Quantifying the relative contributions of environmental fluctuations to the maintenance of a sexually antagonistic polymorphism

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1 Abstract

Sexually antagonistic selection occurs when the selection on the traits or loci differs between the sexes. Sexually antagonistic selection can maintain disadvantageous alleles in a population, which underpins its importance in the maintenance of genetic diversity in populations with separate sexes. Importantly, theoretical studies have shown that environmental fluctuations can significantly increase the expected amount of genetic diversity in a population. Nonetheless, the mechanisms by which environmental fluctuations help maintain polymorphism in a population that experiences sexually antagonistic selection remain unknown. Thus, in this study, we explicitly quantified how temporal fluctuations in population sizes and selection contribute to the maintenance of polymorphism of sexually antagonistic alleles. We do so by adopting an ecological framework that allows the quantification of the relative contributions of environmental fluctuations to species growth rates when rare using simulations. We performed simulations of alleles invading a population while allowing selection and populations sizes to fluctuate over time and quantifying the relative contribution of each type of fluctuation to each alleles' growth rate when rare. Our results showed that both fluctuations in population sizes and selection substantially increased the expected genetic variation under sexually antagonistic selection. Furthermore, our results showed that sexual antagonism creates opportunities for alleles to differentiate in their responses to fluctuations, which promotes the maintenance of polymorphism in analogous ways to the coexistence of species. Our study highlights the importance of quantifying the mechanisms that promote the maintenance

of genetic diversity to understand what environmental drivers play a causal role in explaining levels of diversity that exceed classical theoretical expectations.

2 Introduction

The question of how genetic variation is maintained despite the effects of selection and drift is central within evolutionary biology (Walsh & Lynch, 2018). Classical explanations include overdominance (heterozygote advantage) or frequency-dependent selection (Hedrick, 2007), 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). In species with separate sexes, balancing selection can arise due to sexually antagonistic selection (Connallon & Clark, 2014), 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 maintain genetic variation in a population (Chippindale *et al.*, 2001; Gavrilets, 2014), which in turn can result in phenotypically distinct sexes that express different morphological, physiological, and behavioral traits (Mori *et al.*, 2017; Connallon & Hall, 2018). Nonetheless, the extent to which sexually antagonistic selection can maintain polymorphism in a population is thought to be limited (Connallon & Clark, 2012; Connallon & Hall, 2018). This is because theoretical studies have found that the necessary parameter conditions that give rise to balancing selection are often highly restrictive (Kidwell *et al.*, 1977; Pamilo, 1979; Hedrick, 1999; Curtsinger *et al.*, 1994; Patten *et al.*, 2010; Jordan & Charlesworth, 2012). Importantly, the effect of sexually antagonistic selection generally has been studied under strong simplifying assumptions such as constant population sizes and homogeneous environments (Kidwell

et al., 1977; Pamilo, 1979; Immler *et al.*, 2012; Jordan & Charlesworth, 2012). Studies that have explored the effect of sexually antagonistic selection with more realistic assumptions, such as temporal fluctuations in selection (Connallon *et al.*, 2018) or demographic fluctuations (Connallon & Clark, 2012) have found that polymorphism can be maintained in a much wider set of conditions than classical studies predict. These results suggest that environmental fluctuations are essential to fully understand the effects of sexually antagonistic selection.

The contribution of environmental fluctuations to genetic diversity remains a debated issue in evolutionary biology. Classic theoretical models predict that temporal fluctuations in environmental conditions are unlikely to maintain a genetic polymorphism in haploid populations (Dempster, 1955; Hedrick, 1974; 1986). However, other studies have found that fluctuating selection can maintain genetic variance when populations experience density dependence (Dean, 2005), overlapping of generations (Ellner & Hairston Jr, 1994; Ellner & Sasaki, 1996), or when selection occurs on sex-linked traits (Reinhold, 2000). Similarly, temporal changes in population sizes have been shown to aid in the maintenance of genetic variance (Whitlock, 1992) and to mitigate the effect of genetic drift (Pemberton *et al.*, 1996; Nunney, 2002).

The ways by which environmental fluctuations promote diversity maintenance have been thoroughly studied in ecological contexts (Levins, 1979; Armstrong & McGehee, 1980; Chesson, 2000a; Barabás *et al.*, 2018). From an ecological perspective, polymorphism of sexually antagonistic alleles is equivalent to the coexistence of species, and the fixation of one allele in a population is equivalent to competitive exclusion. Allelic polymorphism

can thus be examined through the same lens as the coexistence of competing species (Ellner & Hairston Jr, 1994; Ellner & Sasaki, 1996; Dean, 2005; Schreiber, 2010). A benefit of analyzing evolutionary dynamics through this lens is that the main theoretical framework used to examine how competing species coexist, Modern Coexistence Theory (Chesson, 2000b; Barabás *et al.*, 2018), allows the explicit quantification of how environmental fluctuations contribute to coexistence. This ecological framework posits that coexistence is promoted by processes that give any species, when rare, an advantage over the existing species in a community (Chesson, 1994; 2000b). The ways upon which fluctuations promote coexistence can be broadly categorized into two mechanisms: *relative non linearity* and *the storage effect*. Relative non linearity arises from competitors responding differently to limiting competitive factors (Chesson, 2000a; Ellner *et al.*, 2016; Zepeda & Martorell, 2019), while the storage effect 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 (Chesson, 2000b; Ellner *et al.*, 2016; Barabás *et al.*, 2018; Schreiber, 2021). Although an exact correspondence to MCT is probably unattainable, there is no study, to our knowledge, that directly quantifies the contributions of environmental fluctuations to the maintenance of a sexually antagonistic polymorphism using this framework.

