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Claire Godineau

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# THÈSE POUR OBTENIR LE GRADE DE DOCTEUR DE L'UNIVERSITÉ DE MONTPELLIER

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Unité de recherche 5554 - ISEM

**Etude théorique des réponses évolutives au changement  
climatique : effets de l'homogamie et des fluctuations de  
l'intensité de la sélection**

**Présentée par Claire GODINEAU**

**Le 25 novembre 2021**

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# Résumé

Les précédentes études théoriques de l'adaptation de populations à un changement environnemental ont identifié la variance génétique comme un élément clef. Le changement climatique contemporain a ravivé l'intérêt de ces études qui tentent maintenant d'intégrer la complexité du vivant et du changement climatique. Des évolutions rapides des dates de floraison ont été documentées chez plusieurs espèces en lien avec le changement climatique. L'appariement entre des individus qui se ressemblent – homogamie – est fréquent, et obligatoire pour la date de floraison, la sélection naturelle peut être différente sur les fonctions mâles et femelles et l'intensité de la sélection entre années diffère selon la durée des saisons. Ces caractéristiques sont généralement ignorées par la théorie sur l'adaptation à un environnement changeant. Dans un premier chapitre, nous avons évalué si l'homogamie peut être responsable des réponses évolutives rapides des phénologies de floraison au changement climatique. Un modèle individu-centré de génétique quantitative simule un changement climatique et l'évolution des dates moyennes de floraison individuelle dans une population isolée. Dans la plupart des scénarios, malgré ses effets négatifs sur le polymorphisme génétique, l'homogamie maintient plus de variance génétique dans un environnement changeant que la panmixie, et permet ainsi aux populations de mieux suivre le changement climatique et d'avoir une valeur sélective plus grande. Un modèle analytique, basé sur le modèle infinitésimal d'héritabilité des traits, confirme ces résultats. Le deuxième chapitre intègre deux éléments supplémentaires, fréquents dans les populations de plantes et d'animaux : la sélection naturelle sexe-spécifique et le dimorphisme sexuel. Le modèle analytique construit est une extension du précédent, et généralise les résultats en incluant le dimorphisme sexuel, et deux modes courants d'homogamie: la préférence homogame des femelles pour certains phénotypes mâles, et, l'homogamie temporelle pour la date de floraison. Le modèle montre que (i) l'homogamie produit de la sélection sexuelle qui intensifie l'effet de la sélection naturelle sur les femelles et diminue celui de la sélection naturelle sur les mâles; (ii) en présence d'un fort dimorphisme sexuel, cette sélection sexuelle engendre une sélection directionnelle sur les mâles, et peut conduire à une évolution de valeurs de traits en dehors de l'intervalle défini par les optimums mâles et femelles; (iii) dans certaines conditions, la mal-adaptation des femelles peut être plus petite dans un environnement changeant que constant, en homogamie comme en panmixie ; (iv) l'homogamie facilite l'adaptation des femelles à un climat changeant, seulement si la sélection sur les femelles est plus forte que celle sur les mâles, et/ou le dimorphisme sexuel n'est pas trop fort et/ou le changement climatique est rapide. La robustesse de ces résultats a été testée à l'aide d'un modèle individu-centré. Le troisième chapitre étudie les effets des fluctuations de l'intensité de la sélection sur les réponses génétiques à long terme des populations. Nous avons utilisé des approximations analytiques et une exploration numérique du modèle infinitésimal. Les fluctuations de l'intensité de la sélection sont modélisées par des fluctuations de la largeur de la fonction de sélection supposée Gaussienne. Ces fluctuations augmentent l'intensité moyenne de la sélection, et diminuent la variance génétique et le retard adaptatif des populations. Les fluctuations ont un coût démographique et diminuent le taux de croissance à long-terme des populations dans la plupart des scénarios. L'ensemble de ces résultats suggère que (i)

l'homogamie ne facilite les réponses évolutives au changement climatique que dans certaines conditions seulement, (ii) des réponses évolutives rapides ne sont pas nécessairement un gage d'atténuation des conséquences démographiques du changement climatique.

**Mots clés :** modélisation, environnement changeant, adaptation, génétique quantitative, sélection sexuelle, phénologie

# Abstract

Previous theory on adaptation to a changing environment has identified genetic variance as a key factor. Contemporary climate change renews the interest for such studies, which now attempt to include the complexity of life and climate change. Rapid evolution of flowering time has been documented in several species as a response to climate change. Assortative mating, i.e. mating restricted to individuals with similar phenotypes, is frequent and obligate for flowering time, the intensity of natural selection can differ between sexes, and the intensity of stabilizing selection on flowering can vary with the duration of seasons, and which fluctuates across years. These features are however largely ignored by extent theory on adaptation to changing environments. In a first chapter, we have studied the effects of assortative mating for flowering date on evolutionary responses to climate change, with the aim to evaluate whether assortative mating, compared to random mating, can explain fast evolutionary responses of flowering phenology to climate change. To this end, an individual-based quantitative genetics model simulates climate change and the evolution of mean individual flowering date in an isolated population. In most scenarios, and despite its negative effect on genetic polymorphism, assortative mating maintains higher genetic variance at equilibrium than random mating, and therefore allows populations to better track climate change and to have a better fitness. An analytic model, based on the infinitesimal model of trait heritability, confirms those results. The second chapter integrates more elements of realism, common in plant and animal populations: sex-specific natural selection and sexual dimorphism. The analytical model is an extension of the previous one, and generalizes results by including sexual dimorphism, and two frequently observed types of assortative mating in animals and plants: assortative preference of females for male phenotypes, and temporal assortative mating for flowering date. The model shows that (i) assortative mating generates sexual selection, which increases the effect of natural selection on females and decreases the effect of natural selection on males, (ii) when sexual dimorphism is large, assortative mating further generates directional sexual selection on male phenotypes, which can lead to the evolution of trait values overshooting the interval between the male and female optima; (iii) in some conditions, which occur both under random and assortative mating, female maladaptation can be smaller in a changing environment than in a constant environment; (iv) assortative mating can help populations to better track climate change than random mating, when selection on females is stronger than that on males and/or sexual dimorphism is not too large, and/or climate change is fast enough. The robustness of results has been tested with an individual-based model. The third chapter studies the effects of the fluctuations of the strength of selection on the long-term responses of populations. To this end, we have used both analytical approximations and a numerical exploration of the infinitesimal model. Fluctuations in the strength of selection are modeled by fluctuations of the width of the fitness function assumed to be Gaussian. Such fluctuations increase the mean strength of selection and therefore decrease genetic variance and adaptive lag. Fluctuations of the strength of selection however have a demographic cost, and decrease the long-run growth rate of populations in most cases. Taken together, these results suggest that: (i) assortative mating improves adaptation to climate change

only under specific circumstances, (ii) rapid evolutionary responses to climate change do not necessarily mitigate its negative consequences on demography.

**Keywords:** modelling, changing environment, adaptation, quantitative genetics, sexual selection, phenology

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# Contents

<b>1</b>	<b>Introduction</b>	<b>13</b>
1.1	Les réponses au changement climatique . . . . .	14
1.1.1	Migration . . . . .	14
1.1.2	Réponses évolutives et plastiques . . . . .	14
1.1.3	Changements de phénologie . . . . .	15
1.2	Comprendre et prédire les réponses évolutives : apport des modèles de sauvetage évolutif . . . . .	15
1.2.1	Déplacement ponctuel de l'optimum . . . . .	16
1.2.2	Déplacement graduel et constant de l'optimum . . . . .	16
1.2.3	Fluctuations de l'optimum . . . . .	17
1.2.4	Preuves empiriques de sauvetage évolutif . . . . .	18
1.3	Homogamie . . . . .	18
1.3.1	Différents types d'homogamie . . . . .	18
1.3.2	Effets de l'homogamie sur les réponses évolutives . . . . .	19
1.4	Effet de la sélection sexe-spécifique sur la phénologie de floraison . . . . .	20
1.5	Objectifs de la thèse . . . . .	20
1.5.1	Effet de l'homogamie sur les réponses évolutives de la phénologie de floraison au changement environnemental . . . . .	21
1.5.2	Effet combiné de l'homogamie et de la sélection sexe-spécifique sur les réponses évolutives . . . . .	21
1.5.3	Effet des fluctuations de la durée de la saison favorable à la floraison sur les réponses évolutives . . . . .	21
	<b>Bibliographie</b>	<b>23</b>
<b>2</b>	<b>Chapitre 1 : Assortative mating can help adaptation of flowering time to a changing climate: Insights from a polygenic model</b>	<b>29</b>
2.1	Main text . . . . .	29
2.2	Appendix 1 . . . . .	48
<b>3</b>	<b>Chapitre 2 : Combined effects of assortative mating and sex-specific natural selection on adaptation of a sexually dimorphic trait to environmental change</b>	<b>71</b>
3.1	Introduction . . . . .	73
3.1.1	Assortative mating facilitates adaptation in a changing environment in the absence of sex-specific selection . . . . .	73

3.1.2	Sex-specific natural selection under random mating and environmental change . . . . .	73
3.1.3	Sexual selection interacts with natural selection . . . . .	74
3.1.4	Aims of the study . . . . .	74
3.2	Methods . . . . .	75
3.2.1	Analytical model . . . . .	76
3.2.1.1	Mating patterns . . . . .	76
3.2.1.2	Genetic architecture . . . . .	77
3.2.1.3	Sex-specific selection and environmental change . . . . .	77
3.2.1.4	Sex-specific trait expression . . . . .	79
3.2.1.5	Metrics recorded . . . . .	79
3.2.1.5.1	Distribution of breeding values after natural selection and reproduction . . . . .	79
3.2.1.5.2	Mismatch of the population mean breeding value to the female optimum . . . . .	80
3.2.2	Individual-based model . . . . .	80
3.2.2.1	Genetic architecture . . . . .	81
3.2.2.2	Matings . . . . .	81
3.2.2.3	Metrics recorded . . . . .	81
3.2.3	Parameter choice . . . . .	82
3.3	Results . . . . .	82
3.3.1	Mismatch to the female optimum under a constant environment . . . . .	83
3.3.1.1	Sexual selection shifts the evolutionary optimum towards the female optimum . . . . .	83
3.3.1.2	Sex-specific selection shifts the evolutionary optimum . . . . .	85
3.3.1.3	Sex-specific trait expression modifies the effect of sexual selection . . . . .	86
3.3.1.4	Sexual selection modifies the effect of the sex-specific natural selection on the adaptive lag . . . . .	87
3.3.2	Total mismatch to the female optimum . . . . .	89
3.3.2.1	Environmental change can decrease the mismatch to the female optimum . . . . .	89
3.3.2.2	Parameter range for which assortative mating helps to track the female optimum . . . . .	90
3.3.3	Genetic variance . . . . .	92
3.3.3.1	Expected effects on genetic variance . . . . .	92
3.3.3.2	Genetic and genic variance in the simulations . . . . .	93
3.3.3.3	Changes in the genic variance do not affect qualitatively the predictions for the mismatch to the female optimum . . . . .	95
3.4	Discussion . . . . .	95
3.4.1	Main results . . . . .	95
3.4.2	Effects of sex-specific natural selection on maladaptation . . . . .	96

3.4.2.1	Assortative mating decreases the maladaptation under constant environment . . . . .	96
3.4.2.2	Beneficial effects of a changing environment . . . . .	96
3.4.3	In most cases, sex-specific trait expression does not help adaptation . . . .	97
3.4.4	Combined effects of sexual and sex-specific natural selection on genetic variance . . . . .	97
3.4.5	Comparison of the types of assortative mating . . . . .	98
3.4.6	Potential demographic consequences . . . . .	99
3.5	Conclusion . . . . .	99

**Bibliography** **101**

3.1	Appendix 2 . . . . .	103
3.1.1	Joint distribution of breeding and phenotypic values under assortative female preference . . . . .	103
3.1.1.1	Initial conditions . . . . .	104
3.1.1.2	Joint distribution of breeding and phenotypic values in the offspring generation . . . . .	104
3.1.1.3	Natural selection . . . . .	105
3.1.1.4	Sexual selection . . . . .	106
3.1.1.5	Joint distribution of breeding and phenotypic values after natural selection . . . . .	106
3.1.1.6	Selection function on males to mate with a given female with phenotype $x$ . . . . .	108
3.1.1.7	Probability that a male has breeding value $g_m$ given the phenotype $x$ of a female in a pair of parents . . . . .	108
3.1.1.8	Probability that a male has breeding value $g_m$ and that a female has breeding value $g_f$ in a pair of parents . . . . .	109
3.1.1.9	Distribution of breeding and phenotypic values in the offspring generation . . . . .	109
3.1.1.10	Mismatch to the female optimum at equilibrium . . . . .	110
3.1.2	Joint distribution of breeding and phenotypic values under random mating	112
3.1.3	Joint distribution of breeding and phenotypic values under temporal assortative mating . . . . .	112
3.1.3.1	Competition between males with different fitness . . . . .	113
3.1.3.2	Competition between males with the same fitness . . . . .	114

**Bibliography** **117**

**4 Chapitre 3 : Fluctuations in selection strength affect the long-term response to climate change** **119**

4.1	Introduction . . . . .	120
4.1.1	Fluctuating selection and its impacts on evolution . . . . .	120
4.1.2	Measuring fluctuating selection . . . . .	121

4.1.3	How can theory clarify the role of the fluctuating selection ? . . . . .	121
4.1.4	Demographic consequences of fluctuating selection . . . . .	122
4.1.5	Questions, methods and main results . . . . .	122
4.2	Methods . . . . .	123
4.2.1	Genetic architecture . . . . .	123
4.2.2	Fluctuating selection . . . . .	123
4.2.3	Adaptive lag . . . . .	123
4.2.4	Population mean fitness and fitness loads . . . . .	124
4.2.5	Distribution of breeding values in the offspring generation . . . . .	124
4.2.6	Analytical approximations . . . . .	125
4.2.7	Numerical investigation . . . . .	125
4.3	Results . . . . .	126
4.3.1	Reference case with a constant fitness width . . . . .	126
4.3.2	Results with variation in the fitness width . . . . .	127
4.3.2.1	Strength of selection . . . . .	127
4.3.2.2	Genetic variance at equilibrium . . . . .	129
4.3.2.3	Adaptive lag . . . . .	130
4.3.2.4	Evolutionary load . . . . .	131
4.3.2.5	Variance load . . . . .	132
4.3.2.6	Geometric mean fitness . . . . .	134
4.4	Discussion . . . . .	134
4.4.1	Main results . . . . .	134
4.4.2	The geometric mean fitness is affected directly and indirectly by the variation in the fitness width . . . . .	135
4.4.3	Interaction between the mean and the variation in the fitness width . . . . .	135
4.4.4	Measuring the position of the population in the fitness landscape . . . . .	136
4.4.5	Phenotypic plasticity and bet-hedging as strategies to cope with fluctuations of the selection strength? . . . . .	136
4.4.6	Usefulness of an individual-based model . . . . .	137
4.4.7	Possible extensions of the model . . . . .	138
4.4.8	Conclusion . . . . .	138
<b>Bibliography</b>		<b>139</b>
4.1	Appendix 3 . . . . .	142
4.1.1	Figures . . . . .	142
4.1.2	Preliminary results from Matthieu Fontaine . . . . .	144
4.1.3	Analytical approximations . . . . .	146
4.1.3.1	Approximation for the strength of selection . . . . .	146
4.1.3.2	Approximation for the adaptive lag . . . . .	146
4.1.3.3	Approximation for the mean fitness . . . . .	147
4.1.3.3.1	Approximation for the variance load . . . . .	148
4.1.3.3.2	Approximation for the evolutionary load . . . . .	149
4.1.3.4	Approximation for the genetic variance . . . . .	149

<b>5 Discussion</b>	<b>151</b>
5.1 Résultats principaux . . . . .	151
5.1.1 Effets de l'homogamie sur les réponses évolutives à la sélection . . . . .	151
5.1.2 Effets de l'homogamie sur la structure de la variance génétique . . . . .	152
5.1.3 Effets de l'homogamie sur l'adaptation . . . . .	154
5.1.4 Effets des fluctuations de l'intensité de la sélection . . . . .	155
5.2 Comment connecter ces résultats théoriques à des résultats empiriques? . . . . .	156
5.3 Perspectives . . . . .	157
5.3.1 Effet de l'autofécondation sur les réponses évolutives au changement climatique . . . . .	157
5.3.1.1 Environnement constant . . . . .	157
5.3.1.2 Environnement changeant . . . . .	158
5.3.2 Effet des variations de la taille de population sur les réponses évolutives au changement climatique . . . . .	158
5.3.3 Effet des stades de vie sur les réponses évolutives au changement climatique	159
5.4 Conclusion générale . . . . .	160
<b>Bibliographie</b>	<b>163</b>

# Introduction

Le changement climatique contemporain se traduit notamment par une augmentation des températures et de leur fluctuation entre années ainsi qu'une augmentation de la fréquence et de l'intensité des épisodes de précipitation ou sécheresse (IPCC, 2014). Le changement climatique modifie dans le temps et l'espace les conditions abiotiques favorables aux espèces, appelées niche climatique. Par exemple, la date de ponte maximisant la valeur sélective des mésanges est de plus en plus précoce (Charmantier et al., 2008). Le changement climatique modifie aussi la durée des saisons de croissance des plantes. Celle-ci augmente car le printemps arrive plus tôt et l'hiver plus tard (Anderson et al., 2012; Bradshaw et Holzapfel, 2008). Les sécheresses en fin de saison peuvent également écourter les saisons de croissance (Hamann et al., 2018). Le changement climatique renforce ainsi la nécessité de comprendre les réponses des populations naturelles à la sélection pour mieux comprendre ses conséquences sur les populations. Parmi les réponses au changement climatique, les changements de phénologie, c'est-à-dire les changements de temporalité des événements répétés dans le cycle de vie des organismes comme la floraison, la migration ou la ponte, sont parmi les plus fréquemment observés (Parmesan, 2007; Parmesan et Yohe, 2003). En particulier les dates de floraison présentent des réponses rapides au changement climatique. Les dates de floraison sont des traits polygéniques sous homogamie temporelle puisque les appariements ne peuvent se produire qu'entre individus ayant des floraisons chevauchantes. L'homogamie crée des associations positives entre effets alléliques augmentant la variance génétique (Crow et Felsenstein, 1968; Crow et Kimura, 1970; Devaux et Lande, 2008; Wright, 1921). L'homogamie pourrait donc expliquer les réponses rapides des phénologies de floraison au changement climatique. La phénologie de floraison a aussi la particularité d'être dimorphique car les floraisons mâles et femelles sont souvent décalées dans le temps (Lloyd et Webb, 1986). Ces différences de floraison entre sexes peuvent être causées par une sélection différente entre les sexes. La sélection sexe-spécifique pourrait contraindre les réponses des phénologies de floraison au changement environnemental. Les réponses des phénologies de floraison au changement environnemental pourraient aussi être contraintes par les fluctuations de la durée de la période favorable à la floraison, accentuées par les effets du changement climatique sur les températures et les précipitations (IPCC, 2014). Au cours de cette thèse nous souhaitons comprendre les effets de l'homogamie, de la sélection sexe-spécifique et des fluctuations de la période favorable à la floraison sur les réponses évolutives des phénologies de floraison.

## 1.1 Les réponses au changement climatique

Le changement climatique menace d'extinction les populations naturelles en modifiant leur niche climatique (Román-Palacios et Wiens, 2020 ; Urban, 2015). Les espèces peuvent éviter l'extinction selon trois voies non mutuellement exclusives : migrer pour suivre le déplacement de leur niche climatique dans l'espace, modifier la temporalité des événements du cycle de vie pour suivre le déplacement de leur niche climatique dans le temps, ou s'adapter aux nouvelles conditions (Bellard et al., 2012 ; Parmesan, 2006). Deux mécanismes déterminent les réponses à la sélection : la plasticité et l'évolution génétique.

### 1.1.1 Migration

Le changement climatique déplace les conditions favorables aux espèces vers les pôles ou vers les sommets (Lenoir et al., 2008). Les preuves que les espèces peuvent suivre le déplacement de leur niche sont nombreuses bien que la migration ne soit pas toujours suffisante pour suivre le déplacement de la niche (Devictor et al., 2012 ; Lenoir et al., 2008 ; Lenoir et al., 2020). Les aires de distributions des espèces terrestres suivent le déplacement de la niche écologique et se déplacent en moyenne à une vitesse de 1.11 km/an ( $\pm 0.96$ ) vers les pôles et de 1.78 km/an ( $\pm 0.41$ ) vers les sommets (Lenoir et al., 2020). Les aires de distribution des espèces marines se déplacent plus rapidement, probablement car les activités humaines contraignent moins le suivi des changements environnementaux (Lenoir et al., 2020). Les réponses au déplacement des niches climatiques peuvent impliquer des changements de la position de l'aire de distribution dans sa globalité (Lenoir et al., 2008). Quand les changements d'aire de distribution ne suffisent pas à suivre le changement rapide de la niche climatique, les populations peuvent décliner à leur marge la plus distante de l'optimum. Par exemple, les populations de pingouin Adélie en Antarctique déclinent à leur marge nord car l'augmentation de la température de surface des océans diminue la quantité de ressources et la qualité des sites de ponte (Cimino et al., 2016).

### 1.1.2 Réponses évolutives et plastiques

Une étude récente estime que les changements d'aire de distribution sont insuffisants et pourraient conduire à l'extinction de 57 à 70% des espèces (Román-Palacios et Wiens, 2020). Cependant, l'impact des déplacements de la niche climatique peut être réduit si les espèces s'adaptent aux nouvelles conditions climatiques locales. La plasticité phénotypique est un mécanisme par lequel un génotype peut ajuster son phénotype en fonction de l'environnement. Elle a été particulièrement étudiée car elle permet de répondre rapidement (à l'échelle d'une génération) à un changement environnemental rapide comme celui causé par le changement climatique. Cependant, plusieurs études suggèrent que la plasticité phénotypique seule n'est pas suffisante pour suivre les changements environnementaux (Etterson, 2004 ; Loeuille, 2019 ; Phillimore et al., 2010). L'évolution génétique peut favoriser une réponse à plus long terme. Bien que l'évolution génétique soit difficile à distinguer de la plasticité (Hoffmann et Sgrò, 2011 ; Merilä et Hendry, 2014), plusieurs études mettent en évidence des réponses évolutives

(Anderson et al., 2012; Etterson, 2004; Franks et al., 2014; Merilä et Hendry, 2014; Pulido et Berthold, 2010; Réale et al., 2003). Par exemple, chez le thym commun (*Thymus vulgaris*), la fréquence des génotypes sensibles au froid augmente en réponse à la diminution de la fréquence des épisodes de froid extrême (Thompson et al., 2013). Les réponses évolutives peuvent ainsi permettre aux populations d'échapper à l'extinction quand la diversité génétique des traits liés au climat est suffisante (Franks et al., 2007; Hamann et al., 2018; Jump et al., 2008; Thompson et al., 2013). C'est ce que l'on appelle le sauvetage évolutif (Carlson et al., 2014; Gomulkiewicz et Holt, 1995).

### 1.1.3 Changements de phénologie

On estime que 59% des espèces présentent des changements de leur phénologie et que les phénologies avancent en moyenne de 2.3 jours par décennie (Parmesan et Yohe, 2003). Les changements de la phénologie de floraison sont particulièrement bien renseignés (Parmesan, 2006). Ces changements sont probablement causés par un avancement du début de la saison de croissance sous l'effet du changement climatique (Anderson et al., 2012; Bradshaw et Holzapfel, 2008; Parmesan, 2006). Par exemple un suivi long terme montre un avancement de la date de floraison de 0.2 à 0.5 jour par génération en réponse à l'augmentation des températures et à la fonte des neiges plus précoce (Anderson et al., 2012). Une fin de saison plus précoce, causée par exemple par la sécheresse, favorise aussi des floraisons précoces permettant de mûrir les graines avant que les conditions environnementales deviennent létales (Hamann et al., 2018). Cependant, les réponses de la phénologie de floraison au changement climatique peuvent être limitées par les interactions biotiques (Loeuille, 2019). Par exemple, si la date de floraison des plantes d'une population avance plus rapidement que la date d'émergence de ses pollinisateurs (Kehrberger et Holzschuh, 2019) alors l'avancement des dates de floraison est limité par l'absence de pollinisateurs pour les plantes les plus précoces. Les réponses des phénologies au changement climatique sont plastiques et/ou génétiques (Anderson et al., 2012; Charmantier et Gienapp, 2014; Franks et al., 2014; Hamann et al., 2018; Merilä et Hendry, 2014; Ramakers et al., 2019; Réale et al., 2003). La plupart des exemples de réponses évolutives au changement climatique concernent les phénologies de floraison (Merilä et Hendry, 2014). Par exemple, Hamann et al. (2018) met en évidence une réponse évolutive rapide du début de floraison en utilisant une approche par résurrection chez *Brassica rapa*. La date de début de floraison avance en moyenne de trois jours en 18 générations en réponse aux changements d'aridité et de précipitations. Ces réponses sont souvent adaptatives c'est-à-dire qu'elles augmentent la valeur sélective des individus (Møller et al., 2008; Radchuk et al., 2019).

