in penetration and survival in ant colonies is chemical mimicry [29,30]. The most important substances which play a role in this process are hydrocarbons [31,32].

Nash et al. [33] found evidence that the greater the match between the surface chemistry of *Maculinea alcon* and two of its host *Myrmica* species, the more easily ant colonies were exploited. The adoption time (*i.e.*, how long a *Myrmica* worker retrieves a *Maculinea* larva once it is found) is a good measure of infectivity of the parasite that combines the speed of retrieval of caterpillars and their initial integration into the ant colony. Considering populations singly, the resident primary host (sensu Thomas et al. [34]) usually retrieves more rapidly the local *Maculinea* caterpillars than other *Myrmica* species do [35]. In Nash studies, chemical similarity was a significant predictor of infectivity, explaining most of the variation in adoption time for the two *Myrmica* species tested (*M. rubra* and *M. ruginodis*) [33].

3. Modeling the dynamics of evolution

3.1. Biological hypotheses

In the process of model-making it is important to clarify the biological hypotheses assumed so that we know exactly what is included and what is not in the dynamics presented by the model. The main biological hypotheses considered in our model are as follows:

1—*In the absence of parasites, ant populations grow logistically*: this reasonable hypothesis is usually included in models for ant population dynamics [36,37], supported by data on the growth of *Myrmica rubra* [38]. Also, it is assumed that the carrying capacity for each ant species is constant during the simulations.

2—*If environmental conditions are constant, butterfly populations grow logistically*: this qualitative behavior is also exhibited by the models developed by Hochberg et al. in both stochastic [37] and deterministic versions [39]. This hypothesis is simply the natural assumption that competition for flower buds and ant nests limits the growth of the parasite population to a carrying capacity.

3—*The survival of Maculinea larvae in the ant nests is mainly dependent on two factors, the host's tolerance and the degree of similarity between the chemical profiles of the parasite larvae and of the ant's brood*: the host's tolerance is a factor that is influenced by the quality of the environment available to the ant colonies. When faced with a very favorable environment, species that normally would not be considered as hosts can raise *Maculinea* larvae with success, as indicated by laboratory experiments [40]. The inclusion of the second factor means that we attribute a higher chance of survival to individuals with a greater degree of chemical similarity with the host ants.

4—*All Maculinea sites have two well-defined regions, an "infection area" and a "non-infection area" (refuge)*: the "infection area" includes the region inside the maximum distance at which nests can be infected by butterfly larvae dropped from food plants and the "non-infection area" (or refuge) is the area beyond this maximum distance, where ant nests cannot be infected by *Maculinea* larvae. The size of those areas may vary according to the site and the ant species [36] but, for the model, the important factor will be the percentage of ant nests inside the infection area, not the actual numerical value of the area.

5—*Inside the infection area, the frequency distribution of the chemical profiles is similar to that of the whole site*: this means that if we took a sample of the chemical profiles inside or outside the infection area we would get similar distributions (*i.e.*, just by accessing the samples one could not determine if they were from the infection or non-infection area). The fact that nuptial flights of the ant colonies can reach up to some hundred meters [36] indicates that this may be a good hypothesis, since we have a genetic flow at a larger scale than the typical infection range (1–10 m).

6—*Ant species and food plants are present in a sufficient number to ensure that there is no risk of butterfly extinction caused by the lack of those resources*: with this we assume that there is no serious shortage of ant nests or food plants during the simulations.

Although there will be situations where these hypotheses do not hold, they are useful simplifications to highlight the factor we want to analyze with the model, that is, the emergence of host specificity patterns. With the simplifying hypotheses we can focus on creating a model for the evolution of the phenotypic characteristics that regulate the dynamics of the *Maculinea–Myrmica* system.

3.2. The phase space

As presented in Section 2, there is evidence that the similarity between the chemical profiles of *Maculinea* larvae and ant species regulate the probability of adoption and survival of the parasite in the colonies of ants. In that sense, we can think of the individual butterflies and ants as having a phenotype that regulates the exploitation probability level achieved by the parasites. The closer the phenotypes, the stronger the exploitation.

It is highly probable that those phenotypes are genetically regulated, and one could think of modeling the gene frequencies in the process of evolution, in which genes for phenotypes "closer" to the host's phenotypes would give a reproductive advantage to the parasitic individuals. Instead, we will use a different approach, modeling the *frequencies of the phenotypes* within populations.

This is justified by the fact that the genetical mechanisms that control morphogenesis and, in turn, the production of hydrocarbon profiles which regulate relations between species, are unknown and probably too complex to be described by the frequencies of a small number of genes. On the other hand, if we choose to model the frequencies of the *phenotypical* characters in the population, we can avoid the complications of relating genes to individual fitness, by looking directly at the phenotype of the individual. This kind of approach has also recently been used to describe the evolution of phenotypes in an influenza virus model [41].

When reasoning for the existence of a coevolutionary arms race between ant species and *Maculinea alcon*, Nash et al. [33] analyzed the hydrocarbon profiles of the individuals, representing populations of *Maculinea alcon* and its hosts as distributions of points in a two-dimensional space. In this representation, each different phenotype is associated with a point in the Cartesian plane, and a population is represented as a cloud of points in the plane. Their results show a correlation between the distance in this phase space with the adoption time by the host species. Barbero et al. [42] presented a similar analysis with respect to sound production in the *Maculinea rebeli—Myrmica schencki* system. Populations of hosts and parasites were represented as clouds of points in two and three dimensional spaces. In this case phenotypic characteristics were the dominant frequency, the pulse lengths and the pulse repetition frequencies of sounds produced by hosts and parasites.

On the basis of those experimental and field observations, we model the populations (ants and butterflies) as distributed in a *phase space* of phenotypes (or *aspect space* in the now classical terminology introduced by Levin and Segel [8]), where the distance between parasite and host is a measure of the parasite exploitation success. In Nash's et al. study [33] the space is simply a subset of R^2. In our model the phase space will be an interval $\Omega = [0,\ L]$ in the real line, each point representing a distinct phenotype. The extension to a phase space with more dimensions is straightforward and should yield the same qualitative results. We reserve for future work its full analysis. Having defined the phase space Ω we proceed to model the *evolution* of the population in this phase space.

3.3. Phenotypic variation and natural selection

To model the dynamics of evolution, we have to make mathematical formulations for two of its basic mechanisms: *phenotypic variation* (existence of distinct phenotypes) in the phase space and *natural selection* (differential reproduction rates) for the individuals with the best phenotypes.

Before modeling the particular case of the *Maculinea–Myrmica* system, we will deduce a general equation for the evolution of the phenotypical distribution of a population on a phase space Ω under the hypothesis that there is a function $f : \Omega \to [0, 1]$, called *fitness function*, that associates each phenotype in phase space to a value in $[0, 1]$. So, if $x \in \Omega$, $f(x)$ is a measure of the reproductive capacity of the individual, for instance, if $f(x) = 1$ phenotype x has the maximum reproductive rate for the species. For a more general model we could think of a function $f : \Omega \to \mathbb{R}^+$ in a sense that there would be no pre-established maximum reproductive rate for the species.

To obtain equations for the evolutionary dynamics we will work with a discretization of Ω. Suppose that Ω is divided in n equal subintervals and $U_i(t) = U_i$, $i = 1, \ldots, n$, is the number of individuals of the population in the interval $[(i-1)\,\triangle x,\ i\triangle x]$, where $\triangle x = L/n$. For modeling purposes we are going to use a continuous domain for the time variable t. So U_i is the number of adult butterflies at time t that have a phenotype in the interval $[(i-1)\,\triangle x,\ i\triangle x]$.

3.3.1. Phenotypic variation

To generate the mechanisms to simulate phenotypic variation in the population, we start by defining r as the reproductive rate for the species. The rate of change of the number of individuals in class i at time instant t could be modeled as rU_i. To create the effect of phenotypic variation (due to diverse factors such as random drift, mutation and recombination) in the population we will suppose that a fraction $v \in (0, 1)$ of the individuals generated by the reproduction of the individuals of class i does not belong to the same class, but to the adjacent classes $i-1$ and $i+1$. This is a way to formulate the common hypothesis that individuals produce offspring with phenotypes similar to their own and v is now a parameter that measures the rate of change of phenotypes in the population through reproduction.

For now, we would have the following equations for the population dynamics:

$$\frac{dU_i}{dt} = \frac{vrU_{i-1}}{2} + (1-v)\,rU_i + \frac{vrU_{i+1}}{2}. \tag{1}$$

3.3.2. Natural selection

To generate a mechanism of natural selection, we must assign higher reproductive rates to the individuals with the best phenotypes. Using the hypothesis that there is a fitness function $f : \Omega \to [0, 1]$ measuring the reproductive capacity of the individual with phenotype x, we define $f_i = f(\bar{x}_i)$ where $\bar{x}_i$ is the midpoint of the interval $[(i-1)\,\triangle x,\ i\triangle x]$. Then f_i is a measure of the reproductive capacity of the individuals in class i. This suggests a modification of Eq. (1) in order to include this differential rate of reproduction.