The use of Modern Coexistence Theory historically required complex mathematical analysis of the models describing the systems dynamics and restrictive assumptions (Barabás *et al.*, 2018); however, recent computational approaches allow the quantification of the relative importance of environmental fluctuations to coexistence using simulations (Ellner

et al., 2016; 2019; Shoemaker *et al.*, 2020). Here, we seek to explicitly quantify how temporal environmental fluctuations contribute to the maintenance of polymorphism under sexually antagonistic selection by applying recent advances in Modern Coexistence Theory. We examined how fluctuations in selection, fluctuations in population sizes, and their interactions can further or hinder the maintenance of polymorphism. In particular, we examined i) Can fluctuations in population sizes and selection allow sexually antagonistic alleles to coexist when differences in their fitness would typically not allow them to? and ii) What are the relative contributions of different types of fluctuations that allow two sexually antagonistic alleles to be maintained in a population? Our study provides the tools to analyze sexual antagonism 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. We then show how we simulated different scenarios of alleles invading a population, where we allowed population sizes, selection, both, or neither to vary. Finally, we detail how we examined the relative contribution of each type of fluctuation to the maintenance or loss of polymorphism.

Population dynamics of sexually antagonistic alleles

Our model examined evolution at a single, biallelic locus. We further assumed the relative fitness of each allele was frequency and density independent. We examined the dynamics of two sexually antagonistic alleles, j and k, that affect fitness in the haploid state. The frequencies of each allele in each sex at the beginning of a life-cycle at generation t are 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 $N_{f,t}$ are the total numbers of males and females in the population at generation t, respectively, while $n_{jf,t}$ 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. Since the loucus is biallelic, the number of males with allele k at generation t is given by $n_{km,t} = N_{m,t} - n_{jm,t}$ and the number of females with allele k by $n_{kf,t} = N_{f,t} - n_{jf,t}$.

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:

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

which upon rearranging and simplifying gives:

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

To illustrate how allele frequencies change through time we use allele j as an example. However, an equivalent expression for allele k can be obtained by interchanging k subscripts for j in Eqn. 5. Selection acts upon these offspring in order to determine the allelic frequencies in females and males in the generation $t + 1$. As an example, the frequency of females with allele j after selection is given by:

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

Changes in alleles frequencies can also be expressed in terms of growth rates. The logarithmic per capita growth rate of allele j in females is therefore given by the number of females carrying 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 logarithmic per capita growth rate of allele j in males m can be obtained by exchanging f for m across the various subscripts in Eqn. 7. Polymorphism in a sexual population, however, is ultimately influenced by growth and establishment of an allele across both sexes. Therefore, the growth rate of allele j across the entire

population of females *and* males is given by:

$$r_{j,t} = \ln\left(\frac{n_{jf,t+1} + n_{jm,t+1}}{n_{jf,t} + n_{jf,t}}\right) \tag{9}$$

An equivalent expression describes $r_{k,t}$, the growth rate of allele k.

Our model further 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 strength of selection against allele j in males is therefore $S_m = 1 - w_{jm}$, and the strength of selection against allele k in females is $S_f = 1 - w_{kf}$. When population sizes and selection are constant, 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; Patten *et al.*, 2010; Connallon *et al.*, 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, polymorphism is maintained in $\approx$ 38% of the parameter space (Kidwell *et al.*, 1977; Pamilo, 1979; Connallon *et al.*, 2018). Nonetheless, it is unrealistic to assume population sizes and selection are constant through time. Temporal changes in population densities are ubiquitous in nature (Whitlock, 1992; Connallon & Clark, 2012; Reinhold, 2000). Similarly, the

effect of sexual selection has been shown to vary through space and time (Kasumovic *et al.*, 2008). If fluctuations in population sizes or selection values affect the coexistence of sexually antagonistic alleles, it should be reflected in increases or decreases of the proportion of the parameter space across which polymorphism is maintained.

Simulations

We examined the effect of fluctuating population sizes and selection in the maintenance of a genetic polymorphism across the selection parameter space ($0 < S_m, S_f < 1$). To do so, we partitioned the parameter space into a 50×50 element grid, which yielded 2500 different pairwise combinations of w_{jm} and w_{kf} values. Henceforth, we will referer to the set of combinations as a grid, which was our unit of replication. For each pairwise combination of w_{jm} and w_{kf} (i.e., element of the grid), as we detail in the next sections, our simulation approach consisted of three main parts. First, we incorporated fluctuations in population sizes and selection into our population dynamics model. Second, we performed simulations to evaluate if both alleles could establish in a population when the environment fluctuated. Finally, we determined the relative contribution of each type of fluctuation to the establishment of each allele.

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

(Table 1). As a control simulation, we set $\sigma_w = 0$ and $\sigma_g = 0$, with no correlation between fluctuations. We ran ten replicates per parameter combination, which resulted in 3780 grids.

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 generations made up of correlated fluctuations of w_{jm} and w_{kf}. We controlled the size of fluctuations in selection (σ_w) and correlation between sexes (ρ_w) by using 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 then, performed a Cholesky decomposition of Eqn. 11 and multiplied it by a (2×500) matrix of random numbers from a normal distribution, which yielded $\gamma_{j,t}$ and $\gamma_{k,t}$. Since fitness values are bounded from zero to one, we added fluctuations in a logistic space. Therefore, we transformed fitness values as $w'_{jm} = \ln \frac{w_{jm}}{1-w_{jm}}$ and $w'_{kf} = \ln \frac{w_{kf}}{1-w_{kf}}$. Finally, we calculated the fitness values at generation t as:

$$w_{jm,t} = \frac{e^{-(w'_{jm}+\gamma_{j,t})}}{(1+e^{-(w'_{jm}+\gamma_{j,t})})^2} \tag{12}$$

$$w_{kf,t} = \frac{e^{-(w'_{kf}+\gamma_{k,t})}}{(1+e^{-(w'_{kf}+\gamma_{k,t})})^2} \tag{13}$$

This approach guaranteed that fluctuations in w_{jm} and w_{kf} were always bounded from zero to one.

Similarly, we generated an independent timeseries of 500 generations made up of correlated fluctuations in population sizes. We again used a Cholesky factorization of the variance-covariance matrix, to control the 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 unit normal random variables, which yielded $\gamma_{m,t}$ and $\gamma_{f,t}$. Finally, we calculated the number of males and females in the population at generation t as $N_{m,t} = N_{m,0} + \gamma_{m,t}$ and $N_{f,t} = N_{f,0} + \gamma_{f,t}$. Therefore, the population sizes in each generation differed from the initial value on the order of σ_g. To avoid extinction due to fluctuations in population sizes, we imposed a lower bound on the population sizes of both sexes of one individual. Note that the scales of σ_g and σ_w are different from each other. While σ_w controls the change in fitness values in logistic space, σ_g controls the number of individuals added or removed to a population.