## 1.2 Comprendre et prédire les réponses évolutives : apport des modèles de sauvetage évolutif

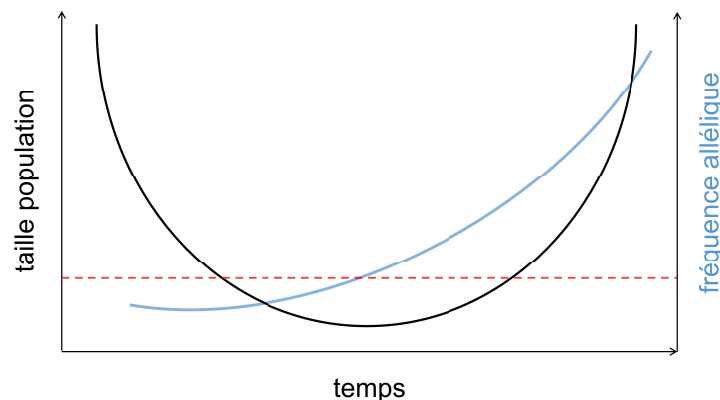
Plusieurs modèles de génétique quantitative ont étudié les conditions de persistance d'une population soumise à un environnement changeant et mimant le changement climatique



(Bürger et Lynch, 1995 ; Charlesworth, 1993 ; Lande et Shannon, 1996 ; Lynch et al., 1991). Ces modèles supposent un trait soumis à la sélection stabilisante de forme Gaussienne : il existe une valeur phénotypique optimale et les individus qui en dévient ont une reproduction ou une survie plus faible. La valeur sélective des individus déviant de l'optimum diminue d'autant plus vite que la fonction de sélection est étroite. Différentes situations biologiques ont été explorées par déplacement de l'optimum au cours du temps. En revanche, les modèles supposent une largeur de la fonction de sélection constante.

### 1.2.1 Déplacement ponctuel de l'optimum

La théorie s'est initialement intéressée à un déplacement ponctuel de l'optimum correspondant par exemple à l'invasion d'un nouvel environnement (Gomulkiewicz et Holt, 1995). Dans ce nouvel environnement, la population n'est pas à son optimum et son taux de croissance diminue (fig 1.1). Dans le même temps, la sélection favorise l'augmentation en fréquence des allèles avantageux et élimine les allèles désavantageux. Pour les populations initialement trop maladaptées, la sélection des allèles avantageux ne suffit pas à compenser la diminution de la taille de population. Une taille de population initialement grande offre plus de diversité génétique et plus de temps pour que le sauvetage évolutif se produise.

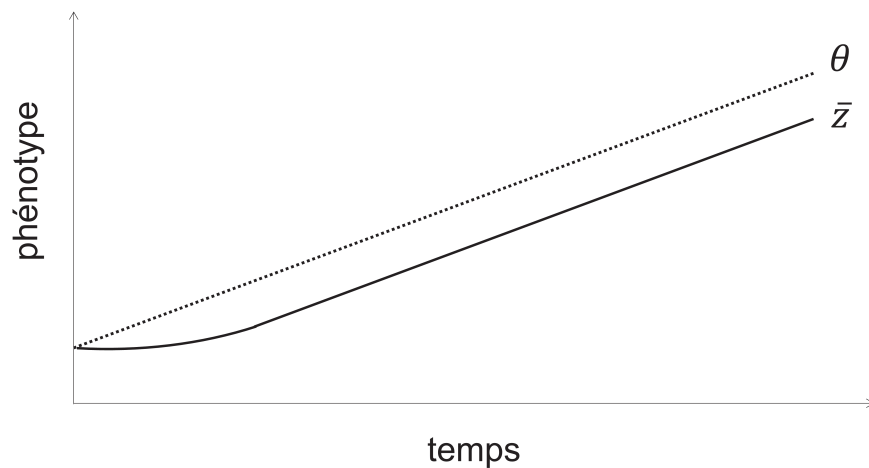


**Figure 1.1:** Taille de population (courbe noire) et fréquence des allèles adaptés (courbe bleue) au cours du temps. Le changement de l'environnement diminue le taux de croissance initial et la population commence à décliner. La sélection naturelle favorise les allèles adaptés qui augmentent en fréquence. Le taux de croissance de la population peut augmenter à nouveau. En dessous d'une certaine taille de population, l'extinction par stochasticité démographique est probable (ligne rouge). Plus la population passe de temps sous cette limite et plus la probabilité d'extinction augmente. Figure adaptée à partir de Loeuille 2019.

### 1.2.2 Déplacement graduel et constant de l'optimum

Certains modèles ont aussi étudié un optimum se déplaçant chaque génération à un taux constant pour modéliser des tendances globales comme l'augmentation des températures (Bürger, 1999 ; Bürger et Lynch, 1995 ; Charlesworth, 1993 ; Lynch et al., 1991 ; Matuszewski et al., 2015 ; Pease et al., 1989). Le gradient de sélection sur le trait augmente quand l'optimum commence à se déplacer. La population répond au déplacement de l'optimum avec un retard jusqu'à atteindre un

régime asymptotique où le phénotype moyen change aussi vite que le déplacement de l'optimum mais conserve toujours un retard adaptatif (Lynch et al. 1991 ; fig 1.2). Le retard adaptatif est d'autant plus grand que le changement environnemental est rapide, que la sélection stabilisante sur le trait est faible et que la variance génétique dans la population est faible (Bürger et Lynch, 1995 ; Charlesworth, 1993 ; Lande et Shannon, 1996 ; Lynch et al., 1991). Cependant, si la vitesse de changement de l'environnement est trop rapide, la diversité génétique initiale peut être insuffisante pour répondre au changement environnemental. Le retard du phénotype moyen à l'optimum augmente, le taux de croissance de la population diminue et la population s'éteint (Bürger et Lynch, 1995 ; Charlesworth, 1993 ; Lynch et al., 1991). On peut ainsi définir une vitesse critique du changement environnemental au-delà de laquelle le risque d'extinction de la population est élevé (Lynch et al. 1991). Des modèles plus récents ont distingué le rôle de la variance génétique pré-existante et des mutations *de novo* dans les réponses évolutives (Anciaux et al., 2018 ; Matuszewski et al., 2015).



**Figure 1.2:** Réponse du phénotype moyen ( $\bar{z}$ ) à un changement graduel et constant du phénotype optimal ( $\theta$ ). La population est en retard sur son optimum jusqu'à atteindre un régime asymptotique où le phénotype moyen change aussi vite que le déplacement de l'optimum mais conserve toujours un retard adaptatif. Figure adaptée à partir de Lynch et al. 1991.

### 1.2.3 Fluctuations de l'optimum

Les modèles de génétique quantitative ont aussi considéré la stochasticité environnementale (Bürger et Lynch, 1995 ; Charlesworth, 1993 ; Lande et Shannon, 1996 ; Lynch et al., 1991). L'optimum fluctue autour d'une valeur constante ou d'une tendance linéaire illustrant par exemple les variations interannuelles de la température autour d'une tendance générale à l'augmentation. Dans un tel environnement, le risque d'extinction augmente avec l'intensité des fluctuations de l'optimum (Bürger et Lynch, 1995). Répondre à la sélection une génération donnée augmente le retard du phénotype moyen à l'optimum à la génération suivante quand les fluctuations de l'optimum sont faibles à modérées et non autocorrélées (bruit blanc). Répondre à la sélection fluctuante augmente donc la valeur sélective moyenne de la population à une génération donnée mais diminue la valeur sélective moyenne de la population à la génération suivante. En moyenne sur plusieurs générations, les populations avec peu de

variance génétique ont alors une meilleure valeur sélective que les populations avec une forte variance génétique (Charlesworth, 1993 ; Lande et Shannon, 1996). En revanche la variance génétique limite le retard à l'optimum quand les fluctuations de l'optimum sont fortes. La valeur sélective moyenne de la population augmente donc avec la variance génétique (Charlesworth, 1993). La variance génétique facilite aussi le suivi d'un optimum aux fluctuations cycliques ou autocorrélées (Charlesworth, 1993 ; Lande et Shannon, 1996 ; Lynch et al., 1991).

#### 1.2.4 Preuves empiriques de sauvetage évolutif

L'ensemble de ces modèles montre la pertinence de la variabilité génétique du trait sous sélection pour quantifier le potentiel évolutif et la persistance des populations dans un environnement changeant. La théorie du sauvetage évolutif est validée par plusieurs données expérimentales (numéro spécial Gonzalez et al., 2013 et revue Carlson et al., 2014). Le sauvetage évolutif est plus difficile à mettre en évidence dans les populations naturelles car les interactions biotiques brouillent le signal du sauvetage évolutif (Carlson et al., 2014). On peut néanmoins citer l'exemple de l'évolution de la résistance à la maladie du tournis (*Myxobolus cerebralis*) chez les populations de truites arc-en-ciel (*Oncorhynchus mykiss*). Cette maladie a entraîné un déclin rapide des populations de truites jusqu'à l'évolution de génotypes résistants à la maladie favorisant le rétablissement des populations (Miller et Vincent, 2008).

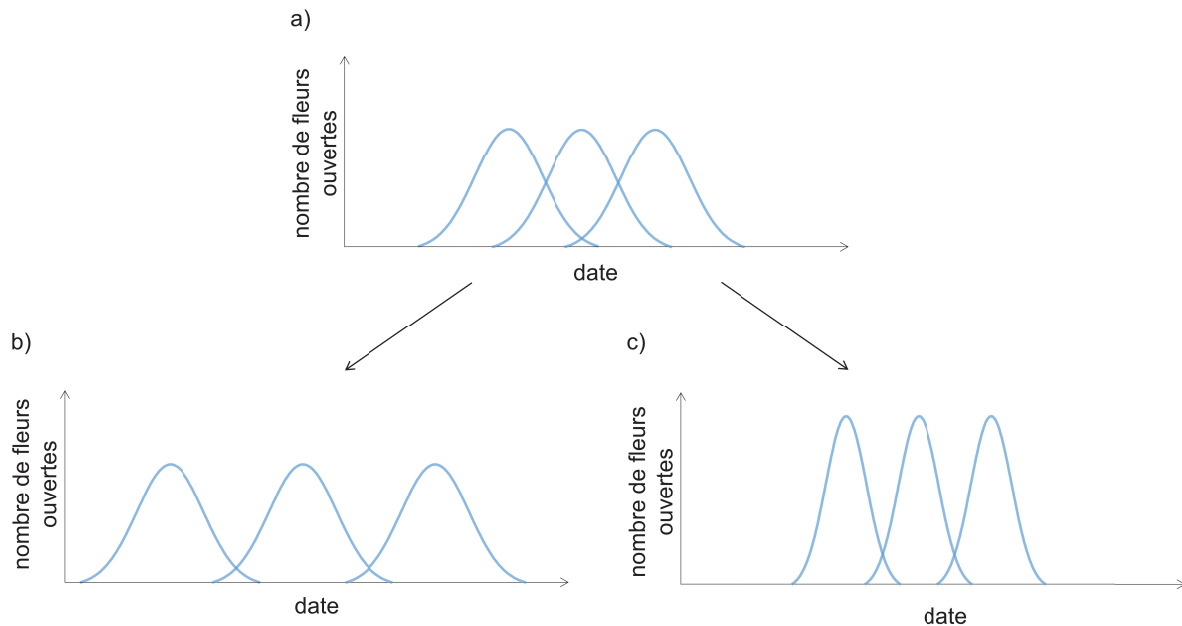
### 1.3 Homogamie

La théorie classique du sauvetage évolutif suppose des appariements panmictiques. Cependant, de nombreux traits quantitatifs sont sous homogamie c'est-à-dire que les appariements se produisent entre individus exprimant des phénotypes similaires (Janicke et al., 2019 ; Jiang et al., 2013).

#### 1.3.1 Différents types d'homogamie

Les mécanismes conduisant à l'homogamie peuvent être classifiés en deux types : l'homogamie par préférence (aussi appelé "Preference/Trait Rules") et l'homogamie par concordance (aussi appelé "Matching Rules" ; Kopp et al., 2018). L'homogamie par préférence se caractérise souvent par une fonction de préférence d'un sexe pour les valeurs phénotypiques de l'autre sexe (Kopp et al., 2018 pour une revue, Rodríguez et al., 2013 et Neelon et al., 2019 pour des estimations empiriques, Sachdeva et Barton, 2017 pour un modèle). Un cas particulier d'homogamie par concordance est l'homogamie par regroupement (appelé "grouping" ; Kopp et al., 2018). A la différence des autres mécanismes, l'homogamie par regroupement n'implique pas de choix. C'est uniquement la valeur du trait qui définit des groupes au sein desquels les appariements sont aléatoires. Les regroupements peuvent avoir lieu dans l'espace ou dans le temps. Par exemple, certains insectes choisissent une plante hôte sur laquelle ils se reproduiront. Les individus qui s'apparient ont donc le même phénotype pour le choix de la plante hôte et le regroupement est spatial. Chez les plantes, la date de floraison implique un regroupement dans

le temps des individus qui s'apparient : les plantes qui fleurissent tôt (ou tard) s'apparient plus fréquemment avec d'autres plantes qui fleurissent tôt (ou tard). L'intensité de l'homogamie augmente avec la variance des dates de floraison dans la population et diminue avec la durée des floraisons individuelles (fig 1.3 ; Fox, 2003 ; Weis et al., 2014 ; Weis et al., 2005).



**Figure 1.3:** Exemples de calendrier d'ouverture des fleurs au cours de la saison. Chaque courbe indique le nombre de fleurs ouvertes par jour au cours de la saison. Un exemple de référence (a) est comparé avec un scénario où l'écart-type des dates de floraison entre individus augmente (b) ou avec une floraison individuelle plus courte (c). Dans ces deux scénarios, l'homogamie augmente par rapport à l'exemple de référence car le chevauchement des floraisons individuelles diminue. Figure adaptée de Weis et al. 2014.

### 1.3.2 Effets de l'homogamie sur les réponses évolutives

L'homogamie affecte la variance génétique et les réponses à la sélection. En effet l'homogamie, en associant des individus avec des phénotypes similaires, crée des associations positives entre effets alléliques similaires (Crow et Felsenstein, 1968 ; Crow et Kimura, 1970 ; Wright, 1921). L'homogamie induit aussi de la sélection sexuelle car les phénotypes rares ont un succès reproducteur plus faible (Fox, 2003 ; Kirkpatrick et Nuismer, 2004 ; Weis et al., 2005). La sélection sexuelle s'ajoute à la sélection naturelle stabilisante et augmente l'intensité totale de la sélection sur les traits sous homogamie par rapport à la panmixie. L'intensité de la sélection stabilisante diminue la variance génétique mesurant le polymorphisme génétique à l'équilibre de liaison et de Hardy-Weinberg. L'homogamie a donc des effets antagonistes sur la variance génétique : elle augmente la variance génétique via son effet positif sur les associations génétiques mais diminue la variance génétique via son effet négatif sur la variance génétique. Les effets de l'homogamie sur la diversité génétique ont été particulièrement étudiés dans le cadre de la spéciation (Devaux et Lande, 2008 ; Kirkpatrick et Nuismer, 2004 ; Kopp et al., 2018 ; Sachdeva et Barton, 2017 ; Smadja et Butlin, 2011). Sous sélection stabilisante, les effets positifs et négatifs de l'homogamie sur la variance génétique se compensent (Lande, 1977). Les

associations positives entre effets alléliques facilitent les réponses évolutives à la sélection directionnelle (voir Fox, 2003 ; O'Donald, 1960 ; Weis et al., 2005 pour des modèles et Baker, 1973 ; de Lange, 1974 ; Shepherd et Kinghorn, 1994 ; Smith et Hammond, 1987 ; Tallis et Leppard, 1987 pour des résultats de sélection artificielle). Cependant, les effets de l'homogamie sur les réponses évolutives à un environnement changeant ne sont pas connus. Si l'homogamie favorisait les réponses évolutives au changement environnemental alors elle pourrait être utilisée comme un levier d'action dans les populations en déclin.

## 1.4 Effet de la sélection sexe-spécifique sur la phénologie de floraison

La sélection sexe-spécifique est fréquente dans les populations naturelles (Cox et Calsbeek, 2009 ; de Lisle et al., 2018). Elle induit un conflit entre les réponses des traits mâles et femelles car la population ne peut pas être à la fois à l'optimum mâle et à l'optimum femelle (Bonduriansky et Chenoweth, 2009). La sélection sexe-spécifique fait donc émerger un phénotype optimal, distinct des optimums mâle et femelle, maximisant la valeur sélective de la population (Lande, 1980). La sélection sexuelle augmente la contrainte sur les réponses évolutives des populations dans un environnement constant (Lande, 1980). Cette sélection sexuelle, qui est notamment générée par l'homogamie pour la date floraison, déplace l'optimum des mâles de leur pic de valeur sélective (Lande, 1980). Les réponses des mâles sont donc un compromis entre la sélection naturelle et la sélection sexuelle. Le conflit entre les réponses des sexes peut être atténué par la diminution des corrélations génétiques entre les traits mâle et femelle faisant ainsi apparaître un dimorphisme sexuel (Bonduriansky et Chenoweth, 2009 ; Lande, 1980 ; Poissant et al., 2010 ; Rhen, 2000). Les effets de la sélection sexe-spécifique dans le cadre du changement environnemental restent peu étudiés. Connallon et Hall (2016) montre que le changement environnemental, en déplaçant les optimums mâle et femelle, peut aligner la direction de la sélection sur les traits mâles et femelles. La réponse de chaque sexe est alors moins contrainte par la réponse de l'autre sexe que dans un scénario où la sélection sur les sexes est opposée. La phénologie de floraison est un trait sous homogamie qui présente fréquemment une différence entre les dates de floraison mâle et femelle (Lloyd et Webb, 1986). Cette asynchronie de floraison peut évoluer en réponse au conflit entre les sexes générés par la sélection sexe-spécifique. Les réponses évolutives de la phénologie de floraison pourraient ainsi être contraintes par la sélection sexe-spécifique et la sélection sexuelle.

## 1.5 Objectifs de la thèse

Cette thèse vise à approfondir la compréhension des réponses évolutives de la phénologie de floraison au changement environnemental en utilisant une approche exclusivement théorique basée sur des observations empiriques. Pour cela, nous nous appuyons sur la littérature théorique des réponses évolutives au changement climatique pour identifier l'effet (i) de l'homogamie, (ii) de

la sélection sexe-spécifique et (iii) des fluctuations de la durée de la période favorable à la floraison sur les réponses évolutives de la phénologie de floraison au changement environnemental.

### 1.5.1 Effet de l'homogamie sur les réponses évolutives de la phénologie de floraison au changement environnemental

Le premier chapitre cherche à comprendre si l'homogamie temporelle pour la date de floraison, via ses effets sur la variance génétique, peut expliquer les réponses rapides de la phénologie de floraison au changement environnemental. Nous utilisons deux modèles complémentaires de génétique quantitative pour identifier les effets de l'homogamie temporelle pour la date de floraison sur la variance génétique dans un environnement changeant par rapport à la panmixie. Le premier modèle est un modèle analytique supposant une taille de population infinie et un grand nombre de loci déterminant la date floraison. La variance génique est constante de telle sorte que la variance génétique évolue sous l'effet des changements d'associations entre effets alléliques. Le second modèle est un modèle individu-centré supposant une taille de population finie, un nombre de loci déterminant la date de floraison limité et une variance génique non constante. Les résultats montrent que l'homogamie temporelle pourrait expliquer les réponses rapides des phénologies de floraison au changement environnemental. Ce chapitre a fait l'objet d'une publication dans "Journal of Evolutionary Biology" dans le numéro spécial intitulé "Assortative mating for labile traits and its fitness consequences in the wild". Cette publication est présentée dans le premier chapitre.

### 1.5.2 Effet combiné de l'homogamie et de la sélection sexe-spécifique sur les réponses évolutives

Dans le deuxième chapitre, nous avons testé la robustesse des conclusions du premier chapitre à la sélection sexe-spécifique. Pour cela, nous avons introduit la sélection sexe-spécifique au modèle analytique précédent et généralisé les résultats en comparant différentes hypothèses sur la façon de modéliser l'homogamie. Dans le prolongement de la littérature théorique sur la sélection sexe-spécifique en environnement constant, nous avons également introduit le dimorphisme sexuel comme un facteur pouvant moduler les conflits sexuels. Les prédictions du modèle analytique sont comparées à des simulations individu-centrées supposant une variance génique non constante. Les résultats, présentés dans le deuxième chapitre, montrent que la gamme de paramètres dans laquelle l'homogamie facilite le suivi de l'environnement par rapport à la panmixie est plus restreinte que celle identifiée dans le chapitre précédent. Le second chapitre sera très prochainement soumis pour publication dans un journal scientifique.

### 1.5.3 Effet des fluctuations de la durée de la saison favorable à la floraison sur les réponses évolutives

Les modèles de génétique quantitative des réponses évolutives au changement environnemental simulent souvent l'augmentation des températures et leurs fluctuations inter-annuelles par un optimum phénotypique mobile. Cependant, ces modèles supposent une

largeur constante de la fonction de sélection. Dans le troisième chapitre, nous avons modélisé les fluctuations de la durée de la saison favorable à la floraison en laissant la largeur de la fonction de sélection varier entre années. Nous avons développé des attendus analytiques pour mieux comprendre l'effet des fluctuations de la durée de la saison favorable à la floraison sur les réponses évolutives et le taux de croissance à long-terme des populations. Les résultats, présentés dans le troisième chapitre, suggèrent que les prédictions de l'adaptation des populations au changement climatique pourraient être améliorées en prenant en compte les fluctuations de la largeur de la fonction de sélection.

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# Chapitre 1 : Assortative mating can help adaptation of flowering time to a changing climate: Insights from a polygenic model

## 2.1 Main text

# Assortative mating can help adaptation of flowering time to a changing climate: Insights from a polygenic model

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## Abstract

Several empirical studies report fast evolutionary changes in flowering time in response to contemporary climate change. Flowering time is a polygenic trait under assortative mating, since flowering time of mates must overlap. Here, we test whether assortative mating, compared with random mating, can help better track a changing climate. For each mating pattern, our individual-based model simulates a population evolving in a climate characterized by stabilizing selection around an optimal flowering time, which can change directionally and/or fluctuate. We also derive new analytical predictions from a quantitative genetics model for the expected genetic variance at equilibrium, and its components, the lag of the population to the optimum and the population mean fitness. We compare these predictions between assortative and random mating, and to our simulation results. Assortative mating, compared with random mating, has antagonistic effects on genetic variance: it generates positive associations among similar allelic effects, which inflates the genetic variance, but it decreases genetic polymorphism, which depresses the genetic variance. In a stationary environment with substantial stabilizing selection, assortative mating affects little the genetic variance compared with random mating. In a changing climate, assortative mating however increases genetic variance compared to random mating, which diminishes the lag of the population to the optimum, and in most scenarios translates into a fitness advantage relative to random mating. The magnitude of this fitness advantage depends on the extent to which genetic variance limits adaptation, being larger for faster environmental changes and weaker stabilizing selection.

## KEYWORDS

fitness, genetic variance, lag, nonrandom mating, phenology, quantitative genetics

## 1 | INTRODUCTION

A change in phenology, which is the timing of recurrent events in the life cycle, is a common response of plant and animal species to current climate change (Merilä & Hendry, 2014; Parmesan & Yohe, 2003).

In particular, flowering time has advanced for many plant populations of temperate zones (Anderson et al., 2012; Franks et al., 2007, 2014; Hamann et al., 2018; Inouye, 2008; Morin et al., 2007) and these changes are partly due to rapid genetic evolution (Ashworth et al., 2016; Franks et al., 2007, 2014; Hamann et al., 2018;

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Lustenhouwer et al., 2018). For example, the resurrection ecology approach used by Hamann et al. (2018) showed that breeding values for flowering onset advanced by an average of three days in 18 generations. In fact, most cases of genetic responses to climate warming concern phenology, and especially flowering time (see the special issue edited by Merilä & Hendry, 2014). Evolution of flowering time is likely affected by assortative mating, that is the positive correlation between mates for flowering time. Assortative mating is obligate in plants, but the phenotypic correlation between mates can range from low to high values, for example 0.05–0.63 within the same old-field community (Weis et al., 2014), depending on the overlap in the flowering phenologies of different individuals in the population (Devaux & Lande, 2008). We here wish to test whether this particular mating pattern in plants can contribute to the rapid observed evolution of flowering time in response to a warming climate.

Available quantitative genetics theory on evolutionary responses to environments changing in time mostly assumes asexual reproduction or sexual reproduction with random mating (Bürger, 1999; Bürger & Lynch, 1995; Charlesworth, 1993; Gomulkiewicz & Holt, 1995; Kopp & Matuszewski, 2014; Lande & Shannon, 1996; Lynch et al., 1991). This theory provides insight on the limits to adaptation to a changing climate. Most models assume stabilizing selection on traits with an optimal value that changes directionally and fluctuates through time. They predict that the population evolves to track the moving optimum, but with some lag. Higher genetic variance for the adaptive traits accelerates the response to selection, decreases the lag of the population to the optimum, which translates into an increased population mean fitness when temporal changes in the optimum are predictable and the lag is not too small (e.g. for a sudden shift in the optimum in Gomulkiewicz & Holt, 1995, a gradual shift in Bürger, 1999; Bürger & Lynch, 1995; Charlesworth, 1993; Kopp & Matuszewski, 2014; Lande & Shannon, 1996; Lynch et al., 1991; Lynch et al., 1993 or cyclic shifts in Bürger, 1999; Charlesworth, 1993; Lande & Shannon, 1996; Lynch et al., 1991). Conversely, increased genetic variance depresses population mean fitness in constant environments, because more individuals depart from the optimal phenotypic value (Lande & Shannon, 1996), and in randomly fluctuating environments because an increased response to selection can move the mean population trait away from the optimum in the next selection episode (Bürger & Lynch, 1995; Charlesworth, 1993; Lande & Shannon, 1996). Whether the quantitative predictions of these theoretical models can be easily extended for adaptive traits under assortative mating is however yet unclear.

Assortative mating has antagonistic effects on the evolution of the genetic variance for adaptive traits. The genetic variance for a polygenic trait can be partitioned into: (a) the genic variance, which is the variance for the trait at linkage and Hardy–Weinberg equilibrium, reflecting the polymorphism at each locus, (b) covariances in allelic effects within and across loci that can be either positive or negative (Bulmer, 1980; Wright, 1921). Assortative mating generates positive associations (covariances) among alleles of similar effects on the trait, both among and within loci, which inflates the genetic variance (Crow & Felsenstein, 1968; Crow & Kimura, 1970; Wright, 1921).

