Coexistence of alleles: insights of Modern Coexistence Theory into the maintenance of genetic diversity

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1   Introduction

The question of how genetic variation is maintained, despite the effects of selection and drift, continues to be central to the study of evolutionary biology (Walsh & Lynch, 2018). Classical explanations include overdominance (heterozygote advantage) or frequency-dependent selection, but in the modern era of genomic data, all patterns of elevated variation than expected under neutrality tend to be categorized broadly as balancing selection, regardless of the evolutionary mechanism (Mitchell-Olds *et al.*, 2007). One of the evolutionary mechanisms coined under balancing selection is sexually antagonistic selection, which occurs when the direction of natural selection on traits or loci differs between the sexes (Connallon & Hall, 2018).

Sexually antagonistic selection has been identified as a powerful engine of speciation that in some cases can mantain polymorphisms of otherwise dis-advantageous alleles in a population (Gavrilets, 2014). The effect of sexually antagonistic selection, however, has been generally studied under strong simplifying assumptions such as constant population sizes and homogeneous environments (Kidwell *et al.*, 1977; Pamilo, 1979; Immler *et al.*, 2012). Few studies have explored the effect of sexually antagonistic selection on the maintenance of polymorphism with more realistic assumptions, such as Connallon *et al.* (2018) that found that classical predictions break down when fluctuations in the environment combined with life-history traits allow local adaptations and promote the maintenance of genetic diversity. The effect of environmental fluctuations without local adaptation, however, has not been studied in the context of sexually antagonistic selec-

tion.

The contribution of environmental fluctuations to genetic variability remains a debated issue in evolutionary biology. Classic theoretical models predict that temporal fluctuations in environmental conditions are unlikely to maintain a genetic polymorphism (Hedrick, 1974; 1986). However, other studies have found that fluctuating selection can maintain genetic variance on sex–linked traits (Reinhold, 2000), or in populations where generations overlap (Ellner & Hairston Jr, 1994; Ellner & Sasaki, 1996). Similarly, temporal changes in population sizes have been shown to mitigate the effect of genetic drift in small populations (Pemberton *et al.*, 1996), and annual plant systems (Nunney, 2002). Thus, both fluctuations in selection and population sizes could dramatically change the effect of sexually antagonistic selection in the maintenance of genetic diversity.

Importantly, progress requires more than just identifying if fluctuations can maintain genetic diversity in a population, but to quantify how exactly they contribute to its' maintenance (Ellner *et al.*, 2016). Modern coexistence theory (MCT) provides a powerful conceptual framework to do so (Chesson, 2000b; 1994; Barabás *et al.*, 2018). Although its core ideas were formalized in an ecological context (Chesson, 1994; 2000a), this framework provides the necessary tools to examine the relative contributions of fluctuations to diversity maintenance, which can also be applied to evolutionary contexts (Ellner & Sasaki, 1996; Reinhold, 2000). From an ecological perspective, polymorphism is equivalent to the coexistence of species, and the fixation of either one of the alleles is equivalent to competitive exclusion. The coexistence of alleles, thus, can be examined through the same lens as the coexistence of competing species.

Here, we seek to explicitly apply recent advances in MCT to the question of how polymorphism is maintained under sexually antagonistic selection. Specifically, we aim to quantify the relative importance of different types of fluctuations to overall stable coexistence, or to exclusion of sexually antagonistic alleles. We extended a conceptualization of MCT (Ellner *et al.*, 2016; 2019) to examine how fluctuations in selection values, fluctuations in population sizes, and their interactions can stabilize or hinder the coexistence of alleles. In particular, we examined i) Can fluctuations in population sizes and selection values allow sexually antagonistic alleles to coexist when differences in their fitness would typically not allow them to? and ii) What is the relative contribution of different types of fluctuations that allow two sexually antagonistic alleles to be maintained in a population? Our study provides the tools to analyze evolutionary dynamics from an ecological perspective and contributes to answering long-lasting questions regarding the effect of non-constant environments on genetic diversity.