Finally, we performed simulations where our population dynamics model (Eqns. 1 to 9) was iterated over 500 generations while allowing selection values and population sizes to fluctuate in each generation. We started each simulation with the initial values

of $N_{m,0} = 200$ and $N_{f,0} = 200$ and equal frequencies of allele j and allele k in each sex. For each generation t in our simulations, the values of $w_{jm,t}$ $w_{kf,t}$, $N_{m,t}$ and $N_{f,t}$ used to calculate allele's frequencies in generation t (e.g., Eqn. 7), corresponded to the t values calculated in each timeseries, as described previously. This approach yielded a final time-series that captured the dynamics of sexually antagonistic alleles with fluctuating values of selection and population sizes.

Invasion simulations

To evaluate if both alleles could establish when the environment fluctuated, we turned towards criteria from Modern Coexistence Theory criteria to evaluate coexistence. 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 abundance 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., in a population with only k alleles, if a random mutation leads to one individual carrying 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 allele's *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 with environmental fluctuations as a template to perform invasion simulations of both alleles. Following the approach of Shoemaker *et al.* (2020), we treated each invasion simulation independently, and hence we performed 500 invasion simulations, one for each generation in our timeseries. We explored all four potential combinations of each allele invading through each pathway (e.g., allele j invading through males, allele k invading through females, and so on). To simulate invasion, we set the density of the invading allele to one individual. Since we treated each invasion simulation as independent, we denoted the initial 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 $n_{jf,i} = 0$. 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 $n_{kf,i} = N_{f,t}$. We then simulated invasion by iterating our population dynamics model one generation, $i + 1$, and calculated the logarithmic growth rate of the invading allele, which in this case would be given by:

$$r_{j,i} = \ln\left(\frac{n_{jm,i+1} + n_{jf,i+1}}{1}\right) \tag{14}$$

Similarly, the logarithmic growth rate of the resident allele would be given by:

$$r_{k,i} = \ln\left(\frac{n_{km,i+1} + n_{kf,i+1}}{n_{km,i} + n_{kf,i}}\right) \tag{15}$$

We performed invasion simulations for each allele invading via each potential pathway. We then calculated the mean invasion growth rate of each allele as an invader as the

average of the 500 invasion growth rates. We also calculated the mean growth rate of each allele as a resident as the average of the 500 resident growth rates. We determined alleles could coexist and therefore polymorphism to be maintained in a point in the parameter space 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 decomposition

Our invasion simulations evaluated whether or not polymorphism can be maintained in a determined point of the selection parameter space when the environment fluctuates. However, we also quantified the relative contributions of fluctuations in selection and population sizes to the predicted coexistence outcome using a *functional decomposition* approach (Ellner *et al.*, 2016; 2019; Shoemaker *et al.*, 2020).

The functional decomposition approach separates 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, a set of two-way interaction terms representing the effect of variables fluctuating simultaneously, and so on (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 a simplified example, if only N_m and N_f were fluctuating, the growth rate of allele j when it is the invader (Eqn. 14) at invasion time i could be decomposed into:

$$r_{j,i}(N_m, N_f) = \mathcal{E}_j^0 + \mathcal{E}_j^{N_m} + \mathcal{E}_j^{N_f} + \mathcal{E}_j^{N_m N_f} \tag{16}$$

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 to their average values. For example, the term $\mathcal{E}_j^{N_m}$ expresses the contribution of fluctuations in N_m when N_f is set to its average, without the contribution when both variables are set to their averages :

$$\mathcal{E}_j^{N_m} = r_{j,i}(N_m, \overline{N_f}) - \mathcal{E}_j^0 \tag{17}$$

If we average Eqn. 16 across generations, we get a partition of the average population growth rate into the variation 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:

$$\bar{r}_j = \mathcal{E}_j^0 + \overline{\mathcal{E}}_j^{N_m} + \overline{\mathcal{E}}_j^{N_f} + \overline{\mathcal{E}}_j^{N_m N_f} \tag{18}$$

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. For simplicity we only show terms related to allele j as an invader, however, the functional decomposition approach can be applied analogously when allele k invades. Note that Table 2 does not include three or four-way interactions (e.g., $\overline{\mathcal{E}}_j^{N_m N_f w_{jm} w_{fk}}$). 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 controlling which variables were allowed to fluctuate. For example, to calculate the value of $\mathcal{E}_j^0$ we performed another 500 simulations of allele j invading but instead of using the values of $w_{jm,i}$ $w_{kf,i}$, $N_{m,i}$ and $N_{f,i}$ used to calculate the frequency of allele j in generation $i+1$, we set the all the variables to their mean values. Then, to calculate the value of $\mathcal{E}_j^{N_m}$, we set all variables except N_m to their mean values and subtracted the value of $\mathcal{E}_j^0$, and so on with subsequent terms.

The functional decomposition approach further allows 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 the maintenance of polymorphism by helping whichever allele is rare, or by hurting whichever allele is common. Therefore, to understand the role of each type of fluctuation, it is necessary to compare how it affects both invader *and* resident growth rates. In the example presented in Eqn. 18, if allele j is invading, then allele k is at it's resident state and there exists an analogous decomposition of $\overline{r}_k$. Therefore we can express the difference between contributions of fluctuations in N_m as:

$$\Delta_j^{N_m} = \overline{\mathcal{E}_j}^{N_m} - \overline{\mathcal{E}_k}^{N_m} \tag{19}$$

If $\Delta_j^{N_m}$ is positive, then fluctuations in the male population size benefit allele j when it is rare more than 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. Therefore, for each allele invading via a different pathway, we calculated 7 separate Δ values, one for each one of the $\mathcal{E}$ terms in Table 2.

