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

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1 Abstract

Sexually antagonistic selection (SAS) occurs when the selection in the traits or loci differs between the sexes. This sexual conflict offers the opportunity for maintaining polymorphism in a population, but it often results in the eventual fixation of the fitter allele. However, the effects of SAS have generally been studied under strong simplifying assumptions, such as constant populations and homogeneous environments, which could considerably change the expected outcomes of SAS. Thus, in this study, we examined how fluctuations in selection and population sizes contributed to the coexistence of sexually antagonistic alleles by adopting an ecological framework that allowed us to examine evolutionary dynamics through the same lens as the coexistence of competing species. We performed simulations of alleles invading a population while allowing selection and populations sizes to fluctuate over time. Then, we quantified coexistence outcomes and the relative contribution of each type of fluctuation to each alleles' invasion growth rate. Our results showed that environmental fluctuations can dramatically increase the expected genetic variation under SAS. The positive contribution of fluctuations, however, depended on the sex and allele where invasion occurred. This study contributes to the growing body of work that shows the importance of non-constant environments on the maintenance of genetic diversity.

2 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 variation that exceed the expected variation 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 (Lande, 1980; Arnqvist & Rowe, 2013).

Sexually antagonistic selection can in some cases can mantain polymorphisms of otherwise dis-advantageous alleles in a population (Gavrilets, 2014), which in turn can result in phenotypically distinct sexes that express morphological, physiological, and behavioral traits to different degrees (Mori *et al.*, 2017; Connallon & Hall, 2018). However, sexually antagonistic selection can only mantain polymorphism in specific scenarios, as classical predictions show that sexual antagonism often results in the fixation of the fitter allele (Kidwell *et al.*, 1977; Pamilo, 1979; Hedrick, 1999; Curtsinger *et al.*, 1994; Patten *et al.*, 2010) . Importantly, the effect of sexually antagonistic selection, has been generally studied under strong simplifying assumptions such as constant population sizes and homogeneous environments (e.g., Kidwell *et al.* (1977); Pamilo (1979); Immler *et al.* (2012)). Few studies have explored the effect of sexually antagonistic selection on the mainte-

nance of polymorphism with more realistic assumptions. Excepctions include Connallon *et al.* (2018) who 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 selection to the best of our knowledge.

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 in 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 environmental 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 (modern coexistence theory) 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 of sexually antagonistic alleles is equivalent to the coexistence of species, and the fixation of either one of the alleles in a population 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 modern coexistence theory to the question of how polymorphism is maintained under sexually antagonistic selection. We examined 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 a novel perspective and contributes to answering long-lasting questions regarding the effect of non-constant environments on genetic diversity.

3 Methods

We first 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 modern coexistence theory.

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 modern coexistence theory lens by calculating the contribution of each of these fluctuations in the coexistence of alleles.

Population dynamics of sexually antagonistic alleles

Our model considered evolution at single, biallelic locus with frequency and density independent effects on the relative fitness of females and males. We examined the dynammics of two sexually antagonistic alleles, j and k, that affect fitness in the haploid state. We assumed allele j always has a high fitness in females ($w_{jf} = 1$), but variable fitness in males ($w_{jm} < 1$); and allele k has a high fitness in males ($w_{km} = 1$), but 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 $S_f = 1 - w_{kf}$.

The frequency of each allele in each sex at the beginning of a life-cycle at time t is given by:

$$p_{jm,t} = \frac{n_{jm,t}}{N_{m,t}} \tag{1}$$

$$p_{jf,t} = \frac{n_{jf,t}}{N_{f,t}} \tag{2}$$

$$p_{km,t} = \frac{N_{m,t} - n_{jm,t}}{N_{m,t}} \tag{3}$$

$$p_{kf,t} = \frac{N_{f,t} - n_{jf,t}}{N_{f,t}} \tag{4}$$

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:

$$p'_{j,t} = \frac{n_{jf}}{N_f}\frac{n_{jm}}{N_m} + \frac{1}{2}\frac{n_{jf}}{N_f}\frac{(N_m - n_{jm})}{N_m} + \frac{1}{2}\frac{(N_f - n_{jf})}{N_f}\frac{n_j m}{N_m}\,, \tag{5}$$

which upon rearranging and simplifying gives:

$$p'_{j,t} = \frac{(N_{m,t}n_{jf,t} + N_{f,t}n_{jm,t})}{2N_f N_m}\,. \tag{6}$$

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:

$$p'_{jf,t+1} = \frac{n_{jf,t+1}}{N'_{f,t+1}} = \frac{p'_j w_{jf}}{p'_j w_{jf} + (1 - p'_j)w_{kf}} \tag{7}$$

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:

$$r_{jf,t} = \ln\left(\frac{n'_{jf,t+1}}{n_{jf,t}}\right) \tag{8}$$

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:

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

An equivalent expression describes r_k, the growth rate of allele k.

Selection mantains both alleles in the population under the condition that:

$$\frac{S_m}{1+S_m} < S_f < \frac{S_m}{1-S_m} \tag{10}$$

(Kidwell *et al.*, 1977; Pamilo, 1979; Connallon & Hall, 2018) Thus, the maintenance of polymorphism of sexually antagonistic alleles is solely determined by the values of S_m and S_f. Note that in our model, the values S_m and S_f are bounded from 0 to 1. Therefore the **parameter space of sexually antagonistic selection** is within the range $0 < S_m, S_f < 1$. Classic theoretical models predict that in constant environments, only in ≈ 0.38 of the selection parameter space alleles can coexist (Kidwell *et al.*, 1977; Pamilo, 1979; Connallon *et al.*, 2018). 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.

Simulations

Typically, would require decomposing alleles' growth rates (e.g., Eqn. 9) analytically to examine the relative contributions of different types of fluctuations to their coexistence (Chesson, 1994; 2000b; Barabás *et al.*, 2018). However an analytical approaches to coexistence often entail complex mathematical analyisis and restrictive assumptions to make mathematics tractable (Ellner *et al.*, 2019). Thus, we opted for an extension of modern coexistence theory that provides the flexibility to examine the contributions of different processes to coexistence using simulations (Ellner *et al.*, 2019; 2016; Shoemaker *et al.*, 2020).

For each simulation, we examined coexistence outcomes across the selection parameter space of sexually antagonistic selection ($0 < S_m, S_f < 1$). To do so, we partitioned the parameter space into a grid of 50×50, which yielded 2500 pairwise combinations of different w_{jm} and w_{kf} values. For each pairwise combination of w_{jm} and w_{kf}, as we detail in the next sections, we iterated our model while controlling the effect size of fluctuations in selection (σ_w) and their correlation (ρ_w), as well as fluctuations in population sizes (σ_g) and their correlation (ρ_g). Then, we performed "invasion simulations" of each allele invading a population, evaluated coexistence outcomes, and determined the relative contribution of each type of fluctuation. Finally, we calculated for each simulation the proportion of the parameter space that allowed alleles to coexist.