2 Methods

We first present the evolutionary consequences of sexually antagonistic selection in constant environments. We then present a model that describes the evolutionary dynamics of sexually antagonistic alleles and show how changes in allele frequencies can be expressed in terms of growth rates, a necessary condition for analyses done using MCT. We continue by simulating different scenarios of alleles invading a population, where we allowed population sizes, selection values, both, or neither to vary. Finally, we examine the results of our simulations through a MCT lens by calculating the contribution of each

of these fluctuations in the coexistence of alleles across the parameter space of sexually

antagonist selection.

Sexually antagonistic selection

Most population genetic models of sex-dependent selection consider evolution at single,

biallelic loci with frequency and density independent effects on the relative fitness of

females and males (Wright, 1942; Kidwell *et al.*, 1977; Immler *et al.*, 2012). Consider a locus

with two alleles, j and k, that affect fitness in the haploid state. Assume allele j always has

a high fitness in females ($w_{jf} = 1$), but has variable fitness in males ($w_{jm} < 1$); and allele k

always has a high fitness in males ($w_{km} = 1$), but has variable fitness in females ($w_{kf} < 1$).

The selection against allele j in males is therefore $S_m = 1 - w_{jm}$, and the selection against

allele k in females is $S_f = 1 - w_{kf}$. Selection mantains both alleles in the population

under the condition that:

$$\frac{S_m}{1 + S_m} < S_f < \frac{S_m}{1 - S_m} \tag{1}$$

(Kidwell *et al.*, 1977; Pamilo, 1979; Connallon *et al.*, 2018). These inequalities can be used to

calculate the proportion of the selection parameter space (within the range $0 < S_m, S_f < 1$)

that leads to polymorphism of sexually antagonistic alleles: in ≈ 0.31 of the parame-

ter space allele j will be fixed, in another ≈ 0.31 of the parameter space allele k will be

fixed, and in ≈ 0.38 of the parameter space polymorphism or coexistence of alleles can be

maintained.

Most of the models used to explore the evolutionary dynamics of sexual antagonism

assume constant population sizes and homogeneous environments (Kidwell *et al.*, 1977; Pamilo, 1979; Immler *et al.*, 2012). In constant environments, the maintenance of polymorphism of sexually antagonistic alleles is solely determined by the values of S_m and S_f. If fluctuations in population sizes or selection values have an effect on the coexistence of sexually antagonistic alleles, it would be reflected in increases or decreases of the proportion of the parameter space of selection where polymorphism is maintained.

Population dynamics of sexually antagonistic alleles

Before introducing fluctuations, as a baseline, we used a model that captures the effects of sexually antagonistic selection. Our model consisted of a population that has discrete generations, and that is subject to the previously described sexual antagonism between allele j and k. The frequency of each allele in each sex at the beginning of a life-cycle at time t is given by:

$$p_{jm,t} = \frac{n_{jm,t}}{N_{m,t}} \tag{2}$$

$$p_{jf,t} = \frac{n_{jf,t}}{N_{f,t}} \tag{3}$$

$$p_{km,t} = \frac{N_{m,t} - n_{jm,t}}{N_{m,t}} \tag{4}$$

$$p_{kf,t} = \frac{N_{f,t} - n_{jf,t}}{N_{f,t}} \tag{5}$$

where $N_{m,t}$ and $N_{t,t}$ are the numbers of males and females in a population at time t, $n_{jf,t}$ is the number of females f with allele j, and $n_{jm,t}$ is the number of males m with allele j at time t, respectively.