In the course of our analysis we noticed that the magnitude of each one of the Δ values could vary considerably across the parameter space. To make them comparable and ease interpretation, 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. 19 would be given by:

$$\delta_j^{N_m} = \frac{\Delta_j^{N_m}}{\sqrt{\sum_{d=1}^{7} (\boldsymbol{\Delta_d})^2}} \tag{20}$$

This normalization bounded δ values from -1 to 1. Similar to the interpretation of Δ terms, positive δ values mean that fluctuations benefit an allele as an invader more than the other allele as a resident and negative δ values imply that fluctuations do not benefit an allele as an invader more than the other allele as a resident.

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 proportion of the parameter space where polymorphism was maintained increased with the effect size of both types of fluctuations (Fig. 1). Increases in the proportion of polymorphism were more likely when fluctuations in selection and population sizes were large, fluctuations in population sizes were negatively correlated, and fluctuations in selection were positively correlated. Importantly, our results show that environmental fluctuations can increase the proportion of allelic polymorphism up to 0.6 (Fig. 1).

Our results matched previous findings that without fluctuations, polymorphism can be maintained in only 0.38 of the parameter space (Fig. 2A). Increments in polymorphism when population sizes fluctuated occurred near the limit of the domain of balancing selection and were particularly pronounced when selection against both alleles was weak (Fig. 2B). When selection against either of the alleles was strong ($S_m, S_f > 0.75$), fluctuations in population sizes did not increase polymorphism compared to the control (Fig. 2B). Similarly, increments in polymorphism when selection fluctuated also occurred near the limit of the domain of balancing selection, however, fluctuations in selection did not affect polymorphism when selection against both alleles was weak ($S_m, S_f < 0.25$) (Fig. 2C). When both population sizes and selection fluctuated, increments in polymorphism occurred near the limit of the domain of balancing selection, regardless of the strength of selection.

The effect of fluctuations in population sizes and selection was not homogeneous across the parameter space. The values of δ^0 followed the effect of selection outlined by Eqn. 10 (Fig. 3). Note that near the limit of the domain of selection, the effect of δ^0 was close to zero. In contrast, the rest of the δ values were generally stronger in magnitude near the limit of the domain of selection (Fig. 3). Despite their similar patterns in the parameter space, the role of each type of fluctuation to the growth rate of alleles when rare depended on the allele and pathway where the invasion took place (Fig. 3).

Fluctuations in population sizes of males and females benefited alleles when alleles invaded via the fluctuating population (Fig. 4). In contrast, fluctuations in the population of one sex made it more difficult for either allele to invade via the other sex (Fig. 4).

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. 4). The relative contribution of both populations fluctuating, $\delta^{N_m N_f}$, was positive when fluctuations were negatively correlated, had a negligible effect when fluctuations were not correlated, and had a negative effect when fluctuations were positively correlated (Fig. 4).

In contrast, the relative contribution of fluctuations in selection depended on the allele where invasion occurred, regardless of the invasion pathway (Fig. 5). For example, $\delta^{w_{jm}}$ which captured the relative contribution of fluctuations in selection against j in males, was always positive when allele k invaded but had negligible effects when allele j invaded (Fig. 5). The relative contribution of fluctuations of both types of selection was negative when fluctuations were negatively correlated, had a negligible effect when fluctuations were not correlated, and had a positive effect when fluctuations were positively correlated (Fig. 5).

5 Discussion

The results of our study provide supporting evidence that environmental fluctuations can substantially increase the expected genetic variance maintained under sexually antagonistic selection (Fig. 1). Perhaps more importantly, our study quantifies exactly how environmental fluctuations help maintain polymorphism. 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). Indeed,

as much as 20% of traits for which data are 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 polymorphism to be maintained in a population in a few scenarios) (Kidwell *et al.*, 1977; Pamilo, 1979; Hedrick, 1999; Curtsinger *et al.*, 1994; Patten *et al.*, 2010). In contrast, our study shows that when we incorporated more realistic assumptions, a sexually antagonistic polymorphism is more likely than not to be maintained in a population (Fig. 1).

The relative contribution of fluctuations in selection

Our simulations indicate that fluctuations in the strength selection can promote allelic coexistence in parts of the parameter space where we would otherwise expect selection to fix one of the alleles (Fig. 2). The effect of fluctuations in selection was generally greater in magnitude near the limit of the domain of selection and where selection against alleles was strong (Fig. 3). In contrast, fluctuations in selection had a minor effect when both alleles had similar fitness, suggesting that fluctuations in selection become advantageous when there exist greater fitness differences between sexually antagonistic alleles (Fig. 3). The effect of fluctuations in selection depended on the identity of the invading allele, as fluctuations in selection contributed positively to the invasion growth rate of the allele that was not directly affected by fluctuations. Fluctuations in selection, however, had a negligible effect in the invasion of the allele which was directly affected by fluctuations (Fig. 5).

The mechanism by which fluctuations in selection promoted coexistence can be under-

stood as *relative non-linearity in response to selection*. 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 parts of the parameter space where we would expect selection to fix the allele with higher fitness, the allele with lower fitness can be maintained in a population if the fitter allele experiences high fluctuations in selection (Fig. 5). 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 allele with lower fitness is the one associated with higher fluctuations in selection, then fluctuations are not likely to promote the maintenance of both alleles in a population (Fig. 5).

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, and it contributed negatively to each allele's growth rate if fluctuations were negatively correlated (Fig. 5). Environmental fluctuations are often correlated (Steele, 1985), and previous studies have shown that positively correlated environmental 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*. The storage effect typically 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). Although a complete correspondence to Modern Coexistence Theory is difficult to obtain, $\delta^{w_{jm},w_{kf}}$ can be understood as a storage effect since it is the contribution to an invasion growth rate of covariance between the environment and competitive factors (Ellner *et al.*, 2016). For an allele invading a population, fluctuations in its fitness can be equivalent to a species experimenting environmental fluctuations, while fluctuations in the fitness of the opposite allele could be understood as fluctuations in competitive factors. Our results show that 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 (Dempster, 1955; 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 heterozygotic 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 fluctuations promote the maintenance of polymorphism when there exist life-history traits that promote local adaptation. Our results provide further evidence that fluctua-

tions 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). The effect of fluctuations in population sizes was generally greater in magnitude near the limit of the domain of selection where both alleles had similar fitness values and had a weaker effect as differences in fitness were larger (Fig. 3). This suggests that fluctuations in population sizes will likely play a smaller role in the maintenance of polymorphism in populations where sexual antagonism is strong. 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 alleles that invaded via the fluctuating population (Fig. 4). If an allele invaded via the non-fluctuating sex, however, fluctuations contributed negatively to its invasion growth rate and thus hampered the maintenance of polymorphism (Fig. 4).