We explored all of the combinations of low ($\sigma_w = (0.1, 0.3)$, $\sigma_g = (1, 10)$), intermediate ($\sigma_w = (0.5, 0.7)$, $\sigma_g = (20, 30, 50)$), and high fluctuations ($\sigma_w = 0.9$, $\sigma_g = 70$) in fitness values and population sizes, with different extents of correlations between fluctuations

(Table 1). As a control simulation, we set $\sigma_w = 0.001$ and $\sigma_g = 0.001$, with no correlation between fluctuations. We ran ten replicates per parameter combination, which resulted in 3780 simulations.

Timeseries

To incorporate the effects of fluctuations into our population dynamics model we generated independent timeseries of fluctuations in selection 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}. We controlled the effect size of fluctuations in selection (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 new fitness values at time $t+1$ as $w_{jm,t+1} = w_{jm}^{\gamma_{j,t}}$ and $w_{kf,t+1} = w_{kf}^{\gamma_{k,t}}$.

Similarly, we generated an independent timeseries of 500 timesteps made up of correlated fluctuations in population sizes. We chose values of $N_m = 200$ and $N_f = 200$ 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 ap-

proach, 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 and females in the population at time $t+1$ as $N_{m,t+1} = N_m + \gamma_{m,t}$ and $N_{f,t+1} = N_f + \gamma_{f,t}$. Therefore, the population sizes in each timestep differed from the inital value of 200 individuals on the order of ρ_g. Note that the scales of σ_g and σ_w are different from each other. While σ_w controls the exponential change in fitness values in each timestep, σ_g controls the number of individuals added to a population in each timestep.

Finally, we performed simulations where our population dynamics model (Eqns. 1 to 9) 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 timestep t (e.g., Eqn. 7), 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 both alleles have positive invasion growth rates.

We used the timeseries that captured the dynamics of our population model as a template to perform invasion simulations of both alleles. We performed 500 independent invasion simulations, one for each timestep in our timeseries. We explored all four potential combinations of each allele invading through each pathway (e.g., allele j invading through males, and allele k invading through females, and so on). To simulate invasion, we set the density of the invading allele to one individual. For example, if allele j was invading via males, then we would set $n_{jm,i} = 1$ and $n_{jf,i} = 0$. Note that each invasion simulation was independent of the iteration that we used to generate the timeseries, therefore we denoted the initial timestep in an invasion simulation with the subscript i. We also set the resident allele, in this case k, to the corresponding value of the timeseries minus one individual, $n_{km,i} = N_{m,t} - 1$ and $n_{kf,i} = N_{f,t}$. Then, we iterated our model one timestep, $i + 1$, 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:

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

Following the approach of Shoemaker *et al.* (2020), we treated each invasion simulation 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 alleles 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 quantified the relative contributions of fluctuating selection and population sizes into the predicted coexistence outcome using a *functional decomposition* approach (Ellner *et al.*, 2016; 2019; Shoemaker *et al.*, 2020).

We applied the functional decomposition approach by 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). In our simulations, 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 variability in N_m and N_f

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

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 2, 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. Note that Table 2 does not include three or four–way interactions (e.g., $\overline{\mathcal{E}}_j^{N_m N_f \bar{w}_{jm} \bar{w}_{fk}}$). This is because in our simulations, we did not allow fluctuations in selection and population sizes to be correlated, therefore their effects are solely captured by the terms in Table 2. We calculated the value of each of the terms in Table 2 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 (i.e., the relative contribution). 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 as Eqn. 16. Therefore we can express the difference between contributions of fluctuations in N_m as:

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

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 7 Δ values, one for each one of the $\mathcal{E}$ terms in Table 2. 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 square root of the sum of the squares of the 7 Δ values. For example, the normalized value of Eqn. 17 would be given by:

$$\delta_j^{N_m} = \frac{\Delta_j^{N_m}}{\sqrt{\sum_{d=1}^{7} (\boldsymbol{\Delta_d})^2}} \tag{18}$$

This normalization bounded δ values from -1 to 1.

4 Results

Our results showed that both fluctuations in selection and population sizes can substantially increase the expected genetic variability under sexually antagonistic selection. The average proportion of coexistence in the selection parameter space increased with the effect size of fluctuations (Fig. 1). Increments in allelic coexistence were more likely when fluctuations were large, and fluctuations in population sizes were negatively correlated, while fluctuations in selection were positively correlated (Fig. 1). Importantly, our results show that environmental fluctuations can more than double the expected genetic variation under sexually antagonistic selection, reaching up to ≈ 0.9 of allelic coexistence in

the selection parameter space (Fig. 1).

Fluctuations increased coexistence by allowing both alleles as invaders to have positive invasion growth rates in instances where selection would typically not allow them to (i.e., they made parts of the parameter space "flip" into coexistence). As a baseline, we show in Fig. 2A the outcome of the control simulation, which matched previous findings that without fluctuations, alleles can coexist in only $\approx$ 0.38 of the selection parameter space (Connallon & Hall, 2018). These "flips" occurred with both types of fluctuations and were more common with larger fluctuations and strongly correlated effects, for which we show examples in Fig. 2A . However, note that there are also parts of the parameter space where coexistence is lost compared to the control simulation, which was more likely when population sizes were fluctuating (Fig. 2A).

Alleles had positive invasion growth rates when positive contributions of fluctuations outweighed the negative contributions of fluctuations. As an example in Fig. 2B we show the functional decomposition of both alleles invading via their favored pathway in parts of the parameter space that "flipped" into coexistence and competitive exclusion (which correspond to the square and triangle in Fig. 2A). Note that each type of fluctuation made similar contributions to each allele, both when they were coexisting or experiencing competitive exclusion (Fig. 2B). However, δ_0, which captures the effect of fluctuations set to their averages, switched between positive and negative contributions for both alleles (Fig. 2B).

The relative contribution of fluctuations in population sizes of males and females benefited alleles when alleles invaded via the fluctuating population (Fig. 2B and Fig. 3). If

alleles invaded via the opposite sex, then fluctuations contributed negatively to their invasion growth rate (Fig. 3). For example, the relative contribution of fluctuations in the male population, δ^{N_m}, was positive for both alleles when they invaded via males and negative when they invaded via females, regardless of the correlation between fluctuations (Fig. 3). The opposite pattern was shown by δ^{N_f}. The relative contribution of both populations fluctuating, $\delta^{N_m N_f}$, was positive when fluctuations were negatively correlated, it had a negligible effect when fluctuations were not correlated, and it had a negative effect when fluctuations were positively correlated (Fig. 3).

In contrast, fluctuations in selection benefited the allele that was unaffected by selection. For example, $\delta^{w_{jm}}$ which captured the relative contribution of fluctuations in selection against j in males, contributed negatively to allele j´s invasion growth rate but benefited allele k's invasion, regardless of the correlation between fluctuations or the sex where invasion occurred (Fig. 4). The opposite pattern was shown by $\delta^{w_{kf}}$. The relative contribution of both types of selection fluctuating, was negative when fluctuations were negatively correlated, it had a negligible effect when fluctuations were not correlated, and it had a positive effect when fluctuations were positively correlated (Fig. 4).