The individuals in the population mate at random before selection occurs, and therefore the frequency of offspring with allele j after mating, $p'_{j,t}$ can be expressed as:

$$p'_j = \frac{n_{jf,t}}{N_{f,t}}\frac{n_{jm,t}}{N_{m,t}} + \frac{1}{2}\frac{n_{jf,t}}{N_{f,t}}\frac{(N_{m,t}-n_{jm,t})}{N_{m,t}} + \frac{1}{2}\frac{(N_{f,t}-n_{jf,t})}{N_{f,t}}\frac{n_jm,t}{N_{m,t}}, \tag{6}$$

which upon rearranging and simplifying can be written as:

$$p'_{j,t} = \frac{(N_{m,t}n_{jf,t} + N_{f,t}n_{jm,t})}{2N_f N_m}. \tag{7}$$

Selection acts upon these offspring in order to determine the allelic frequencies in females and males in the next generation, $t+1$. As an example the frequency of females with allele j after selection is given by:

$$p'_{jf,t+1} = \frac{n_{jf,t+1}}{N'_{f,t+1}} = \frac{p'_j w_{jf}}{p'_j w_{jf} + (1-p'_j)w_{kf}} \tag{8}$$

The logarithmic growth rate of j in females, is therefore given by the number of females with allele j after selection, divided by the original number of females carrying allele j:

$$r_{jf,t} = \ln\left(\frac{n'_{jf,t+1}}{n_{jf,t}}\right) \tag{9}$$

An equivalent expression for the per capita growth rate of allele j in males m can be obtained by exchanging f for m across the various subscripts in this expression.

Allelic coexistence in a sexual population, however, is ultimately influenced by growth

and establishment of an allele across both sexes. Therefore, the full growth rate of allele j across the entire population of females *and* males is given by:

$$r_j = \ln \left(\frac{n'_{jf,t+1} + n'_{jm,t+1}}{n_{jf,t} + n_{jf,t}} \right) . \tag{10}$$

Equivalently, there exists an expression for r_k.

Simulations

Our simulation approach first consisted of incorporating fluctuations in selection and population sizes into our population dynamics model of sexually antagonistic selection. For reasons that will hopefully become clear later, we then performed simulations of each allele invading a population that experiences the aforementioned fluctuations. Finally, we looked at the relative contributions of each type of fluctuation that allowed each allele to establish in a population. We performed simulations across the selection parameter space of sexually antagonistic selection ($0 < S_m, S_f < 1$). For simplicity, we initially present our approach focusing on a fixed point in the selection parameter space.

Timeseries

We first incorporated the effects of fluctuations into our population dynamics model. To do so, we generated independent timeseries of fluctuations in fitness values and population sizes. In the case of fluctuations in selection values, for a given value of w_{jm} and w_{kf} (i.e., a fixed point in the selection parameter space), we generated a timeseries of 500 timesteps made up of correlated fluctuations of w_{jm} and w_{kf}. Following the approach of

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Shoemaker *et al.* (2020) we controlled the effect size of fluctuations in fitness values (σ_w) and its´ correlation (ρ_w) by using the Cholesky factorization of the variance-covariance matrix:

$$C_w = \begin{bmatrix} \sigma_w^2 & \rho_w \sigma_w^2 \\ \rho_w \sigma_w^2 & \sigma_w^2 \end{bmatrix} \tag{11}$$

We multiplyed Eqn. 11 by a (2×500) matrix of random numbers from a normal distribution with mean 0 and unit variance, which yielded γ_j and γ_k. Then, we calculated the value of w_{jm} at time $t+1$ as $w_{jm,t+1} = w_{jm}^{\gamma_{j,t}}$. We calculated the value of $w_{kf,t+1}$ analogously.

Similarly, we generated a timeseries of 500 timesteps made up of correlated fluctuations in population sizes. We chose values of N_m and N_f of 200 individuals each as the initial value of population sizes throughout our simulations. We performed a Cholesky factorization of the variance-covariance matrix, controlling the effect size of fluctuations in population sizes with σ_g and their correlation with ρ_g. Similar to our previous approach, we multiplied this factorization by a random matrix of uncorrelated random variables, which yielded γ_m and γ_f. Finally, we calculated the number of males in the population at time $t+1$ as $N_{m,t+1} = N_m + \gamma_{m,t}$. We calculated the value of $N_{f,t+1}$ analogously.