Our results suggest that in parts of the parameter space where we would expect selection to fix the allele with higher fitness, the allele with lower fitness could achieve 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 respond differently to fluctuations, and thus, promotes the maintenance of polymorphism. 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 maintenance of genetic diversity, while positively correlated fluctuations will likely impair it (Fig. 4). 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).

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 a decrease in the overall fitness of a population (Connallon & Hall, 2018). Sexually antagonistic selection necessarily involves a mismatch between the traits a population expresses 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 alleles with lower fitness 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 multiple alleles in a population, even when selection against some of those alleles is strong (Fig. 2A). Thus, the maintenance of genetic diversity promoted by fluctuations 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 the maintenance of genetic diversity of sexually antagonistic alleles without the need for local adaptations or life-history stages that involve overlapping generations. The environmental drivers that maintain sexually antagonistic traits are still poorly understood (Connallon & Hall, 2018), and it is essential that we understand how diversity might respond to rapid environmental change (Tylianakis *et al.*, 2008).

Figures and tables

Table 1: Parameters used in our simulations to control the effect sizes of fluctuations in population sizes (σ_g) and selection values (σ_w) and their respective correlations (ρ_g and ρ_w). We ran ten replicates for each 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 defined in Eqn. (18), the partition of the average population growth rate is made up of the variation free growth rate ($\mathcal{E}_j^0$), main effects of each variable fluctuating, and the interaction between fluctuations in correlated variables.

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 new 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}

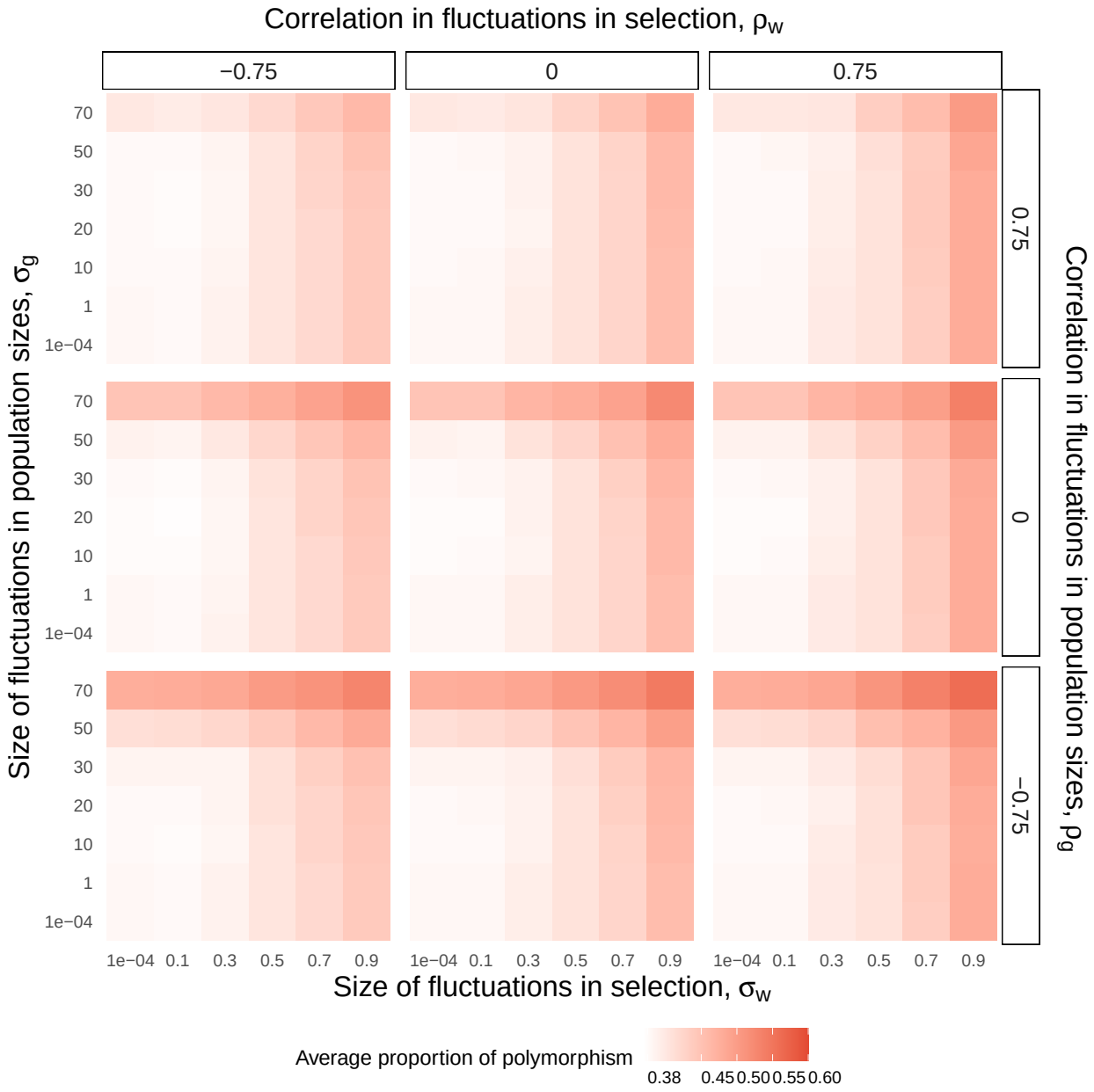

Figure 1: The average proportion of polymorphism maintained in the selection parameter space. For all parameter combinations in our simulations, we show the average proportion of polymorphism in our simulation grids, for all replicates and invasion scenarios (each allele invading a different sex). Each panel corresponds to a different combination of correlations between fluctuations and rows and columns within a pannel show the size of fluctuations in population sizes and in selection, respectively. 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 polymorphism (0.38) as white in our color scheme.