The overall average effect of fluctuations, δ^0, was at first glance unpredictable when fluctuations were incorporated (Fig. 2B). Recall that δ_0 captured the relative contribution of fluctuations set to their mean. Without fluctuations, δ^0 captured exclusively the effect of selection and had positive values in parts of the parameter space where each allele could invade (Fig S1 Supporting Information). Fluctuations in population sizes generated stochastic changes of δ^0 across the parameter space (Fig. S1). In contrast, fluctuations in

selection slightly changed the magnitude of δ^0, which was heightened in parts of the parameter space that were on the limit of the coexistence region (Fig. S1). When both selection and population sizes fluctuated, the value of δ_0 varied greatly across the selection parameter space (Fig. S1).

Nonetheless, changes in δ^0 can be explained by how we implemented the functional decomposition. Quantifying δ^0 required calculating invader and resident growth rates when fluctuations are set to their mean. However, fluctuations set to their mean do not necessarily equal the mean values when there are no fluctuations. For example, we chose an initial value of 200 male and female individuals for all of our simulations. Without fluctuations in population sizes, the mean population size for each sex was 200 individuals. In contrast, when we incorporated large fluctuations in population sizes ($\sigma_g = 70$), the realized mean of population sizes used to calculate mean invasion growth rates ranged from 100 to 600 individuals (Fig. S2). Effectively, this caused the numbers of males and females to become skewed when we calculated mean invasion growth rates. Changes in δ_0 compared to the control simulation were partly determined by the effective number of females and males in a population when fluctuations were set to their mean (Fig. S3 and S4). The value of δ_0 tended to 'flip" from positive to negative compared to the control simulation if an allele invading via males was introduced to a population predominantly made up of male individuals and to change from positive to negative is the population was predominantly female (Fig.S3). The opposite pattern was shown when alleles invaded via females (Fig.S4).

5 Discussion

The results of our study provide supporting evidence that environmental fluctuations can increase the expected genetic variance maintained under sexually antagonistic selection. Antagonistically selected alleles are an important component of genetic variation for many species (Foerster *et al.*, 2007; Van Doorn, 2009; Bonduriansky & Chenoweth, 2009; Innocenti & Morrow, 2010). Furthermore, as much as 20% of traits for which data is available are thought to be under sexually antagonistic selection (Morrissey, 2016). Yet, a large body of work suggests that the criteria for maintaining antagonistic genetic variation are very restrictive (i.e., we would expect the coexistence of alleles in few scenarios) (Kidwell *et al.*, 1977; Pamilo, 1979; Hedrick, 1999; Curtsinger *et al.*, 1994; Patten *et al.*, 2010). Our study shows that incorporating more realistic assumptions, such as non-constant selection and population sizes can more than double the expected genetic variation under sexually antagonistic selection (Fig. 1).

The relative contribution of fluctuations in selection

Our simulations indicate that fluctuations in selection can promote allelic coexistence in parts of the parameter space where we would typically expect selection to fix one of the alleles (Fig. 2). Fluctuations in selection had positive contributions to the invasion growth rate of the allele that was unaffected by selection and were dis-advantageous if the fluctuations directly affected the invading allele (e.g., $\delta_{w_{jm}}$ contributed positively to the growth rate of k and negatively to the growth rate of j, Fig. 4). The mechanism by which fluc-

tuations in selection promoted coexistence can be understood as *relative non-linearity in response to selection* and arises because fluctuations in w_{jm} and w_{kf} do not affect both alleles equally, and thus each allele responds differently to them.

The term *relative non-linearity* refers to fluctuation-dependent coexistence mechanisms that arise from competitors responding differently to limiting competitive factors (Chesson, 2000a; Ellner *et al.*, 2016; Zepeda & Martorell, 2019). Our results suggest that in the case of sexually antagonistic alleles, a non-advantageous allele can be maintained in a population if the favored allele experiences high fluctuations in selection (Fig. 4). This could be the case, for example, if traits associated with sexual dimorphism like ornaments or bright colors are also associated with higher predator rates (Bildstein *et al.*, 1989; Götmark *et al.*, 1997) or sex-biased mortality (Promislow *et al.*, 1992). However, if the non-advantageous allele is the one associated to higher fluctuations in selection, then fluctuations will likely erode genetic diversity.

The interactive effect of fluctuations in selection, $\delta_{w_{jm},w_{kf}}$, accounts for the additional change in alleles' growth rates when both w_{jm} and w_{kf} vary, beyond the contribution of each effect varying on its own. This term only promoted allelic coexistence when fluctuations were positively correlated, while it contributed negatively to each allele's growth rate if fluctuations were negatively correlated (Fig. 4). Environmental fluctuations are often correlated (Steele, 1985), and previous studies have shown that positively correlated fluctuations can increase the invasion growth rate of a species when there are species-specific environmental responses and there is buffered population growth where species are shielded from competition (Schreiber, 2021). This coexistence mechanism is often

referred to as the *storage effect* and it arises when fluctuations that alternate between favorable conditions to one species and those favorable to another, there exists a life-history stage for each species to survive unaffected by the unfavorable conditions, such as long–term dormancy (Chesson, 2000b; Ellner *et al.*, 2016; Barabás *et al.*, 2018; Schreiber, 2021). In the case of sexually antagonistic alleles, a "species-specific" environmental response arises from the fact that each allele responds differently to fluctuations in w_{jm} and w_{kf}, while population buffering occurs because one allele is unaffected by fluctuations in selection in the other allele. Thus, in environments where selection on both alleles fluctuates simultaneously, only positively correlated fluctuations benefit the maintenance of genetic diversity in a population. This could arise, for example, in environments where sexual selection on both sexes is stronger when climatic conditions are favorable and becomes negligible in stressful conditions (Cockburn *et al.*, 2008).

Temporal fluctuations in selection were initially thought to be of limited importance to the maintenance of polymorphism (Hedrick, 1974; 1986). However, some studies have shown that in populations with overlapping generations (Ellner & Sasaki, 1996; Sasaki & Ellner, 1995), sex-limited traits (Reinhold, 2000), or heterozygote individuals carrying rare alleles (Schreiber, 2020), fluctuations in selection over time can maintain allelic coexistence due to the storage effect. Similarly, Connallon & Hall (2018) found that when fluctuations in selection promote local adaptations due to life-history traits, the expected proportion of allelic coexistence in the selection parameter space can increase significantly. Our results provide further evidence that fluctuations in selection can promote the maintenance of genetic diversity, as sexual antagonism requires selection to differentially affect

the alleles involved and thus promote non-linear responses to fluctuations, as well as a storage effect when fluctuations are positively correlated.