Finally, we performed simulations where our population dynamics model (Eqns. 2 to 10) iterated over 500 timesteps while allowing selection values and population sizes to fluctuate in each timestep. We started each simulation with the initial values of N_m and N_f described before and equal frequencies of allele j and allele k in each sex. For

each timestep t in our simulations, the values of w_{jm} w_{kf}, N_m and N_f used to calculate allele´s frequencies in in timestep t (e.g., Eqn. 8), corresponded to the t values calculated in each timeseries, as described previously. This approach yielded a final timeseries that captured the dynamics of sexually antagonistic alleles, with fluctuating values of selection and population sizes.

Invasion simulations

Modern coexistence theory has shown that coexistence is promoted by mechanisms that give species a population growth rate advantage over other species when they become rare (Chesson, 1982; 2003; Barabás *et al.*, 2018). Typically, one species is held at its *resident* state, as given by its steady-state abundances while the rare species is called the *invader*. In the context of alleles in a population, an allele is an *invader* when a mutation occurs that introduces that allele into a population in which it is absent (e.g., if in a population with only k alleles, a random mutation made one individual carry the j allele). Within sexually antagonistic selection, each allele has two pathways of invasion, depending on whether the mutation arises in a female or in a male. If an alleles' *invasion growth rate* (or the average instantaneous population growth rate when rare) is positive, it buffers it against extinction, maintaining its persistence in the population. Coexistence, and hence polymorphism, occurs when all of the alleles in a population have positive invasion growth rates.

To study the dynamics of sexually antagonistic alleles through this framework, we used the timeseries that captured the dynamics of our population model as a template

to perform invasion simulations of both alleles. We allowed each allele to invade via two different pathways males and females, and we explored all potential combinations of each allele invading through a different pathway (e.g., allele j invading through males, and allele k invading through females, and so on). For every point in the parameter space of sexually antagonistic selection, therefore, we explored four different types of invasion.

For each timestep in the timeseries, we performed simulations of the two alleles invading separately via their respective pathway. To simulate invasion, we set the density of the invading allele to one individual, while the resident allele was set to the corresponding value of the timeseries, and iterated our model one timestep. Note that this iteration of our model was independent of the previous iteration that we used to generate the timeseries, therefore we denoted a timestep in an invasion simulation with the subscript i. For example, if allele j was invading via males, then we would set $n_{jm,i} = 1$ and $n_{jf,i} = 0$, while the allele k would be the resident. The abundance of the resident before the invasion was determined by its abundance at timestep t of the timeseries. Then, we iterated our model one timestep and calculated the logarithmic growth rate of j allele invading as:

$$r_j = \ln \left(\frac{n_{jm,i+1} + n_{jf,i+1}}{1} \right) \tag{12}$$

Correspondingly, the logarithmic growth rate of the k allele as a resident would be given by:

$$r_k = \ln \left(\frac{n_{km,i+1} + n_{kf,i+1}}{N_{m,t} + N_{f,t}} \right) \tag{13}$$

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We treated each timestep of the timeseries independently, and hence we performed 500 invasion simulations. We then calculated, for each allele invading via a different pathway, its mean invasion growth rate as the average of the 500 invasion growth rates. We also calculated the mean growth rate of the resident allele as the average of the 500 resident growth rates. We determined alleles to be coexisting if both of them, invading via their respective pathway, had positive mean invasion growth rates, which is often referred to as the mutual invasibility criterion (Barabás *et al.*, 2018).

Functional decompostion

Our invasion simulations tell us whether or not sexually antagonistic alleles can coexist in a determined point of the selection parameter space. However, we also wanted to quantify the relative contributions of fluctuating selection and population sizes into the predicted coexistence outcome. Therefore, we turned towards an extension of modern coexistence theory (Ellner *et al.*, 2019) that provides the flexibility to analyze the contributions of different processes to coexistence using *functional decomposition*. This approach applies to any collection of two or more processes, mechanisms, or species differences affecting population growth rate (Ellner *et al.*, 2016; 2019), and has been used to show the relative contribution of variable temperature and silicate to the coexistence of algal species (Ellner *et al.*, 2016) and to quantify the relative importance of environmental fluctuations and variation in predator abundances to the coexistence of intertidal species (Shoemaker *et al.*, 2020).