The reproduction rate of individuals in class i would then be given by rf_i and the equation for the population dynamics becomes:

$$\frac{dU_i}{dt} = \frac{vrf_{i-1}U_{i-1}}{2} + (1-v)\,rf_iU_i + \frac{vrf_{i+1}U_{i+1}}{2}. \tag{2}$$

Up to now, the model has two components of natural selection, that is, higher reproduction rates to the "best" phenotypes and phenotypic variation. Another important feature of natural selection is that limited natural resources impose serious restrictions to population growth. Eqs. (1) and (2) represent a situation where growth is unlimited. To add a limiting mechanism to the model we suggest including a Verhulst-type term in the equation in the form:

$$T = -\frac{rU_i \sum_{j=1}^{n} U_j}{K} \tag{3}$$

where the parameter K is the environment carrying capacity of the species. Including this in the model we obtain:

$$\frac{dU_i}{dt} = \frac{vr}{2}\left(f_{i-1}U_{i-1} - 2f_iU_i + f_{i+1}\,U_{i+1}\right) + rU_i\left(f_i - \frac{\sum_{j=1}^{n} U_j}{K}\right). \tag{4}$$

If we now abandon the discretization of the phenotype domain in classes, defining $u(x, t)$ as the *density* of individuals with phenotype x at time t, Eq. (4) suggests the following dynamics for u:

$$\frac{\partial u}{\partial t} = \overline{v}\,\overline{r}\,\frac{\partial^2 [f(x)u]}{\partial x^2} + \overline{r}u\left(f(x) - \int_\Omega u\,dx \Big/ K\right). \tag{5}$$

The model obtained in (5) is a reaction–diffusion-type equation for the evolution of $u(x, t)$, with a non-constant diffusion coefficient $D(x) = \overline{v}\,\overline{r}f(x)$. From now on we drop the notation $\overline{v}\,\overline{r}$ using v and r instead for a cleaner presentation.

The dependence of $D(x)$ on r is expected, since the rate of reproduction defines the rate of generation of new phenotypes and therefore the propagation speed of the population along the phase space. Also, dependence on the function $f(x)$ has the clear meaning that in regions where the individuals reproduce more, diffusion will be faster, since "movement" in phase space, in this case, is related to generation of new phenotypes and, hence, reproduction. Finally $\sqrt{v}$ is a measure of change in the population in the phase space in the time scale of reproduction. In other words, it measures how much a population could change in the phase space through diffusion in a time scale compatible with observable changes in population numbers. The second term in the equation is a Verhulst-type dynamics with two modifications. First, the maximum reproductive rate is dependent on the position x in phase space. Second, the usual term in a Verhulst-type dynamics is N/K, N being the total number of individuals competing for the limited resources, in this case, since the population is distributed along the phase space, N is given by $N = \int_\Omega u\, dx$.

3.4. Fitness functions for the Maculinea–Myrmica system

Eq. (5) provides the dynamics of a population under the effects of natural selection and those that generate phenotypic variation (mutation, for instance) as long as the *fitness function* $f : \Omega \to [0, 1]$ that regulates the reproduction of the species is known. In the case of the *Maculinea–Myrmica* system the fitness of an individual butterfly is dependent on the *distribution* of the phenotypes of the ant hosts. In this sense, the fitness functions in this case will be represented by *functionals* since they are dependent not only on the phenotype x of the individual, but also on a function that represents the distribution of the phenotypes. This is in agreement with the biological intuition, since the fitness of an individual with phenotype x is dependent on the *distribution* of host and parasite phenotypes. As an example we can think of a parasite with phenotype x, if the whole population of hosts has a phenotype that is "far" from x, the fitness of the parasite should be very low, while if the host population is "near" x the fitness should be high.

We will start creating fitness functions for a system with just one parasite species and one host species.

3.4.1. Single host model

Defining $P \equiv P(x, t)$ as the parasite density with phenotype x at time t and $H \equiv H(x, t)$ as the host density with phenotype x, we must define a fitness functional $g(H, x)$ for the dynamics of P and a functional $f(H, P, x)$ for the dynamics of H.

The best possible phenotype for the parasite is the one which is "closest" to the phenotypes of the host species. In this sense we include a parameter ε that defines the maximum vicinity in which a phenotype can be considered to be subject to exploitation by a parasite. If a parasite has a phenotype x, $(x - \varepsilon, x + \varepsilon)$ is the vicinity in the phase space that contains all host individuals that can be exploited by this particular parasite.

The best possible situation for a parasite occurs when all the population of hosts is *inside* the vicinity $(x - \varepsilon, x + \varepsilon)$. The worst possibility is when no host is found within the boundaries of the vicinity. This suggests that we could model the fitness of a parasite by the fraction of the population of hosts that is inside the vicinity $(x - \varepsilon, x + \varepsilon)$:

$$g\,(H, x) = \frac{\int_{x-\varepsilon}^{x+\varepsilon} H\,(x, t)\, dx}{\int_\Omega H\,(x, t)\, dx}. \tag{6}$$

Now to model the fitness of the host species we must take into account two important factors:

1—Recognition *among* individuals of the host species is an important factor for reproduction since in many species a new queen does not always start a new colony on her own, but enters one that already exists, sometimes being rejected and sometimes being accepted. In this sense, it favors the host to have a phenotype similar to the majority of the population of hosts. Of course, feeding larvae is another activity in which recognition among individuals plays an important role.

2—Not all colonies of hosts lie within the infection range (*i.e.*, some may be far from a butterfly host plant, thus creating a *refuge* for the host species) thus not all are subjected to the possibility of exploitation by parasites. This is, clearly, a very important biological information since it is related to the selective pressure that the parasite population can exert over the host population. Since this fraction of the colonies within the infection range will vary, we include a parameter γ that is related to the fraction of the population that *is* exposed to the parasite (the fraction outside the refuge).

As in the fitness function for the parasite, factor 1 could be modeled as the fraction of hosts in the vicinity $(x - \varepsilon, x + \varepsilon)$. If all the population of hosts are inside the vicinity, the whole of the population recognizes the individual as "similar", thus maximizing its fitness. On the other hand, being similar to the parasite is not advantageous to the host, so a measure of fitness with relation to the parasites could be 1 minus the fraction of parasites in the vicinity $(x - \varepsilon, x + \varepsilon)$. But we must include also the selection pressure on the fitness function, even if the host population is similar to the whole of the parasite species, if the selective pressure is very low (in other words, the chance that the colony is exposed to the parasite is very low), that should not have a great influence on the fitness of the individual. With those aspects in mind, we suggest the following as fitness function for the host species:

$$f\,(H, P, x) = \frac{\int_{x-\varepsilon}^{x+\varepsilon} H\,(x, t)\, dx}{\int_\Omega H\,(x, t)\, dx} \left(1 - \gamma \frac{\int_{x-\varepsilon}^{x+\varepsilon} P\,(x, t)\, dx}{\int_\Omega P\,(x, t)\, dx}\right). \tag{7}$$

The model for one host species and one parasite is given by the following system of partial differential equations:

$$\begin{aligned}\frac{\partial H}{\partial t} &= v_H r_H \frac{\partial^2 \left[f\left(H,P,x\right)H\right]}{\partial x^2} + r_H H\left(f\left(H,P,x\right) - \int_\Omega H\,dx \Big/ K_H\right)\\ \frac{\partial P}{\partial t} &= v_P r_P \frac{\partial^2 \left[g(H,x)P\right]}{\partial x^2} + r_P P\left(g(H,x) - \int_\Omega P\,dx \Big/ K_P\right)\end{aligned} \tag{8}$$

where v_H is the rate of phenotypic change of the host species, r_H its reproduction rate and K_H the carrying capacity. By analogy v_P, r_P and K_P are the parameters relative to the parasite species.

3.4.2. Multiple host model

The most significant modification with respect to the model of one host species is that the host species are present inside the infection area with different frequencies. There are two factors that contribute to the frequency observed inside the infection area: the carrying capacity of the environment with respect to each host species and the percentage of nests of the host nests that are inside the infection area. For example: even if the carrying capacity of the site with respect to one host species A is small when compared to host species B, if most of the A nests are inside the infection area (meaning that the parasite exerts a high selection pressure on species A) then species A could be more abundant to the parasite than species B. A measure of the abundance of the host species A to the parasite is given by $\gamma_A K_A$, the percentage of nests inside the infection area times the carrying capacity of the site with respect to species A.