Assortative mating can however decrease the polymorphism maintained at mutation-selection equilibrium at each locus, that is the genic variance. It does so because it decreases the reproductive success of individuals with a rare phenotype, which generates stabilizing sexual selection (Kirkpatrick & Nuismer, 2004). Compared with random mating, assortative mating can therefore either increase or decrease the genetic variance for polygenic traits (Kirkpatrick & Nuismer, 2004). A number of studies have found that the predicted genetic variance is higher under assortative than under random mating (e.g. for neutral quantitative traits see Crow & Felsenstein, 1968; Crow & Kimura, 1970; Devaux & Lande, 2008; Wright, 1921, for traits under disruptive or directional selection see Crosby, 1970; Devaux & Lande, 2008; Fox, 2003; Kopp & Matuszewski, 2014; Sachdeva & Barton, 2017; Weis et al., 2005). This past theory suggests that the positive effects of assortative mating on associations among allelic effects often dominate the negative effects of depressing genetic variance. Lande (1977) however predicts no effect of assortative mating on the equilibrium genetic variance for a trait under constant or fluctuating stabilizing selection, suggesting a perfect compensation of its antagonistic effects on the components of genetic variance. Predictions for the antagonistic effects of assortative mating on the genetic variance for a polygenic trait are however lacking when the optimal trait changes with a trend through time, as imagined under climate change.

As a consequence of its effect on the evolution of the genetic variance, assortative mating can either facilitate, or constrain, the response to disruptive selection, the evolution of genetic divergence in spatially heterogeneous environments, and sympatric or parapatric speciation (Devaux & Lande, 2008; Kirkpatrick & Nuismer, 2004; Kopp et al., 2018; Sachdeva & Barton, 2017; Servedio, 2016; Smadja & Butlin, 2011). Assortative mating can facilitate the response to directional selection, because higher homozygosity increases the genetic variance exposed to selection (Fox, 2003; O'Donald, 1960; Weis et al., 2005). The latter models (Fox, 2003; O'Donald, 1960; Weis et al., 2005) considered a single biallelic locus determining the trait in a population subject to directional selection. Quantitative genetics models for polygenic traits predict that assortative mating can increase genetic responses to specific forms of artificial selection, but only if heritability is high and selection is weak (Baker, 1973; De Lange, 1974; Shepherd & Kinghorn, 1994; Smith & Hammond, 1897; Tallis & Leppard, 1987). These predictions about response to selection remain to be tested for polygenic traits under natural stabilizing selection with an optimum varying in time.

Our goal was to test the ideas that (a) assortative mating for a polygenic trait increases genetic variance in a changing climate despite its antagonistic effects on the genic variance and the associations among allelic effects and (b) an increased genetic variance then improves adaptation to climate change. We thus compare the genic and genetic variance, lag to the optimum, and mean fitness of an isolated population, under either random or assortative mating, and in different scenarios of environmental change affecting the optimal value for a polygenic trait. The evolving trait under assortative mating is flowering time, and the strength of assortative mating evolves



with the degree of overlap in flowering among individuals in the population. Both the genic variance and the associations among allelic effects are shaped by drift, mutation, selection and the mating pattern. To better understand the mechanisms explaining the effects of assortative mating on the evolutionary responses of flowering time to climate change, we compare our simulation results with analytical predictions. First, we extend to the case of assortative mating the model of Bürger and Lynch (1995), relating the genetic variance, lag and mean fitness in a changing environment. Second, we adapt the predictions of Sachdeva and Barton (2017) for the evolution of the associations among allelic effects to our scenarios of selection and assortative mating for flowering time. Our simulations and analytical model both confirm that assortative mating can help adaptation of flowering time to a changing climate through its positive effect on the evolution of the genetic variance. This adaptive advantage of assortative mating can however be small and increases with the speed of climate change.

## 2 | METHODS

Our individual-based model assumes discrete and nonoverlapping generations and a constant population size with no migration. We model the evolution of peak flowering time, which is under assortative mating because flowering must overlap between mates. Flowering time is assumed to be under stabilizing selection around an optimal time, which varies with climate and thus among years. For comparison, we also consider a scenario with adaptation to climate change for a trait under random mating. All notations are defined in Table 1.

### 2.1 | Flowering phenology and mating

The plant is annual, bears hermaphroditic flowers, with synchronous male (pollen release) and female (stigma receptivity) phase. The plant is self-compatible without inbreeding depression. We consider that flowers are open for a single day, but the reasoning would be the same for different flower longevities (be it hours or days), as long as fitness varies on the same time scale. Each flower produces a large number of ovules and thus a large number of seeds. The reproductive success of mothers does not depend on access to mates (no pollen limitation or interference). A flower can only be fertilized by a pollen grain emitted by a flower open the same day. Pollen grains compete to fertilize ovules only within days. We assume that peak flowering time is genetically variable and can thus be different among plants; in contrast we assume that the duration of flowering is the same for all plants. The distribution of open flowers through days for all plants is approximately Gaussian with a constant variance  $\alpha^2$ . Each plant opens exactly 20 flowers during 9 days with the following sequence for the number of open flowers per day: {1,2,2,3,4,3,2,2,1} (Table 2), corresponding to  $\alpha^2 = 4.5$ . The peak flowering date  $Z_j$  (integer number) for individual  $j$  can be positive or negative, describing, respectively, later or earlier flowering compared with an arbitrary

reference date set to 0 in year 0. The strength of assortative mating for peak flowering date is measured by the phenotypic correlation between mates:

$$\rho = \frac{\text{cov}(Z_x, Z_y)}{\sqrt{\text{Var}(Z_x)\text{Var}(Z_y)}} \quad (1)$$

with  $Z_x$  and  $Z_y$  respectively, the peak flowering dates of a mother and a father mating together and contributing to the next generation.

In our model with constant duration of flowering, the strength of assortative mating is solely affected by the spread, that is variance, in peak flowering dates among plants. Relaxing this assumption by making duration of flowering variable among individuals, but with identical mean duration and spread of flowering dates, would not affect the strength of assortative mating. As variance of flowering time evolves in our model, so does the strength of assortative mating across years. Note that, even if mating among flowers is strictly assortative between days, it is random among flowers within days.

### 2.2 | Selection

The number of viable seeds produced by a flower depends on the day at which its ovules are fertilized. Early flowers may never produce viable seeds because of frost, whereas late flowers may not have enough time to mature their seeds (Chuine, 2010; Inouye, 2008; Morin et al., 2007; O'Neil, 1997). In year  $i$ , the probability that a fertilized ovule will turn into a viable seed is maximal at time  $\theta_i$  (real number) within the year; this optimal flowering time can vary across years. Seed viability declines as a Gaussian function with distance to the optimal flowering time  $\theta_i$ . We assume that  $\omega^2$  the width of this Gaussian function remains constant through years. The female fitness  $w_{ij}$  of a plant  $j$  in year  $i$  is measured by summing, over all days in the year, its number of open flowers multiplied by the probability that each will produce viable seeds.

A new generation of  $N$  plants ( $N = 1,000$ ) is formed by randomly sampling  $N$  fertilized ovules among all flowers open in the year, according to their probability of producing a viable seed. Each of these ovules is then paired to a pollen grain sampled at random among all flowers open the same day as the flower bearing the ovule.

### 2.3 | Genetic architecture

This expected phenotypic value  $z_j$  of a plant  $j$  is defined by:

$$z_j = g_j + e_j \quad (2)$$

with  $g_j$  the breeding value, and  $e_j$  the micro-environmental effect for peak flowering time associated with plant  $j$ , drawn from a Normal distribution  $\mathcal{N}(0, V_e = 4)$ . Note that peak flowering time is not plastic in our model (but see Discussion).

TABLE 1 List of symbols with their description

Symbol	Description
$Z_j$	Peak flowering date (integer) for plant $j$ ( $x$ for mother and $y$ for father)
$\bar{Z}_i$	Population mean peak flowering date in year $i$
$z_j$	Peak flowering time (real) for plant $j$
$e_j$	Micro-environmental effect for plant $j$ drawn from a Normal distribution $\mathcal{N}(0, V_e)$
$V_e$	Variance for environmental effects on peak flowering time
$g_j$	Breeding value of peak flowering time for plant $j$
$\bar{g}_i$	Population mean breeding value of peak flowering time in year $i$
$V_{g_i}$	Genetic variance for peak flowering time in year $i$ measured in the simulations before selection
$V_g$	Expected genetic variance
$V[\bar{g}_i]$	Variance, among realizations of the stochastic evolutionary trajectories, in $\bar{g}_i$ the mean breeding value in generation $i$
$V_{LEi}$	Genic variance for peak flowering time at Hardy-Weinberg and linkage equilibrium in year $i$ measured in the simulations before selection
$V_{LE}$	Expected genic variance at Hardy-Weinberg and linkage equilibrium
$L$	Number of loci determining peak flowering time
$L_e$	Effective number of loci
$\sigma_l$	Standard deviation in allelic effects for locus $l$
$a_{xjl}$ (resp. $a_{yjl}$ )	Maternal (resp. paternal) allelic effect at locus $l$ for plant $j$
$\bar{a}_{xl}$ (resp. $\bar{a}_{yl}$ )	Maternal (resp. paternal) mean allelic effect at locus $l$
$\bar{a}_l$	Population mean allelic effect at locus $l$
$\mu$	Allelic mutation rate
$U$	Genomic mutation rate
$V_m$	Mutational variance for peak flowering time
$h^2$	Heritability of peak flowering time
$\alpha^2$	Individual variance for flowering time, which links to the duration of flowering for individual plants
$\rho$	Phenotypic correlation between mates
$\theta_i$	Optimal flowering time in year $i$
$\omega^2$	Width of the Gaussian fitness function relating seed viability with time
$k$	Speed of the optimum change per generation
$\varepsilon_i$	Deviation of the optimal flowering time in year $i$ drawn from a Normal distribution $\mathcal{N}(0, V_\theta)$
$V_\theta$	Variance among year in the optimal flowering time
$w_{ij}$	Female fitness of a plant $j$ in year $i$
$\bar{w}_i$	Population mean fitness in year $i$
$\bar{w}$	Expected population mean fitness
$\delta_i$	Phenotypic lag of the population to the optimal flowering time measured in year $i$ in the simulations
$\delta$	Expected phenotypic lag of the population to the optimal flowering time
$N$	Population size
$N_e$	Effective population size
$V_s$	Width of the Gaussian fitness function relating fitness to breeding values for peak flowering time
$\tilde{V}$	Width of the Gaussian function relating the expected population mean fitness to the expected phenotypic lag
$s_n$	Strength of natural selection on breeding values for peak flowering time
$s_s$	Strength of sexual selection on breeding values for peak flowering time

The actual peak flowering date of individual  $j$  in the simulations is an integer value  $Z_j$  obtained by rounding the expected phenotypic value  $z_j$  (real number) up or down to the next integer, depending on the random draw from a Bernoulli distribution of parameter equals to the fractional part of  $z_j$ .

The breeding value for peak flowering time is determined by  $L$  additive and freely recombining loci:

$$g_j = \sum_{l=1}^L (a_{xjl} + a_{yjl}) \quad (3)$$

**TABLE 2** Parameter values for all scenarios tested, with corresponding literature references for the number of loci  $L$ , the individual variance in flowering time  $\alpha^2$  (in days<sup>2</sup>), the width of the Gaussian function for stabilizing selection  $\omega^2$  (in days<sup>2</sup>), the speed of the optimum change  $k$  (in days/generation), the variance in the fluctuations in the optimum  $V_\theta$  (in days<sup>2</sup>), the genomic mutation rate  $U$ , and the mutational variance  $V_m$  (in days<sup>2</sup>)

Case	Number of loci, $L$	Individual variance in flowering time, $\alpha^2$	Width of the Gaussian fitness function, $\omega^2$	Speed of the optimum change, $k$	Variance in the fluctuations in the optimum, $V_\theta$	Genomic mutation rate, $U$	Mutational variance, $V_m$
Reference	5 (Putterill et al., 2004)	4.5 (Primack et al., 2004)	400 (Gauzere et al., 2020)	0, -0.1, -0.2, -0.3, -0.4, -0.5 (Hamann et al., 2018)	100 (Gauzere et al., 2020)	0.1 (Russell et al., 1963)	0.04 (Lynch, 1988)
Constant environment			0, 20, 50, 100, 400, 1,000, 10,000, 100,000, infinite	0	0		
Constant environment and higher number of loci	50		0, 20, 50, 100, 400, 1,000, 10,000, 100,000, infinite	0	0		
Stationary environment				0	0, 5, 25, 100, 400, 900		
No fluctuations					0		
Weaker assortative mating		22.7					
Higher number of loci	50						
Stronger stabilizing selection			50				
Rarer mutations of smaller effects, higher number of loci and stronger stabilizing selection	50		50			0.01	0.004

with  $a_{xjl}$  (resp.  $a_{yjl}$ ) the maternal (resp. paternal) allelic effect at locus  $l$  for plant  $j$ .

Mutations occur at rate  $\mu = U/(2L)$  per allele with  $U$  the genomic mutation rate, whereas the mutational effects are sampled in a Normal distribution  $\mathcal{N}(0, V_m/U)$  with  $V_m$  the mutational variance for the evolving trait introduced by mutation each generation.

## 2.4 | Metrics recorded in the simulations

We record several metrics for the genetic diversity of peak flowering time. The (additive) genetic variance  $V_{gi}$  of the trait in year  $i$  among plants before selection is measured in the simulations by:

$$V_{gi} = \frac{1}{N} \sum_{j=1}^N \left( \sum_{l=1}^L (a_{xjl} + a_{yjl}) - \bar{g}_i \right)^2 \quad (4)$$

with  $a_{xjl}$  (resp.  $a_{yjl}$ ) the maternal (resp. paternal) allelic effect at locus  $l$  for plant  $j$ , and  $\bar{g}_i$  the population mean breeding value of the trait in year  $i$ .

We also record the genic variance  $V_{LE}$ , that is genetic variance at linkage and Hardy-Weinberg equilibrium.  $V_{LE}$  is a measure of genetic polymorphism for loci affecting peak flowering time and is defined in year  $i$  as:

$$V_{LEi} = \frac{1}{N} \sum_{j=1}^N \left[ \sum_{l=1}^L (a_{xjl} - \bar{a}_{xl})^2 + (a_{yjl} - \bar{a}_{yl})^2 \right] \quad (5)$$

with  $\bar{a}_{xl}$  (resp.  $\bar{a}_{yl}$ ) the population mean of maternal (resp. paternal) allelic effects at locus  $l$  in year  $i$ .

To measure the (mal)adaptation of the population, we also record in each year the population mean fitness  $\bar{w}_i$ , as well as the phenotypic lag of the population mean flowering date  $\bar{Z}_i$  to the optimal flowering time  $\theta_i$ ,  $\delta_i = \bar{Z}_i - \theta_i$ . Without lack of generality, we assume that the optimal flowering time decreases across years in our scenarios mimicking climate change (as in Franks et al., 2007). In this case, the population lag  $\delta_i$  is expected to be on average positive whenever the population lags behind the optimal flowering time.

## 2.5 | Simulated environments

Climate change is characterized by a trend of increasing temperature with strong fluctuations of temperature between years (IPCC, 2007) and is modelled here with the following changes in the optimal flowering time:

$$\theta_i = \theta_0 + k \times i + \varepsilon_i \quad (6)$$

where  $k < 0$  measures the speed of the optimum change (day/year) and  $\varepsilon_i$  is sampled independently for each year  $i$  in a Normal distribution  $\mathcal{N}(0, V_\theta)$ . The initial optimal flowering time  $\theta_0$  is, without loss of generality, set to 0. The variance  $V_\theta$  here scales the amplitude of the stochastic fluctuations in optimal flowering time across years.

Several scenarios (Table 2) are compared to understand the effects of assortative mating on adaptation: (a) scenarios in which peak flowering time is neutral (i.e.  $\omega^2$  is infinitely large), (b) scenarios with stabilizing selection in a constant environment ( $k = 0$  and  $V_\theta = 0$ ), (c) scenarios with uncorrelated fluctuations around a constant optimal flowering time in a stationary environment ( $k = 0$  and  $V_\theta > 0$ ), (d) scenarios with a linear trend through years in the optimal flowering time with no fluctuations ( $k < 0$  and  $V_\theta = 0$ ) and finally (e) scenarios including both a linear trend and fluctuations in the optimal flowering time ( $k < 0$  and  $V_\theta > 0$ ). Each scenario is replicated 10 times. Initial genotypes of plants at the start of each simulation depend on the scenario. Simulations with no selection (neutral scenario) last for 10,000 generations and start with a monomorphic population with all allelic effects set to 0 (corresponding to  $\theta_0$ ). For scenarios with constant stabilizing selection ( $k = 0$  and  $V_\theta = 0$ ), each replicate lasts for 2,000 generations and starts with the genotypes of individuals recorded at the last generation of one of the 10 replicated populations in the neutral scenario. For scenarios with environmental change ( $k < 0$  or  $V_\theta > 0$ ), each simulation lasts 2,000 generations and starts with the genotypes of individuals recorded at the last generation of one of the 10 replicated populations obtained under the same and constant strength of stabilizing selection. Population genetic variance  $V_{gi}$ , genic variance  $V_{LEi}$ , lag  $\delta_i$  and mean fitness  $\bar{w}_i$  are averaged over the last 1,000 generations of each simulation (and the last 6,000 generations for the neutral scenario), and over the 10 replicate simulations. Averaging over generations allows discarding expected fluctuations due to random genetic drift and random fluctuations of the optimal flowering time. We checked that the genetic variance measured in the simulations has approximately reached equilibrium, as indicated by its small average change per generation over the last 1,000 generations (from -0.02% to 0.03%). We report in the figures the confidence intervals for those metrics, based on their variance among the 10 replicate simulations.

## 2.6 | Simulated scenarios and parameter choice

We define a reference parameters set, for which the number of loci, the genomic mutation rate and the mutational variance correspond to published empirical estimates for flowering time (Table 2; see also references in Devaux & Lande, 2008). This parameter choice assumes that mutations are frequent with small effects on the trait and fitness, a situation for which we expect allelic effects to be distributed as a Gaussian at each locus (see comparison to analytical predictions). Empirical estimates for parameters describing selection on flowering time and how it changes with climate change are hard to obtain. The default values for the width of the fitness function and the amplitude of the fluctuations among years in the optimal time are similar to the predictions obtained from a mechanistic model (Gauzere et al., 2020) that simulates variation in natural selection on plant spring phenology along climatic gradients. The range of values for the speed of the optimum change originates from observed change in breeding values for flowering date in annual

plant populations subject to increasing drought (Franks et al., 2007; Hamann et al., 2018). We also vary the strength of stabilizing selection in a constant environment and the amplitude of the stochastic fluctuations around the optimal flowering time in fluctuating but stationary environments (Table 2). We test the robustness of our conclusions when increasing the number of loci affecting the trait, but keeping the same input of phenotypic variance through mutation per generation, both in a constant and changing climate (Table 2). In the climate change scenarios, we increase the strength of stabilizing selection and we test for the effects of variation in the mutational parameters by combining increased strength of selection, increased number of loci and rarer mutations of larger effects, a situation for which we expect the Stochastic House of Card approximation to better describe the genetic variance than does the Gaussian model (Turelli, 1984). We finally decrease the strength of assortative mating by extending the duration of flowering for each plant: each plant produces 40 flowers over 21 days with the following sequence for the number of open flowers per day {1,1,1,1,1,2,2,3,3,3,4,3,3,3,2,2,1,1,1,1,1} and corresponding to  $\alpha^2 = 22.7$ . All combinations of parameters tested are summarized in Table 2.

## 2.7 | Simulations under random mating

Our analytical predictions (see next section and Appendix S1 for their derivations) suggest that under assortative mating for flowering time the response to selection acting only on the female fitness equals that of a population under random mating with the same genetic variance, but with selection acting on both the female and male fitness. Assortative mating indeed generates indirect (sexual) selection on flowering time through the male fitness: plants flowering closer to, rather than further from, the optimal time that maximizes maternal fitness (seed viability) also sire more offspring as fathers, because they mate preferentially with more fecund mothers. Indirect selection on the male fitness due to sexual selection through assortative mating, under this selection scheme is exactly equal to direct selection on the female fitness (see Appendix S1). This result conveniently allows disentangling different effects of assortative mating on the evolution of flowering time. By comparing our simulations for the evolution of flowering time in which natural selection acts directly, and only, on the female fitness to simulations under random mating and the same selection on both the male and female fitness, we can contrast situations for which the same level of genetic variance is expected to induce the same response to selection under both assortative and random mating. Through this comparison, we can therefore test the idea that assortative mating accelerates adaptive responses through its effect on the evolution of the genetic variance.

To simulate scenarios of random mating, we assume that a given flower can be fertilized by any pollen grain emitted by flowers open any day of the year. We assume that pollen production per flower varies with days within years, with the same optimal flowering time that maximizes both pollen production and seed viability. Mothers and fathers of the next generation are drawn independently, but

randomly according to the same selection function, and then paired randomly, that is independently of day. Apart from the mating pattern and the direct selection acting on the male fitness, all other aspects of the simulations are identical in scenarios of random and assortative mating. We test for the effect of assortative mating on adaptation to changing environments by comparing the genetic variance, the lag of the population and the population mean fitness under random and assortative mating for each scenario of environmental change.

## 2.8 | Comparisons of the simulations with extant analytical predictions

To validate the simulation model, results are compared with previous analytical predictions about the genetic variance at equilibrium in a constant environment, with or without stabilizing selection. We are not aware of similar, closed-form, analytical predictions for the genetic variance in a changing environment.

### 2.8.1 | Genetic variance in the neutral case

When the trait is neutral, the genetic variance measured in the simulations is compared with its expectation at mutation-random genetic drift equilibrium under random mating (Lynch & Hill, 1986):

$$V_g = V_{LE} = 2N_e V_m \quad (7)$$

with  $N_e$  the effective population size expected to equal  $N$  the census population size.

Under assortative mating, the expected genetic variance (Devaux & Lande, 2008) is:

$$V_g = \frac{V_{LE}}{1 - h^2 \rho \left(1 - \frac{1}{2L_e}\right)} \quad (8)$$

with  $h^2 = V_g / (V_e + V_g)$  the heritability of the trait,  $\rho$  the strength of assortative mating (Equation 1),  $V_{LE}$  the genic variance at linkage equilibrium and  $L_e$  the effective number of loci for the quantitative trait,  $L_e = (\sum_{l=1}^L \sigma_l)^2 / (\sum_{l=1}^L \sigma_l^2)$  with

$$\sigma_l = \sqrt{\left(\sum_{j=1}^N (a_{xjl} - \bar{a}_l)^2 + \sum_{j=1}^N (a_{yjl} - \bar{a}_l)^2\right) / 2N}$$

the standard deviation in allelic effects for locus  $l$  (as defined in Crow & Felsenstein, 1968) and  $\bar{a}_l$  the population mean allelic effect at locus  $l$ .

### 2.8.2 | Genetic variance under random mating and stabilizing selection in a constant environment

Extant predictions about the amount of genetic variance maintained at equilibrium with Gaussian stabilizing selection, random mating, mutation and random genetic drift are approximations that depend

on assumptions about the mutation regime. The distribution of allelic effects at each locus is expected to be approximately Gaussian (Turelli, 1984) when:

$$\frac{V_m}{U} < 9\mu V_s, \quad (9)$$

with  $V_s$  the variance of the Gaussian fitness function relating fitness and breeding values for the trait.

Assuming that allelic effects are normally distributed at each locus and neglecting linkage disequilibrium in a randomly mating population, Latter (1970) predicts that the genetic variance for a polygenic trait should be well approximated by:

$$V_g(G) = \frac{L}{2N_e} \left( \sqrt{V_s} \sqrt{16N_e^2 \mu \frac{V_m}{U} + V_s} - V_s \right) \quad (10)$$

With a different mutational regime, in which rare mutations have large effects on the quantitative trait, the genetic variance is predicted by the Stochastic House of Cards approximation (Bürger, 1999):

$$V_g(\text{SHC}) = \frac{2N_e V_m}{1 + \frac{V_m N_e}{U V_s}} \quad (11)$$

According to Turelli (1984), we expect the latter approximation to be accurate when:

$$\frac{V_m}{U} > 20\mu V_s \quad (12)$$

We compare these predictions of the genetic variance from Equations 10 and 11 to that measured in our simulations, in a constant environment with random mating, varying the strength of stabilizing selection over a large range; in Figure 1, we show only the prediction that best fits the genetic variance in the simulations. We estimate  $V_s$  in our simulations assuming that the distribution of flowerers through days for any individual plant is exactly Gaussian with variance  $\alpha^2$  (see Appendix S1 and Table 2):  $V_s = V_e + \omega^2 + \alpha^2$ . We expect the genetic variance to converge towards neutral expectations (see Equations 7 and 8 above) when  $\omega^2$ , and thus  $V_s$ , are very large and stabilizing selection is very weak. We therefore vary the value of  $\omega^2$  in a constant environment to test for this prediction (Table 2).

## 2.9 | New analytical predictions for the evolution of flowering time under assortative mating in a changing environment

### 2.9.1 | Relationships between genetic variance, lag to the optimum and population mean fitness

Bürger and Lynch (1995) predict that when the population size is finite, and the optimum fluctuates around a linear trend, as in our

simulations, the lag of the mean phenotype to the optimum fluctuates from generation to generation. Yet, the expected value of this lag  $\delta$  after a large number of generations in a changing environment can be predicted as:

$$\delta = E [\bar{g}_i - \theta_i]_{i \rightarrow \infty} \rightarrow \frac{-k}{s_n} \quad (13)$$

with  $s_n = V_g / (V_g + V_s)$  a measure of the strength of stabilizing selection on the trait (see Appendix S1).