The functional decomposition approach focuses on any biotic or abiotic fluctuations

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affecting a population´s growth rate. In our case, it consists of breaking up the average growth rate of each allele into a null growth rate in the absences of fluctuations in all selected variables, a set of main effect terms that represent the effect of only one variable fluctuating, and a set of two-way interaction terms representing the effect of variables fluctuating simultaneously (Ellner *et al.*, 2019). This is a function of four variables: the number of males in the population (N_m), the number of females in the population (N_f), the fitness of allele j in males (w_{jm}), and the fitness of allele k in females (w_{kf}). As an example, if only N_m and N_f were fluctuating, the growth rate of allele j when it is the invader at timestep t could be decomposed into:

$$r_{j,t}(N_m, N_f) = \mathcal{E}_j^0 + \mathcal{E}_j^{N_m} + \mathcal{E}_j^{N_f} + \mathcal{E}_j^{N_m N_f} \tag{14}$$

Where $\mathcal{E}^0$ is the null growth rate when N_m and N_f are set to their averages. Terms with superscripts represent the marginal effects of letting all superscripted variables vary while fixing all the other variables at their average values. For example, the term $\mathcal{E}^{N_m}$ expresses the contribution of fluctuations in N_m when N_f is at its average, without the contribution when both variables are set to their averages :

$$\mathcal{E}_j^{N_m} = r_{j,t}(N_m, \overline{N_f}) - \mathcal{E}_j^0 \tag{15}$$

If we average Eqn. 14 across the timesteps in our simulation, we get a partition of the average population growth rate into the variance–free growth rate, the main effects of variability in N_m, the main effects of variability in N_f, and the interaction between

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variability in N_m and N_f

$$\bar{r}_j = \mathcal{E}_j^0 + \overline{\mathcal{E}}_j^{N_m} + \overline{\mathcal{E}}_j^{N_f} + \overline{\mathcal{E}}_j^{N_m N_f} \tag{16}$$

However, in our simulations w_{jm} and w_{kf} also fluctuated, therefore the full functional decomposition of the growth rate of allele j as an invader is found in Table 1, as well as a brief description of the meaning of each term. The implementation and interpretation of the functional decomposition of the invasion growth rates of each allele are identical to each other. We calculated the value of each of the terms in Table 1 by performing another set of invasion simulations as described previously, but instead of allowing all variables to fluctuate, systematically setting the required variables to their means and subtracting the corresponding $\mathcal{E}$ values.

The functional decomposition approach further requires the *comparison* of each term, to understand if how it affects invaders and residents. This is because fluctuations can promote coexistence by helping whichever allele is rare, or they can hurt whichever allele is common. Therefore, to understand the role of each type of fluctuation, it is necessary to compare how it affects invader *and* resident growth rates. In the example presented in Eqn. 16, if allele j is invading, then allele k is at it's resident state and there exists an analogue decomposition of $\bar{r}_k$ with the exact same terms. Therefore we can express the difference between contributions of fluctuations in N_m as:

$$\Delta_j^{N_m} = \overline{\mathcal{E}}_j^{N_m} - \overline{\mathcal{E}}_k^{N_m} \tag{17}$$

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If $\Delta_j^{N_m}$ is positive, then fluctuations in the male population benefit allele j when it is rare more than what they benefit k as a resident. If $\Delta_j^{N_m}$ is negative, then fluctuations benefit k as a resident more than j as an invader, and if it is minimal, then fluctuations have an equal effect in j and k. Therefore, for each allele invading via a different pathway, we calculated 16 Δ values, one for each one of the $\mathcal{E}$ terms in Table 1. However, since the magnitude of each one of these values could vary considerably across the parameter space of selection, to make them comparable, we normalized each Δ value by dividing it by the length of the $\boldsymbol{\Delta}$ vector. For example, the normalized value of Eqn. 17 would be given by:

$$\Delta_j^{N_m,n} = \frac{\Delta_j^{N_m}}{\sqrt{\sum\limits_{i=1}^{16}(\boldsymbol{\Delta_i})^2}} \tag{18}$$

This normalization bounded Δ values from -1 to 1.