The dynamics for each host species is directly generalized from the system of Eqs. (8), the main modification concerning the parasite's fitness function. If $H_i(x,t),\ i = 1,\ldots,n$ is the distribution of each host species, then the fraction of host individuals within the vicinity of phenotype x is given by:

$$g\left(H_1,\ldots,H_n,x\right) = \frac{\sum_{i=1}^{n} \gamma_i \int_{x-\varepsilon}^{x+\varepsilon} H_i\left(x,t\right)dx}{\sum_{i=1}^{n} \gamma_i \int_\Omega H_i\left(x,t\right)dx}. \tag{9}$$

Again, maximum fitness (with numerical value of 1) for the parasite is obtained when all host populations are within the vicinity $(x-\varepsilon, x+\varepsilon)$ and zero fitness is obtained when no host is found in it.

Finally we obtain the system of equations for n hosts:

$$\begin{aligned}\frac{\partial H_i}{\partial t} &= v_i r_i \frac{\partial^2 \left[f_i\left(H_i,P,x\right)H_i\right]}{\partial x^2} + r_i H_i\left(f_i\left(H_i,P,x\right) - \int_\Omega H_i\,dx \Big/ K_i\right), \quad i = 1,\ldots,n\\ \frac{\partial P}{\partial t} &= v_P r_P \frac{\partial^2 \left[g(H_1,\ldots,H_n,x)P\right]}{\partial x^2} + r_P P\left(g(H_1,\ldots,H_n,x) - \int_\Omega P\,dx \Big/ K_P\right)\end{aligned} \tag{10}$$

with the obvious meanings for the parameters.

3.4.3. Adimensional parameters

For the time variable we adopt $t^* = r_P t$, the time scale of the parasite reproduction dynamics, $x^* = x/\sqrt{v_P}$ is the adimensional variable for the phase space, $h_i(x,t) = \sqrt{v_P}H_i/K_i,\ i = 1,\ldots,n$ the densities of the host species and $p(x,t) = \sqrt{v_P}P/K_P$ the parasite density. Dropping the stars, the adimensional system of equation is:

$$\begin{aligned}\frac{\partial h_i}{\partial t} &= \theta_i\lambda_i \frac{\partial^2 \left[f_i\left(h_i,p,x\right)h_i\right]}{\partial x^2} + \lambda_i h_i\left(f_i\left(h_i,p,x\right) - \int_\Omega h_i\,dx\right), \quad i = 1,\ldots,n\\ \frac{\partial p}{\partial t} &= \frac{\partial^2 \left[g(h_1,\ldots,h_n,x)p\right]}{\partial x^2} + p\left(g(h_1,\ldots,h_n,x) - \int_\Omega p\,dx\right)\end{aligned} \tag{11}$$

where $\theta_i = v_i/v_P$, $\lambda_i = r_i/r_P$ and $\overline{\Omega} = [0, L/\sqrt{v_P}]$. θ_i is a measure of the ratio of the speed of change in phase space between the host species i and the parasite, $\theta_i > 1$ means that the host population i changes faster than the parasite. λ_i is a measure of how fast the host species i reproduces, $\lambda_i > 1$ means that the host species reproduces faster than the parasite.

The fitness functions can be written in terms of the adimensional variables, yielding:

$$\begin{aligned}f_i(h_i,p,x) &= \frac{\int_{x-\delta}^{x+\delta} h_i\left(x,t\right)dx}{\int_{\overline{\Omega}} h_i\left(x,t\right)dx}\left(1 - \gamma_i \frac{\int_{x-\delta}^{x+\delta} p\left(x,t\right)dx}{\int_{\overline{\Omega}} p\left(x,t\right)dx}\right)\\ g\left(h_1,\ldots,h_n,x\right) &= \frac{\sum_{i=1}^{n} \gamma_i\sigma_i \int_{x-\delta}^{x+\delta} h_i\left(x,t\right)dx}{\sum_{i=1}^{n} \gamma_i\sigma_i \int_{\overline{\Omega}} h_i\left(x,t\right)dx}\end{aligned} \tag{12}$$

Table 1
Summary of model parameters and their biological meaning.

Symbol	Meaning
$H_i(x, t)$	Density of hosts of species i in the phenotypic position x at time instant t
$P(x, t)$	Density of parasites the phenotypic position x at time instant t
v_i	Coefficient of generation of distinct phenotypes by host species i through reproduction
v_P	Coefficient of generation of distinct phenotypes by parasite i through reproduction
r_i	Maximum reproduction rate for host species i
r_P	Maximum reproduction rate for parasite species
K_i	Carrying capacity of the environment with respect to host species i
K_P	Carrying capacity of the environment with respect to the parasite species (related to the abundance of food plants)
ε	Maximum distance in aspect space between "similar" phenotypes
Dimensionless symbols	
$h_i(x, t)$	Density of hosts of species i in the phenotypic position x at time instant t
$p(x, t)$	Density of parasites the phenotypic position x at time instant t
θ_i	Ratio of the coefficient of generation of distinct phenotypes by host species i and the parasite's coefficient
λ_i	Ratio of Maximum reproduction rate for host species i and the maximum reproductive rate of the parasite species
γ_i	Selection pressure over host species i (percentage of study area occupied by food plants)
$\sigma_i = K_i/K_1$	Relative abundance of host species i with respect to host species 1
$f_i(h_i, p, x)$	Fitness function for host species i
$g\,(h_1, \ldots, h_n, x)$	Fitness function for the parasite species
$\delta = \varepsilon/\sqrt{v_P}$	Ratio between a distance of significant change in the phenotype space and the distance traveled through the generation of new phenotypes in the time scale of parasite reproduction

where $\delta = \varepsilon/\sqrt{v_P}$ and $\sigma_i = K_i\ /\ K_1$. From now on we suppose that the host species are ordered from the most abundant one H_1 to the less abundant H_2 so each σ_i is in the interval $[0, 1]$.

To have a clear biological interpretation of parameter δ, we will begin by exposing the meaning of parameters ε and $\sqrt{v_P}$. Parameter ε is the maximum distance in phase space in which a host individual can be exploited by a parasite, so if the distance between the parasite and the host is greater than ε, that individual cannot be exploited by the parasite. In this sense, ε is a measure of significant *change* in phase space. In reaction–diffusion phenomena, the speed of propagation of the population can be approximated by $2\sqrt{UD}$ where U is the reaction rate and D is the diffusion coefficient. In the present model, the diffusion coefficient is given by $r_P v_P$ while the "rate of reaction" is the population reproduction rate r_P which leads to the speed: $V = 2r_p\sqrt{v_P}$. If we now multiply this speed by the time scale of the reproduction $1/\,r_p$ we obtain the distance traveled by the population in the reproduction time scale: $2\sqrt{v_P}$. Now $\delta = \varepsilon/\sqrt{v_P}$ is the ratio between a distance of significant change in the phenotype space and the distance traveled by the population in the time scale of parasite reproduction. With this $\delta \ll 1$ means that the parasite population can change significantly (in phenotypic space) in the time scale of reproduction (of course if $\theta_i \cong 1$ so do the host populations), while $\delta \gg 1$ means that phenotypic changes in the population occur slowly.

In Table 1 we present a summary with all the parameters and their biological meaning.

3.4.4. Boundary conditions

The phase space $\Omega = [0,\ L]$ is the space of phenotypic characteristics and, as such, it is an abstraction useful to model and understand some aspects of the evolution of the *Maculinea–Myrmica* system. Different types of boundary conditions could be assigned, for instance, the homogeneous Dirichlet boundary condition $u(0, t) = u(L, t) = 0$ would represent a situation where there are lethal phenotypes at the boundaries and any individual born with a phenotype outside $\Omega = [0,\ L]$ would die before reproducing. Zero flux conditions at the boundaries would mean that no further phenotypic variation is allowed beyond the boundaries of the domain. Although those are interesting conditions and raise some other interesting biological questions, we will focus our simulations on situations where the boundaries do not affect the qualitative results, in other words, ideally we would be simulating the phase space $\Omega^* = (-\infty, +\infty)$.

4. Simulations

4.1. Numerical methods, presentation format of the simulation results and initial conditions

To solve the system we employed the Crank–Nicholson finite difference scheme [43], with approximation for the functionals f and g as $f^{n+1} \cong f^n$ and $g^{n+1} \cong g^n$. Two more routines were implemented: a simple forward-scheme and a Runge–Kutta type scheme to check the results. All methods provided very similar results.

The solutions for the system of differential equations are $n + 1$ functions of two variables: $h_i(x, t),\ i = 1, \ldots, n$ and $p(x, t)$. To optimally present the results a movie resource would be necessary. Thus to create clear and concise images of the results we will restrict the figures to bidimensional graphics. In order to do that, we observe that $h_i(x, t)$, in most simulations

are close to bell-shaped distributions in the variable x, moving in phase space as t changes. Similarly $p(x, t)$ has also a bell-shaped distribution in x in most simulations, sometimes splitting into two or more bell-shaped curves with distinct averages.