The model by Bürger and Lynch (1995) assumes random mating, a constant genetic variance and that the distribution of breeding values and phenotypic values are Gaussian. In Appendix S1, we show that the same prediction holds under (a) assortative mating, (b) the specific assumptions of our individual-based model of flowering time and (c) a Gaussian distribution of phenotypes and breeding values (which is a good approximation in our simulations, both under random and assortative mating). Equation 13 should predict the lag to the optimum, knowing the genetic variance for the adaptive trait, under both random and assortative mating, in the simulations. We therefore compare the prediction for the lag in Equation 13 to the lag averaged over the last 1,000 generations and the 10 replicate simulations in a changing environment, whereas replacing the genetic variance  $V_g$  by its averaged value over the last 1,000 generations and the 10 replicate simulations.

Bürger and Lynch (1995) also predict the relationship between  $\bar{w}$ , the expected population mean fitness asymptotically reached in a changing environment, and the population genetic variance  $V_g$ :

$$\bar{w} = E [\bar{w}_i]_{i \rightarrow \infty} \rightarrow \sqrt{\frac{\omega^2}{\bar{V}}} e^{-\frac{k^2}{2\bar{V}\bar{V}}} \quad (14)$$

with  $\bar{V} = V_g + V_s + V_\theta + V[\bar{g}_i]$ ,  $V[\bar{g}_i]$  the variance, among realizations of the stochastic evolutionary trajectories, in  $\bar{g}_i$  the mean breeding value in generation  $i$ , and again  $s_n = V_g / (V_g + V_s)$ .

To compare  $\bar{w}$  with the measured population mean fitness, we replace in Equation 14 the genetic variance  $V_g$  by its averaged value over the last 1,000 generations and the 10 replicate simulations.  $V[\bar{g}_i]$  caused by random genetic drift and environmental fluctuations in selection is replaced by the variance among the 10 replicate populations in  $\bar{z}_i$  in generation  $i$  averaged over the last 1,000 generations.  $V[\bar{g}_i]$  could be different between random and assortative mating, but the measured difference between mating patterns is here small (see Figure S1); to make the figures more readable, predictions of Equation 14 are drawn only for random mating.

### 2.9.2 | Components of the genetic variance under assortative and random mating under environmental change

We use the infinitesimal model of trait inheritance (Appendix S1, see a review in Barton et al., 2017) to predict the structure of the

genetic variance at equilibrium in our simulations, that is the relative contribution of genic variance to genetic variance. The infinitesimal model assumes that, conditional on the breeding values of the male and female parents, the distribution of breeding values in the family is distributed as a Gaussian, with a mean equal to the averaged breeding values of the two parents and with a fixed variance equal to half the genic variance in the population. The infinitesimal model should approximate well this distribution when the evolving trait is controlled by a large number of loci, each of small additive effect (Barton et al., 2017). In Appendix S1, we also assume that the distribution of breeding values in the population is Gaussian and further assume that the population is large enough to neglect random genetic drift. We modify a model by Sachdeva and Barton (2017) about the effect of assortative mating on the evolution of the genetic variance to fit the assumptions of our individual-based simulations for the evolution of flowering time. Derivations of these analytical predictions under assortative mating are presented in Appendix S1. The predicted structure of the genetic variance for flowering time under assortative mating is measured by the ratio of genic to genetic variance and is predicted to be, for all scenarios of environmental change:

$$\frac{V_{LE}}{V_g} = 1 + 2s_n - s_s \quad (15)$$

where  $s_s = V_g / (V_g + V_e + \alpha^2)$  is a measure of the strength of sexual selection.

This ratio varies between 0 and 2 in the case of assortative mating.

In comparison, with random mating, this ratio is:

$$\frac{V_{LE}}{V_g} = 1 + s_n, \quad (16)$$

and which varies between 1 and 2.

When  $V_{LE}/V_g$  is close to one, associations among allelic effects within and across loci make a small contribution to the genetic variance, as expected under random mating and weak selection. When  $V_{LE}/V_g$  is larger than one, negative associations among allelic effects depress the genetic variance below the genic variance. When  $V_{LE}/V_g$  is smaller than one, positive associations among allelic effects inflate the genetic variance above the genic variance. Note that our analytical model makes no prediction about the genic variance at equilibrium  $V_{LE}$ , whereas  $V_{LE}$  can evolve in the simulations. We replace in the expression of  $s_n$  and  $s_s$  the genetic variance  $V_g$  by its averaged value over the last 1,000 generations and the 10 replicate simulations and use Equations 15 and 16 to compare the predicted structure of the genetic variance to that measured in the simulations with the ratio  $V_{LE}/V_g$ . Due to the assumptions of the infinitesimal model, we expect these predictions to be more accurate when the number of loci is large.

### 3 | RESULTS

#### 3.1 | Strength of assortative mating in the simulations

The mean overlap of flowering times among plants decreases with increased variance in peak flowering dates among plants, that is spread of peak flowering dates within the year, and with decreased duration of flowering for individual plants  $\alpha^2$ . Because flowering duration for each plant is constant in our simulations, assortative mating, as measured by the phenotypic correlation of peak flowering dates among mates, varies with the evolving genetic variance for flowering times. In the simulations with assortative mating, the strength of assortative mating thus ranges from 0.45 to 1 when  $\alpha^2 = 4.5$ , and from 0.33 to 0.39 when  $\alpha^2 = 22.7$  (Table S1). As expected, the measured strength of assortative mating is null in the simulations with random mating (results not shown).

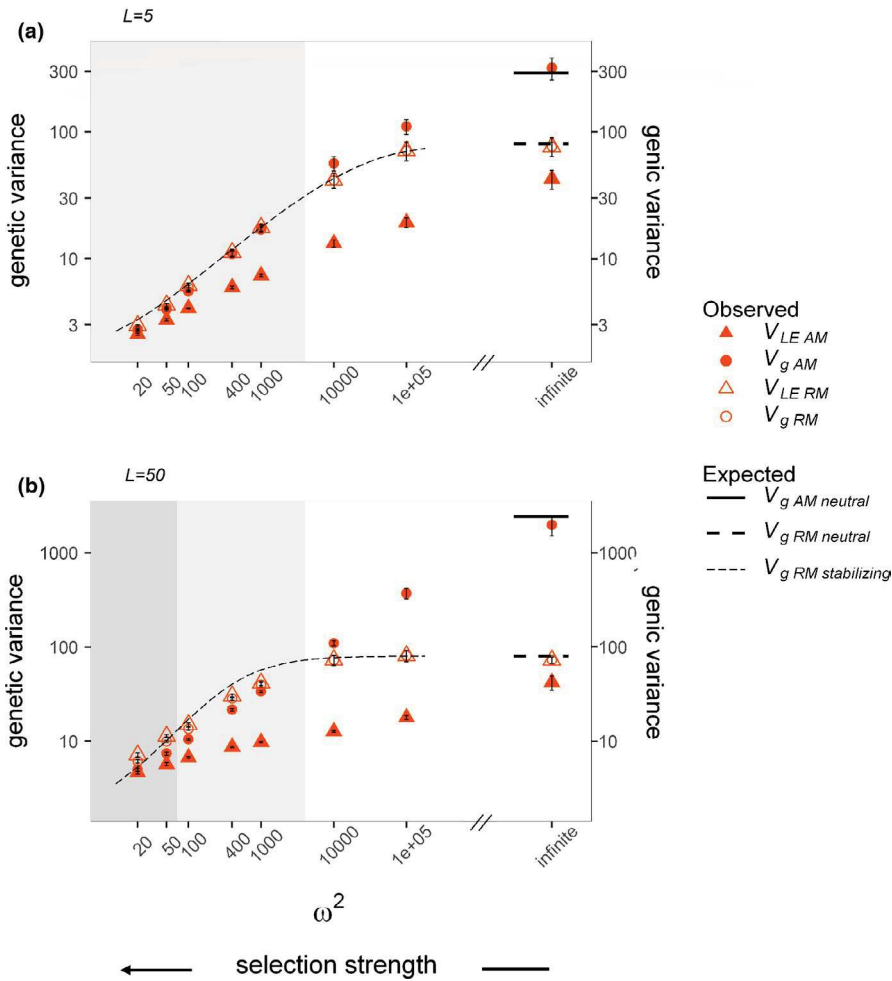
#### 3.2 | Evolution of the genetic variance in a constant environment

Overall, the genetic variances in the simulations with a constant environment match well their analytical predictions at equilibrium without selection under both mating patterns (Equations 7 and 8), and with stabilizing selection under random mating (Equation 10 for Figure 1a and Equation 11 for Figure 1b, Table 2), thus validating the code for the model.

Consistently with predictions (Equations 7 and 8 and bold dashed and solid lines in Figure 1), the genetic variance for a neutral trait is much larger under assortative than under random mating (Figure 1). For both mating patterns, the genetic variance decreases with increased stabilizing selection (lower  $\omega^2$ ). For weak stabilizing selection (i.e. strength of natural selection  $s_n < 0.01$ ; Equation 13), the genetic variance remains higher under assortative than under random mating (Figure 1). For moderate stabilizing selection ( $0.01 \leq s_n \leq 0.1$ ), the difference in genetic variance between the two mating patterns vanishes (Figure 1). For stronger stabilizing selection ( $0.1 < s_n$ ), the genetic variance in the simulations is slightly smaller under assortative than under random mating (Figure 1b).

#### 3.3 | Evolution of the genetic variance in a changing environment

Random fluctuations in the optimum have a weak effect on the evolution of the genetic variance, under both assortative and random mating (Figure 2). An optimum moving at a constant speed across generations however has noticeable effects on the evolution of the genetic variance (Figure 3). For all scenarios with a directional change in the optimum, the genetic variance is higher (up to 90%) under assortative than under random mating (Table S1). For both random and



**FIGURE 1** Mean genic (triangles, right y-axis) and genetic (circles, left y-axis) variance under assortative (filled symbols) versus random (open symbols) mating as a function of  $\omega^2$ , the width of the Gaussian function for stabilizing selection in a constant environment, and for (a) the reference genetic architecture or (b) a higher number of loci (Table 2). Symbols are means over the 10 replicate simulations whereas vertical bars are confidence intervals at 95% based on the inter-simulation variance. Predictions for the neutral case (infinite  $\omega^2$ ) are represented by the bold dashed line for random mating (Equation 7) and the solid bold line for assortative mating (Equation 8); the thin dashed line represents the prediction for random mating only from Equation 10 in (a) and Equation 11 in (b). Dark grey corresponds to  $s_n > 0.1$ , grey to  $0.01 \leq s_n \leq 0.1$  and white to  $s_n < 0.01$  (Equation 13)

assortative mating, the genetic variance peaks at some intermediate speed of the optimum change, but this peak is higher and reached for faster optimum change under assortative than under random mating (Figure 3).

### 3.4 | Components of the genetic variance

Our analytical predictions for the ratio  $V_{LE}/V_g$  of the genic to the genetic variance (Equations 15 and 16) based on the infinitesimal model of trait inheritance match relatively well the simulation results, but as expected, with more accuracy when the number of loci determining the trait is larger (Figure S2).

The structure of the total genetic variance depends on the mating pattern. Under random mating, the genetic variance is essentially composed of genic variance (Figures 1, 3 and Figure S2), as shown by the ratio  $V_{LE}/V_g$  being greater but close to one (Equation 16; see also predictions in Appendix S1). The genetic variance is indeed slightly smaller than the genic variance because of weak negative genetic associations between allelic effects within and across loci generated by stabilizing selection (Figure S2). The contribution of these negative genetic associations increases, as predicted, with increased strength of stabilizing selection, but remains small in all simulations (Figures 1,

3 and Figure S2). In contrast,  $V_{LE}/V_g$  is smaller than one in populations under assortative mating (Figure S2); the genetic variance is then larger than the genic variance because of positive genetic associations among allelic effects both within and among loci generated by assortative mating. The contribution of those associations can be large under assortative mating but it decreases, as predicted, with increased strength of stabilizing selection (Figures 1, 3 and Figure S2). Interestingly, the structure of the genetic variance is more sensitive to the strength of stabilizing selection under assortative than random mating (Figure S2; compare Equations 15 and 16). As expected, the contribution of positive genetic associations among allelic effects to the genetic variance is smaller when the duration of individual flowering phenology is longer ( $\alpha^2 = 22.7$ ) and assortative mating is weaker ( $\rho \sim 0.3$ , compare squares and circles in Figure S2).

The speed of change and the fluctuations of the optimum barely affect the structure of the genetic variance as reflected by  $V_{LE}/V_g$  (Figure S2), as also predicted by our analytical model (Equations 15 and 16). The genic variance  $V_{LE}$  is however always lower under assortative than under random mating (Figures 1 and 3), but the difference in  $V_{LE}$  between the mating patterns shrinks with faster change in the optimum (Figure 3). For both random and assortative mating, the genic variance  $V_{LE}$  peaks at the same speed of the optimum change as does the genetic variance (Figure 3).



### 3.5 | Relationship between genetic variance and mean fitness in a stationary environment

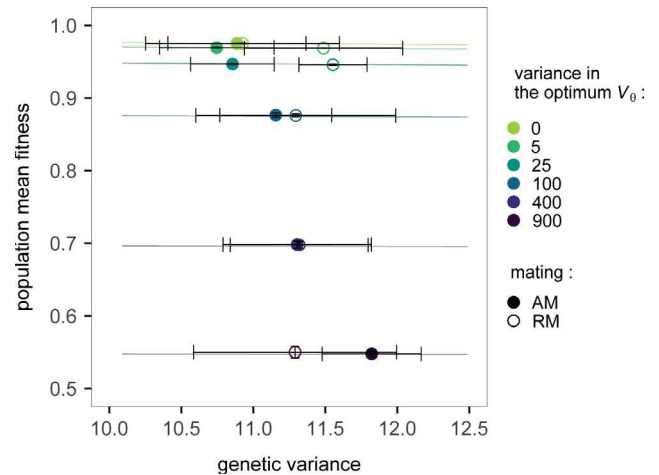
With no directional environmental change ( $k = 0$ ), the average lag of the population to the optimum is null in the simulations (results not shown), as predicted by Equation 13. In this stationary environment, the expected population mean fitness  $\bar{w}$  is predicted to decrease with higher genetic variance  $V_g$ , amplitude of the fluctuations  $V_\theta$  in the optimum, stochastic variations in mean breeding value  $V[\bar{g}_t]$  or strength of stabilizing selection (Equation 14). Again, population mean fitnesses in the simulations match closely these analytical predictions (Figure 2). Genetic variance is weakly affected by the mating pattern in a stationary environment; consequently, for a given  $V_\theta$ , the population mean fitness is similar under assortative and random mating (Figure 2 and Table S1).

### 3.6 | Relationship between genetic variance, lag and mean fitness in a directionally changing environment

The lag of the population to the optimum is predicted to decrease nonlinearly with higher genetic variance  $V_g$  (Equation 13). In particular, when the genetic variance is already high relative to  $V_s = V_e + \omega^2 + \alpha^2$ , it has little effect on the lag. For a given genetic variance, the lag is expected to increase with faster optimum change and weaker stabilizing selection (higher  $\omega^2$ ). For both random and assortative mating, the lags measured in the simulations match well their analytical predictions (Figure 4). The quality of the fit varies, but maximal differences are ~5% under random mating and ~22% under assortative mating (Figure 4). These differences are consistent with those mentioned—for random mating only—in the discussion of Bürger and Lynch (1995).

The predicted relationship between the population mean fitness and the genetic variance is nonmonotonic in a changing environment (Equation 14). Small genetic variance strongly limits adaptation and results in a large lag to the optimum; in this case, a larger genetic variance greatly decreases the lag, as well as greatly increases the mean fitness. As the lag to the optimum shrinks and is less sensitive to the genetic variance, the beneficial effect of a larger genetic variance on population mean fitness is decreased. For a very small lag to the optimum, the population mean fitness eventually decreases with larger genetic variance, as in a stationary environment. Fit between predictions and measured fitness in the simulations varies but mismatches remain small (Figure 5).

The higher genetic variance observed in the simulations under assortative mating compared to random mating allows populations to better track the optimum, as the lag to the optimum is always smaller under assortative than under random mating (Figure 4 and Table S1). In most scenarios with a directional change in the optimum, the smaller lag for populations under assortative mating results in higher population mean fitness, compared with random mating (Figure 5). This fitness advantage increases with faster optimum change (Figure 5 and Table S1). These conclusions are generally



**FIGURE 2** Population mean fitness as a function of genetic variance under assortative (filled symbols) or random (open symbols) mating for a stationary environment with  $\omega^2 = 400$ . A line is the expected relationship between fitness and genetic variance under random mating from Bürger and Lynch (1995, Equation 14) for a given variance of the optimum  $V_\theta$ . Symbols are means over the 10 replicate simulations. Horizontal and vertical bars are confidence intervals at 95% based on the inter-simulation variance. Confidence intervals for population mean fitness decreases as variance of the optimum  $V_\theta$  decreases and can be smaller than the symbol. Colours of symbols and lines change with the variance of the optimum  $V_\theta$  from 0 to 900

robust to changes in the strength of assortative mating and stabilizing selection, the number of loci and the mutational input in the trait, but the sizes of the lag and the fitness advantage of assortative mating compared with random mating vary (Figures 4 and 5): this fitness advantage ranges from -0.7% to almost 100% (Table S1). The size of the fitness advantage of assortative mating compared with random mating depends on whether genetic variance under random mating strongly limits adaptation (Figure 5 and Table S1). For example, under strong stabilizing selection, the lag to the optimum is small even for small genetic variance, and assortative mating provides a modest advantage in adaptation to a changing environment compared to random mating (Figures 4e,f, 5e,f and Table S1). When the genetic variance is large, such as with a large number of loci determining the trait, and when the optimum changes slowly, an increase in genetic variance actually depresses population mean fitness, as predicted (Equation 14). In this case, mean fitness can be smaller in populations under assortative compared with random mating, but differences are very small (0.7%; Figures 4d, 5d and Table S1).

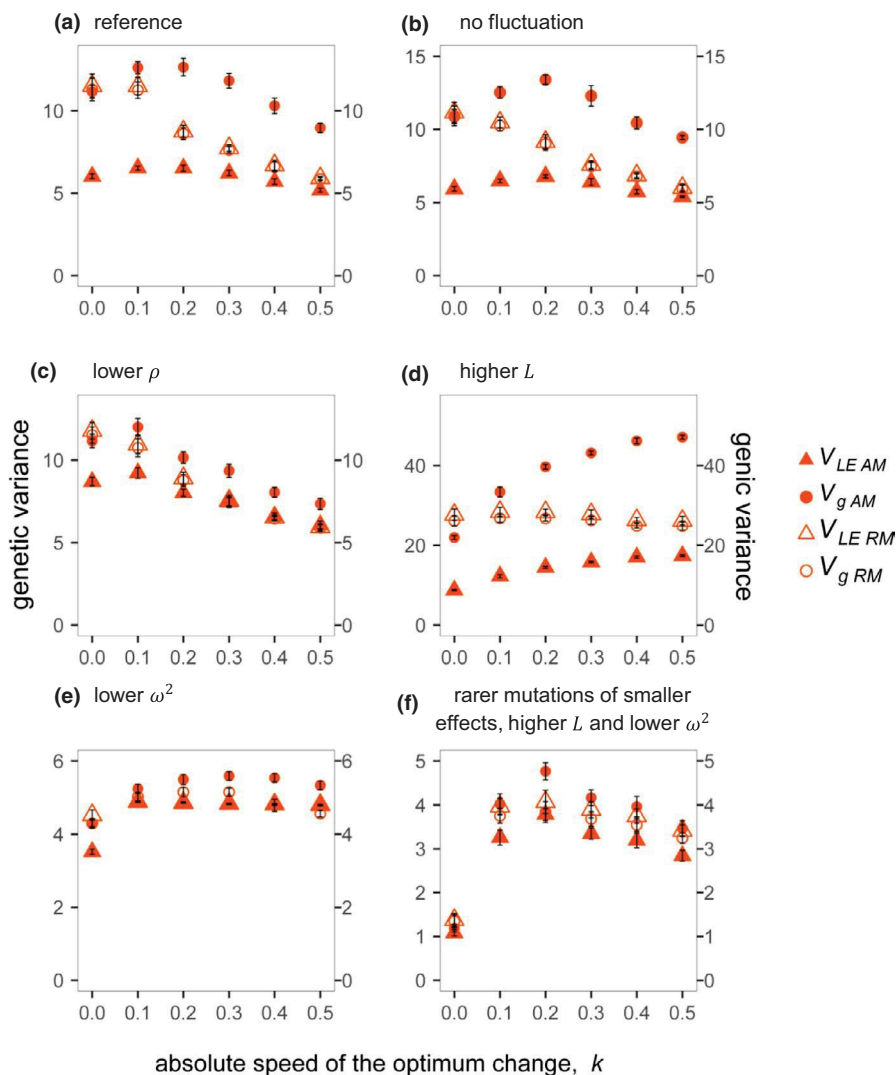
## 4 | DISCUSSION

Rapid evolutionary changes in flowering phenology in response to climate change have been repeatedly reported (Ashworth et al., 2016; Franks et al., 2007, 2014; Hamann et al., 2018;

Lustenhouwer et al., 2018; Merilä & Hendry, 2014). We here test whether assortative mating for traits affecting flowering phenology could be responsible for these observations. Our simulations show that assortative mating has antagonistic effects on the evolution of the genetic variance whenever there is stabilizing selection: compared with random mating, assortative mating depresses the level of genetic polymorphism at each locus (i.e. the genic variance) but generates positive genetic associations among allelic effects both among and within loci. In a stationary environment with substantial stabilizing selection, the genetic variance at equilibrium is comparable between random and assortative mating, and assortative mating provides no or little adaptive advantage. When the environment changes with a trend, genetic variance is larger under assortative than under random mating and this reduces the lag of the population to the optimum and improves its mean fitness, with only a few exceptions. We discuss below the mechanisms explaining these patterns, and the implications for our understanding of adaptive responses of flowering time and other traits to climate change.

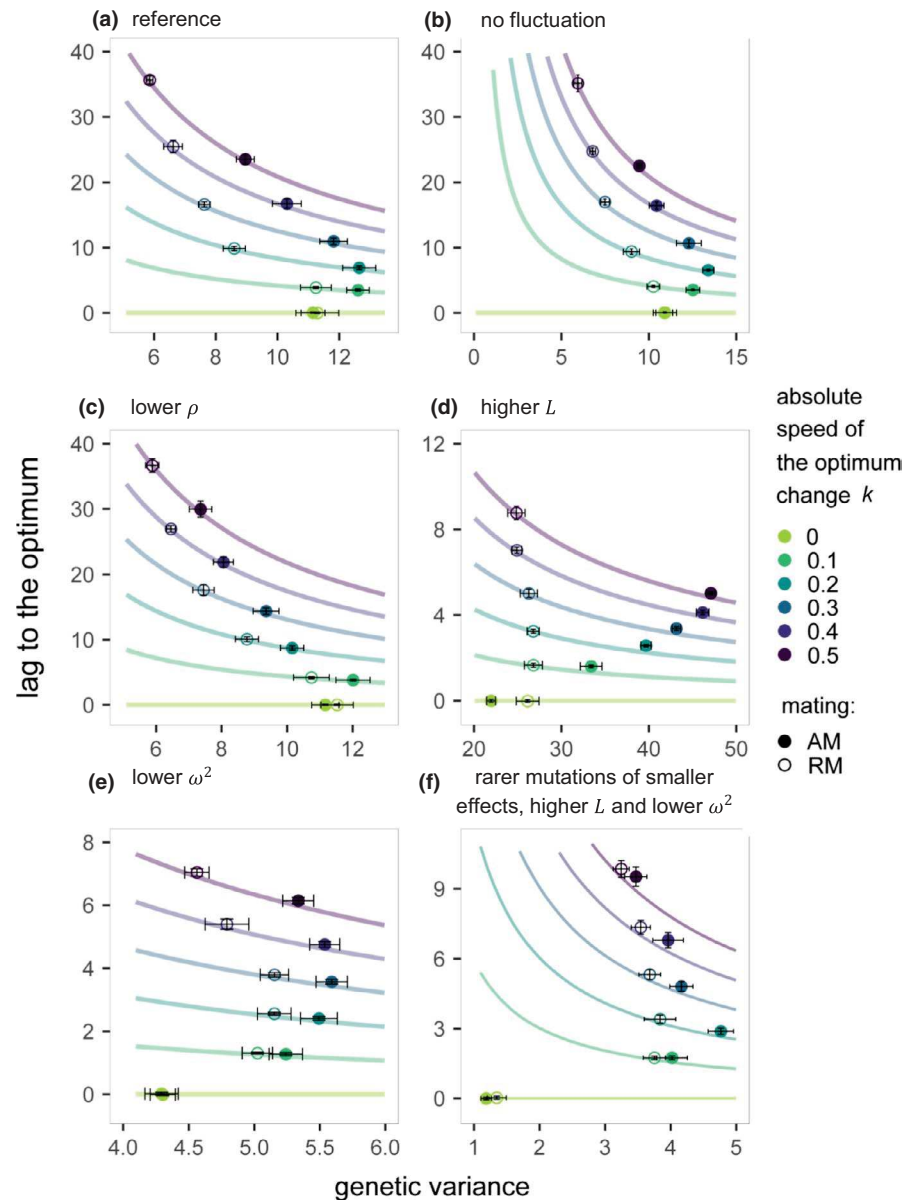
#### 4.1 | The structure of genetic variance is affected differently by assortative and random mating

Our simulations and analytical model show that genic variance composes a smaller part of genetic variance under assortative than under random mating, especially when stabilizing selection is weak and assortative mating is strong. Such results are consistent with previous theory (Crow & Felsenstein, 1968; Crow & Kimura, 1970; Devaux & Lande, 2008; Fox, 2003; Lande, 1977; Weis et al., 2005; Wright, 1921). Our analytical model shows, surprisingly, that the contribution of genic to genetic variance is little affected by the speed of the optimum change (Equation 15 and Figure S2). Genetic variance under assortative mating is much larger than genic variance, because it is mainly composed of positive associations between allelic effects both across (positive linkage disequilibrium) and within (heterozygote deficit) loci. For random mating, genic variance can be larger than genetic variance because of negative associations between allelic effects generated by stabilizing selection (Bulmer effect, Bulmer, 1971). Our analytical model shows that assortative



**FIGURE 3** Mean genic (triangles; right y-axis) and genetic (circles; left y-axis) variance as a function of the absolute speed of the optimum change  $k$  (days per generation), under assortative (filled symbols) and random (open symbols) mating for (a) the reference case, (b) no fluctuations in the optimum ( $V_0 = 0$ ), (c) weaker assortative mating  $\rho$  ( $\alpha^2 = 22.7$ ), (d) higher number of loci  $L$  ( $L = 50$ ), (e) stronger stabilizing selection with lower  $\omega^2$  ( $\omega^2 = 50$ ) and (f) rarer mutations of smaller effects, higher  $L$  and lower  $\omega^2$  ( $U = 0.01$ ;  $V_m = 0.004$ ;  $L = 50$ ;  $\omega^2 = 50$ ; see Table 2)

**FIGURE 4** Lag of the population to the optimum under assortative (filled symbols) and random mating (open symbols) as a function of the genetic variance for (a) the reference case, (b) no fluctuations in the optimum ( $V_{\theta} = 0$ ), (c) weaker assortative mating  $\rho$  ( $\alpha^2 = 22.7$ ), (d) higher number of loci  $L$  ( $L = 50$ ), (e) stronger stabilizing selection with lower  $\omega^2$  ( $\omega^2 = 50$ ) and (f) rarer mutations of smaller effects, higher  $L$  and lower  $\omega^2$  ( $U = 0.01$ ;  $V_m = 0.004$ ;  $L = 50$ ;  $\omega^2 = 50$ ; see Table 2). A line is the expected relationship between lag and genetic variance under random mating from Bürger and Lynch (1995, Equation 13) for a given absolute speed of the optimum change  $k$ . The same prediction holds for assortative mating. Symbols are means over the 10 replicate simulations. Horizontal and vertical bars are confidence intervals at 95% based on the inter-simulation variance. Colours of lines and symbols change with the absolute speed of the optimum change  $k$ . Note the differences in scales for the y-axes and the x-axes among panels



mating also generates stabilizing sexual selection by favouring fathers with flowering times most similar to that of the most fecund mothers. This stabilizing sexual selection adds up to stabilizing natural selection and reduces the contribution of positive associations among allelic effects to genetic variance (Equation 15 and Equation S27 of Appendix S1). Our analytical model however shows that positive effects of assortative mating dominate its negative effects on the relative contribution of these associations when assortative mating is strong compared with stabilizing natural selection, as observed in the simulations. Kirkpatrick and Nuismer (2004) have also found that assortative mating generates additional stabilizing selection, which is caused in their model by the limited mating potential for individual with rare phenotypes, and that sexual stabilizing selection constrains the contribution of positive associations to the genetic variance.