The relative contribution of fluctuations in population sizes

Fluctuations in population sizes caused overall increases in the proportion of coexistence compared to the control simulation (Fig. 1). Similar to fluctuations in selection, fluctuations in population sizes had positive contributions to the invasion growth rate of alleles due to *relative non-linearity in response to population sizes*. In contrast to fluctuations in selection, fluctuations in population sizes benefited the allele that was directly affected by fluctuations (e.g., δ_{N_m} benefited the invasion of growth rate of an allele if it invaded via males Fig. 3). If an allele invaded via the non-fluctuating sex, however, fluctuations contributed negatively to its invasion growth rate (Fig. 3).

Therefore, our results suggest that a non-advantageous allele could have a positive invasion growth rate if it invaded via a population experiencing temporal changes in its size. Temporal changes in population sizes of males and females can arise due to sex differences in movement (e.g., if males immigrate to higher quality areas (Matter & Roland, 2002)), development (e.g., females requiring more time to mature than males (Kasumovic *et al.*, 2008)), and behavior (e.g., cannibalistic mating (Elgar *et al.*, 2003)). When males and females experience different population dynamics, sexual antagonism allows alleles to differentiate in their response to fluctuations, and thus, promote allelic coexistence. The interactive effect of fluctuations in males and females, δ_{N_m,N_f}, shows that if both populations fluctuate, then negatively correlated fluctuations promote the main-

tenance of genetic diversity, while positively correlated fluctuations will likely impair it (Fig. 3). These insights offer an exciting avenue of research to understand if sexually selected traits are often found in populations that experience negatively correlated temporal changes in population sizes, and could help explain the high heritabilities of those traits (Reinhold, 2000).

Nonetheless, fluctuations in population sizes also caused competitive exclusion in some parts of the parameter space where we would expect selection to maintain both alleles (Fig. 2A). These "flips" into competitive exclusion were driven by changes to δ_0 compared to the control simulation when fluctuations in population sizes were incorporated (Fig. 2B). The stochastic changes in δ_0 were caused by changes to the mean number of males and females used to calculate the value of each allele's mean invasion growth rate. Our results suggest that invasion is less likely to occur when an allele is introduced to a population via a sex that is overrepresented in a population (Figs. S3 and S4). These results highlight the fact that antagonistic selection is often ineffective in the face of genetic drift (Connallon & Clark, 2012), and that in populations with sex-skewed ratios the effect of sexually antagonistic selection might be lost. Indeed, previous studies have found that the impact of drift is particularly pronounced at or near the parameter domain for balancing selection (Connallon & Clark, 2012), which we also found in our simulations (Fig. S1). Thus, although fluctuations can promote allelic coexistence due to differential responses to fluctuations, the criteria for maintaining sexually antagonistic alleles in a population is sensitive to sex ratios which can result in the loss of polymorphism.

Allelic coexistence and sexual conflict

Our study exclusively focused on the maintenance of polymorphism in a population understood as the coexistence of alleles. However, maintaining non-advantageous alleles in a population is costly, and can result in the overall fitness of a population to decrease (Connallon & Hall, 2018). Sexually antagonistic selection necessarily involves a mismatch between the traits a population express and the optimal expression of those traits, and it is often resolved once members of both sexes express traits that match the sex-specific optima (i.e., when non-advantageous alleles are eliminated from a population) (Lande, 1980). Our results show that large fluctuations in selection and population sizes can impede the resolution of sexual conflict by maintaining both alleles in a population, even when selection against those alleles is strong (Fig. 2A). The maintenance of genetic diversity promoted by fluctuations, thus, might involve trade-offs in the fitness and evolution of a population that might not be feasible in nature.

Conclusion

Our study contributes to the growing body of work that shows that the criteria for maintaining genetic variation under sexually antagonistic selection are overly conservative (Connallon & Clark, 2012; Connallon *et al.*, 2018). Processes like recurrent mutations (Radwan, 2008), genetic drift (Connallon & Clark, 2012), local adaptations (Connallon *et al.*, 2018), and alleles that experience seasonal changes in dominance (Wittmann *et al.*, 2017) have been shown to dramatically change the levels of sexually antagonistic variance in natural populations. Our study shows that non-constant environments might promote

481 the maintenance of genetic diversity of sexually antagonistic alleles without the need for
482 local adaptations or life-history stages that involve overlapping generations. However,
483 our study also shows that fluctuations can also erode genetic diversity, especially when
484 fluctuations in population sizes are involved. The environmental drivers that maintain
485 sexually antagonistic traits are still poorly understood (Connallon & Hall, 2018), and it is
486 essential that we understand how diversity might respond to rapid environmental change
487 (Tylianakis *et al.*, 2008). Our study provides an interdisciplinary example of how to imple-
488 ment an ecological framework to study how genetic diversity is maintained and responds
489 to environmental fluctuations.

Figures and tables

Table 1: 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). We ran ten replicates for each one of the factorial combinations of the following parameters, which yielded a total of 3780 simulations.

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, 70	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

Table 2: Functional decomposition of the growth rate of allele j. As we exemplify in Eqn. 16, each term captures the contribution of fluctuations to an alleles' invasion growth rate.

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 selection 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}