Given the above considerations, we will display only the trajectory of the *average phenotypes* in each of the curves $h_i(x, t)$, $p(x, t)$ as long as those distributions do not split in two. In the rare cases when such splitting occurs we will plot the split of the average phenotype into two other average phenotypes. To obtain the average phenotype of $h_i(x, t)$ and $p(x, t)$, we use the formula:

$$\begin{aligned} \overline{h_i}(t) &= \frac{\int_\Omega x h_i\,(x, t)\,dx}{\int_\Omega h_i\,(x, t)\,dx} \\ \overline{p}(t) &= \frac{\int_\Omega x p\,(x, t)\,dx}{\int_\Omega p\,(x, t)\,dx}. \end{aligned} \tag{13}$$

Results of the simulations will be presented as graphs of $\overline{h_i}(t)$ and $\overline{p}(t)$. Unless stated otherwise it will be implicit that $h_i\,(x, t)$ and $p\,(x, t)$ are in fact bell-shaped distributions around the averages $\overline{h_i}(t)$ and $\overline{p}(t)$. From now on we also drop the bars on $\overline{h_i}(t)$ and $\overline{p}(t)$ for a cleaner look.

To construct the initial host and parasite distributions, we first used a basic distribution

$$S(x) = (1 + \xi_i)e^{-((x-m)/v)^2} \tag{14}$$

where m is the average of the initial distribution, v the initial variance and ξ_i is a random variable uniformly distributed in $[0,\ 1/10]$. The index i for the random variable is associated with each point of the discretization grid used in the numerical approximations for the solution. For each host and parasite species a basic distribution $S(x)$ was constructed with its initial variances and averages, each distribution was them normalized to produce coherent initial population numbers:

$$Q(x) = 0.4\,S(x) \Big/ \int S(x)dx. \tag{15}$$

The qualitative results of the simulations were, for the most part, independent of the initial conditions. The cases in which the final qualitative behavior depended on the initial conditions are mentioned explicitly.

4.2. Simulation results

4.2.1. Results for a single host species

For the scenarios with a single host species there are two qualitative behaviors that can be displayed: an arms race between parasite and host or a stationary distribution of phenotypes, meaning that the coevolution of phenotypes comes to a halt. The arms race behavior is observed in two different dynamics called by us: (i) continuous and (ii) complex. In the continuous arms race, both species evolve in a fixed direction, while in the complex regimen the direction can change and the population can go through cycles with regular periods before evolving to a different cycle. Fig. 1 presents the qualitative behaviors observed in the simulations of the model.

As we conducted an exploration of the parameter space $(\delta, \theta, \gamma, \lambda)$, the stationary distributions presented three important types of results, which we shall define as two peaks, flat and one peak. Fig. 2 presents examples of such stationary distributions.

We should note that the two peaked stationary distributions show a sign of maladaptation by the parasite. In fact it can be shown [44] that this is related with the magnitude of the phenotypic variation coefficient and the scale of change of the fitness function. We shall not enter the details of the result in this paper, but we mention that this maladaptation is analogous to the ones presented in models with a high mutation rate.

In Fig. 3 we present the qualitative results obtained from 630 simulations.

Parameter γ: As expected, low selection pressures ($\gamma < 0.2$) tend to lead to a stationary behavior. Of course this is coherent with the theoretical expectations about coevolution of host and parasite, because under very weak selection pressures (on phenotypic similarity with the parasite) all phenotypes have similar reproduction rates, and the effect of natural selection cannot change significantly the distribution of phenotypes in the population. We must remark that in the model individuals very different from the average phenotype are penalized, if this factor is removed, even very low selection pressures will lead to changes in the phenotypic distribution of the host population. A high selection pressure alone does not lead to a consistent arms race regimen in the model, as one could expect. In part this is because hosts, in this case, are penalized if they differ too much from their own population, while parasites do not face this difficulty (here we must stress that we are focusing the model on the larvae phenotypes for being adopted by the hosts, while certainly it is important for the adult butterfly to be similar to its own species population). As a result, we could say that hosts might be in a situation where it is difficult for them to evolve countermeasures against the parasite (in this case, to evolve phenotypes very different from the parasite) because this also imposes a penalty on the host's own reproduction rate. Note that this reasoning is also valid for the evolution of a behavior that makes individuals less tolerant to slightly different phenotypes, since they would also tend to be less tolerant also with individuals of their own species. Of course these considerations are valid *if hosts are*

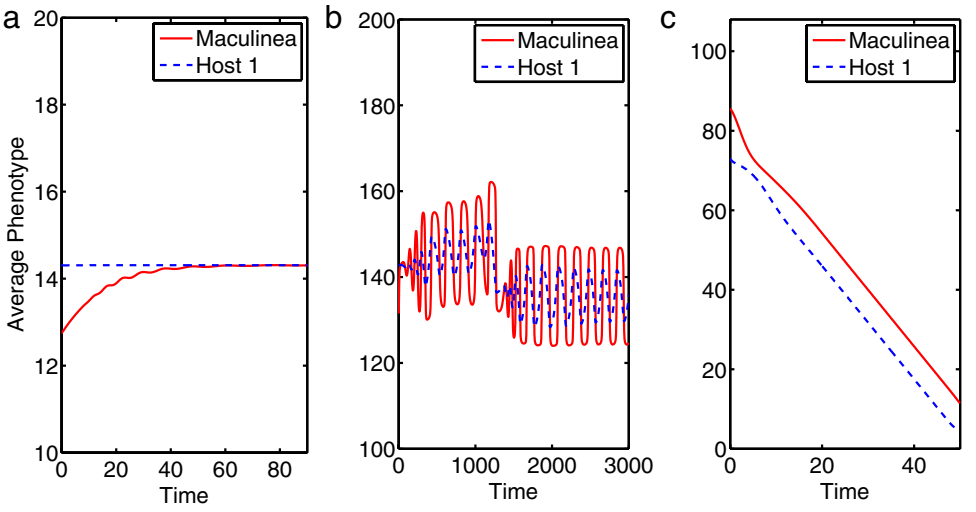

Fig. 1. Average phenotypes $h(t)$ (host) and $p(t)$ (parasite). (a) Stationary distribution of phenotypes. ($\theta = 0.24, \lambda = 0.33, \gamma = 0.89, \delta = 0.27$) (b) Complex arms race between host and parasite. ($\theta = 0.27, \lambda = 0.10, \gamma = 0.97, \delta = 8.74$) (c) Arms race with both species changing their average phenotypes in the same direction. ($\theta = 0.36, \lambda = 10.00, \gamma = 0.40, \delta = 5.40$).

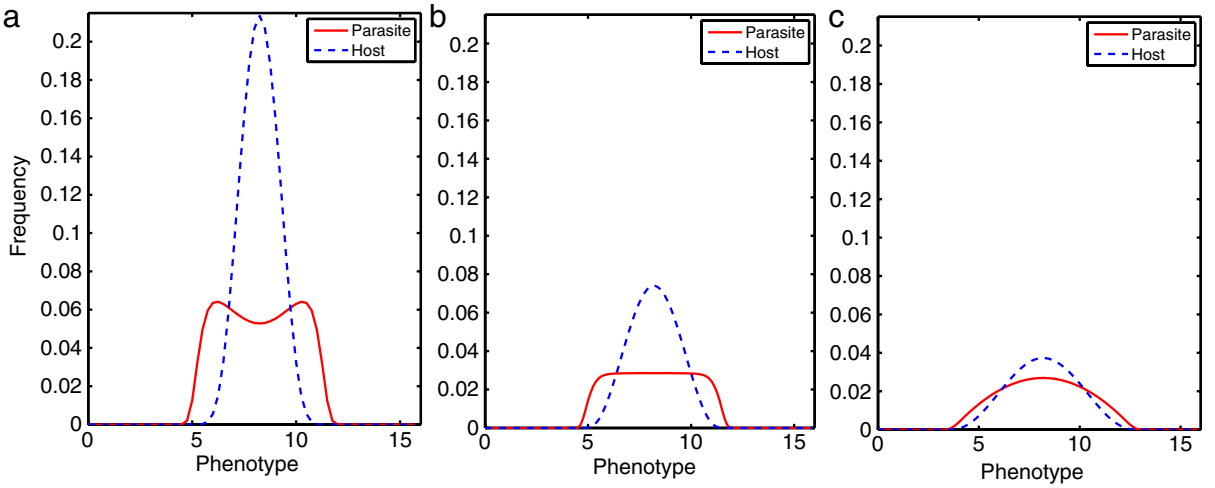

Fig. 2. Examples of stationary distributions. (a) Two peaks: $\theta = 0.125, \lambda = 1, \gamma = 0.60, \delta = 1$. (b) Flat: $\theta = 0.25, \lambda = 1, \gamma = 0.60, \delta = 0.5$. (c) One peak: $\theta = 0.5, \lambda = 1, \gamma = 0.60, \delta = 0.5$.

really penalized when they have a very distinct phenotype from the average (with the usual assumption that the population distribution is a bell-shaped curve). Self-recruitment of new queens might play a relevant role in this dynamics but it would be necessary to describe also the *spatial distribution* of colonies, adding further complexities to the model.