If genetic variance were constant and comparable for the two mating patterns, as assumed in our analytical model, in all scenarios

genetic variance would be higher under assortative than under random mating (Equations S27 and S28 of Appendix S1). Such a prediction is however not observed in the simulations. Genetic variance in the simulations is smaller under assortative than under random mating and is affected by the scenario of optimum change (Figures 1 and 3). Fully understanding the evolution of genetic variance under assortative mating thus requires understanding the evolution of genic variance. Mechanisms explaining the evolution of genic variance under assortative mating in constant and changing environments are discussed in the next sections below.

#### 4.2 | Assortative mating has little effects in a constant or stationary environment

The lower genetic variance under assortative than random mating observed in the simulations with a constant environment likely

results from the additional stabilizing sexual selection on males that decreases the level of polymorphism at mutation–selection–drift balance, as shown in Kirkpatrick and Nuismer (2004). Given that the part of the genetic variance due to associations among allelic effects scales to the genic variance (Equation 15), a reduced polymorphism depresses both components of the genetic variance. In a constant environment with strong stabilizing selection ( $s_n > 0.1$ ; Equation 13), the genetic variance under assortative mating is slightly smaller than that under random mating (Figure 1b), because the negative effects of assortative mating on genic variance are not entirely compensated by its positive effects on associations among allelic effects. For moderate stabilizing selection ( $0.01 \leq s_n \leq 0.1$ ), the genetic variance under assortative mating is similar to that under random mating, showing that negative and positive effects of assortative mating balance each other. The same conclusion holds in a stationary environment in which the optimum fluctuates around a constant value across generations. These findings are consistent with the predictions of Lande (1977) that assortative mating does not affect the genetic variance at mutation–selection equilibrium, as long as assortative mating and linkage disequilibrium are not too strong, and stabilizing selection is not too weak. In a constant environment, the genetic variance under assortative mating is much larger than that under random mating only when stabilizing selection is weak ( $s_n < 0.01$ ) and thus approaching neutral conditions (Crow & Kimura, 1970; Devaux & Lande, 2008; Wright, 1921). We further find that the population mean fitness in a constant or stationary environment is little affected by the mating pattern, either because the genetic variances are comparable between the two mating patterns (when selection is moderate), or because the load due to stabilizing selection is very small when selection is weak.

#### 4.3 | Genetic variance is larger for assortative than for random mating in a changing environment

In a changing environment, the genetic variance is systematically larger, and the genic variance smaller, under assortative than under random mating. Genic and genetic variances peak at different speeds of the optimum change for random and assortative mating (Figure 3). The bell-shaped relationship between genetic variance and speed of the optimum change has already been described in simulations with random mating (Bürger, 1999; Bürger & Lynch, 1995). The increase of genic variance in slowly changing environments has been interpreted as resulting from increasing frequency of initially rare beneficial alleles (Bürger, 1999). With assortative mating, this increase of genic variance is amplified by the production of positive associations among allelic effects, resulting in larger genetic variance than under random mating. The decline in genic variance for rapidly changing environments and random mating is interpreted as a consequence of increased random genetic drift in declining populations lagging far behind their optimum (Bürger, 1999; Bürger & Lynch, 1995). The effective

population size may then be affected by the lag to the optimal value, which is larger under random than under assortative mating (see next section); it could explain that the genetic variance declines for slower change in the optimum for random mating than it does for assortative mating.

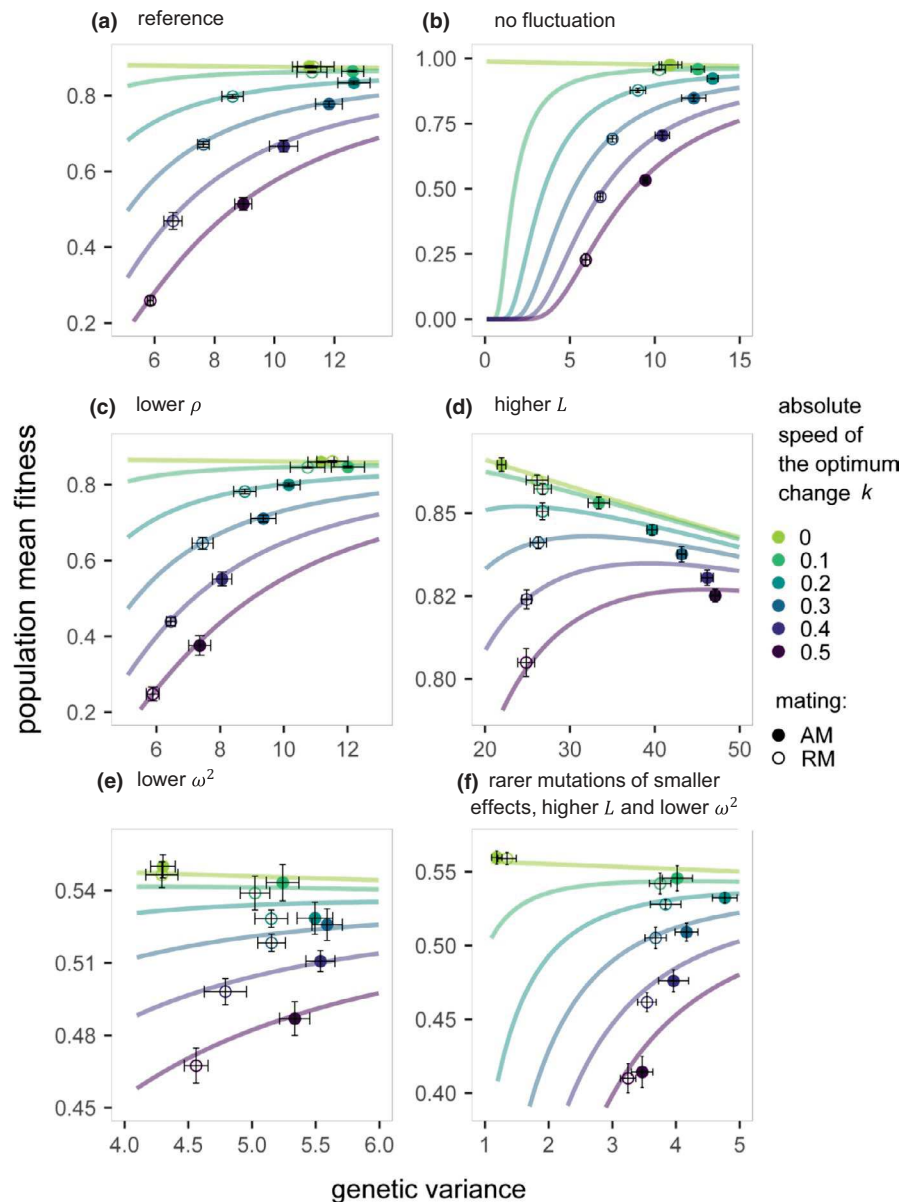
#### 4.4 | Increased genetic variance explains the fitness advantage of assortative mating in a changing environment

In a constantly changing environment with a linear trend, previous theory predicts that populations track the moving optimum, but with a constant lag at equilibrium (Bürger, 1999; Bürger & Lynch, 1995; Charlesworth, 1993; Kopp & Matuszewski, 2014; Lande & Shannon, 1996; Lynch et al., 1991, 1993). This equilibrium lag is larger for faster environmental change, but also when weaker stabilizing selection or lower genetic variance decreases the response to selection. Our analytical model for flowering time with assortative mating shows that selection acting on female fecundity generates indirect selection on male fitness (additional stabilizing sexual selection). Interestingly in this model, the equilibrium lag has the same expression and dependence on genetic variance as in a model with random mating and direct selection on both male and female fitness (Equation 13 or Equation S22, Appendix S1). As the genetic variance is systematically higher under assortative than under random mating in a directionally changing environment in our simulations, this expression (Equation 13) predicts a smaller lag in adaptation for a trait under assortative mating than for a trait under random mating; our simulation results match this prediction very well (Figure 4). This finding thus generalizes to a polygenic trait the conclusion that temporal assortative mating accelerates response to directional selection obtained by Fox (2003) and Weis et al. (2005) in single-locus models.

The difference in lag between random and assortative mating is often small whenever the lag is already small, that is when the optimum change is slow, genetic variance is large or stabilizing selection is strong (Figure 4 and Table S1). Previous analytical models of artificial directional selection on a quantitative trait predict that assortative mating has small effects on the genetic response to selection compared to random mating, when selection is strong, but, contrary to this study, also when heritability is low (Baker, 1973; De Lange, 1974; Shepherd & Kinghorn, 1994; Smith & Hammond, 1897; Tallis & Leppard, 1987). The gain of genetic response under assortative mating compared with random mating was also found to be small in experiments of artificial selection, and significant only in a few studies (Breese, 1956; McBride & Robertson, 1963, but not in Campo & Garcia, 1994; Garcia & Sanchez, 1992; Sutherland et al., 1968; Wilson et al., 1965).

There is an optimal genetic variance that maximizes population mean fitness in a changing environment (Figure 5). On one hand, mean fitness increases with genetic variance because increased genetic variance reduces the lag between the mean phenotype

**FIGURE 5** Population mean fitness under assortative (filled symbols) and random mating (open symbols) as a function of the genetic variance for (a) the reference case, (b) no fluctuations in the optimum ( $V_{\theta} = 0$ ), (c) weaker assortative mating  $\rho$  ( $\alpha^2 = 22.7$ ), (d) higher number of loci  $L$  ( $L = 50$ ), (e) stronger stabilizing selection with lower  $\omega^2$  ( $\omega^2 = 50$ ) and (f) rarer mutations of smaller effects, higher  $L$  and lower  $\omega^2$  ( $U = 0.01$ ;  $V_m = 0.004$ ;  $L = 50$ ;  $\omega^2 = 50$ ; see Table 2). A line is the expected relationship between fitness and genetic variance under random mating from Bürger and Lynch (1995, Equation 14) for a given absolute speed of the optimum change  $k$ . The expected relationship between fitness and genetic variance under assortative mating is not displayed because the difference among predicted fitness under random and assortative mating is very small. Symbols are means over the 10 replicate simulations. Horizontal and vertical bars are confidence intervals at 95% based on the inter-simulation variance. Colours of lines and symbols change with the absolute speed of the optimum change  $k$ . Note the differences in scales for the y-axes and the x-axes among panels



and the optimum (Lande & Shannon, 1996). On the other hand, population mean fitness decreases as genetic variance increases, because of a load induced by individuals deviating from the optimum (Bürger & Lynch, 1995; Lande & Shannon, 1996). In most scenarios of environmental change explored, the beneficial effects of assortative mating compared with random mating dominate the evolution of population mean fitness. The difference in population mean fitness between assortative and random mating increases with faster optimum change and weaker stabilizing selection (Figure 5). In a rapidly warming climate with a longer favourable season (Barichivich et al., 2013; Vitasse et al., 2011), and thus weaker stabilizing selection, the fitness advantage conferred by assortative mating compared to random mating could be large, and critical to population persistence. In the scenarios for which genetic variance does not limit adaptation, and the lag is already small under random mating, the beneficial effects of assortative mating on mean fitness can be small.

#### 4.5 | Limitations of the model

Flowering time is typically a plastic trait, responsive to temperature (Anderson et al., 2012; Nicotra et al., 2010). This plasticity is partially adaptive, helping flowering time to get closer to the optimum (Donohue et al., 2000; Franks et al., 2014), and plasticity can evolve (Franks et al., 2014; Hamann et al., 2018). We here do not model explicitly such plastic responses, but our conclusions should still hold if we assume partially adaptive plastic responses to environmental cues indicative of optimal phenotypes, with no genetic variation for the slope of the reaction norm (making flowering of all genotypes respond similarly to the cue). Such a change simply amounts to rescaling the optimal value for the trait and interpreting it as the optimal breeding value (rather than the optimal phenotypic value) for flowering time.

Our model of flowering time evolution assumes temporal assortative mating, the same flowering duration for all plants,

synchronous male and female flowering, female fitness varying through time within years and no direct selection on male flowering. Under these assumptions, our analytical derivations highlight that the response to sexual selection on fathers is exactly equal to the response to natural selection on mothers (Equation S20, Appendix S1). The same prediction would hold if selection on male fitness also varied with time within the year (Equation S14 and see discussion in Appendix S1), since fathers compete to access mates only with other fathers flowering at the same time. Although we expect the fitness advantage of assortative mating in a changing environment to hold for reproductive time in animals (fishes: Kirkpatrick & Selander, 1979; birds: Friesen et al., 2007; corals: Tomaiuolo et al., 2007), we believe that changing the type of assortative mating, the life history traits affected by selection and the difference in phenology between male and female organs could however alter our quantitative conclusions about the lag to the optimum and the genetic variance maintained under assortative mating. Temporal assortative mating, in which males compete only with those flowering at the same time, was claimed to maintain higher genetic variance for flowering time than for traits under other types of assortative mating (Fox, 2003; Kopp et al., 2018; Weis et al., 2005).

#### 4.6 | Biological implications

Climate change is expected to affect individual flowering phenologies and their distributions in populations (Anderson et al., 2012; Franks et al., 2007, 2014; Hamann et al., 2018; Inouye, 2008; Morin et al., 2007) and thus to affect the strength of assortative mating in natural populations (Devaux & Lande, 2008; Weis et al., 2005). Results here suggest that decreasing the strength of assortative mating, for example by lengthening individual flowering duration, would rapidly decrease genetic variance by shrinking positive associations among allelic effects, but would eventually maintain increased genetic polymorphism for flowering time. In contrast, increasing assortative mating, for example by shortening individual flowering duration, would rapidly increase genetic variance by creating large positive associations among allelic effects. But in the long term, increased assortative mating would depress genetic polymorphism. In the context of evolutionary rescue, in which a population avoid extinction by adapting to a stressful environment causing its decline (Gomulkiewicz & Holt, 1995), increasing genetic variance in the short term by increasing assortative mating over a few generations should be greatly beneficial.

## 5 | CONCLUSIONS

Assortative mating increases genetic responses of flowering time to climate change as compared to a trait under random mating. This mating pattern in plants may explain why fast genetic evolution

of flowering time has been repeatedly measured in response to a warming climate, whereas empirical evidence for genetic adaptation to climate change remains in general rare (Merilä & Hendry, 2014). The fitness advantage conferred by assortative mating for flowering time compared with random mating in an isolated population is explained by the increase of the population genetic variance in scenarios of climate change tested here. The magnitude of this fitness advantage is however quite variable and can be small in some scenarios, casting doubt on the general ability of assortative mating for flowering time to rescue populations from extinction in a changing climate. Our results also suggest that theory on adaptive responses to selection under random mating could be used for traits under assortative mating to predict the fate of natural populations, as done by Gienapp et al. (2013) for the evolution of laying date in birds under different scenarios of climate change. This would allow knowing whether genetic variance limits adaptation, without having to know the strength of assortative mating.

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#### CONFLICT OF INTEREST

The authors have no conflict of interest to declare.

#### AUTHOR CONTRIBUTIONS

All authors designed the study, contributed to the interpretation of results and CG wrote the paper with the contribution from CD and OR. CD and CG wrote the simulation script and CG run simulations. OR wrote the analytical model with the contribution from CD and CG.

#### DATA AVAILABILITY STATEMENT

The script (.cdf) simulating the evolution of populations under assortative and random mating is available at: <https://doi.org/10.5061/dryad.zs7h44j85>. Please contact the corresponding author for the notebook (.nb).

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## SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section.

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## 2.2 Appendix 1

## 1 SUPPORTING INFORMATIONS

2 **Table S1: Parameter values and metrics measured in the simulations under assortative (AM) and random (RM) mating.** Number of loci  $L$ , individual  
 3 variance in flowering time  $\alpha^2$  (in  $days^2$ ), width of the Gaussian function for stabilizing selection  $\omega^2$  (in  $days^2$ ), speed of the optimum change  $k$  (in  
 4  $day/generation$ ), variance in the fluctuations of the optimum  $V_\theta$  (in  $days^2$ ), genomic mutation rate  $U$ , and mutational variance  $V_m$  (in  $days^2$ ); grey cells  
 5 correspond to the same value as for the reference case (first line). Mean phenotypic correlation among mates  $\rho$  measured under AM, differences between RM  
 6 and AM in genetic variance ( $dV_g$ ), lag ( $d\delta$ ), and population mean fitness ( $d\bar{w}$ ) scaled by the corresponding value for RM; advantages for AM correspond to  
 7 positive values for  $dV_g$  and  $d\bar{w}$ , but negative values for  $d\delta$ . \* indicates that for  $k = 0$ , lags  $\delta$  are close to zero compared to  $\omega^2$ , such that  $d\delta$  can be artificially  
 8 large and are thus not reported.

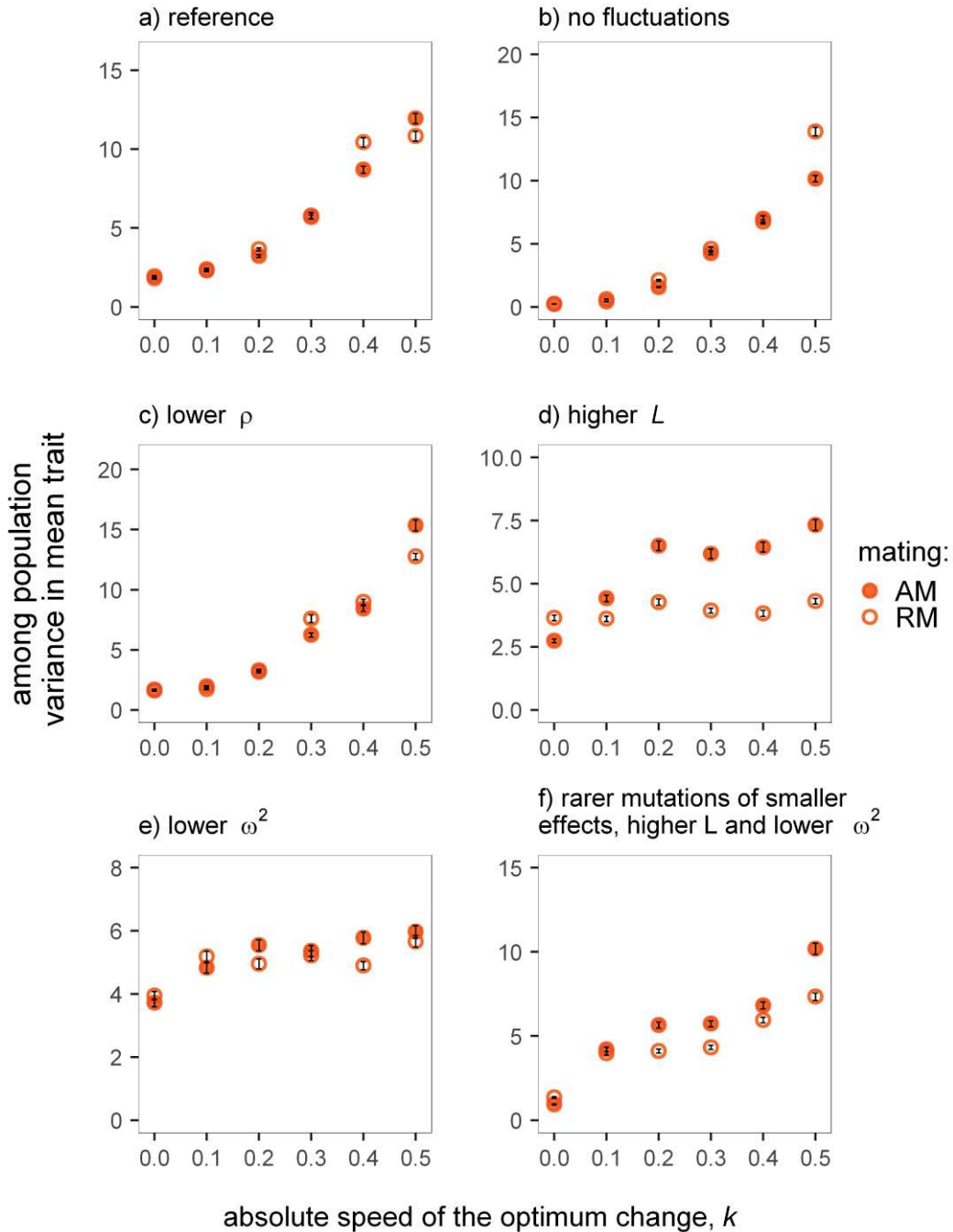
case	number of loci $L$	individual variance in flowering time $\alpha^2$	width of the Gaussian fitness function $\omega^2$	speed of the optimum change $k$	variance in the fluctuations of the optimum $V_\theta$	genomic mutation rate $U$	mutational variance $V_m$	phenotypic correlation among mates under AM $\rho_{AM}$	differences between RM and AM in genetic variance $dV_g$ (%)	differences between RM and AM in lag $d\delta$ (%)	differences between RM and AM in population mean fitness $d\bar{w}$ (%)
reference	5	4.5	400	0	100	0.1	0.04	0.75	-1.2	*	0.0
				-0.1				0.78	12.1	-10.1	0.2
				-0.2				0.78	47.1	-30.1	4.5
				-0.3				0.77	54.9	-33.9	15.9
				-0.4				0.75	55.8	-34.3	42.0
				-0.5				0.73	53.0	-34.0	98.5
constant environment	grey	grey	20	0	0	grey	0.45	-2.5	*	0.1	
			50				0.56	-3.3	*	0.1	
			100				0.63	-6.8	*	0.2	
			400				0.74	-0.4	*	0.0	
			1000				0.80	-2.8	*	0.0	
			10000				0.92	35.8	*	-0.1	
			100000				0.95	54.8	*	-0.1	
			infinite				0.97	317.2	*	0.0	