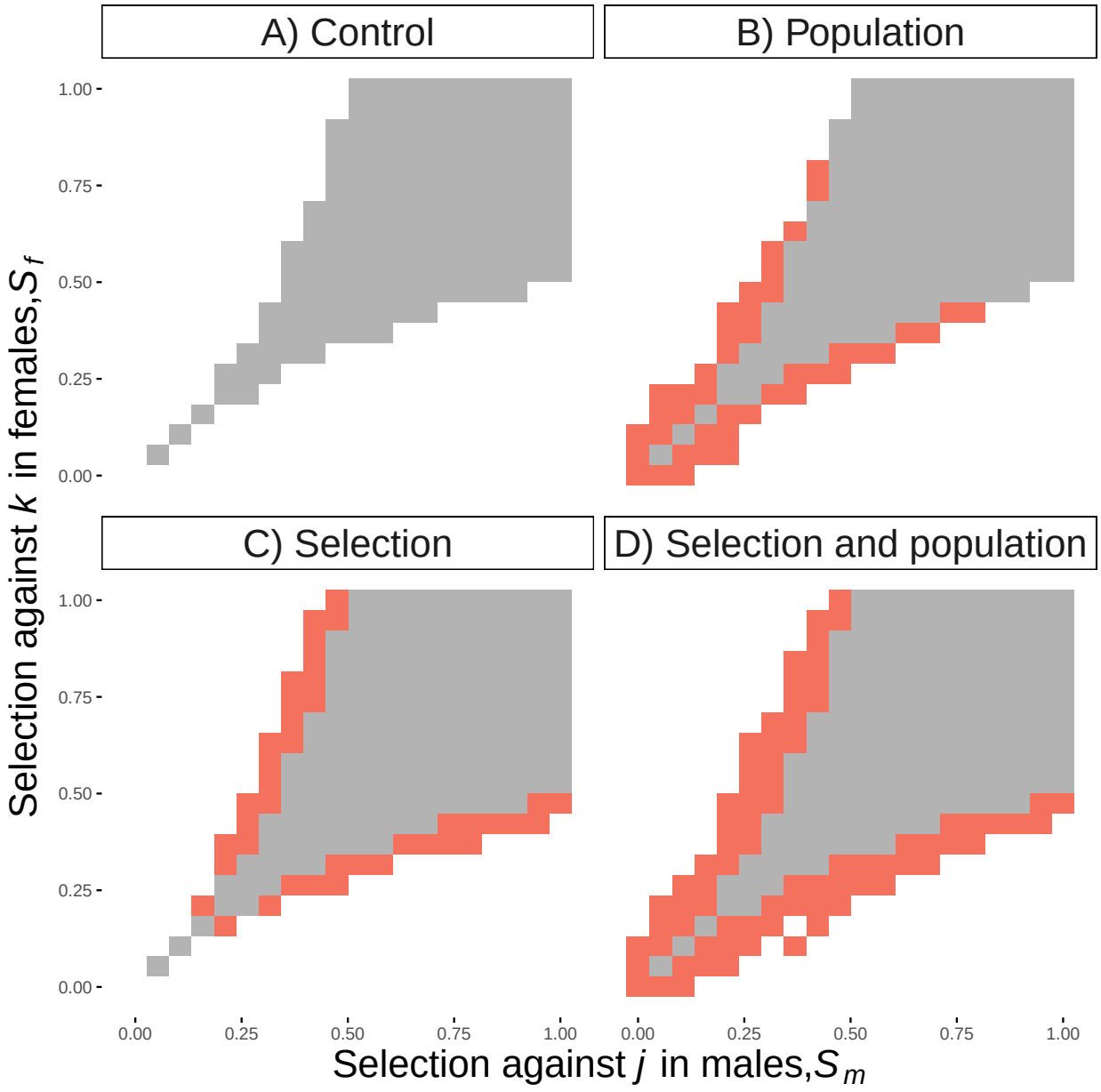

Figure 2: Polymorphism in the parameter space. We show the outcomes of our invasion simulations when j invaded via males and k invaded via females. As a reference, j is favored in females and k is favored in males. Each panel corresponds to a different replicate of our simulation grids. Grey areas indicate parts of the selection parameter space where polymorphism can be maintained without fluctuations, while white areas indicate parts of the parameter space that correspond to the fixation of one of the alleles (following Eqn.10). Red areas indicate parts of the parameter space where polymorphism can be maintained when fluctuations were incorporated. In A) we show the outcomes of our simulations in the control grid ($\sigma_g = 0.0001$, $\rho_g = 0$, $\sigma_w = 0.0001$, $\rho_w = 0$). In the B) we show the outcomes when we incorporated high fluctuations in population sizes that were negatively correlated ($\sigma_g = 70$, $\rho_g = -0.75$, $\sigma_w = 0.001$, $\rho_w = 0$). In C) we show the outcomes when we incorporated fluctuations in selection that were positively correlated ($\sigma_g = 0.0001$, $\rho_g = 0$, $\sigma_w = 0.9$, $\rho_w = 0.75$). Finally, in D) we show the outcomes when both population sizes and selection fluctuated ($\sigma_g = 70$, $\rho_g = -0.75$, $\sigma_w = 0.9$, $\rho_w = 0.75$).