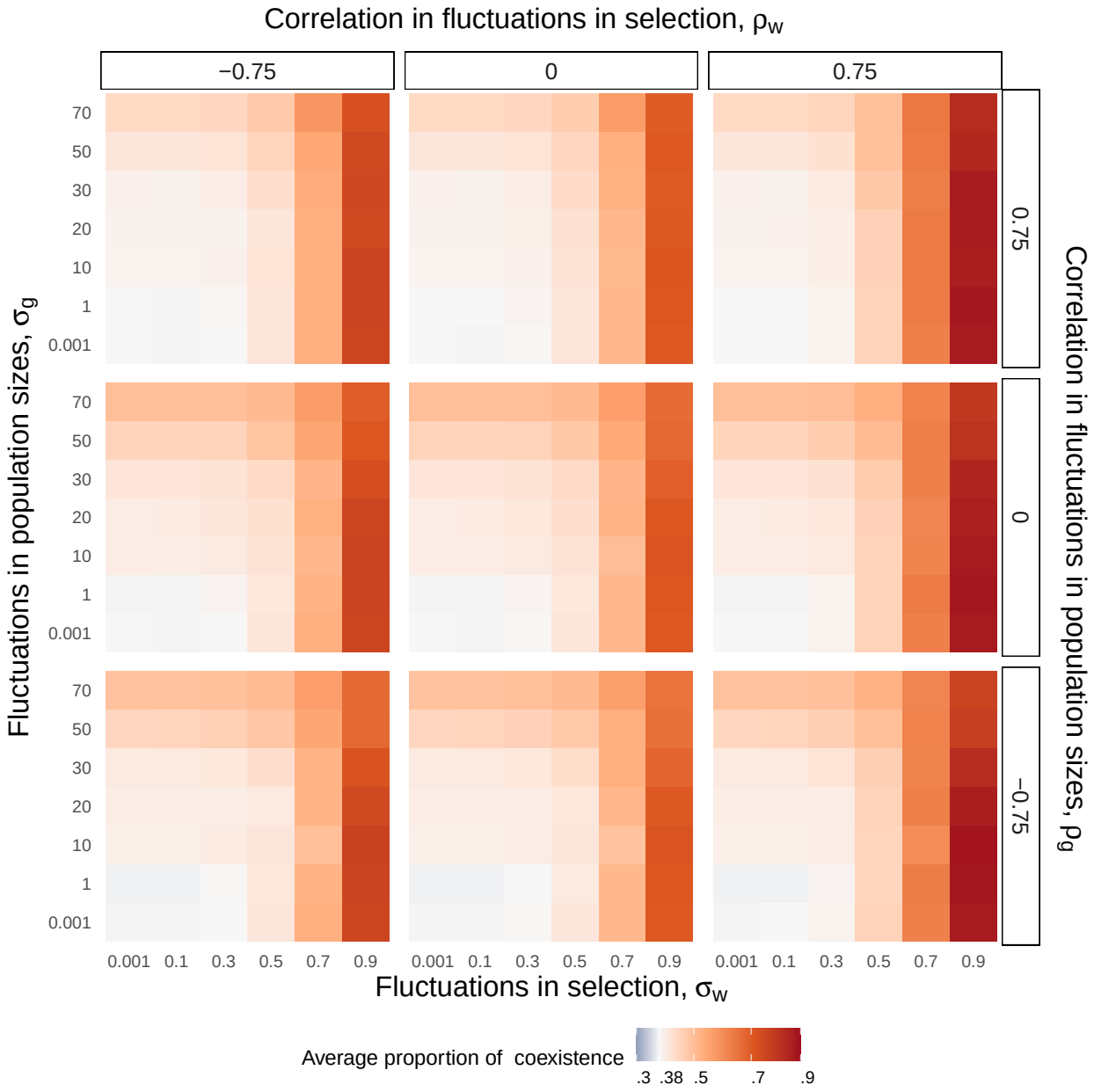

Figure 1: The average proportion of coexistence across the selection parameter space. For all parameter combinations in our simulations, we show the average proportion of coexistence for all replicates and invasion scenarios (each allele invading a different sex). Each panel corresponds to a different combination of correlations between fluctuations. Labels on top indicate the correlation between fluctuations in selection ρ_w, while labels on the right show the correlation in fluctuations between fluctuations in population sizes ρ_g. As a basis of comparison, we show the expected proportion of coexistence (0.38) as white in our color scheme.

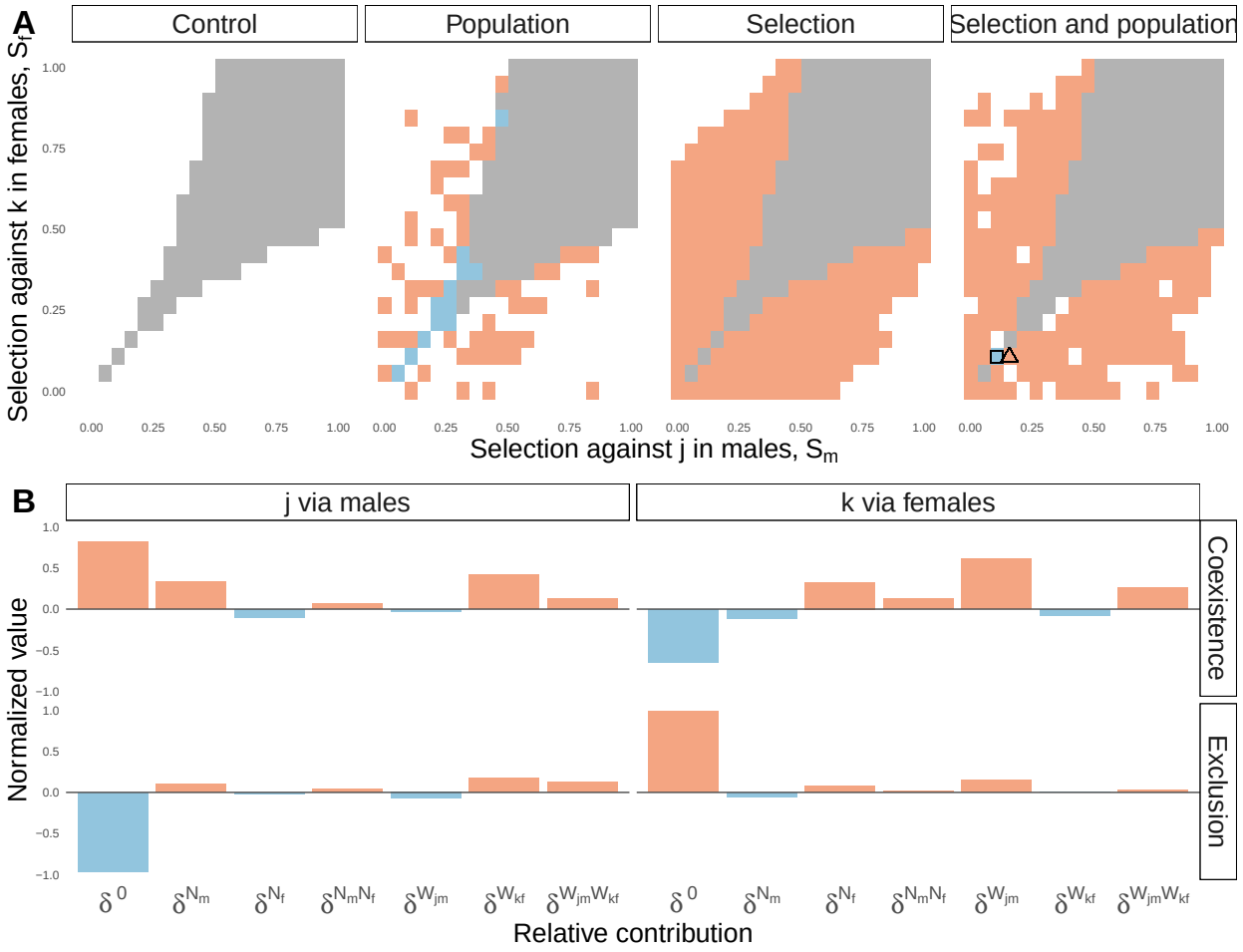

Figure 2: Coexistene outcomes and functional decomposition. In A) each panel corresponds to one replicate of a different type of simulation. All panels show the coexistence outcomes in the selection parameter space when j invaded via males and k invaded via females. As a reference, j is favored in females and k is favored in males. In the Control panel ($\sigma_g = 0.001$, $\rho_g = 0$, $\sigma_w = 0.001$, $\rho_w = 0$) grey areas indicate parts of the selection parameter space where alleles can coexist, while white areas indicate parts of the parameter space that correspond to competitive exclusion (following Eqn.10). In the Population ($\sigma_g = 70$, $\rho_g = -0.75$, $\sigma_w = 0.001$, $\rho_w = 0$), Selection ($\sigma_g = 0.001$, $\rho_g = 0$, $\sigma_w = 0.9$, $\rho_w = 0.75$), and Selection and population ($\sigma_g = 0.9$, $\rho_g = -0.75$, $\sigma_w = 0.9$, $\rho_w = 0.75$) panels, red areas indicate parts of the parameter space that "flipped" into coexistence, while blue areas show changes into competitive exclusion. We highlighted two points in the parameter space in the Selection and popualtion panel that corresponded to changes into coexistence (triangle) and into competitive exlcusion (square). In B) we show the functional decomposition of the coexistence and competitive exclusion points highlighted in A). Each panel corresponds to each allele invading via their respective pathway and shows the bar plots of the different δ values that made up the functional decomposition of each allele as an invader. Red colors indicate positive δ values that benefited that allele as an invader more than the other allele as a resident, while blue colors indicate negative δ values that benefited a resident more than the invader.