9

case	number of loci $L$	individual variance in flowering time $\alpha^2$	width of the Gaussian fitness function $\omega^2$	speed of the optimum change $k$	variance in the fluctuations of the optimum $V_\theta$	genomic mutation rate $U$	mutational variance $V_m$	phenotypic correlation among mates under AM $\rho_{AM}$	differences between RM and AM in genetic variance $dV_g$ (%)	differences between RM and AM in lag $d\delta$ (%)	differences between RM and AM in population mean fitness $d\bar{w}$ (%)	
constant environment and higher number of loci	50		20	0	0			0.53	-19.1	*	1.7	
			50					0.65	-25.8	*	1.9	
			100					0.73	-22.5	*	1.3	
			400					0.83	-23.9	*	0.8	
			1000					0.89	-15.3	*	0.3	
			10000					0.96	51.2	*	-0.2	
			100000					0.99	359.2	*	-0.1	
infinite	1.00	2623.8	*	0.0								
stationary environment				0				0	-0.4	*	0.0	
								5	0.74	-6.5	*	0.0
								25	0.74	-6.0	*	0.1
								100	0.75	-1.2	*	0.1
								400	0.75	-0.1	*	0.1
								900	0.75	4.7	*	-0.4
no fluctuations				0	0			0.74	-0.4	*	0.0	
				-0.1				0.76	22.1	-12.8	0.2	
				-0.2				0.77	48.8	-30.3	5.1	
				-0.3				0.75	64.0	-37.2	22.6	
				-0.4				0.73	53.8	-33.7	50.1	
				-0.5				0.72	59.0	-35.9	134.7	
weaker assortative mating		22.7		0				0.38	-3.1	*	-0.2	
				-0.1				0.39	11.8	-9.2	0.1	
				-0.2				0.36	15.7	-13.3	2.3	
				-0.3				0.35	25.6	-18.4	10.1	
				-0.4				0.34	24.9	-18.8	25.6	
				-0.5				0.33	25.1	-18.3	51.6	

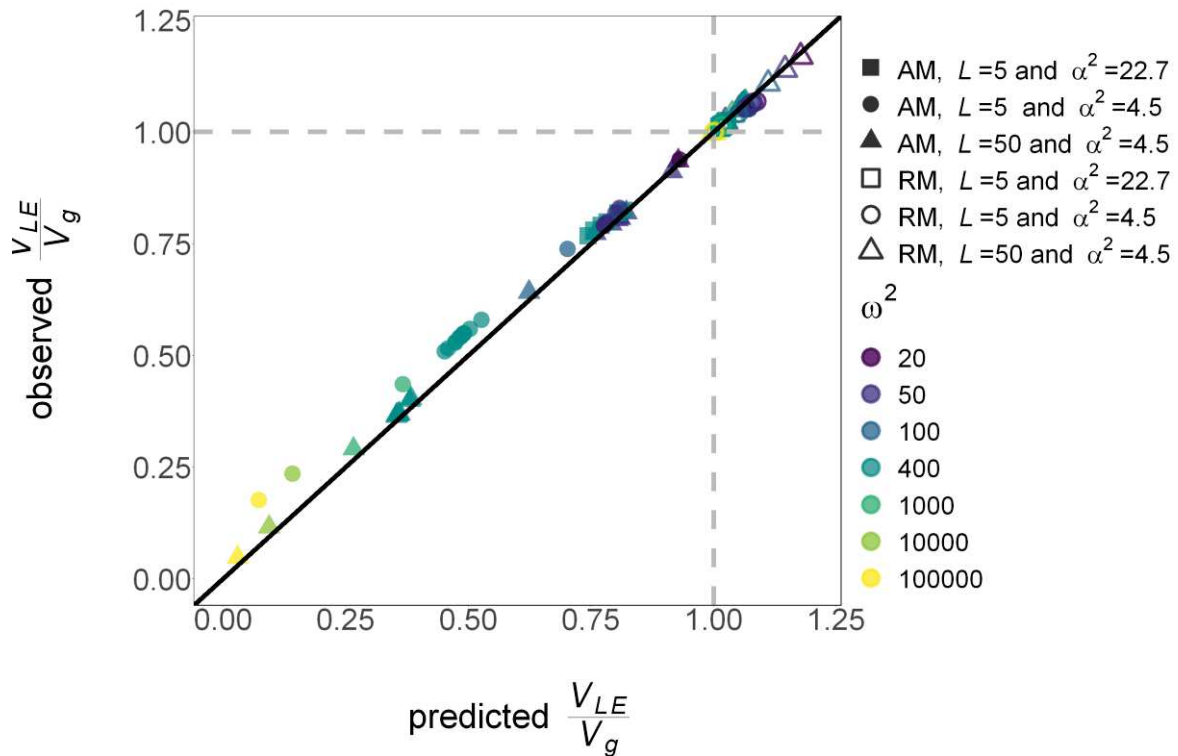
case	number of loci $L$	individual variance in flowering time $\alpha^2$	width of the Gaussian fitness function $\omega^2$	speed of the optimum change $k$	variance in the fluctuations of the optimum $V_\theta$	genomic mutation rate $U$	mutational variance $V_m$	phenotypic correlation among mates under AM $\rho_{AM}$	differences between RM and AM in genetic variance $dV_g$ (%)	differences between RM and AM in lag $d\delta$ (%)	differences between RM and AM in population mean fitness $d\bar{w}$ (%)
higher number of loci	50			0				0.83	-16.0	*	0.5
				-0.1				0.87	24.7	-3.4	-0.5
				-0.2				0.89	48.2	-20.7	-0.7
				-0.3				0.90	64.2	-32.7	-0.4
				-0.4				0.90	85.3	-41.4	0.8
				-0.5				0.90	89.5	-42.6	2.5
stronger stabilizing selection			50	0				0.58	0.2	*	0.6
				-0.1				0.60	4.3	-2.5	0.8
				-0.2				0.60	6.6	-5.8	0.0
				-0.3				0.61	8.5	-5.8	1.5
				-0.4				0.61	15.6	-11.9	2.5
				-0.5				0.60	16.9	-12.7	4.2
rarer mutations of smaller effects. higher number of loci and stronger stabilizing selection	50		50	0		0.01	0.004	0.51	-12.0	*	0.1
				-0.1				0.60	7.2	-0.2	0.7
				-0.2				0.61	24.2	-15.3	0.8
				-0.3				0.60	13.2	-9.6	0.8
				-0.4				0.60	11.8	-7.5	3.1
				-0.5				0.59	6.8	-3.4	1.1

12



13

14 **Figure S1:** Among population variance in mean trait under assortative (filled symbols) and random  
 15 (open symbols) mating as a function of the absolute speed of the optimum change per generation  $k$ , for  
 16 (a) the reference case, (b) no fluctuations of the optimum ( $V_\theta = 0$ ), (c) weaker assortative mating  $\rho$   
 17 ( $\alpha^2 = 22.5$ ), (d) higher number of loci  $L$  ( $L = 50$ ), (e) lower  $\omega^2$  and thus increased stabilizing selection  
 18 ( $\omega^2 = 50$ ), (f) rarer mutations of smaller effects, higher  $L$  and lower  $\omega^2$  ( $U = 0.01$ ,  $V_m = 0.0004$ ,  $L = 50$ ,  
 19  $\omega^2 = 50$ , see Table 2). This variance is the average (over the last 1000 generations) variance of the  
 20 mean trait  $z$  among the 10 replicate populations. It depends on the speed of the optimum change and  
 21 is little affected by the mating pattern. Vertical bars correspond to confidence interval at 95% based on  
 22 the inter-population variance.



23

24 **Figure S2:** Observed (measured) versus predicted ratio of the genic to the genetic variance  $V_{LE}/V_g$ ; note  
 25 that  $V_g$  measured in the simulated populations is used to compute the strength of stabilizing selection  
 26 and heritability in the predicted ratio  $V_{LE}/V_g$  (eqn 15 and eqn 16). Filled or open symbols for assortative  
 27 or random mating, respectively; squares for number of loci  $L = 5$  and low assortative mating ( $\alpha^2 = 22.7$   
 28 days<sup>2</sup>), circles for number of loci  $L = 5$  and strong assortative mating ( $\alpha^2 = 4.5$  days<sup>2</sup>), and triangles for  
 29 number of loci  $L = 50$  and strong assortative mating ( $\alpha^2 = 4.5$  days<sup>2</sup>). Colours correspond to different  
 30 widths of the Gaussian function for stabilizing selection  $\omega^2$ . Estimates for simulations with different  
 31 speeds of the optimum change are indicated with the same symbol and colour; note that they overlap  
 32 in many instances and hide that  $V_{LE}/V_g < 1$  for assortative mating. Black line for  $x = y$  and grey dashed  
 33 lines for  $V_{LE} = V_g$ .

**Table S2: List of symbols for the Appendix S1 with their description**

Symbol	Description
$z$ ( $x$ or $y$ )	phenotypic value (for females or males) drawn from a Normal distribution of expectation $\bar{z}$ and variance $V_z = V_g + V_e$
$e$	micro-environmental effect drawn from a Normal distribution of null expectation and variance $V_e$
$g$	breeding value drawn from a Normal distribution of expectation $\bar{g}$ and variance $V_g$
$\bar{g}'_{im}$ (or $\bar{g}'_{if}$ )	population mean breeding value of mothers (or potential fathers in the pollen pool) after natural selection in year $i$
$V'_{gm}$ (or $V'_{gf}$ )	genetic variance among potential mothers (or fathers) in the population after natural selection in year $i$
$cov'_m(g, z)$	covariance between breeding $g$ and phenotypic $z$ values among selected mothers
$r_{gf, z_f}$	regression coefficient of the breeding value over the phenotypic value, in males
$p_i(g, z)$	joint distribution of breeding $g$ and phenotypic $z$ values in year $i$ with mean $\mu_i$ and variance $\Sigma$
$p'_{im}(g_m, x)$	joint distribution of breeding $g_m$ and phenotypic $x$ values in year $i$ for mothers after natural selection
$p_i(g, \cdot)$	marginal distribution of genotypes $g$ in the population in year $i$
$p_i(\cdot, z)$	marginal distribution of phenotypes $z$ in the population in year $i$
$v_i(t)$	probability to produce viable seeds at day $t$ and year $i$ with optimum $\theta_i$ and width $\omega^2$
$w_i(x)$	total female fitness in year $i$ of a plant with peak flowering time $x$
$\bar{w}_{im}$	population mean fitness of mothers in year $i$
$S_n$	a measure of the strength of natural selection on peak flowering time
$S_s$	a measure of the strength of sexual selection on peak flowering time
$S_m$	a measure of the strength of natural selection on reproductive phenologies of individual mothers
$s_n$	a measure of the strength of natural selection on breeding values for peak flowering time
$s_s$	a measure of the strength of sexual selection on breeding values for peak flowering time
$\bar{s}$	a measure scaling the ratio of genic to genetic variance, which depends on both natural and sexual selection
$\Phi(t z)$	flowering phenology at day $t$ of a plant with peak flowering time $z$
$\tilde{\Phi}_{im}(t x)$	reproductive phenology at day $t$ of a plant with peak flowering time $x$
$\Psi_i(y x)$	probability that a plant with peak flowering time $y$ mates with, and sires, an offspring of a mother with peak flowering time $x$
$R(g g_m, g_f)$	probability that an offspring has a breeding value $g$ given the breeding values $g_m$ and $g_f$ of its mother and father
$\eta(z g)$	conditional distribution of phenotypes $z$ given the breeding values $g$
$\varphi_i(g_m, g_f)$	joint distribution of breeding values $g_m$ for mothers and $g_f$ for fathers of the same offspring in year $i$
$h_i(g_f x)$	probability that the father has the genotype $g_f$ given the phenotype $x$ of the mother in year $i$
$\gamma_i(g_f y)$	probability that, in year $i$ , a father has a breeding value $g_f$ given its phenotype $y$

36 **Appendix S1: Changes in genetic variance and mean flowering time under the**  
37 **infinitesimal model with a Gaussian distribution of breeding values**

38 We derive predictions for the deterministic changes across generations in mean and  
39 variance in flowering time, assuming a Gaussian distribution of breeding values and  
40 phenotypes, and an infinitesimal model of trait inheritance. We use these equations to  
41 predict at equilibrium and for several patterns of environmental changes: (i) the mean  
42 lag of the population to the optimal flowering time, given its genetic variance, (ii) the  
43 population mean fitness, given its genetic variance, (iii) the level of genetic variance in  
44 flowering time, given the genic variance (the genetic variance at linkage and Hardy-  
45 Weinberg equilibrium) in the population. We here make the same assumptions about  
46 patterns of selection on flowering time as in our individual-based simulations, and build  
47 on the model in the Appendix S4 of Sachdeva & Barton (2017) by including the  
48 following new components: (i) partial heritability of the phenotype, (ii) temporal  
49 assortative mating, (iii) different effects of selection on male and female fitness, (iv)  
50 fitness of mothers varying with time within a year, and (v) changes in selection across  
51 years. Our derivations also extend to a scenario of temporal assortative mating the  
52 predictions from Bürger & Lynch (1995) for the lag and the population mean fitness  
53 obtained under random mating in a changing environment.

54 In our model, individuals are hermaphrodite, male and female flowering are  
55 synchronous within individuals, each generation is completed within a single year, and  
56 climatic conditions within that year determine the optimal flowering time, which varies  
57 across years. In the following, we derive the changes from one generation to the next  
58 in the distribution of the breeding values for peak flowering time due to (i) natural  
59 selection on seed viability, (ii) sexual selection generated by assortative mating  
60 between mates, and (iii) recombination. If we assume that the distributions of breeding



61 and phenotypic values are initially Gaussian, under the assumptions of our model and  
62 the infinitesimal model of trait inheritance, we show that these distributions remain  
63 Gaussian through the life cycle and in the next generation. Natural and sexual selection  
64 have distinct effects on the fitness of mother and father individuals that contribute to  
65 the next generation: the distribution of breeding and phenotypic values of peak  
66 flowering time can thus differ between mothers and fathers of the next generation;  
67 changes in these distributions are thus derived separately. We use the indices  $m$  and  
68  $f$  throughout the derivations to denote mothers and fathers, respectively. All notations  
69 and their description are summarized in Table 1 and S2.

70

### 71 ***Effective female reproductive phenology***

72 The flowering phenology of an individual is described by the number of flowers it opens  
73 each day  $t$  within a year; this number is assumed to be distributed through time as a  
74 Gaussian function with peak flowering time  $z$  (phenotypic value consisting of a real  
75 number) and variance  $\alpha^2$ . The proportion of open flowers on day  $t$  for an individual with  
76 peak flowering time  $z$  is then:

$$77 \quad \Phi(t|z) = \frac{1}{\sqrt{2\pi\alpha^2}} e^{-\frac{(t-z)^2}{2\alpha^2}} \quad \text{S1}$$

78 where  $\alpha$  scales the duration of the individual flowering phenology. If each individual  
79 flowers for a longer time ( $\alpha$  is larger), chances that flowering will overlap between  
80 individuals with different peak flowering times are higher and assortative mating will be  
81 weaker.

82 As in the individual-based simulations, each flower produces a different number  
83 of viable seeds depending on the day  $t$  it is open and pollinated. The optimal flowering  
84 time that maximizes the number of viable seeds per flower can vary between years;  
85 this number of viable seeds is proportional to:

86 
$$v_i(t) = e^{-\frac{(t-\theta_i)^2}{2\omega^2}}$$
 S2

87 where  $\theta_i$  is the optimal flowering time in year  $i$  and  $\omega$  is related to the duration of the  
88 favourable flowering season, which is here assumed to be constant across years.

89 Given its flowering phenology  $\Phi(t|x)$ , an individual plant with a peak flowering  
90 time  $x$  has a total female fitness proportional to the total number of seeds it produces  
91 in year  $i$ , which depends on  $x$  :

92 
$$w_i(x) = \int v_i(t)\Phi(t|x)dt = \sqrt{\frac{\omega^2}{\omega^2+\alpha^2}} e^{-\frac{(x-\theta_i)^2}{2(\alpha^2+\omega^2)}}$$
 S3

93

94 Eqn S3 can be derived using results about products of Gaussian functions. The  
95 variation in seed production per flower with day  $t$  within years affects the reproductive  
96 phenology of a mother contributing to the next generation, i.e. modifies the effective  
97 reproductive phenology. Let  $\tilde{\Phi}_{im}(t|x)$  be the distribution of viable seeds through days  
98  $t$  produced by a mother with peak flowering time  $x$  in year  $i$ :

99 
$$\tilde{\Phi}_{im}(t|x) = \frac{v_i(t)\Phi(t|x)}{w_i(x)}$$
 S4

100 This function  $\tilde{\Phi}_{im}(t|x)$  has also a Gaussian shape with mean and variance:

101 
$$E_{\tilde{\Phi}_{im}}(t|x) = x + S_m(\theta_i - x)$$
 S5

102 
$$V_{\tilde{\Phi}_{im}}(t|x) = \alpha^2(1 - S_m)$$
 S6

103 with  $S_m = \frac{\alpha^2}{\omega^2+\alpha^2}$  a measure of the strength of the effect of natural selection on the  
104 individual reproductive phenology of mothers.

105 Variation through days  $t$  in the number of viable seeds per flower thus results in  
106 (i) a shift in the effective peak of reproduction away from the flowering peak of the  
107 mother and closer to the optimal flowering time (eqn S5), and (ii) a reduction in the

108 duration of the effective reproduction for the mother plant compared to the duration of  
 109 its flowering (eqn S6).

110 As in our individual-based simulations, we assume that the quantity and quality  
 111 of pollen emitted by a single flower does not change with days within years.

112

113 ***Effect of natural selection on the distributions of breeding values and***  
 114 ***phenotypes***

115 Peak flowering time is a quantitative trait, such that the phenotypic value  $z$  of an  
 116 individual is the sum of its breeding value and a random environmental effect:

$$117 \quad z = g + e \quad \text{S7}$$

118

119 We assume a Gaussian distribution of breeding values for peak flowering time in the  
 120 population in year  $i$ , with mean  $\bar{g}_i$  and variance  $V_g$ . We also assume that the genetic  
 121 variance  $V_g$  for peak flowering time has reached equilibrium in the population and is  
 122 constant across years, but it varies within a year as a result of natural selection,  
 123 assortative mating and recombination. At birth, environmental effects are distributed  
 124 as a Gaussian with mean 0 and variance  $V_e$ . We therefore have a joint Gaussian  
 125 distribution of phenotypic and breeding values, with a probability density function

$$126 \quad p_i(g, z), \text{ with mean } \mu_i = \begin{pmatrix} \bar{g}_i \\ \bar{g}_i \end{pmatrix} \text{ and variance-covariance matrix } \Sigma = \begin{pmatrix} V_g & V_g \\ V_g & V_g + V_e \end{pmatrix} \text{ at the}$$

127 beginning of year  $i$ . Under these assumptions and using results about the product of  
 128 Gaussian functions, the mean fitness of mothers in the population is (see also e.g.  
 129 Bürger & Lynch, 1995; Cotto & Chevin, 2020):

$$130 \quad \bar{w}_{im} = \int w_i(z) p_i(\cdot, z) dz = \sqrt{\frac{\omega^2}{V_g + V_e + \omega^2 + \alpha^2}} e^{-\frac{(\bar{z}_i - \theta_i)^2}{2(V_g + V_e + \omega^2 + \alpha^2)}} \quad \text{S8}$$

131 with  $p_i(\cdot, z) = \int p_i(g, z)dg$  the marginal distribution of phenotypic values  $z$  in the  
 132 population in year  $i$ .

133 Recall that we assume that all males contribute equally to the pollen pool. After  
 134 natural selection, the joint distribution of breeding and phenotypic values for peak  
 135 flowering time is Gaussian for both mothers ( $p'_{im}(g, z)$ ) and potential fathers ( $p'_{if}(g, z)$ )  
 136 contributing to the next generation, but they have different means and variances.  
 137 These means and variances can be computed from linear and quadratic selection  
 138 gradients, as classically done in quantitative genetics models assuming a Gaussian  
 139 distribution of phenotypes and breeding values (see **proof 1** at the end of the  
 140 Appendix). Let  $S_n = \frac{V_g + V_e}{V_g + V_e + \omega^2 + \alpha^2}$  be a measure of the strength of stabilizing selection  
 141 on peak flowering time, and  $h^2 = V_g / (V_g + V_e)$  the heritability of the trait. We find that,  
 142 after natural selection, the mean breeding value of mothers among parents in the next  
 143 generation is:

$$144 \quad \bar{g}'_{im} = \bar{g}_i + h^2 S_n (\theta_i - \bar{z}_i)$$

145 Noting that, initially,  $\bar{z}_i = \bar{g}_i$ , the mean breeding value of mothers is:

$$146 \quad \bar{g}'_{im} = \bar{g}_i + h^2 S_n (\theta_i - \bar{g}_i) \quad \text{S9}$$

147 The variance of breeding values among mothers is:

$$148 \quad V'_{gm} = V_g (1 - h^2 S_n) \quad \text{S10}$$

149 The mean phenotypic value and phenotypic variance of mothers are:

$$150 \quad \bar{z}'_{im} = \bar{g}_i + S_n (\theta_i - \bar{g}_i) \quad \text{S11}$$

$$151 \quad V'_{zm} = (V_g + V_e) (1 - S_n) \quad \text{S12}$$

152 The covariance of breeding and phenotypic values among selected mothers is:

$$153 \quad cov'_m(g, z) = V_g (1 - S_n) \quad \text{S13}$$

154 For potential fathers in the pollen pool, we have:

$$155 \quad \bar{g}'_{if} = \bar{z}'_{if} = \bar{g}_i, V'_{gf} = V_g, V'_{zf} = V_g + V_e, \text{ and } cov'_f(g, z) = V_g$$

156 ***Assortative mating through flowering phenology***

157 Mating occurs at random between flowers that are open on the same day. Competition  
158 between potential fathers for access to mothers thus occurs within days. The  
159 probability that a plant with peak flowering time  $y$  sires an offspring of a mother with  
160 peak flowering time  $x$  is:

161 
$$\Psi_i(y|x) = \int \tilde{\Phi}_{im}(t|x) \frac{p_i(\cdot, y)\Phi(t|y)}{\int p_i(\cdot, z)\Phi(t|z)dz} dt \quad \text{S14}$$

162 where  $p_i(\cdot, z) = \int p_i(g, z)dg$  is the marginal distribution of phenotypes  $z$  among plants  
163 in the population in year  $i$ . The term  $\tilde{\Phi}_{im}(t|x)$  describes the proportion of all seeds  
164 mothered by a plant with peak flowering time  $x$  that is fertilized on day  $t$ . The fraction  
165 describes the probability that a father with phenotype  $y$  fertilizes any of these seeds.  
166 The denominator describes that all fathers with open flowers on that day  $t$  are in  
167 competition for pollinating the open flowers of the mother. Plants with more open  
168 flowers on that day  $t$  have a higher chance to sire the seeds fertilized on that day.

169 Note that eqn S14 would also hold if the quantity or quality of pollen emitted by a  
170 flower depended on days within years, considering that all flowers open on the same  
171 day emitted the same quantity and quality of pollen. The dependence of the male  
172 fitness with days would appear in both the numerator and denominator of the fraction  
173 in eqn S14 and would thus cancel out. Therefore, the results below not only apply to  
174 the case of no direct natural selection on male flowering phenology, but also to a case  
175 with selection on male fitness similar to that assumed for female fitness, which gives  
176 more generality to our conclusions. Ultimately, this is due to the fact that the seeds  
177 produced by all mothers along all days within the year compete to establish in the  
178 population, and form the next generation, while pollen grains compete to pollinate  
179 flowers only with other pollen grains emitted on the same day. The total pollen  
180 production of a plant over the whole year becomes irrelevant whenever competition for

181 mates occurs only within a single day and male fitness varies with day. Results would  
 182 however differ if pollen production per flower would differ among plants that flower on  
 183 the same day.

184 Given that the distribution of peak flowering times among fathers is Gaussian,  
 185 using results about Gaussian function products, we can show that  $\Psi_i(y|x)$  has a  
 186 Gaussian shape with mean and variance:

$$187 \quad E_{\Psi_i}(y|x) = \bar{g}_i + S_s(x + S_m(\theta_i - x) - \bar{g}_i) \quad \text{S15}$$

$$188 \quad V_{\Psi_i}(y|x) = (V_g + V_e)(1 - S_s)(1 + S_s(1 - S_m)) \quad \text{S16}$$

189 with  $S_s = \frac{V_g + V_e}{V_g + V_e + \alpha^2}$  a measure of the strength of sexual selection acting on peak  
 190 flowering times of the fathers.

191 Increasing  $\alpha^2$  increases the duration of flowering and thus the overlap in  
 192 flowering among individuals. As a consequence increasing  $\alpha^2$  decreases the strength  
 193 of sexual selection  $S_s$ . Fathers mated with mothers with a given flowering time have a  
 194 mean flowering time shifted towards the peak of the effective reproductive phenology  
 195 of the mothers (eqn S15). The variance in peak flowering time among those fathers  
 196 mated with a mother with a given peak flowering time is furthermore reduced by sexual  
 197 selection  $S_s$  (eqn S16). This effect is stronger when the duration of flowering is shorter  
 198 (i.e.  $S_s$  is larger) and when the duration of the effective reproduction of mothers is even  
 199 shorter due to natural selection (i.e. when  $S_m$  is larger). For comparison, in the absence  
 200 of natural selection on seed viability (i.e. with  $S_m = 0$ ), we would have  $V_{\Psi_i}(y|x) = (V_g +$   
 201  $V_e)(1 - S_s^2)$ .

202

### 203 ***Distribution of breeding values in the offspring generation***

204 The infinitesimal model assumes that, conditional on the breeding value of the father  
 205 and mother parents, the distribution of breeding values in the family is distributed as a

206 Gaussian, with a mean equal to the averaged breeding values of the two parents, and  
 207 a fixed variance equal to half the genic variance in the population  $V_{LE}/2$ . The  
 208 infinitesimal model approximates well the distribution of breeding values within a family  
 209 when the evolving trait is controlled by a large number of loci, each of small additive  
 210 effects (Bulmer, 1980; Tufto, 2000 and see a review in Barton et al. 2017). The  
 211 probability that an offspring has a breeding value  $g$  given the breeding values  $g_f$  and  
 212  $g_m$  of its father and mother, respectively, is then:

$$213 \quad R(g|g_m, g_f) = \frac{1}{\sqrt{\pi V_{LE}}} e^{-\frac{\left(g - \frac{g_m + g_f}{2}\right)^2}{V_{LE}}} \quad S17$$

214 At the scale of the population, we obtain the joint distribution of breeding and  
 215 phenotypic values of all offspring in the next generation as:

$$216 \quad p_{i+1}(g, z) = \eta(z|g) \iint R(g|g_m, g_f) \varphi_i(g_m, g_f) dg_f dg_m \quad S18$$

217 where  $\eta(z|g)$  is the probability that the offspring has phenotype  $z$  given its breeding  
 218 value  $g$  (which is distributed as a Gaussian centered on  $g$  with variance  $V_e$ ), and  
 219  $\varphi_i(g_m, g_f)$  is the joint distribution of the breeding values of the mother and father of the  
 220 same offspring in year  $i$ . With assortative mating, the breeding values of parents  
 221 covary.