The parameter space of sexually antagonist selection

To evaluate if fluctuations in fitness values and population sizes allow sexually antagonistic alleles to coexist when selection would typically not allow them to, we applied the approach presented so far to the whole parameter space of selection ($0 < S_m, S_f < 1$). To do so, we partitioned the parameter space in 2500 parts, each one a combination of different w_{jm} and w_{kf} values. For each parameter combination, we separately calculated each allele's mean invasion growth rate when invading through males and females, as well as its functional decomposition. Then, we determined coexistence outcomes using

the mutual invasibility criterion. Finally, we calculated the proportion of the parameter

space that allowed alleles to coexist, for each allele invading via a different sex.

We explored all of the combinations of low , intermediate and high fluctuations in fitness values and population sizes, with different extents of correlations between fluctuations (Table 2). As a control simulation, we set $\sigma_w = 0.001$ and $\sigma_g = 0.001$, with no correlation between fluctuations. For each one of the factorial combinations of σ_g, σ_w, ρ_g and ρ_w (Table 2), we performed invasion simulations across the parameter space of selection. We did three replicates per parameter combination, which resulted in 432 simulations.

Figures and tables

Table 1: Functional decomposition of the growth rate of allele j.

Term	Formula	Meaning
$\mathcal{E}_j^0$	$\overline{r}_j(\overline{N_m}, \overline{N_f}, \overline{w_{jm}}, \overline{w_{kf}})$	Growth rate at mean population size and fitness values.
$\overline{\mathcal{E}}_j^{N_m}$	$\overline{r}_j(N_m \overline{N_f}, \overline{w_{jm}}, \overline{w_{kf}}) - \mathcal{E}_j^0$	Main effect of fluctuations in N_m
$\overline{\mathcal{E}}_j^{N_f}$	$\overline{r}_j(\overline{N_m}, N_f, \overline{w_{jm}}, \overline{w_{kf}}) - \mathcal{E}_j^0$	Main effect of fluctuations in N_f
$\overline{\mathcal{E}}_j^{w_{jm}}$	$\overline{r}_j(\overline{N_m}, \overline{N_f}, w_{jm}, \overline{w_{kf}}) - \mathcal{E}_j^0$	Main effect of fluctuations in w_{jm}
$\overline{\mathcal{E}}_j^{w_{kf}}$	$\overline{r}_j(\overline{N_m}, \overline{N_f}, \overline{w_{jm}}, w_{kf}) - \mathcal{E}_j^0$	Main effect of fluctuations in w_{kf}
$\overline{\mathcal{E}}_j^{N_m,N_f}$	$\overline{r}_j(N_m, N_f, \overline{w_{jm}}, \overline{w_{kf}}) - [\mathcal{E}_j^0 + \overline{\mathcal{E}}_j^{N_m} + \overline{\mathcal{E}}_j^{N_f}]$	Interaction of fluctuations in N_m and N_f
$\overline{\mathcal{E}}_j^{w_{jm},w_{kf}}$	$\overline{r}_j(\overline{N_m}, \overline{N_f}, w_{jm}, w_{kf}) - [\mathcal{E}_j^0 + \overline{\mathcal{E}}_j^{w_{jm}} + \overline{\mathcal{E}}_j^{w_{kf}}]$	Interaction of fluctuations in w_{jm} and w_{kf}