Parameter θ: With moderate to strong selection pressure ($0.4 < \gamma < 0.8$), high values of $\theta (>10)$ will lead to an arms race regimen. This means that the host's rate of change in the phase space would have to be faster than the parasite's in order for the host to create a permanent arms race with the parasite. Again, this is related to the hypothesis that the host might be in conflict with its own population when changing phenotypes. Values of θ near unity and below lead to a stationary regimen, where the parasite exploits the host without any evolution of countermeasures (in terms of phenotypes). Of course this is also in agreement with what is expected from the theoretical point of view, if a parasite can change faster than the host, it will be hard for the host population to escape exploitation by evolving its phenotype distribution in phase space.

Parameter λ: Just as for parameter θ moderate to strong selection pressure in combination with high values of $\lambda (>5)$ can lead to a permanent arms race regimen. As observed in the modeling section, the diffusion coefficient is dependent on the reproduction rates of the populations. $\lambda > 1$ means that hosts can reproduce faster than the parasites (comparing the best possible phenotypes). So a consequence of $\lambda > 1$ is that the host population can evolve faster than the parasite population, creating the permanent regimen of arms race. When $\lambda \leq 1$ the parasite population evolves faster than hosts and the system displays a stationary behavior.

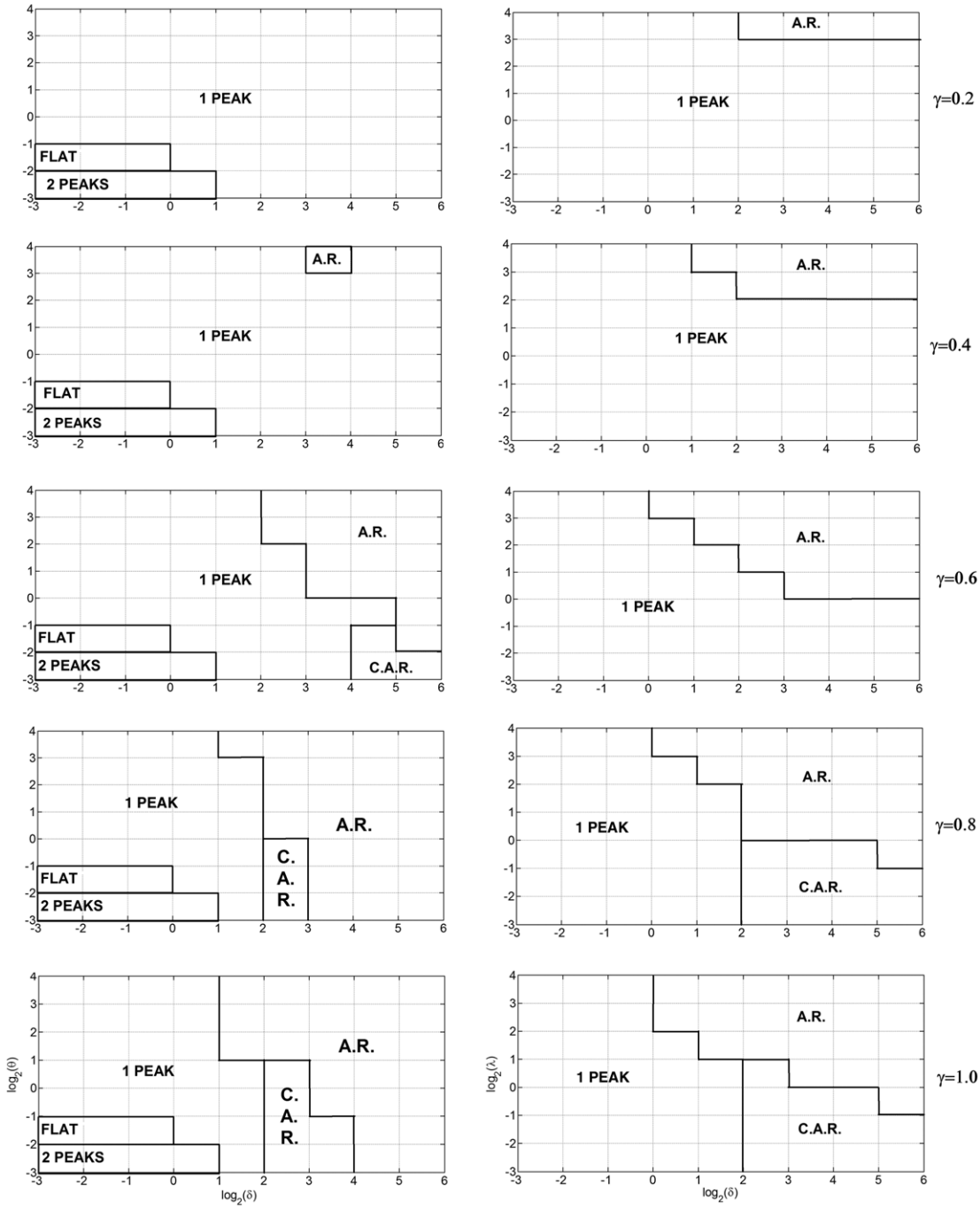

Fig. 3. Qualitative results from simulations. Each rectangle represents the result of the simulation with the parameters in its lower left corner. 2 PEAKS: stationary distribution as in Fig. 2-(a). FLAT: stationary distribution as in Fig. 2-(b). 1 PEAK: stationary distribution as in Fig. 3-(c). A.R: arms race regimen as in Fig. 1-(c). C. A. R: complex arms race regimen as in Fig. 1-(b).

Parameter δ: Again, a moderate to strong selection pressure is necessary for the system to display a permanent arms race behavior. When $\delta \gg 1$ we are simulating a situation where populations change very slowly in the phase space. In opposition to what we could intuitively expect, this condition leads to a permanent arms race behavior. Also, this was the only scenario in which the complex arms race, presented in Fig. 1(b), occurred. The opposite situation, $\delta \ll 1$, when populations change very quickly in phase space, tends to lead to stable stationary distributions.

Since we have four parameters in the model, exploring the possible outcomes of the simulations becomes a challenging task. It is not feasible to explore in detail the whole space of possible combinations. As complement to the simulations

Table 2
Taking a random sample of the parameter space (as described in this section) in 27% of the simulations we observed an arms race behavior. All other simulations displayed a convergent stationary behavior.

Qualitative behavior	Frequency (%)
Arms race	27
Stationary	73

presented in Fig. 3, we also took *random samples* from the parameter space and analyze the frequency of the observed behaviors.

Since each of the parameters has a clear biological meaning, we should not (in the absence of precise information on the biological system) assign a greater probability to particular biological situations when taking a random sample from the parameter space. For example, parameter λ defines the rate of reproduction of the host in relation to the parasite, when $\lambda > 1$ the host reproduces faster than the parasite and when $\lambda < 1$ the parasite reproduces faster. Those two are very clear and distinct biological situations and we do not want to assign a greater probability to any of them. So our procedure will be to create a set of possible values for the parameter λ: $I_\lambda = [1/10,\ 1] \cup [1,\ 10]$, and assign a probability of 0.5 to $\lambda \in I_1 = [1/10,\ 1]$, and a probability of 0.5 to $\lambda \in I_2 = [1,\ 10]$. Once defined which interval λ is in, I_1 or I_2, we sample λ from a uniform distribution within the interval. We apply the same reasoning for parameters δ and θ, obtaining: $\theta \in I_\theta = [1/10, 1] \cup [1, 10]$ and $\delta \in I_\delta = [1/10, 1] \cup [1, 10]$.

Parameter γ is the selection pressure, the fraction of the population that is inside the infection area. To create the random sample of the parameter space, γ is sampled in the interval $I_\gamma = [0, 1]$ with a uniform distribution. While initial conditions may play a role in some aspects of the dynamics of the system, the qualitative behavior is not usually affected by the initial distributions. Both parasite and host initial distributions are Gaussian curves to which some noise is added.

In Table 2 we present the percentage of each qualitative behavior in 100 simulations with parameters sampled as defined above.

4.2.2. Results for two host species

One of the main objectives for the creation of the mathematical model for the evolution of phenotypic characters in the *Maculinea–Myrmica* system was to afford the possibility to simulate the evolution of multiple/single host behavior over long time ranges.

To explore the possible outcomes in terms of host specificity behavior, we ran simulations both systematically exploring the parameter space and taking random samples of it. We begin by presenting the simulations from the random samples of the parameter space.