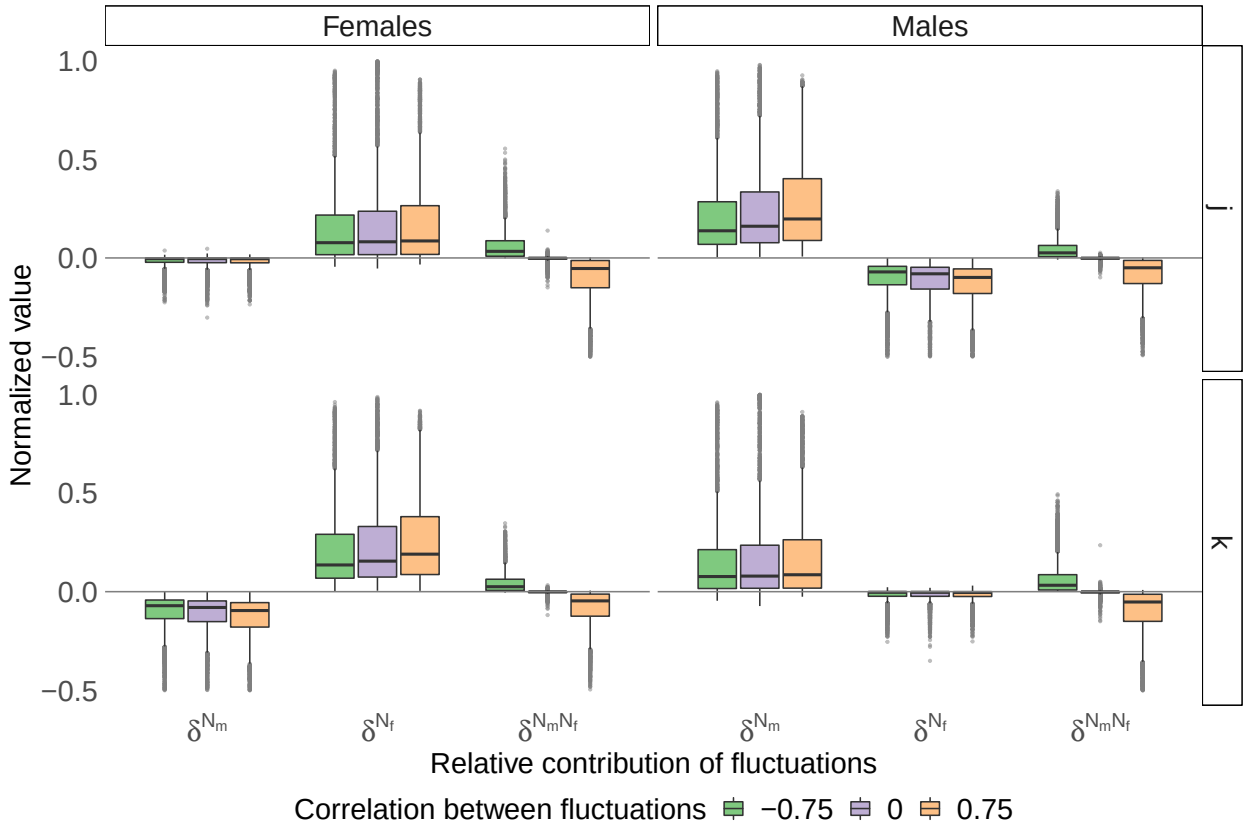

Figure 3: The relative contributions of fluctuations in population sizes. As a reference, positive δ values imply that the corresponding fluctuation benefits that allele as an invader more than the other allele as a resident, negative δ values indicate fluctuations benefit the residents more than the invader, and δ values close to zero indicate that the corresponding fluctuation has equal contributions to invaders and residents. Each panel corresponds to each allele invading via a different pathway, for which we show the boxplots of the three distinct δ values that captured the effects of fluctuations in population sizes, for all of the replicates in our simulation in which $\sigma_g = 70$. Each color corresponds to a different correlation between fluctuations in population sizes (ρ_g), as the legend indicates. Each box plot extends from the first to third quantiles of the corresponding posterior distribution of parameter values, and the line inside the the box indicates the median.