222 After integration (see **proof 2** at the end of the Appendix), we obtain that the joint  
 223 distribution of breeding and phenotypic values in the next generation is Gaussian with  
 224 mean breeding and phenotypic values as:

$$225 \quad \bar{g}_{i+1} = \bar{z}_{i+1} = \frac{1}{2} \bar{g}'_{im} + \frac{1}{2} \left( \bar{g}'_{if} + h^2 S_s (\bar{z}'_{im} + S_m (\theta_i - \bar{z}'_{im}) - \bar{z}'_{if}) \right) \quad S19$$

226 The mean breeding values of offspring is the average of the breeding values of mothers  
 227 (first term) and fathers (second term) after natural and sexual selection. The sexual  
 228 selection term (in factor of  $S_s$ ) only affects the mean breeding value of fathers, and  
 229 depends on the difference between the mean flowering peak of potential fathers ( $\bar{z}'_{if}$ )

230 and the time at which a maximal number of viable seeds will be produced by mothers  
 231 (as given by  $\bar{z}'_{im} + S_m(\theta_i - \bar{z}'_{im})$ ). We show in **proof 2** at the end of the Appendix that,  
 232 once we replace mean breeding and phenotypic values of fathers and mothers after  
 233 natural selection, eqn S19 can be rearranged as:

$$234 \quad \bar{g}_{i+1} = \bar{z}_{i+1} = \frac{1}{2}(\bar{g}_i + h^2 S_n(\theta_i - \bar{g}_i)) + \frac{1}{2}(\bar{g}_i + h^2 S_n(\theta_i - \bar{g}_i)) \quad \text{S20}$$

235 Eqn S20 shows that indirect selection on fathers due to sexual selection through  
 236 temporal assortative mating, under our selection scheme, results in a selection  
 237 response in fathers (second term) exactly equal to the selection response in mothers  
 238 (first term), despite the fact that we have no natural selection affecting male fitness.

239 We thus have at the beginning of the next year:

$$240 \quad \bar{g}_{i+1} = \bar{g}_i + s_n(\theta_i - \bar{g}_i) \quad \text{with } s_n = h^2 S_n \quad \text{S21}$$

241 The change in mean breeding values is therefore the same as under random mating  
 242 with the same selection on male and female fitness, although here mating is assortative  
 243 and natural selection only acts on female fitness. Note that this conclusion would not  
 244 necessarily hold with other forms of selection on flowering time (e.g. if flowering time  
 245 affected the total fecundity of females, but not the distribution of viable seeds during a  
 246 female reproductive season, results not shown).

247

### 248 ***Evolutionary lag and mean fitness at equilibrium***

249 Eqn S21 is the same as eqn 4 in Bürger & Lynch (1995), which was derived under the  
 250 assumption of random mating. As in our individual-based simulations, Bürger & Lynch  
 251 (1995) consider a population evolving in an environment in which the optimal  
 252 phenotype fluctuates stochastically around a linear trend through generations. They  
 253 assume constant genetic variance and constant strength of stabilizing selection around  
 254 the optimum. The similarity of eqn S21 with their result for the expected change in



255 breeding values across generations allows the use of their predictions for the expected  
 256 asymptotic lag of the population (between mean and optimal phenotype; equation 8b  
 257 in Bürger & Lynch, 1995) and for the expected population mean fitness, once in its  
 258 asymptotic regime (equation 9 in Bürger & Lynch, 1995).

259 As in the individual-based simulations (and as in Bürger & Lynch, 1995), we  
 260 assume that  $\theta_i = k \times i + \varepsilon_i$ , with  $\varepsilon_i$  drawn in a Gaussian distribution with mean 0 and  
 261 variance  $V_\theta$ . We can then predict that the expected asymptotic lag for peak flowering  
 262 time in our model is the same as the one in a randomly mating population with selection  
 263 acting both on male and female fitness, given the genetic variance at equilibrium:

$$264 \quad \delta = E[\bar{g}_i - \theta_i] \xrightarrow{i \rightarrow \infty} \frac{-k}{s_n} \quad \text{S22}$$

265 In the asymptotic regime, the lag fluctuates around this expected value among different  
 266 stochastic realizations of the evolutionary trajectories.

267 The expected mean population fitness is predicted to be:

$$268 \quad \bar{w} = E[\bar{w}_i] \xrightarrow{i \rightarrow \infty} \sqrt{\frac{\omega^2}{\tilde{V}}} e^{-\frac{k^2}{2s_n^2 \tilde{V}}} \quad \text{S23}$$

269 with  $\tilde{V} = V_g + V_e + \alpha^2 + \omega^2 + V_\theta + V[\bar{g}_i]$ , and  $V[\bar{g}_i]$  the variance, among realizations of  
 270 the stochastic evolutionary trajectories, in the mean breeding value in year  $i$ .

271

## 272 **Genetic variance at equilibrium**

273 By integrating eqn S18 we also find the genetic variance among the offspring in the  
 274 next generation (see **proof 2** at the end of the Appendix):

$$275 \quad V_g'' = \frac{V_{LE}}{2} + \frac{V'_{gm}}{4} + \frac{V'_{gf}}{4} - \frac{h^4(V'_{zf} - V_{\psi_i}(y|x))}{4} + \frac{h^4 S_s^2 (1 - S_m)^2 V'_{zm}}{4} + \frac{h^4 S_s (1 - S_m) V'_{zm}}{2} \quad \text{S24}$$

276 Sexual selection  $S_s$  decreases the genetic variance in fathers contributing to the next  
 277 generation, as shown by the fourth negative term, which depends on the difference in  
 278 phenotypic variance among fathers before and after sexual selection. Yet, assortative

279 mating also inflates the genetic variance (due to positive genetic correlations between  
 280 mates), as shown by the last two positive terms in the sum, and which depend on the  
 281 phenotypic variance among mothers. In the absence of sexual selection ( $S_s = 0$ ) and  
 282 as expected, the genetic variance in the next generation is simply  $V_g'' = \frac{V_{LE}}{2} + \frac{V_{gm}'}{4} + \frac{V_{gf}'}{4}$ .

283 Replacing  $V_{\psi_i}(y|x)$  and the variances after selection by their values (eqn S10,  
 284 S12, and S16), and noting that  $S_s(S_n + (1 - S_n)S_m) = S_n$ , we find the genetic variance  
 285 in the next generation:

$$286 \quad V_g'' = \frac{V_{LE}}{2} + \frac{V_g}{2}(1 - \tilde{s}) \quad \text{with } \tilde{s} = h^2(2S_n - S_s) \quad \text{S25}$$

287 For comparison, the change in genetic variance under random mating and selection  
 288 on male and female fitness is:

$$289 \quad V_g'' = \frac{V_{LE}}{2} + \frac{V_g}{2}(1 - s_n) \quad \text{S26}$$

290 Assortative mating for flowering time in our model has therefore antagonistic effects  
 291 on the evolution of the genetic variance: the first positive term in  $\tilde{s}$  suggests that  
 292 assortative mating enhances the effect of natural stabilizing selection of decreasing  
 293 the genetic variance, by doubling its intensity in comparison to random mating with  
 294 selection on both sexes ( $2S_n$ ); the second negative term in  $\tilde{s}$ , which depends directly  
 295 on the strength of sexual selection ( $S_s$ ), suggests an effect of assortative mating of  
 296 increasing genetic variance.

297 At equilibrium, we have  $V_g'' = V_g$  and thus the part of the genetic variance made of  
 298 genic variance (the genetic variance at linkage equilibrium) is:

$$299 \quad \frac{V_{LE}}{V_g} = 1 + \tilde{s} \quad \text{S27}$$

300 This equation defines the genetic variance only implicitly as  $\tilde{s}$  also depends on  $V_g$ . It  
 301 however suggests that the structure of the genetic variance (the fraction of the total  
 302 genetic variance made of genic variance) should be similarly shaped by the strength

303 of natural and sexual selection independently of the scenario for the environmental  
304 change. With random mating and selection on male and female fitness this ratio  
305 equals:

$$306 \quad \frac{V_{LE}}{V_g} = 1 + s_n \quad \text{S28}$$

307 As  $s_n$  is strictly positive, the ratio is always above one under random mating and  
308 stabilizing selection: the genic variance exceeds the genetic variance, because of the  
309 negative associations between allelic effects generated by stabilizing selection.  
310 Conversely under assortative mating,  $\tilde{s}$  can become negative, which predicts that the  
311 genetic variance can be larger than the genic variance, because of the positive  
312 associations between allelic values across and within loci. The coefficient  $\tilde{s}$  is negative  
313 when  $V_g + V_e + \alpha^2 < \omega^2$ , which happens if (i) the duration of the favorable flowering  
314 season, as scaled by  $\omega^2$ , is long and natural selection on flowering time not very strong,  
315 and (ii) the duration of individual flowering, as scaled by  $\alpha^2$ , is short and sexual  
316 selection is strong.

317 **Proof 1:**

318 To derive eqn S9-13, we use the following results (see e.g. Cotto & Chevin, 2020) for

319 the means  $\mu'_i$  and variance-covariance matrix  $\Sigma'$  after an episode of selection:

$$320 \mu'_i = \mu_i + \Sigma \beta_i \text{ with } \beta_i = \begin{pmatrix} \frac{\partial \ln(\bar{w}_i)}{\partial \bar{g}_i} \\ \frac{\partial \ln(\bar{w}_i)}{\partial \bar{z}_i} \end{pmatrix} \text{ and } \Sigma' = \Sigma + \Sigma^T H_i \Sigma \text{ with } H_i = \begin{pmatrix} \frac{\partial^2 \ln(\bar{w}_i)}{\partial \bar{g}_i^2} & \frac{\partial^2 \ln(\bar{w}_i)}{\partial \bar{z}_i \partial \bar{g}_i} \\ \frac{\partial^2 \ln(\bar{w}_i)}{\partial \bar{z}_i \partial \bar{g}_i} & \frac{\partial^2 \ln(\bar{w}_i)}{\partial \bar{z}_i^2} \end{pmatrix}.$$

321

322 **Proof 2:**

323 We can rewrite eqn S18 as:

$$324 p_{i+1}(g, z) = \eta(z|g)p_{i+1}(g, \cdot)$$

325 with the marginal distribution of breeding values in the offspring generation given as:

$$326 p_{i+1}(g, \cdot) = \iint R(g|g_m, g_f) \varphi_i(g_m, g_f) dg_f dg_m$$

327 where  $R(g|g_m, g_f)$  is the probability that an offspring has a breeding value  $g$  given the

328 breeding values  $g_m$  and  $g_f$  of its mother and father, and  $\varphi_i(g_m, g_f)$  is the joint

329 distribution of the breeding values of the mothers and fathers of the same offspring in

330 year  $i$ . Under assortative mating,  $\varphi_i(g_m, g_f)$  can be written as:

$$331 \varphi_i(g_m, g_f) = \int p'_{im}(g_m, x) h_i(g_f|x) dx$$

332 where  $p'_{im}(g_m, x)$  is the probability that a mother after natural selection has breeding

333 value  $g_m$  and phenotypic value  $x$  (see eqn S9 to S12 for moments of this joint

334 distribution of breeding and phenotypic values), and  $h_i(g_f|x)$  is the probability that a

335 father has the genotype  $g_f$  given the phenotype  $x$  of a mother in a pair of parents. The

336 latter probability is given by:

$$337 h_i(g_f|x) = \int \gamma_i(g_f|y) \Psi_i(y|x) dy$$

338 where  $\Psi_i(y|x)$  is the probability that a plant with a peak flowering time  $y$  sires an  
 339 offspring of a mother with a peak flowering time  $x$  (see eqn S14), and  $\gamma_i(g_f|y)$  is the  
 340 probability that, in year  $i$ , a father has a breeding value  $g_f$  given its phenotype  $y$ . The  
 341 conditional distribution  $\gamma_i(g_f|y)$  has a Gaussian shape with mean and variance for  
 342 fathers:

$$343 \quad E_{\gamma_i}(g_f|y) = \bar{g}'_{if} + r_{g_f,z_f}(y - \bar{z}'_{if})$$

$$344 \quad V_{\gamma_i}(g_f|y) = V'_{gf} - r_{g_f,z_f}^2 V'_{zf}$$

345 where  $r_{g_f,z_f}$  is the regression coefficient of the breeding values over the phenotypic  
 346 values in potential fathers, which is also more simply the heritability:

$$347 \quad r_{g_f,z_f} = \frac{cov'_f(g, z)}{V'_{zf}} = \frac{V_g}{V_z} = h^2$$

348 Given that  $\Psi_i(y|x)$  has also a Gaussian shape, using results about the product of  
 349 Gaussian functions, we can show that  $h_i(g_f|x)$  also corresponds to the probability  
 350 density function of a Gaussian distribution with mean and variance:

$$351 \quad E_{h_i}(g_f|x) = \bar{g}'_{if} + h^2(E_{\Psi_i}(y|x) - \bar{z}'_{if})$$

$$352 \quad V_{h_i}(g_f|x) = V'_{gf} - h^4 V'_{zf} + h^4 V_{\Psi_i}(y|x),$$

353 which, after replacing the mean and variance in  $\Psi_i(y|x)$  by their expression in eqn S15  
 354 and S16, become:

$$355 \quad E_{h_i}(g_f|x) = \bar{g}_i + h^2 S_s (S_m \theta_i + x(1 - S_m) - \bar{g}_i)$$

$$356 \quad V_{h_i}(g_f|x) = V_g \left( 1 - h^2 (1 - (1 - S_s)(1 + (1 - S_m)S_s)) \right)$$

357 Using results about the product of Gaussian functions with similar reasoning to  
 358 compute  $\varphi_i(g_m, g_f)$  and  $p_{i+1}(g, \cdot)$ , we finally find that the distribution of offspring  
 359 breeding values in the next generation is Gaussian with a mean and variance equal to:

$$360 \quad \bar{g}_{i+1} = \frac{1}{2} \bar{g}'_{im} + \frac{1}{2} \left( \bar{g}_i + h^2 S_s (S_m \theta_i + \bar{z}'_{im}(1 - S_m) - \bar{g}_i) \right)$$

$$V_g'' = \frac{V_{LE}}{2} + \frac{V_{gm}'}{4} + \frac{V_{gf}'}{4} - \frac{h^4(V_{zf}' - V_{\psi_i}(y|x))}{4} + \frac{h^4 S_s^2 (1 - S_m)^2 V_{zm}'}{4} + \frac{h^4 S_s (1 - S_m) V_{zm}'}{2}$$

If there is no direct natural selection on male fitness and only natural selection on female fitness, one can replace the means and variances in breeding values and phenotypic values in mothers after selection by their expressions (eqn S9-12) and obtain:

$$\bar{g}_{i+1} = \frac{1}{2}(\bar{g}_i + h^2 S_n (\theta_i - \bar{g}_i)) + \frac{1}{2}(\bar{g}_i + h^2 S_s (S_n + (1 - S_n) S_m) (\theta_i - \bar{g}_i))$$

$$V_g'' = \frac{V_{LE}}{2} + \frac{V_g}{2} - \frac{h^2 V_g}{2} + \frac{h^2 V_g (1 - S_s) (1 + (1 - S_m) S_s)}{4} + \frac{h^2 V_g (1 - S_n) (1 + (1 - S_m) S_s)^2}{4}$$

Note that, when replacing the selection coefficients by their values we have  $S_s (S_n + (1 - S_n) S_m) = S_n$  and thus:

$$\bar{g}_{i+1} = \bar{z}_{i+1} = \frac{1}{2}(\bar{g}_i + h^2 S_n (\theta_i - \bar{g}_i)) + \frac{1}{2}(\bar{g}_i + h^2 S_n (\theta_i - \bar{g}_i)),$$

or

$$\bar{g}_{i+1} = \bar{g}_i + s_n (\theta_i - \bar{g}_i) \quad \text{with } s_n = h^2 S_n$$

and

$$V_g'' = \frac{V_{LE}}{2} + \frac{V_g}{2} (1 - \tilde{s})$$

$$\text{with } \tilde{s} = h^2 (2S_n - S_s) = 2s_n - s_s \quad \text{with } s_s = h^2 S_s$$

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## Chapitre 2 : Combined effects of assortative mating and sex-specific natural selection on adaptation of a sexually dimorphic trait to environmental change

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## Abstract

Assortative mating is common in plant and animal natural populations. It can facilitate adaptation to environmental change, as it can increase genetic variance compared to random mating. In contrast, sex-specific natural selection, which is also frequent in natural populations, can hamper population adaptation. In particular, intersexual conflict in selection may cause the mean phenotype in the population to depart from that maximizing female fitness with potential demographic consequences in species where male fitness does not limit the population growth. The combined effects of assortative mating and sex-specific natural selection on evolutionary responses remain unexplored under environmental change, and are here studied. We built an analytical model based on the infinitesimal model of trait inheritance to evaluate the combined effect of sex-specific natural selection, sexual selection and sex-specific trait expression on the genetic responses in a changing environment. We model and compare predictions for (i) the genetic variance and (ii) the mismatch of the population mean breeding value to the female optimum, for traits under (a) female preference, (b) temporal assortative mating, and (c) random mating. Analytical predictions are validated by an individual-based model, which relaxes the assumption of an infinite number of loci and infinite population size. We showed that the mismatch of the population mean breeding value to the female optimum is composed of two parts: (i) a mismatch caused by sex-specific natural selection, sex-specific trait expression and sexual selection in a constant environment, and (ii) an adaptive lag caused by environmental change. Sex-specific trait expression combined with sexual selection generated by assortative mating induces a runaway of the mean breeding value, constrained by natural selection, which can lead to the evolution of trait values overshooting the interval between the male and female optima. In some conditions, which occur both under random and assortative mating, female maladaptation can be smaller in a changing environment than in a constant environment. Assortative mating can help populations to better track climate change than random mating, when selection on females is stronger than that on males and/or sexual dimorphism is not too large, and/or climate change is fast enough. The robustness of results has been tested with an individual-based model. It is therefore important to take into account sex-specific responses of traits under assortative mating to improve our understanding of the adaptation of populations to environmental change.

## 3.1 Introduction

### 3.1.1 Assortative mating facilitates adaptation in a changing environment in the absence of sex-specific selection

Assortative mating for a trait, which is measured by the phenotypic correlation for this trait between mates, is common in natural populations (see reviews in Jiang et al., 2013 and Janicke et al., 2019). Assortative mating has antagonistic effects on the genetic variance of populations. It increases genetic variance by creating positive associations among allelic effects within and among loci (Crow and Felsenstein, 1968; Crow and Kimura, 1970; Devaux and Lande, 2008; Fox, 2003; Weis et al., 2005; Wright, 1921). It also induces variation in the reproductive success of males, and favors males with phenotypes that resemble the phenotype of most common and/or successful females in the population. Stabilizing sexual selection generated by assortative mating adds to stabilizing natural selection, which decreases genetic polymorphism and therefore genetic variance (Godineau et al., 2021; Kirkpatrick and Nuismer, 2004). The size of positive and negative effects of assortative mating on the genetic variance under stabilizing selection depends on the strength of selection (Godineau et al., 2021; Lande, 1977). The positive effects of assortative mating on genetic variance generally exceed the negative ones under directional or disruptive selection (Crosby, 1970; Devaux and Lande, 2008; Fox, 2003; Godineau et al., 2021; Sachdeva and Barton, 2017; Weis et al., 2005). The positive effects of assortative mating on genetic variance were recently shown to help populations track environmental change, increasing their mean fitness in a model for the evolution of flowering time under climate change (Godineau et al., 2021). Those effects of assortative mating on evolutionary responses to environmental change however have been studied in the absence of sex-specific natural selection, although it is frequent in natural populations (Cox and Calsbeek, 2009; de Lisle et al., 2018). Sex-specific natural selection can generate conflicts between sexes and impede evolutionary responses to a changing environment (Connallon and Hall, 2016; Lande, 1980). We here investigated the combined effects of assortative mating and sex-specific selection on adaptation to a constant or changing environment.

### 3.1.2 Sex-specific natural selection under random mating and environmental change

Males and females experience different selection strength and optima under sex-specific natural selection. When sex-specific natural selection appears on a trait that is perfectly correlated between sexes and shows no sex-specific expression, males and females have the same phenotype and neither males nor females can simultaneously be at their optimum (Bonduriansky and Chenoweth, 2009). The selection is antagonistic because selection on males and on females is in opposite direction. There is a conflict between sexes to reach their own optimum and the evolutionary trajectory of the trait under sex-specific natural selection is thus constrained by a trade-off between selection on males and females (Lande, 1980). A reduced genetic correlation between males and females can solve the conflict between sexes (Bonduriansky and Chenoweth, 2009). Male and female traits are then free to evolve towards their respective

optimum (Bonduriansky and Chenoweth, 2009; Lande, 1980; Poissant et al., 2010; Rhen, 2000). An alternative way to solve sexual conflicts is to evolve sex-specific trait expression, which is a form of plasticity where the same genotype results in different phenotypic values in males and females. Traits (such as size in many animals) showing sex-specific expression despite being genetically correlated between sexes are very common (Cox and Calsbeek, 2009). When conflicts caused by sex-specific natural selection are only partially solved, because of genetic constraints and imperfect plasticity, the mismatch between the female optimum and the phenotypes of females is likely to contribute to a genetic load. This genetic load depresses the population growth rate, and impacts the demography and persistence of the population in a changing environment. Female fitness, and not male fitness, indeed constrains demography in populations in which male gametes are sufficiently abundant to fertilize all female gametes, and in which parental care is provided mostly by females.

In a model of sex-specific adaptation to environmental change, Connallon and Hall (2016) showed that in many instances signals of antagonistic selection between sexes may vanish in a changing environment. Continuously changing environment can also maintain antagonistic sex-specific selection and prevent the resolution of conflicts, on the contrary to a constant environment in which conflicts can be solved (Connallon and Hall, 2016). A recent model of sex-specific selection on a single bi-allelic locus under environmental change suggests that environmental change maintains more genetic variance than a constant environment, especially when stabilizing selection is strong (Connallon et al., 2019).

### 3.1.3 Sexual selection interacts with natural selection

Those models of sex-specific selection do not consider the effect of sexual selection, although it can modify the selection on males. Lande (1980) indeed shows that the phenotype of males at equilibrium is shifted away from the adaptive peak by sexual selection generated by assortative mating, even in the absence of genetic correlation between sexes. Our previous study investigated the effect of sexual selection, through temporal assortative mating for flowering time, on the evolutionary responses under environmental change (Godineau et al., 2021). Despite the absence of natural selection on males, we found that assortative mating generated indirect selection on males, of the same strength as natural selection on female fitness.

### 3.1.4 Aims of the study

Here we investigate how assortative mating modifies sex-specific natural selection and thus affects the evolutionary responses of a trait with sex-specific expression under environmental change. We develop an analytical model for the evolution of a quantitative trait based on the infinitesimal model of trait inheritance, under assortative mating and sex-specific natural selection, *i.e.* with different male and female optima, and with different levels of sexual conflicts, *i.e.* different values of sex-specific trait expression. We assume a single population of infinite size and a constant genic variance, *i.e.* the genetic variance at linkage and Hardy-Weinberg equilibrium, and thus assume that the evolution of the genetic variance for the trait results

only from changes in associations between allelic effects within and among loci. We focused on female maladaptation, which often constrains more population demography than does male maladaptation. We evaluate both the mismatch of the population mean breeding value to the female optimum, and the genetic variance at equilibrium for three types of mating: female assortative preference, temporal assortative mating, and random mating. We show that this mismatch is composed of two parts: (i) a mismatch caused by sex-specific natural selection, sex-specific trait expression and sexual selection in a constant environment, and (ii) an adaptive lag caused by environmental change. We validate our analytical results using an individual-based model with a finite and constant population size, a limited number of loci and in which the genetic variance evolves. The mismatch and the genetic variance in the simulated populations fit the predictions of the analytical model.

## 3.2 Methods

We develop an analytical model (based on the model in the Appendix S1 of Godineau et al., 2021) for the evolution of a quantitative trait under assortative mating, sex-specific natural selection and sex-specific trait expression, similar to flowering time in species for which male and female flowering phenology differ. We compare the effects on genetic adaptation of female assortative preference (preference for males resembling them), which is assumed in models of assortative mating (e.g. Sachdeva and Barton, 2017), temporal assortative mating, which is typical for reproductive time (Devaux and Lande, 2008; Fox, 2003; Godineau et al., 2021; Morbey and Ydenberg, 2001; Weis et al., 2005), and random mating. This allows disentangling the effects of sex-specific natural selection and sexual selection generated by assortative mating on genetic responses. We model both a constant and a changing environment, defined by the optimal trait values, which maximize female and male fitness, shifting at the same and constant speed  $k < 0$  over time for both males and females. We use an analytical model for the deterministic change over time of a quantitative trait based on the infinitesimal model of trait inheritance. This model assumes an isolated population of infinite size, a large number of loci and a constant genetic variance (i.e. the genetic variance at linkage and Hardy-Weinberg equilibrium and a measure of genetic polymorphism). For the sake of simplicity, we will describe the life cycle of a dioecious population with separated sexes but the model can also apply to hermaphroditic organisms for which the female trait (e.g. the date of stigmat receptivity) and the male trait (e.g. the date of pollen release) are perfectly genetically correlated, and affect respectively the male and female fitness components. Natural selection, mating and recombination occur successively within a generation. Generations are non-overlapping and occur within a year. The reproductive success of females is not limited by their access to males, and the population growth rate is thus measured by the mean fitness of females in the population. Females compete with all others in the population regardless of the mating pattern. The siring success of a male depends on its fitness, but also on the probability to mate with many and very fit females. With assortative mating, this mating probability depends on the female phenotypic distribution and their degree of choosiness, as well as the intensity of male-male competition, which also varies with the female phenotype. A population is expected to produce more offspring and to be less subject to extinction whenever

its mean phenotype is closer to the female optimum compared to a population with a mean phenotype more distant to the female optimum. We therefore measure genetic adaptation by the mismatch, at equilibrium, of the population mean breeding value to the optimum for the female trait. The robustness of the analytical results is tested with an individual-based model in which the genic variance evolves, the number of loci is limited, the population size is finite and constant, and the organisms are all hermaphroditic.