The simulations with two host species clearly increase the number of parameters. In this case, instead of having only one parameter λ we have λ_1, representing the rate of reproduction of host 1 and λ_2 the rate of reproduction of host 2. By analogy, we also have parameters γ_1, γ_2 (selection pressures), θ_1, θ_2 (host rate of change in phase space), σ_1, σ_2 (abundance of hosts in population site). In our simulations, the parameter δ is unaltered by the number of host species (although one could think of a situation where the parameter δ is different for each host species).

As in the simulations for one host species we sampled the parameter space following exactly the same rules defined in Section 4.2.1. The parameters σ_i, as defined in Section 3.4.3 are given by K_i/K_1 where K_i is the carrying capacity of the population site for species i (*i.e.*, a measure of the abundance of species i) and host 1 is the most abundant species at the site of study, not necessarily in the infection area. As a consequence, each σ_i, is in the interval $(0, 1]$, for our simulations $\sigma_1 = 1$ and σ_2 has a uniform distribution in $(0, 1]$.

The same qualitative behaviors observed in the one host simulations and their relation with the parameters are also present in the simulations for two hosts. The difference of the dynamics is that the parasite can now exhibit multiple host behavior, referred to as MHB for the rest of this paper. Single host behavior shall be referred to as SHB.

MHB was present in the simulations both as a *transient* and as a *stable* behavior. *Transient* MHB occurs when the parasite population, while evolving, goes through a stage where it exploits more than one host simultaneously, but, as time passes, it becomes more and more specialized in just one of the host species. *Stable* MHB is the behavior displayed by the parasite when it does not concentrate in just one host species and, independently of how long we simulate the system, exploits *both* host species. In Fig. 4 we present simulations of both situations.

Transient MHB can happen in almost any situation. Host populations may have, by chance, similar initial phenotypic distributions. The parasite population may have to travel through phase space to exploit the most abundant species, crossing in its path the region where the population of a less abundant species lies. This is especially true in the case of a one-dimensional phase space model, since there is only one path connecting two distinct points in the phenotypic space. The duration of a transient MHB is dependent on the parameters of the model which represent selection pressure, rate of change in phenotypic space, species abundance and other factors. Given the above considerations, we will consider only *stable* MHB as "true" multiple host behavior.

We ran 100 simulations of the two host model, and in 19 the parasite displayed MHB behavior. In 16 of those 19 simulations, the parasite population splits in two subpopulations, each one adapting to the phenotype of one host species;

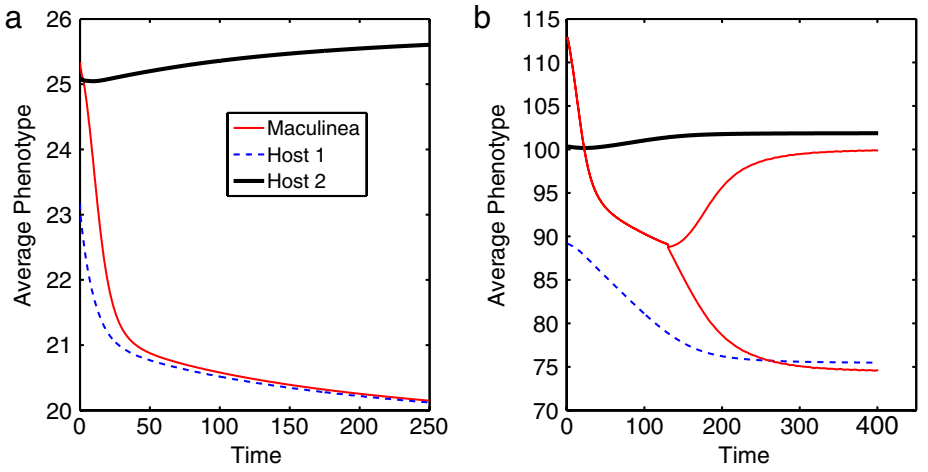

Fig. 4. Average phenotypes of hosts and parasite. (a) Evolution of single host behavior, the parasite population concentrates its average phenotype in only one host species. (b) Stable multiple host behavior. Parasite population splits into two subpopulations, each with an average phenotype similar to one host species.

in the other 3 the parasite population created a valley (*i.e.*, two close peaks in the distribution) that imprisoned both host species within it, in these 3 special cases, results of MHB behavior were strongly dependent on the initial conditions.

Another important point related to host specificity is the question whether the parasite population will always converge to the most abundant host species in the infection area. In our model the abundance of host i in the infection area can be approximated by $\gamma_i K_i$, the population abundance in the whole site times the percentage of nests *inside* the infection area.

In each simulation, we observed which was the host species the parasite population converged to. In the SHB simulations the classification of convergence for each case was obvious, and in 76.5% (62 of 81 SHB cases) of the simulations the parasite converged to the host most abundant in the infection area. In most cases where the parasite population converged to the less abundant species there was a common factor: the rate of change in phase space of the more abundant species was greater than unity and the less abundant was lower than unity. In biological terms this implies at least two important features: the more abundant species evolves faster and also has a greater population variance. If the host species evolves faster, it makes it harder for the parasite population to adapt and pursue its phenotype in phase space, creating a favorable scenario for the parasite to converge to a less abundant host species (that does not evolve so quickly). Contributing in the same direction, the less abundant species has a smaller rate of change in the aspect space, evolving slower and having a smaller population variance. This smaller variance creates a peak of adaptation for the parasite species, since the majority of the host individuals can be found in a smaller vicinity around the average. This also contributes for the parasite population to converge to the less abundant species because although the species is less abundant it could provide the most common *phenotype* in the host population as a whole.

Convergence to less abundant species has been observed in field studies [45] and, of course, there are other conceivable explanations besides the one exposed above. One possible explanation is simply transient state of evolution, host species densities are changing with time and the parasite does not had yet the time to adapt to the most abundant one. Another possible explanation is just distance in phase space. If the most abundant host species is too "far away" in phase space from the parasite population, it is possible that there is no feasible way for evolution to create a "jump" in adaptation for the parasite population to exploit the most abundant species. In other words, the parasite species is separated from a peak of higher fitness by a long valley of low adaptation (the region between the host species where there are no exploitable phenotypes).

In the cases where MHB was observed classification of convergence to the most abundant species became more subjective, since the parasite population now divides its phenotypic distribution between two host species. Even so there were cases where we could identify convergence of the majority of the parasite population to one host species, in 78.9% (15 of 19 cases) the convergence was to the most abundant host species. In the 4 cases where convergence was not clear the population divided almost equally between the host species.

Arms race behavior was more frequent with two host species being present in 39% of the simulations. It occurred in 30% (25 of 81) of the cases of SHB, a percentage similar to the one host simulations, and in 73% (14 of 19) of the cases of MHB. In the MHB cases the parasite could be in an arms race with one of the host species while in stationary regimen with the other. In such cases the regimen was counted as an arms race.

In Table 3 we present a summary of the two-host simulation results.

All the above considerations are related to the simulations in which we took a *random* sample of the parameter space. To explore further the relations of parasite and host when there are 2 host species, we also ran systematic simulations,

Table 3
Summary of results for the two-host simulations.

Behavior	Frequency (%)	Arms race (%)	Convergence to most common host (%)
MHB	19	74	79
SHB	81	30	77

Table 4
Simulation results for $\delta = 8, \lambda = 0.5$. S—Stationary distribution, AR—simple arms race regimen, CAR—complex arms race. For example: H1-AR/H2-S means that the parasite population subdivided into two subpopulations, having as primary host H1 and is in an arms race with it;—host H2 is a secondary host and the parasite subpopulation has a stationary distribution. The other combinations have analogous interpretation: H2-S/H1-CAR. The "=" sign means that the parasite subdivides into subpopulations of equal number of individuals.