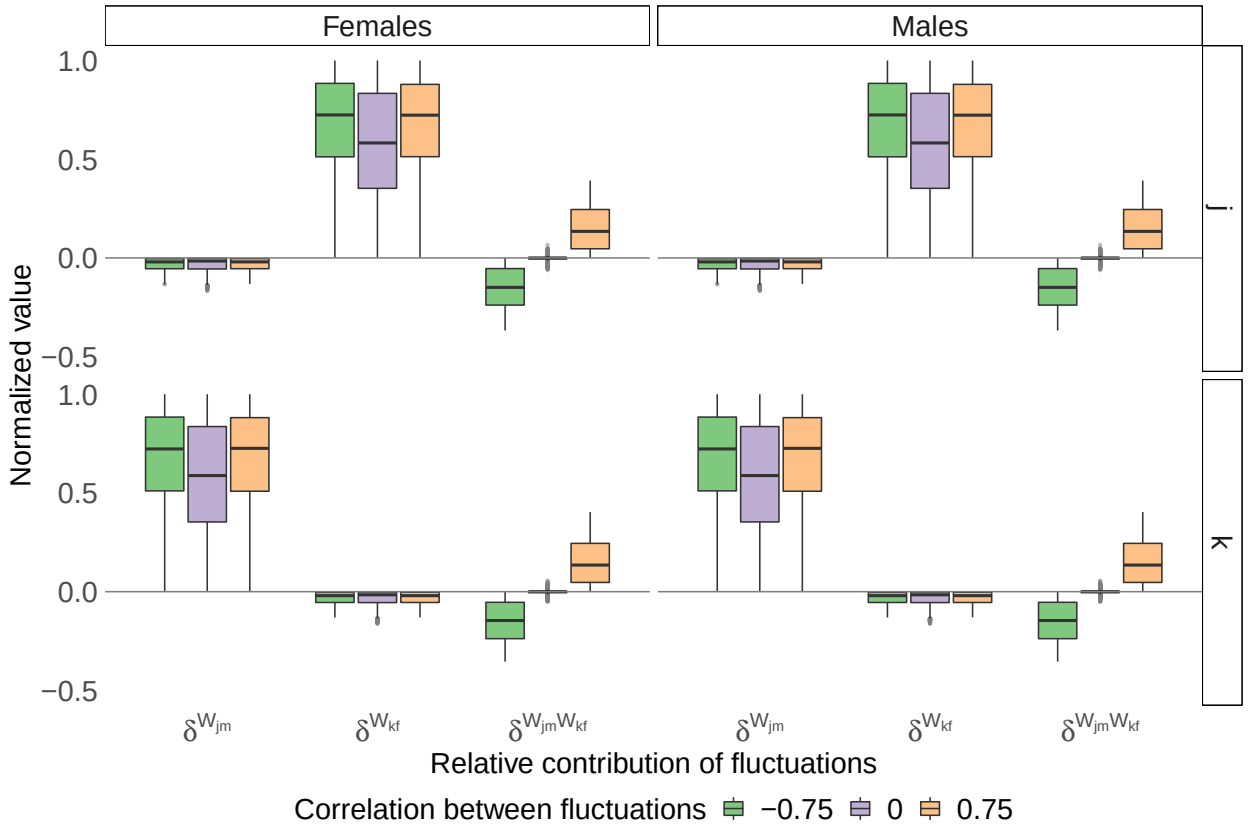

Figure 4: The relative contributions of fluctuations in selection. As a reference, positive δ values imply that the corresponding fluctuation benefits that allele as an invader more than the other allele as a resident, negative δ values indicate fluctuations benefit the residents more than the invader, and δ values close to zero indicate that the corresponding fluctuation has equal contributions to invaders and residents. Each panel corresponds to each allele invading via a different pathway, for which we show the boxplots of the three distinct δ values that captured the effects of fluctuations in population sizes, for all of the replicates in our simulation in which $\sigma_w = 0.90$. Each color corresponds to a different correlation between fluctuations in population sizes (ρ_w), as the legend indicates. Each box plot extends from the first to third quantiles of the corresponding posterior distribution of parameter values, and the line inside the the box indicates the median.

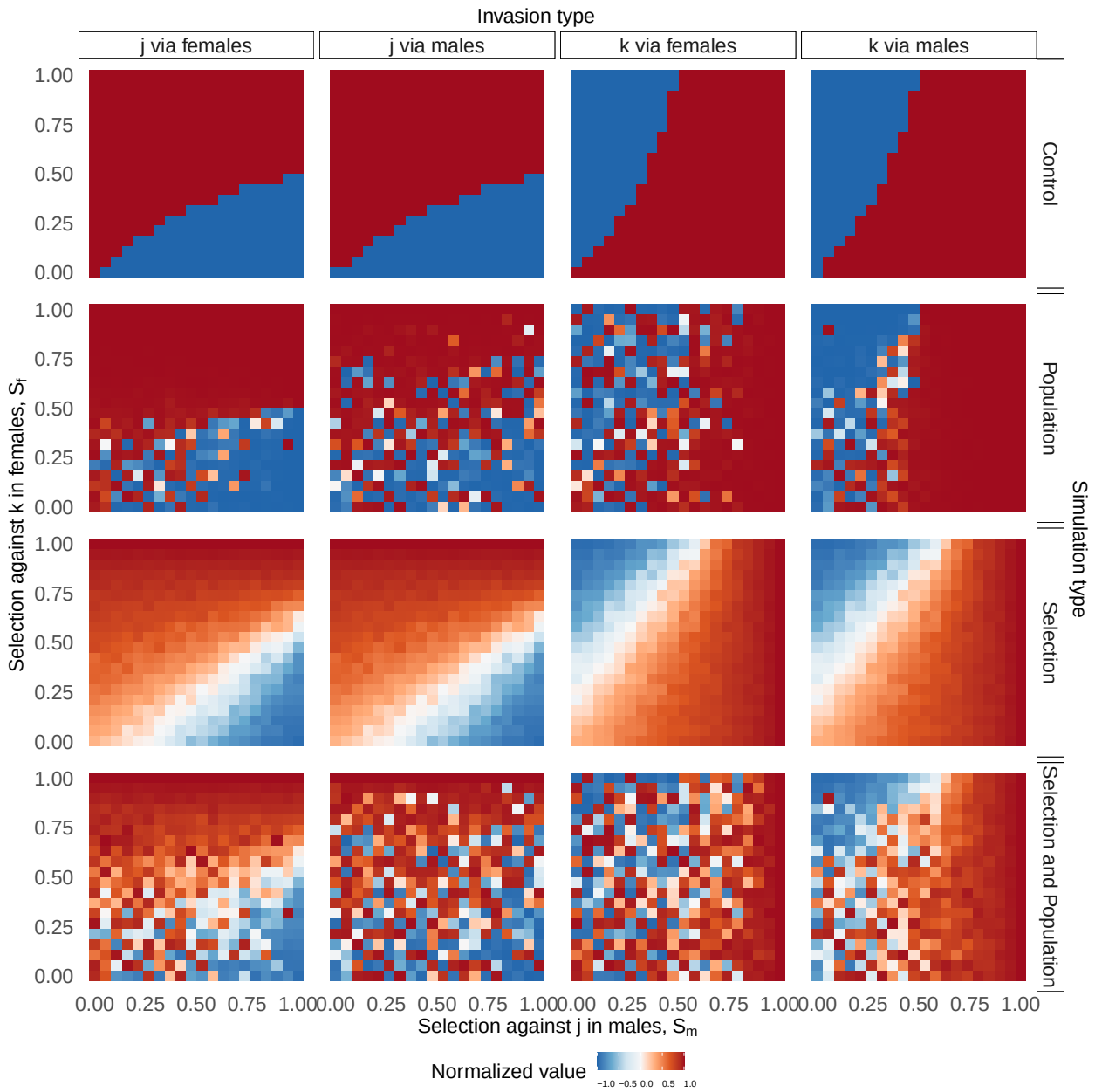

Figure 5: The normalized value of δ^0 in the selection parameter space. Each row in this figure corresponds to one replicate of a different type of simulation, while each column corresponds to a different type of invasion. In the Control simulation ($\sigma_g = 0.001$, $\rho_g = 0$, $\sigma_w = 0.001$, $\rho_w = 0$), δ^0 had a normalized value of 1 in parts of the parameter space where selection allows each allele to be fixed in a population, and a value of -1 where each allele can not be mantained in a population. The Population simulation corresponds to a replicate of a simulation where only population sizes fluctuated, without correlation between fluctuations ($\sigma_g = 70$, $\rho_g = 0$, $\sigma_w = 0.001$, $\rho_w = 0$). The Selection simulation corresponds to a replicate of a simulation where only selection fluctuated, without correlations betweeen fluctuations ($\sigma_g = 0.001$, $\rho_g = 0$, $\sigma_w = 0.9$, $\rho_w = 0$). Finally, the Selection Population simulation corresponds to a replicate of a simulation where both selection and population sizes fluctuated, without correlations between fluctuations ($\sigma_g = 70$, $\rho_g = 0$, $\sigma_w = 0.9$, $\rho_w = 0$).