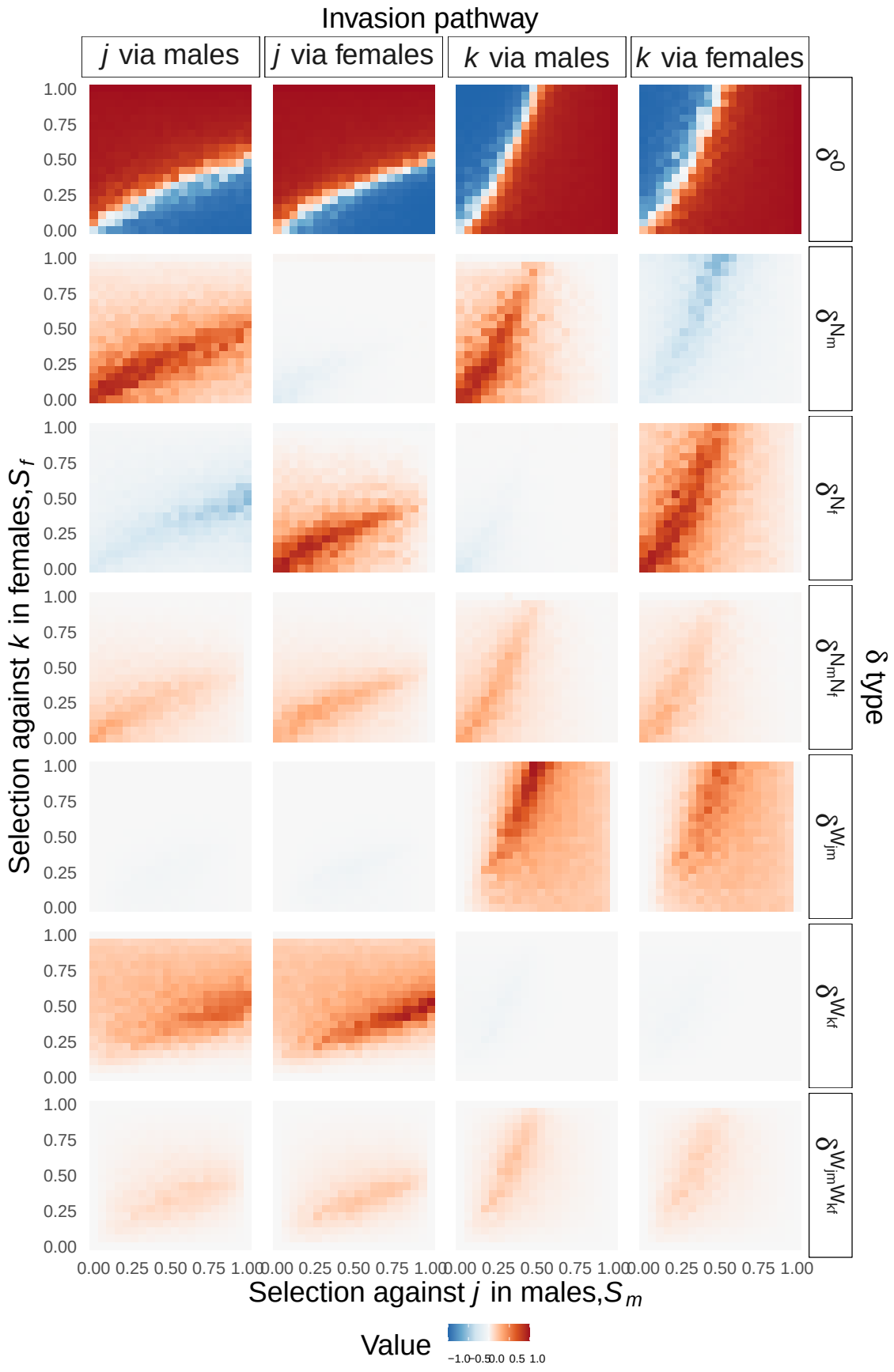

Figure 3: Distribution of δ values across the parameter space. Caption continued in next page.

Figure 3: We show the results of the functional decomposition approach for one replicate of our simulation grids where both population sizes and selection fluctuated with correlated effects ($\sigma_g = 70$, $\rho_g = -0.75$, $\sigma_w = 0.9$, $\rho_w = 0.75$). Each row corresponds to a different type of δ value, as indicated with labels on the right. Each column corresponds to an allele invading a different pathway, as indicated with labels on top. Areas in red correspond to δ values that contributed positively to each allele's invasion growth rate, while blue areas denote points in the parameter space where fluctuations had a negative contribution to invasion growth rates.