$\overline{\mathcal{E}}_j^{N_m w_{jm}}$	$\overline{r}_j(N_m, \overline{N_f}, w_{jm}, \overline{w_{kf}}) - [\mathcal{E}_j^0 + \overline{\mathcal{E}}_j^{N_m} + \overline{\mathcal{E}}_j^{w_{jm}}]$	Interaction of fluctuations in N_m and w_{jm}
$\overline{\mathcal{E}}_j^{N_m w_{kf}}$	$\overline{r}_j(N_m, \overline{N_f}, \overline{w_{jm}}, w_{kf}) - [\mathcal{E}_j^0 + \overline{\mathcal{E}}_j^{N_m} + \overline{\mathcal{E}}_j^{w_{kf}}]$	Interaction of fluctuations in N_m and w_{kf}
$\overline{\mathcal{E}}_j^{N_f w_{jm}}$	$\overline{r}_j(\overline{N_m}, N_f, w_{jm}, \overline{w_{kf}}) - [\mathcal{E}_j^0 + \overline{\mathcal{E}}_j^{N_f} + \overline{\mathcal{E}}_j^{w_{jm}}]$	Interaction of variation in N_f and w_{jm}
$\overline{\mathcal{E}}_j^{N_f w_{fk}}$	$\overline{r}_j(\overline{N_m}, N_f, \overline{w_{jm}}, w_{kf}) - [\mathcal{E}_j^0 + \overline{\mathcal{E}}_j^{N_f} + \overline{\mathcal{E}}_j^{w_{kf}}]$	Interaction of fluctuations N_f and w_{kf}
$\overline{\mathcal{E}}_j^{N_m w_{jm} w_{fk}}$	$\overline{r}_j(N_m, \overline{N_f}, w_{jm}, w_{kf}) - [\mathcal{E}_j^0 + \overline{\mathcal{E}}_j^{N_m} + \overline{\mathcal{E}}_j^{w_{jm}} + \overline{\mathcal{E}}_j^{w_{kf}}]$	Interaction of fluctuations in N_m, w_{jm}, and w_{kf}
$\overline{\mathcal{E}}_j^{N_f w_{jm} w_{fk}}$	$\overline{r}_j(\overline{N_m}, N_f, w_{jm}, w_{kf}) - [\mathcal{E}_j^0 + \overline{\mathcal{E}}_j^{N_f} + \overline{\mathcal{E}}_j^{w_{jm}} + \overline{\mathcal{E}}_j^{w_{kf}}]$	Interaction of fluctuations in N_f, w_{jm}, and w_{kf}
$\overline{\mathcal{E}}_j^{N_m N_f w_{jm}}$	$\overline{r}_j(N_m, N_f, w_{jm}, \overline{w_{kf}}) - [\mathcal{E}_j^0 + \overline{\mathcal{E}}_j^{N_m} + \overline{\mathcal{E}}_j^{N_f} + \overline{\mathcal{E}}_j^{w_{jm}}]$	Interaction of variation in N_m, N_f, and w_{jm}
$\overline{\mathcal{E}}_j^{N_m N_f w_{fk}}$	$\overline{r}_j(N_m, N_f, \overline{w_{jm}}, w_{kf}) - [\mathcal{E}_j^0 + \overline{\mathcal{E}}_j^{N_m} + \overline{\mathcal{E}}_j^{N_f} + \overline{\mathcal{E}}_j^{w_{kf}}]$	Interaction of fluctuations in N_m, N_f, and w_{kf}
$\overline{\mathcal{E}}_j^{N_m N_f w_{jm} w_{fk}}$	$\overline{r}_j(N_m, N_f, w_{jm}, w_{kf}) - [\mathcal{E}_j^0 + \overline{\mathcal{E}}_j^{N_m} + \overline{\mathcal{E}}_j^{N_f} + \overline{\mathcal{E}}_j^{w_{jm}} + \overline{\mathcal{E}}_j^{w_{kf}}]$	Interaction of variation in N_f, N_m, w_{jm}, and w_{kf}

Table 2: Parameters used in our simulations to control the effect size of fluctuations in population sizes (σ_g) and selection values σ_w, as well as their respecitve correlations (ρ_g and ρ_w).

Parameter	Values	Description
σ_w	0.001, 0.1, 0.3, 0.5, 0.7, 0.9	Effect size of fluctuations in fitness values
σ_g	0.001, 1, 10, 20, 30, 50	Effect size of fluctuations in population sizes
ρ_w	-0.75, 0, 0.75	Correlation between fluctuations in fitness values
ρ_g	-0.75, 0, 0.75	Correlation between fluctuation in population sizes

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