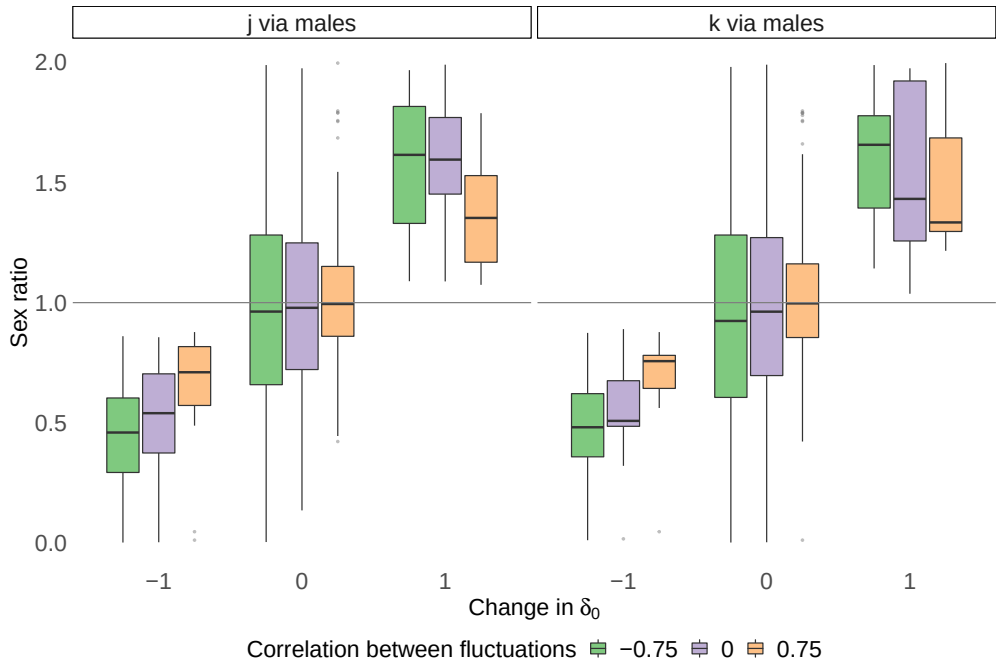

Figure 6: This is a caption

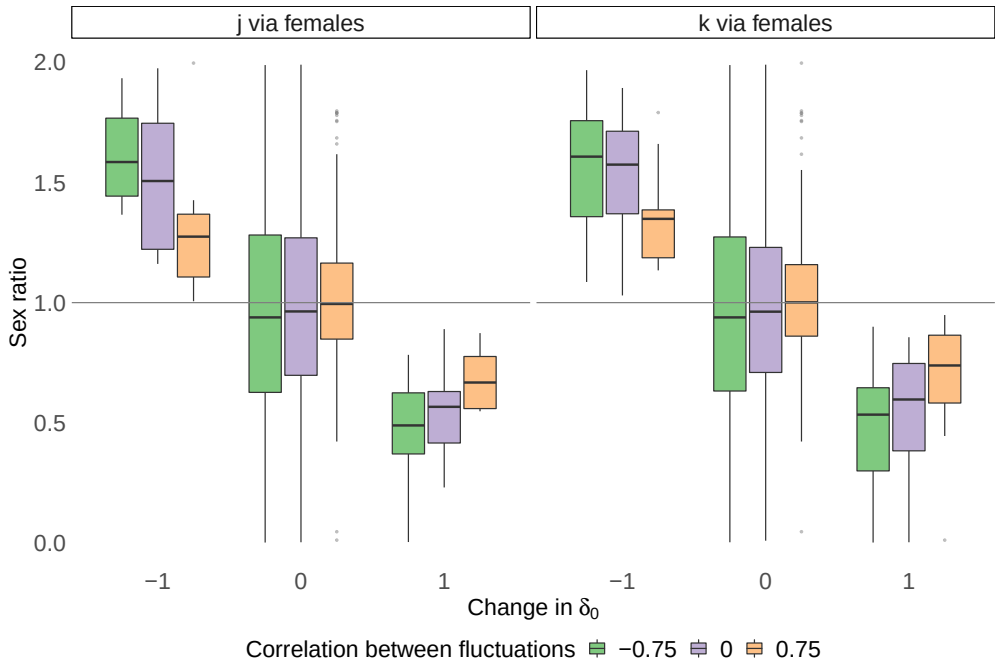

Figure 7: This is a caption

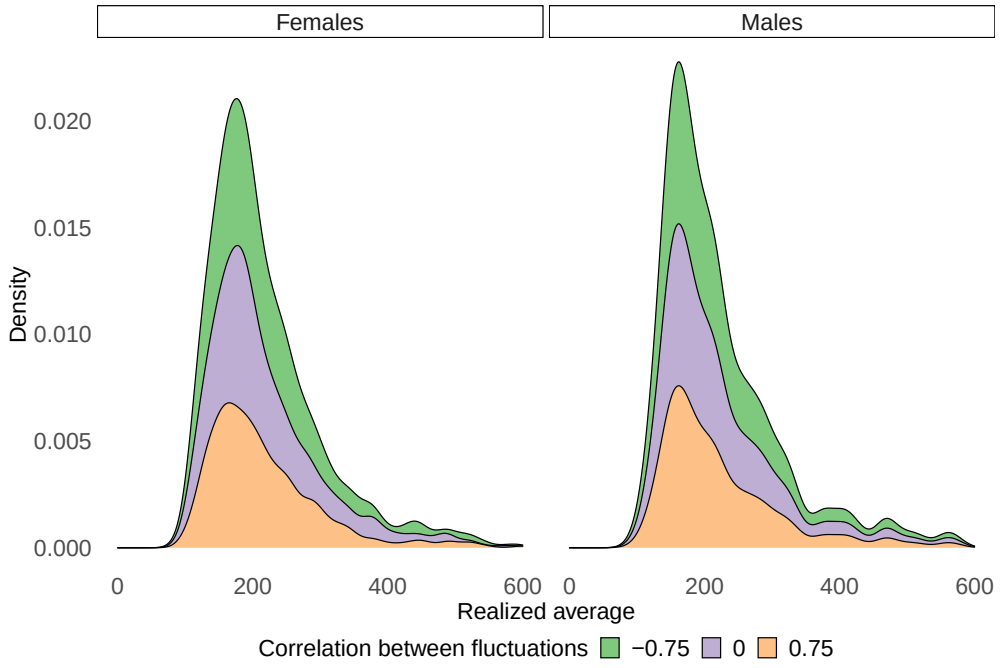

Figure 8: This is a caption

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