$\sigma_2 = 1$						
	γ_2					
		0.2	**0. 4**	**0.6**	**0.8**	**1**
γ_1	**0.2**	H1-S = H2-S	H2-S	H2-AR	H2-AR	H2-AR/H1-S
	0.4	H1-S	H1-S = H2-S	H2-S/H1-S	H2-S/H1-S	H2-AR/H1-S
	0.6	H1-AR	H1-S/H2-S	H1-S = H2-S	H2-S/H1-S	H2-S/H1-S
	0.8	H1-AR	H1-S/H2-S	H1-S/H2-S	H1-S = H2-S	H1-S = H2-S
	1	H1-AR/H2-S	H1-AR/H2-S	H1-S/H2-S	H1-S = H2-S	H1-S = H2-S
$\sigma_2 = 0.5$						
	γ_2					
		0.2	**0.4**	**0.6**	**0.8**	**1**
γ_1	**0.2**	H1-S	H1-S/H2-S	H2-S/H1-S	H2-S/H1-S	H2-AR/H1-S
	0.4	H1-S	H1-S	H1-S/H2-S	H1-S/H2-S	H1-S/H2-S
	0.6	H1-AR	H1-AR	H1-S/H2-S	H1-S/H2-S	H1-S/H2-S
	0.8	H1-AR	H1-AR	H1-AR/H2-S	H1-S/H2-S	H1-S/H2-S
	1	H1-CAR	H1-AR/H2-S	H1-AR/H2-S	H1-AR/H2-S	H1-AR/H2-S
$\sigma_2 = 0.25$						
	γ_2					
		0.2	**0.4**	**0.6**	**0.8**	**1**
γ_1	**0.2**	H1-S	H1-S	H1-S	H1-S/H2-S	H1-S/H2-S
	0.4	H1-S	H1-S	H1-S	H1-S	H1-S/H2-S
	0.6	H1-CAR	H1-AR	H1-AR	H1-AR	H1-S/H2-S
	0.8	H1-CAR	H1-AR	H1-AR	H1-AR	H1-AR/H2-S
	1	H1-CAR	H1-CAR	H1-AR	H1-AR/H2-S	H1-AR/H2-S
$\sigma_2 = 0.125$						
	γ_2					
		0.2	**0.4**	**0.6**	**0.8**	**1**
γ_1	**0.2**	H1-S	H1-S	H1-S	H1-S	H1-S
	0.4	H1-S	H1-S	H1-S	H1-S	H1-S
	0.6	H1-CAR	H1-CAR	H1-AR	H1-AR	H1-AR
	0.8	H1-CAR	H1-CAR	H1-CAR	H1-AR	H1-AR
	1	H1-CAR	H1-CAR	H1-CAR	H1-CAR	H1-CAR

restricting the parameter space with the following assumptions:

(a) All populations have similar phenotypic variation coefficients, *i.e.*, $\theta_i = 1$. This assumes that the rate of phenotypic change in the populations due to mutation, recombination and other factors are similar for all species.

(b) The maximum reproduction rates $\lambda_i = \lambda$ are equal for each host species and are smaller or equal 1. This assumes that the timescale of population dynamics is similar for all host species and slower than the parasite timescale (*i.e.*, the population of parasite exhibit significant changes faster than the hosts). This could be indeed a good hypothesis, since parasite and host generation times are distinct, the parasite's being significantly shorter.

To explore the occurrence of stable multiple host behavior we ran 1200 simulations, corresponding to the combination of parameters $\delta \in \{0.125, 1, 8\}$, $\lambda \in \{0.125, 0.25, 0.5, 1\}$, $\sigma_2 = K_2/K_1 \in \{0.125, 0.25, 0.5, 1\}$ and $\gamma_{1,2} \in \{0.2, 0.4, 0.6, 0.8, 1\}$.

Since parameter λ did not affect much the results in the simulations with two hosts, we present variation in terms of δ, σ_2 and $\gamma_{1,2}$. Results are shown in Tables 4–6.

Table 5
Simulation results for $\delta = 1$, $\lambda = 0.5$. S—Stationary distribution, AR—simple arms race regimen, CAR—complex arms race. For example: H1-AR/H2-S means that the parasite population subdivided into two subpopulations, having as primary host H1 and is in an arms race with it;—host H2 is a secondary host and the parasite subpopulation has a stationary distribution. The other combinations have analogous interpretation: H2-S/H1-CAR. The "=" sign means that the parasite subdivides into subpopulations of equal number of individuals.

$\sigma_2 = 1$						
	γ_2					
		0.2	**0.4**	**0.6**	**0.8**	**1**
γ_1	**0.2**	H1-S = H2-S	H2-S	H2-S	H2-S	H2-S
	0.4	H1-S	H1-S = H2-S	H2-S	H2-S	H2-S
	0.6	H1-S	H1-S	H1-S = H2-S	H2-S/H1-S	H2-S
	0.8	H1-S	H1-S	H1-S/H2-S	H1-S = H2-S	H2-S/H1-S
	1	H1-S	H1-S	H1-S	H1-S/H2-S	H1-S = H2-S
$\sigma_2 = 0.5$						
	γ_2					
		0.2	**0.4**	**0.6**	**0.8**	**1**
γ_1	**0.2**	H1-S	H1-S	H2-S	H2-S	H2-S
	0.4	H1-S	H1-S	H1-S	H1-S/H2-S	H2-S/H1-S
	0.6	H1-S	H1-S	H1-S	H1-S	H1-S/H2-S
	0.8	H1-S	H1-S	H1-S	H1-S	H1-S
	1	H1-S	H1-S	H1-S	H1-S	H1-S
$\sigma_2 = 0.25$						
	γ_2					
		0.2	**0.4**	**0.6**	**0.8**	**1**
γ_1	**0.2**	H1-S	H1-S	H1-S	H1-S/H2-S	H2-S/H1-S
	0.4	H1-S	H1-S	H1-S	H1-S	H1-S
	0.6	H1-S	H1-S	H1-S	H1-S	H1-S
	0.8	H1-S	H1-S	H1-S	H1-S	H1-S
	1	H1-S	H1-S	H1-S	H1-S	H1-S
$\sigma_2 = 0.125$						
	γ_2					
		0.2	**0.4**	**0.6**	**0.8**	**1**
γ_1	**0.2**	H1-S	H1-S	H1-S	H1-S	H1-S
	0.4	H1-S	H1-S	H1-S	H1-S	H1-S
	0.6	H1-S	H1-S	H1-S	H1-S	H1-S
	0.8	H1-S	H1-S	H1-S	H1-S	H1-S
	1	H1-S	H1-S	H1-S	H1-S	H1-S

From Tables 4–6, we can draw the following conclusions:

(a) As the parameter δ increases, the frequency of MHB also increases. This is to be expected, since one possible biological interpretation of high values of δ is that the host populations are very tolerant (*i.e.*, phenotypical changes that occur in the timescale of reproduction are not noticed by the host population).
(b) The parasite population goes through a transition of host specificity as the relative abundance of host changes. Such gradual shift can be seen in Table 5, when $\sigma_2 = 1$ and $\gamma_2 = 0.8$, as γ_1 changes, the parasite population shifts its primary host.

As an example of the host transition in Fig. 5 we present the fraction of the total parasite population exploiting host 1 as γ_1 changes ($\delta = 1$, $\lambda = 0.5$, $\gamma_2 = 0.6$, $\sigma_2 = 0.5$).

4.2.3. More than two hosts

Preliminary exploration of the parameter space in the cases of three or more hosts indicates that the same qualitative behaviors are expected. In particular, stable multiple host behavior with more than two hosts was observed in simulations of random exploration of the parameter space, the key factor for multiple host behavior being, as before, the relative abundance of hosts.

5. Conclusions

Below we present a summary of the conclusions obtained through the simulations of the model and discussed in detail in the previous section.

Table 6
Simulation results for $\delta = 0.125$, $\lambda = 0.5$. S—Stationary distribution, AR—simple arms race regimen, CAR—complex arms race. For example: H1-AR/H2-S means that the parasite population subdivided into two subpopulations, having as primary host H1 and is in an arms race with it;— host H2 is a secondary host and the parasite subpopulation has a stationary distribution. The other combinations have analogous interpretation: H2-S/H1-CAR. The "=" sign means that the parasite subdivides into subpopulations of equal number of individuals.

$\sigma_2 = 1$						
	γ_2					
		0.2	**0.4**	**0.6**	**0.8**	**1**
γ_1	**0.2**	H1-S = H2-S	H2-S	H2-S	H2-S	H2-S
	0.4	H1-S	H1-S = H2-S	H2-S	H2-S	H2-S
	0.6	H1-S	H1-S	H1-S = H2-S	H2-S	H2-S
	0.8	H1-S	H1-S	H1-S	H1-S = H2-S	H2-S
	1	H1-S	H1-S	H1-S	H1-S	H1-S = H2-S
$\sigma_2 = 0.5$						
	γ_2					
		0.2	**0.4**	**0.6**	**0.8**	**1**
γ_1	**0.2**	H1-S	H1-S/H2-S	H2-S	H2-S	H2-S
	0.4	H1-S	H1-S	H1-S	H1-S/H2-S	H2-S
	0.6	H1-S	H1-S	H1-S	H1-S	H1-S
	0.8	H1-S	H1-S	H1-S	H1-S	H1-S
	1	H1-S	H1-S	H1-S	H1-S	H1-S
$\sigma_2 = 0.25$						
	γ_2					
		0.2	**0.4**	**0.6**	**0.8**	**1**
γ_1	**0.2**	H1-S	H1-S	H1-S	H1-S/H2-S	H2-S
	0.4	H1-S	H1-S	H1-S	H1-S	H1-S
	0.6	H1-S	H1-S	H1-S	H1-S	H1-S
	0.8	H1-S	H1-S	H1-S	H1-S	H1-S
	1	H1-S	H1-S	H1-S	H1-S	H1-S
$\sigma_2 = 0.125$						
	γ_2					
		0.2	**0.4**	**0.6**	**0.8**	**1**
γ_1	**0.2**	H1-S	H1-S	H1-S	H1-S	H1-S
	0.4	H1-S	H1-S	H1-S	H1-S	H1-S
	0.6	H1-S	H1-S	H1-S	H1-S	H1-S
	0.8	H1-S	H1-S	H1-S	H1-S	H1-S
	1	H1-S	H1-S	H1-S	H1-S	H1-S