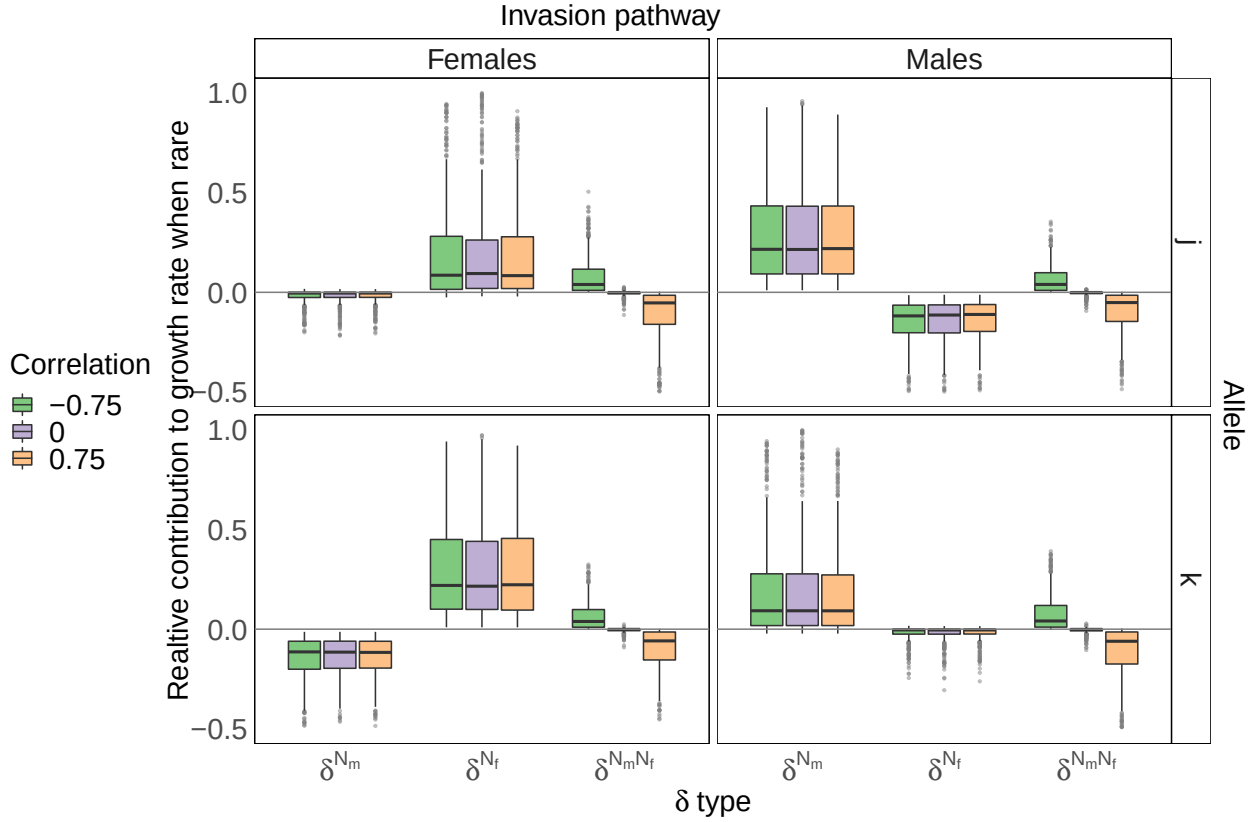

Figure 4: The relative contributions of fluctuations in population sizes to alleles' growth rates when rare. Positive δ values imply that the corresponding fluctuation benefits that allele as an invader more than the other allele as a resident while negative δ values indicate fluctuations benefit the residents more than the invader. Each panel corresponds to the result of simulations where each allele invaded via a different pathway, as indicated by top and right labels. 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. Box plots extend from the first to third quantiles of the corresponding δ values, and the line inside the the box indicates the median. The upper whisker extends to the largest value no further than 1.5 times the inter-quantile range (IQR, or the distance between the first and third quartiles); the lower whisker extends to the smallest value at most 1.5 times the IQR. Data beyond the end of the whiskers are determined to be outliers and are plotted individually with solid grey points.

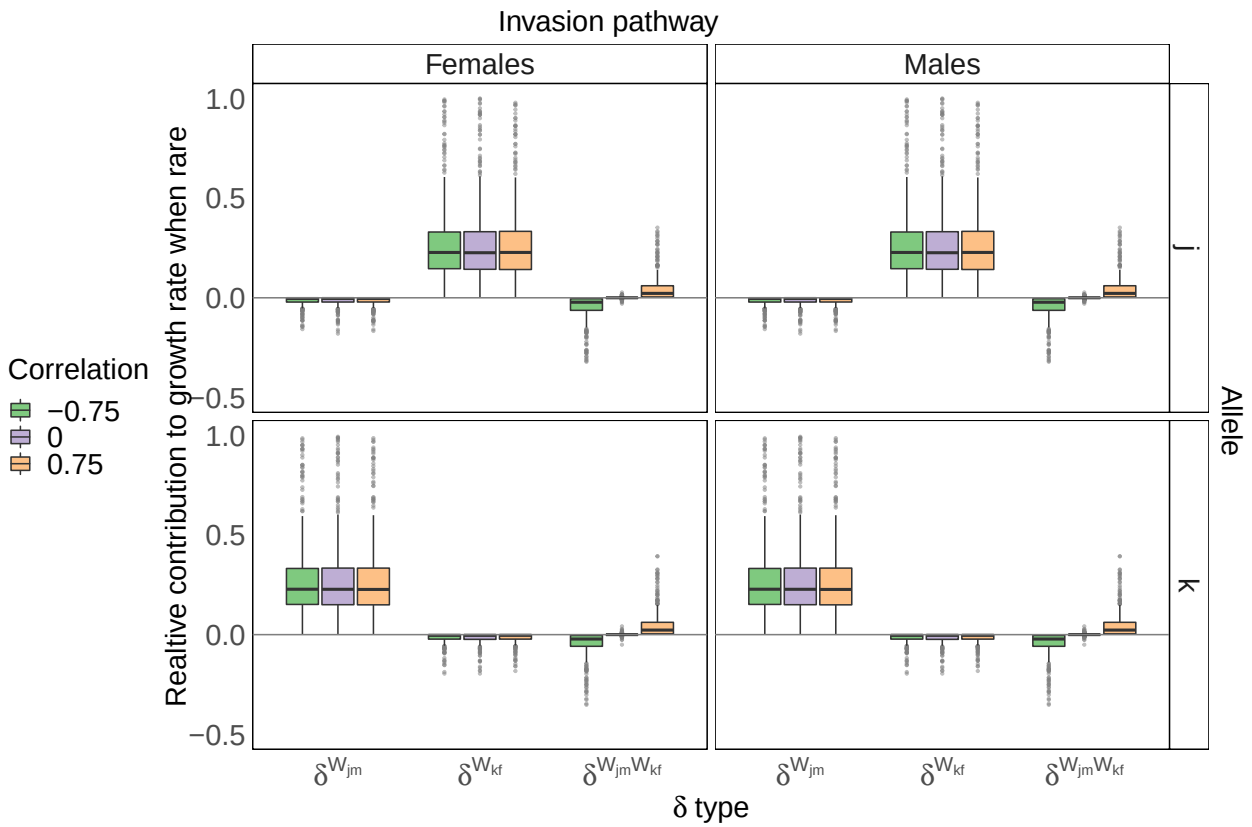

Figure 5: The relative contributions of fluctuations in selection to alleles' growth rates when rare. Positive δ values imply that the corresponding fluctuation benefits that allele as an invader more than the other allele as a resident while negative δ values indicate fluctuations benefit the residents more than the invader. Each panel corresponds to the result of simulations where each allele invaded via a different pathway, as indicated by top and right labels. We show the boxplots of the three distinct δ values that captured the effects of fluctuations in selection, for all of the replicates in our simulation in which $\sigma_w = 0.9$. Each color corresponds to a different correlation between fluctuations in population sizes (ρ_w), as the legend indicates. Box plots extend from the first to third quantiles of the corresponding δ values, and the line inside the the box indicates the median. The upper whisker extends to the largest value no further than 1.5 times the inter-quantile range (IQR, or the distance between the first and third quartiles); the lower whisker extends to the smallest value at most 1.5 times the IQR. Data beyond the end of the whiskers are determined to be outliers and are plotted individually with solid grey points.

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