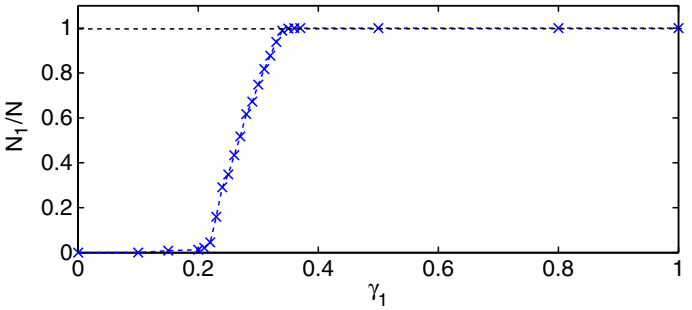

Fig. 5. Host transition simulations with parameters $\delta = 1, \lambda = 0.5, \gamma_2 = 0.6, \sigma_2 = 0.5$. As γ_1 increases, the parasite population gradually shifts from host 2 to multiple host behavior and then finally to host 1. The fraction N_1/N is the parasite population exploiting host 1 divided by the total parasite population.

5.1. Multiple host behavior

Our simulations show that multiple host behavior can occur in both transient and stable forms during the evolution of the system. In transient MHB we should expect superposition of phenotypes while in stable MHB we would expect to observe distinct phenotypes for the exploited populations, with the parasite population splitting into two subpopulations each exploiting one host species. These are testable conclusions in long term studies, being necessary to monitor the evolution of

phenotypes at sites where this behavior was observed. Our simulations also point out that SHB is expected to be the most common behavior, with the parasite exploiting the most common host species.

Thomas et al. [34] point out four possible scenarios to explain MHB in *Maculinea* populations. We discuss them below in light of the mathematical model:

1—*Benign environmental conditions*: non-host species can raise *Maculinea* larvae under favorable conditions. In the model, this could be interpreted as a momentary (= temporal) change in the parameter δ (more precisely, it would be a change in the ε parameter which defines δ), making it possible for the parasite population to exploit the host populations that were beyond reach under a smaller value of δ. Results presented in Table 4 of this paper indicate that high values of tolerance can lead to MHB. This transient MHB effect referred to by Thomas is also present in our model but one should not use the same explanation for MHB if this is too often recorded in different places and environmental conditions.

2—*Polymorphic larvae of Maculinea may be adapted to MHB*: our simulations results point out that, in stable MHB, parasite populations would split in subpopulations each adapting to a different host. In our model we are not investigating the possibility of a speciation event, but it is clear that this divergence of adaptation might lead to the rise of two distinct species. If we interpret the result as one species with two different phenotypes, we will have the polymorphic scenario. On the other hand, if we look at the results with the possibility of a speciation event we get the next possible scenario.

3—*Mixed-host populations* may *occupy habitats in areas that are on biogeographic boundaries like between single-host M. alcon and M. rebeli areas*: in this case we would be looking at two distinct species (their separation in good species is also questioned by the Tartally et al. [24] each one having a SHB behavior). This scenario is analogous to the population subdividing into two subpopulations and considering this as a speciation event.

4—*Truly generalist Maculinea populations may show phenotypic adaptations to more than one host*: in this last scenario we are considering the case where hosts have very distinct phenotypes and, yet, the parasite is adapted to both. The current version of the model does not include this possibility since we would have to consider a distinct topology in the phase space (a parasite could have a phenotype x that is both close to z and y while z and y themselves are far apart).

Note that transient MHB behavior is also another possible scenario to explain observations of MHB behavior.

5.2. Exploitation of the most abundant hosts

As expected, our simulations indicate that parasite populations should typically adapt to exploit the most common host species inside the infection area. On the other hand, special combinations of parameters (rates of reproduction and evolution) can lead to the exploitation of less abundant species. Transient exploitation of less abundant species is also observed in the simulations with more than two host species, the populations of the two less abundant species can create a "common phenotype" attracting the parasite population as though both were just one population. Finally, convergence to a less abundant species was observed in the simulations if the distance in phase space from the parasite population to the abundant host was too great for the population to reach it. Indirect evidence of these conclusions could be obtained by checking particular cases where the parasite converged to the less abundant species: if the cause of this convergence was due to faster evolution of the most abundant species, we would expect to observe a greater variance of phenotypes in the most abundant species than in the less abundant one. If the cause of the convergence was distance in phase space, distance in phenotypic space can also be measured, and we could expect the distance from host to non-host to be greater in this case than in other regular cases (*i.e.*, when the parasite population converged to the most abundant host). Finally, if the case was the one in which the parasite exploits the two less abundant species instead of exploiting the most abundant one, we would expect to see a superposition of the phenotypes of the less abundant species.

5.3. Arms race

The simulations indicate that arms race between host and parasite is expected to occur under the combination of certain factors. For instance, strong selective pressure and hosts with a higher rate of reproduction or mutation than parasites. Multiple host simulations indicate that, under certain conditions, the parasite may be in an arms race with one of the hosts while being in stable stationary state with respect to the other. This kind of dynamics may be suggested as a mechanism for a speciation event, leading to two distinct parasite species.

5.4. Relations to other models and approaches

The approach we have used to model the evolutionary dynamics of parasite–host interactions and the general model presented in Section 3.4 is, to the best of our knowledge, a novelty and presents significant differences from other models that use EES concepts or classic models from population genetics based on the dynamics of gene frequencies. As both approaches refer to the same phenomena, connections and certain equivalences between them are expected to occur. For example we quote [6], whose general results are very similar to our one host simulations:

> "[…]. Victim–exploiter coevolution can lead to four qualitatively different dynamic regimes. If the victim is under strong stabilizing selection, the outcome is similar to the one of a mutualistic interaction. If this is not the case and if the exploiter can evolve faster than the victim, the system may reach a stable equilibrium where the victim is

> trapped at a fitness minimum and experiences disruptive selection. If, in contrast, the victim can evolve faster than the exploiter, both species may undergo coevolutionary cycles [...]".

It is beyond the scope of this paper to explore the full range of correspondences between the approaches.

5.5. Cautionary remarks

Since the model presented was not designed to fit any specific data it is natural that it should raise some skepticism and doubt by those who wish to understand the dynamics of the real biological system. About this specific point we would like to call the attention to the following point: when trying to draw a general evolutionary explanation for the patterns observed in the host specificity behavior of the *Maculinea–Myrmica* system the scientist has access only to few field studies of the process of evolution, usually from different places. Each field study can be thought of as a "snapshot" of the distributions of the populations in the phenotypic phase space and offers some information about the process as it is occurring in modern times in some specific places. From those few studies the scientist may try to create a logic explanation of the patterns observed, and they usually take the form of a linguistic sequence such as (Jackson [46]):

> [...]. Clearly selection must favour highly virulent parasites, those which more quickly exploit host resources, but this process might lead to the death of all potential hosts [...].

Of course this is just an example, but in any such evolutionary reasoning we would see that the reasoning is much less precise than the mathematical model. In the model, at least all the hypotheses are explicit and the parameters explained while in the linguistic sequence much is left undefined and referred to only in a subjective way. Just to be clear, however, it is far from our objective to diminish the importance and value of the expert advice in the understanding of the evolutionary processes or to criticize this particular article/reasoning. Our point is to stress that the model may be a valuable tool for the theoretical biologist who wishes to try his ideas in a controlled "environment", where all hypotheses are clear even though the model is not meant to fit any specific quantitative data (also the logical reasoning in not adjustable to fit quantitative data). We think that a two-dimensional version of the model, given enough field studies, could be developed to fit evolutionary data, although we observe that the amount of necessary data could make the project infeasible. From this point of view, the limitation in data-fitting would come not from the model, but from the difficulty of gathering enough data to fit the model.

With this in mind, we observe that the percentage of observed behaviors in the model should not be used to validate or invalidate the model, because it is dependent on the probability distribution of the parameters in the natural system. For example, in some simulations, there are host species that reproduce four times faster than others. While this may be possible under certain conditions and at specific places this may not be probable. In our sampling of the parameters space we took a uniform distribution over [1, 10], while the distribution of the parameter in the natural system may not be uniform. The percentage of observed behaviors do indicate that some will require a special combination of factors to occur, for instance, stable MHB and convergence to less abundant species. To state clearly, we expect the *qualitative results* drawn from the percentages observed in the simulations to be reflected in the real biological system.

Finally we cite Oster and Wilson [47, preface pp. 8–9] to illustrate our view of the model's main relevance:

"[...]. The most important role models plays in science is to help us to perceive a problem more clearly and to generate thoughts that might not otherwise have occurred [...]".

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