### 3.2.1 Analytical model

We derive predictions for the deterministic changes across generations in the mean and variance of a trait for individuals with separate sexes, assuming a Gaussian distribution of breeding and phenotypic values, and an infinitesimal model of trait inheritance. The details of the model are provided in the Appendix.

#### 3.2.1.A Mating patterns

Assortative mating is first modeled by the preference of females for male phenotypes similar to their own phenotype (*i.e.* trait value). The relative siring success of a male with phenotype  $y$  that mates with a female with phenotype  $x$  is:

$$\psi_P(y|x) = A(y|x) \frac{w_m(y)}{\iint p_m(g_m, z) w_m(z) A(z|x) dg_m dz} \quad (3.1)$$

with  $p_m(g_m, z)$  the joint distribution of breeding and phenotypic values of males before natural selection and reproduction,  $A(y|x)$  the preference function of a female with phenotype  $x$  for males centered on  $x$  and with a preference range  $\alpha^2$  such as:

$$A(y|x) = \exp\left(-\frac{(x-y)^2}{2\alpha^2}\right) \quad (3.2)$$

and  $w_m(y)$  the quantity of viable gametes produced by a male with phenotype  $y$  such as:

$$w_m(z) = \exp\left(-\frac{(z-\theta_m)^2}{2\omega_m^2}\right) \quad (3.3)$$

The strength of stabilizing selection applying on the male phenotypes to match the female phenotype  $x$  increases with the strength of the assortment measured as  $1/\alpha^2$  (first term of eqn 3.1). The relative siring success of a male increases with its fitness and is measured by its relative fitness compared to the other males in the population and by the female preference (the ratio in eqn 3.1).

Assortative mating is also modelled for traits linked to reproductive time, and is thus temporal: mating occurs only if the reproductive period of both males and females overlap (*e.g.*

overlap in flowering phenologies). The relative siring success of a male with phenotype  $y$  that mates with a female with phenotype  $x$  within a year is then:

$$\psi_T(y|x) = \int \phi_f(t|x) \phi_m(t|y) \frac{w_m(y)}{\iint p_m(g_m, z) w_m(z) \phi_m(t|z) dg_m dz} dt \quad (3.4)$$

with  $\phi_f(t|x)$  and  $\phi_m(t|y)$  the proportion of female (respectively male) gametes produced at time  $t$ , given the phenotype  $x$  (respectively  $y$ ) of the female (respectively male), which declines as a Gaussian function with mean  $x$  (and respectively  $y$ ) and width  $\alpha^2$ .

The relative siring success of a male with phenotype  $y$  that mates with a female with phenotype  $x$  at a given time  $t$  under temporal assortative mating depends on the number of gametes produced by the female and the male at time  $t$  within the year (first two terms), and the fitness of the male compared to the other males in the gamete pool at time  $t$  in the population (third term in eqn 3.4). In other words, the relative siring success depends on the quality of the male and the overlap between its reproductive period and the reproductive period of a female at time  $t$  compared to the other males. In contrast to female preference, competition between males for the access to a particular female varies with time.

Under random mating, females can mate with any male in the population and individuals (males and females) compete with all others of the same sex in the population to produce offspring. The success of a mating depends only on the fitness of the mates.

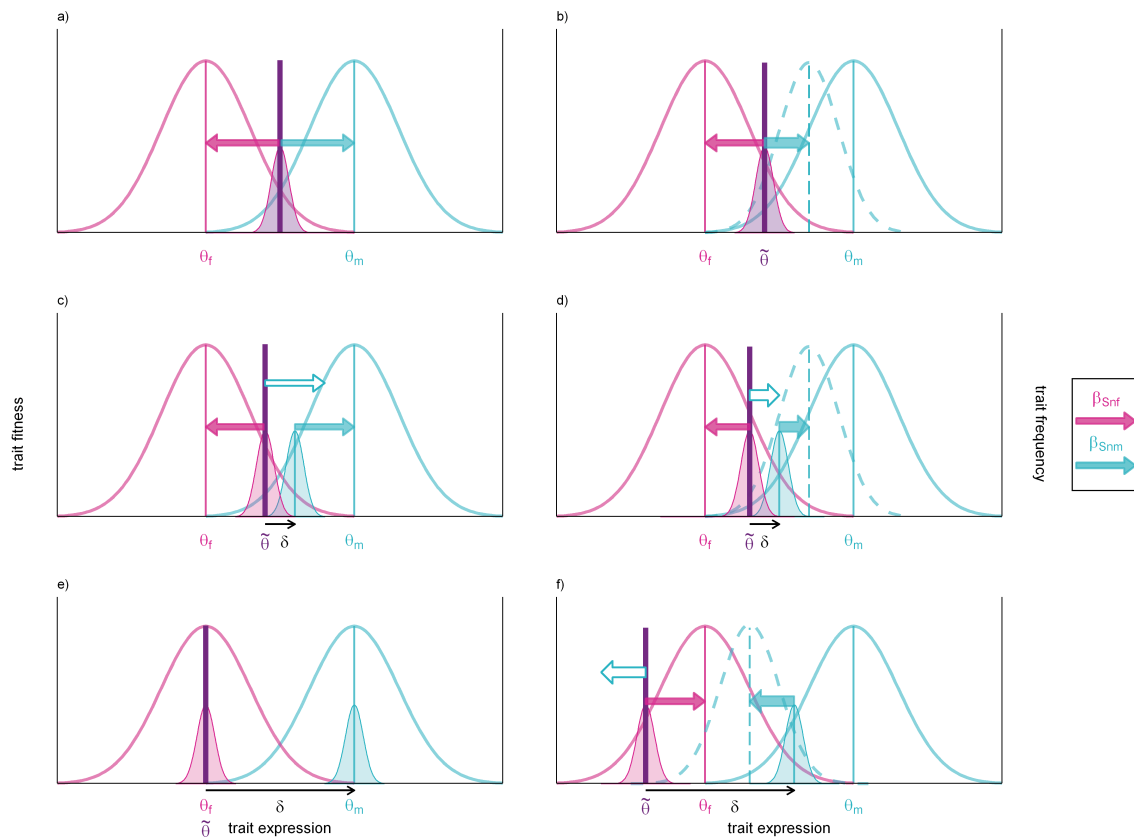
### 3.2.1.B Genetic architecture

We assume a single quantitative trait determined by a large number of loci with additive effects. The distribution of breeding values is Gaussian with mean  $\bar{g}$  and variance  $V_g$ , while environmental effects are distributed as a Gaussian with mean  $\bar{e} = 0$  and variance  $V_e$ . The phenotype (trait value) of a female is determined by the sum of its breeding value and its environmental effect,  $z = g + e$ . The phenotype of a male depends on sex-specific trait expression  $\delta$  with  $z = g + e + \delta$ . Under these assumptions, at the beginning of each generation, female (respectively male) phenotypes are distributed as a Gaussian with mean  $\bar{z}_f = \bar{g}$  (respectively  $\bar{z}_m = \bar{g} + \delta$ ) and variance  $V_z = V_g + V_e$ . We use an infinitesimal model of trait inheritance assuming that, conditional on the breeding value of the father and the mother, the distribution of breeding values of the offspring in any family is distributed as a Gaussian with mean value equal to the average breeding values of the two parents, and a variance equal to half the genic variance  $V_{LE}$ . Here the genic variance is assumed to be constant, and the genetic variance can thus only evolve through changes in negative or positive associations among allelic effects.

### 3.2.1.C Sex-specific selection and environmental change

Natural selection is sex-specific and affects fertility (*i.e.* the production of gametes) or survival. The male and female fitness decline with the distance to the optimal phenotype as a Gaussian function. The (optimal) phenotype that maximizes female fitness  $\theta_f$  is different from

the one that maximizes male fitness  $\theta_m$ , and the width of the selection function on the female fitness  $\omega_f$  can be different from that on the male fitness  $\omega_m$  (e.g. fig 3.1a). The widths of selection functions are constant over time. In a constant environment, the optimal phenotypes for males and females are constant, while in a changing environment, the optimal phenotypes change with a decreasing linear trend over generation and at the same speed  $k < 0$ . This environmental change can depict optimal flowering date for plants (Franks et al. 2007, Hamman et al. 2018). Male and female fitness are therefore year-specific even if, in the following, the year dependency in the equations is ignored for readability.



**Figure 3.1:** Position of the evolutionary optimum for breeding values compared to male and female optimal phenotypes, under random (first column) versus assortative mating (second column). The sex-specific phenotypic expression is absent (first row), smaller than the difference between the male and female optimal phenotypes (second row) or equal to the difference between the male and female optima (last row). Filled Gaussian distributions are the phenotypic distributions of female (pink) and male (blue) traits. The pink and blue Gaussian curves show natural selection acting on female and male fertility, respectively. The dashed blue Gaussian curve is the result of both natural and sexual selection on males. The mean breeding value of the population is assumed to be at its evolutionary optimum. The filled pink (blue) arrow is the selection gradient on female (male) traits after natural (and sexual) selection. The selection gradient is measured on the mean phenotype but its effect is on the mean breeding value, which is shifted from the mean male phenotype due to sex-specific trait expression. The effect on the mean breeding value of the selection gradient on males is represented by an empty blue arrow. The length of the arrows indicates the part of the selection gradient caused by a shift from the optimal phenotype, while their thickness indicates the part of the selection gradient caused by the strength of natural and/or sexual selection.

### 3.2.1.D Sex-specific trait expression

Sex-specific trait expression, denoted  $\delta$ , shifts the male phenotype away from its breeding value, and it is constant over time; sex-specific trait expression modeled here is thus similar to constant phenotypic plasticity. For example, body size frequently differs between sexes, and is under sex-specific selection (Shine, 1989). For hermaphroditic individuals, an example is dichogamy, *i.e.* the temporal shift between pollen release and stigma receptivity. We here explore value of  $\delta$  that should attenuate sexual conflicts, with the male phenotypes being closer to their optimal value, *i.e.* for  $\theta_m - \theta_f \geq \delta \geq 0$  if  $\theta_m > \theta_f$  or  $\theta_m - \theta_f \leq \delta \leq 0$  if  $\theta_m < \theta_f$ .

### 3.2.1.E Metrics recorded

#### 3.2.1.5.a Distribution of breeding values after natural selection and reproduction

We derive the joint distribution of phenotypic and breeding values after natural selection and reproduction. This derivation requires successive integrations presented in the Appendix, and involves the probability that a father has breeding value  $g_m$  given the phenotype  $x$  of the mother and the probability that an offspring has a breeding value  $g$  given the breeding values  $g_m$  and  $g_f$  of its father and mother. Given the joint distribution of phenotypic and breeding values of the parents,  $p_f(g_f, x)$  and  $p_m(g_m, y)$ , one can find the joint distribution after natural selection  $p'_f(g_f, x)$  and  $p'_m(g_m, y)$  and the joint distribution after natural selection and reproduction  $p''_f(g_f, x)$  and  $p''_m(g_m, y)$ . The mean breeding value after natural selection and reproduction  $\bar{g}''$  are presented in table 3.1. The strength of natural selection on females and males is measured as:

$$S_{nf} = \frac{V_z}{V_z + \omega_f^2} \quad (3.5)$$

and

$$S_{nm} = \frac{V_z}{V_z + \omega_m^2} \quad (3.6)$$

The strength of sexual selection on males is measured as:

$$S_s = \frac{V_z(1 - S_{nm})}{V_z(1 - S_{nm}) + \alpha^2} \quad (3.7)$$

The genetic variance after natural and sexual selection  $V_g''$  is:

$$V_g'' = \frac{V_{LE}}{2} + \frac{V_g}{2}(1 - h^2 S^*) \quad (3.8)$$

with  $S^*$  presented in table 3.2 and varying with the mating pattern. Note that the first term on the right side of eqn 3.8 is constant under the assumption of the infinitesimal model of trait inheritance, and that the second term is determined by the stabilizing effect of both natural and sexual selection on the trait and by the genetic associations among allelic effects generated by the mating pattern.



At equilibrium the genetic variance is constant ( $V_g'' = V_g$ ) and is:

$$V_g = \frac{V_{LE}}{1 + h^2 S^*} \quad (3.9)$$

**Table 3.1:** Mean breeding value after natural and sexual selection. The expression of the mean breeding value is the same under assortative female preference and temporal assortative mating for a given genetic variance. A indicates assortative mating while R indicates random mating.

assortative mating: female preference or temporal assortative mating	$\bar{g}_A'' = \bar{g} + \frac{h^2}{2} [S_{nf}(\theta_f - \bar{g})(1 + S_s) + S_{nm}(\theta_m - \bar{g} - \delta)(1 - S_s) - \delta S_s] \quad (3.10)$
random mating	$\bar{g}_R'' = \bar{g} + \frac{h^2}{2} [S_{nf}(\theta_f - \bar{g}) + S_{nm}(\theta_m - \bar{g} - \delta)] \quad (3.11)$

**Table 3.2:**  $S^*$  under all mating patterns. P indicates female preference, T temporal assortative mating and R random mating.

female preference	$S_P^* = \frac{S_{nf}(1 + S_s)^2 + S_{nm}(1 - S_s) - S_s(1 + S_s)}{2} \quad (3.12)$
temporal assortative mating	$S_T^* = \frac{S_{nf}(1 + S_s)^2 + S_{nm}(1 - S_s^2) - 2S_s}{2} \quad (3.13)$
random mating	$S_R^* = \frac{S_{nf} + S_{nm}}{2} \quad (3.14)$

### 3.2.1.5.b Mismatch of the population mean breeding value to the female optimum

As female maladaptation is a predictor of threats on the population growth rate, we evaluate the mismatch  $M$  of the population mean breeding value  $\bar{g}$ , not to both the male and female optima, but only to the female optimum  $\theta_f$  with:

$$M = \theta_f - \bar{g} \quad (3.15)$$

## 3.2.2 Individual-based model

The predictions of the mismatch to the female optimum and the genetic variance obtained by the analytical model are compared to results of simulations from an individual-based model (modified from Godineau et al. 2021). This model is built with the same assumptions as for the

analytical model except that (i) population size is finite and constant ( $N = 1000$ ), (ii) the number of loci determining the quantitative trait is limited ( $L = 5$ ), (iii) both the genic and genetic variances for the trait can vary over generations/years, (iv) the individuals are all hermaphroditic, and (vi) mutation and segregation of loci are explicitly modeled.

### 3.2.2.A Genetic architecture

Mutations occur at a rate  $\mu = U/2L$  with  $L$  the number of loci for both the female and male traits, and  $U = 0.1$  the genomic mutation rate. The effects of mutations are sampled in a Normal distribution of null expectation and variance equal to the mutational variance introduced by mutation each generation  $V_m = 0.04$ .

### 3.2.2.B Matings

Regardless of the mating pattern,  $N$  females are sampled in the population according to their fitness. Under random mating,  $N$  males are sampled in the population according to their fitness and then randomly paired with the females. Under female preference, the probability for a male to mate with a given female is the product of its fitness and the preference function of the female. Under temporal assortative mating, the probability for a male to mate with a given female at time  $t$  is proportional to the product of the quantity of its gametes produced at time  $t$  and its fitness defined by its trait value.

### 3.2.2.C Metrics recorded

We record the mismatch of the population mean breeding value to the female optimum as  $M$  with eqn 3.15.

The genetic variance  $V_g$  is measured before selection as:

$$V_g = \frac{1}{N} \sum_{j=1}^N \left( \sum_{l=1}^L (a_{xjl} + a_{yjl}) - \bar{g} \right)^2 \quad (3.16)$$

with  $a_{xjl}$  (resp.  $a_{yjl}$ ) the maternal (resp. paternal) allelic effect at locus  $l$  for individual  $j$  and  $\bar{g}$  the population mean breeding value of the trait.

The genic variance, a measure of genetic polymorphism, of the trait is measured as:

$$V_{LE} = \frac{1}{N} \sum_{j=1}^N \left( \sum_{l=1}^L (a_{xjl} - \bar{a}_{xl})^2 + (a_{yjl} - \bar{a}_{yl})^2 \right) \quad (3.17)$$

with  $\bar{a}_{xl}$  (resp.  $\bar{a}_{yl}$ ) the population mean maternal (resp. paternal) allelic effect at locus  $l$ .

Simulations last for 3 000 generations. In a constant environment, simulations start with allelic effects sampled in a Normal distribution of null expectation and variance equal to four. In a changing environment, simulations start with the genotypes of the last generation produced

under constant environment. The average change of the genetic variance per generation over the last 1 000 generations is small (from  $-0.03$  to  $0.05\%$ ) and we thus assume that the population has reached an equilibrium. Mismatch to the female optimum and genetic and genic variances are averaged over the last 1 000 generations to discard expected fluctuations due to random genetic drift. We did not replicate simulations because results fit well their expectation.

### 3.2.3 Parameter choice

We use the same parameter values for the sex-specific trait expression and sex-specific selection in the analytical and the individual-based models. We compare three ratios of the width of the natural selection function on males to that on females and keep their harmonic mean constant to 100 with  $\frac{\omega_m^2}{\omega_f^2} = \frac{300}{60}; \frac{100}{100}; \frac{60}{300}$ . We also compare three absolute differences between the female and male optimal phenotypes:  $|\theta_m - \theta_f| = 0, 3, 10$  and up to four absolute values for sex-specific phenotypic expression:  $|\delta| = 0, 1, 2, 3$ . The speeds of the environmental change tested here are  $k = 0, -0.1, -0.2, -0.3, -0.4, -0.5$ . The female preference ranges  $\alpha^2$  are set to 4.5, 10, 20 and 50.

## 3.3 Results

We derive, from the analytical model, the mismatch of the mean breeding value of the population ( $\bar{g}$ ) to the phenotype maximizing the female fitness ( $\theta_f$ ), *i.e.* a measure of the adaptation of the population when the population growth rate is assumed to be driven only by females. We show that, for a given amount of genetic variance, the mean breeding value at equilibrium, and thus the mismatch to the female optimum, is the same under temporal assortative mating (*A*) and female preference (*P*), but differs from that under random mating (*R*, 3.10 and 3.11). The mismatch to the female optimum for mating pattern ( $\cdot$ ) is:

$$M_{\cdot} = M_{\cdot 0} + M_{\cdot k} = (\theta_f - \bar{\theta}_{\cdot}) + \frac{k}{h^2 \bar{s}_{\cdot}} \quad (3.18)$$

with  $M_{\cdot 0} = \theta_f - \bar{\theta}_{\cdot} = \theta_f - \bar{g}_{\cdot}$  the mismatch under constant environment,  $\bar{\theta}_{\cdot}$  the evolutionary optimum for breeding values, and  $M_{\cdot k} = \frac{k}{h^2 \bar{s}_{\cdot}}$  the mismatch between the mean breeding value of the population and the evolutionary optimum under a changing environment.

This mismatch to the female optimum depends on the balance between the two parts it is composed of (eqn 3.18 and fig 3.2). The first part  $M_{\cdot 0}$  is caused by sex-specific trait expression, sex-specific selection and sexual selection in a constant environment; it accounts for the deviation from the female optimal phenotype  $\theta_f$  of the evolutionary optimum  $\bar{\theta}_{\cdot}$ , which corresponds to the population breeding value that maximizes the population mean fitness. The second part  $M_{\cdot k}$  is caused by the shift through years, at the same speed  $k$ , of the optimal female and male phenotypes, and is called the adaptive lag. Below we show that the mating patterns quantitatively affect both parts of the mismatch to the female optimum. We first describe specific cases (*e.g.* no sex-specific trait expression or equal strength of selection on both males and females) to help

understand what drives the mismatch to the female optimum. Recall that we address only cases for which the sex-specific trait expression does not evolve, is smaller and of the same sign than the difference between the optimal values for males and females.

### 3.3.1 Mismatch to the female optimum under a constant environment

At equilibrium and in a constant environment, the evolutionary optimum for the population mean breeding value is for the two types of assortative mating modelled here:

$$\bar{\theta}_A = \frac{1}{2\bar{s}_A} (S_{nf}(1 + S_s)\theta_f + S_{nm}(1 - S_s)(\theta_m - \delta) - S_s\delta) \quad (3.19)$$

with the optimal breeding value of males corresponding to  $\theta_m - \delta$ , and

$$\bar{s}_A = \frac{S_{nf}(1 + S_s) + S_{nm}(1 - S_s)}{2} \quad (3.20)$$

The mismatch to the female optimum under a constant environment and assortative mating thus becomes:

$$M_{A0} = \frac{S_{nm}(1 - S_s)(\theta_f - (\theta_m - \delta)) + S_s\delta}{S_{nf}(1 + S_s) + S_{nm}(1 - S_s)} \quad (3.21)$$

If the strength of sexual selection  $S_s$  (eqn 3.7) tends towards zero (*i.e.*  $\alpha^2 \rightarrow \infty$ ), we obtain the evolutionary optimum for the population mean breeding value under random mating :

$$\bar{\theta}_R = \frac{1}{2\bar{s}_R} (S_{nf}\theta_f + S_{nm}(\theta_m - \delta)) \quad (3.22)$$

with

$$\bar{s}_R = \frac{S_{nf} + S_{nm}}{2} \quad (3.23)$$

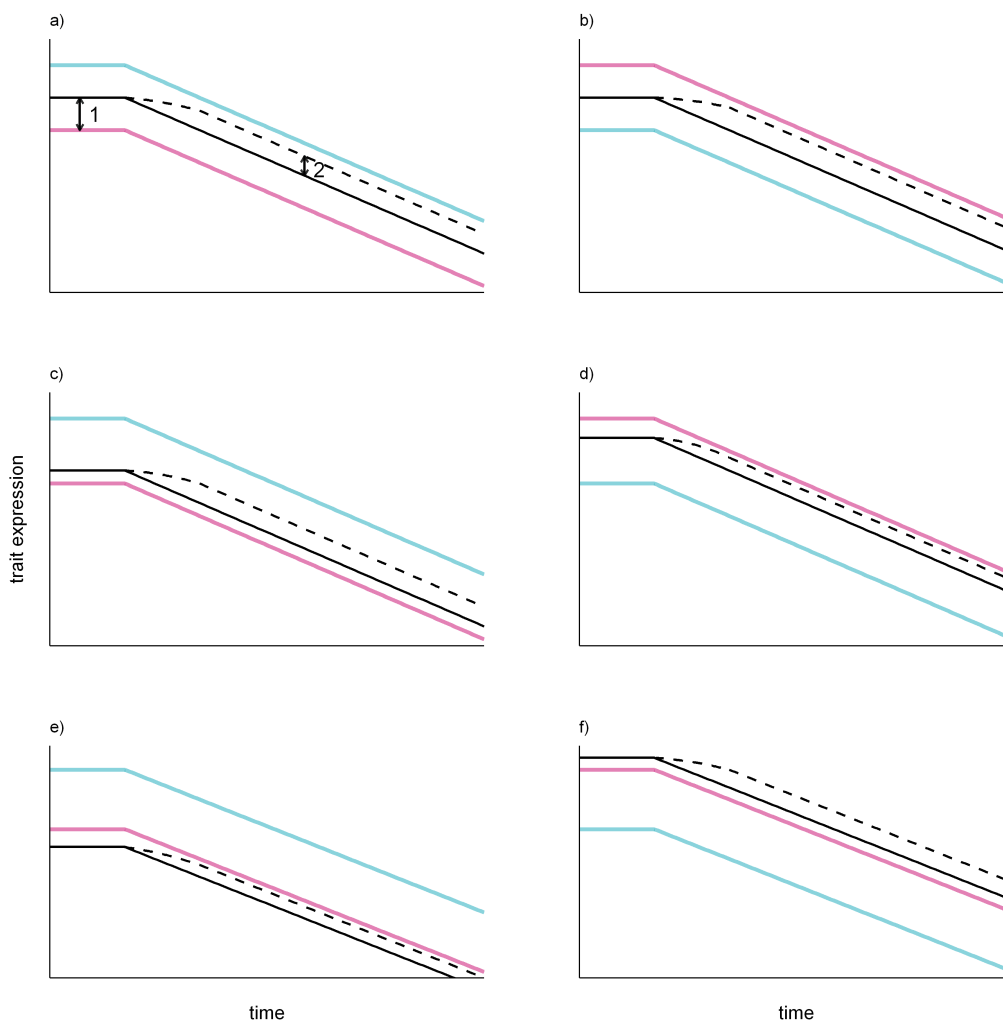
Note that  $\bar{s}_R$  is also referred as  $S_R^*$  (eqn 3.14).

The mismatch under random mating then becomes:

$$M_{R0} = \frac{S_{nm}(\theta_f - (\theta_m - \delta)) + S_s\delta}{S_{nf} + S_{nm}} \quad (3.24)$$

#### 3.3.1.A Sexual selection shifts the evolutionary optimum towards the female optimum

With no sex-specific trait expression ( $\delta = 0$ ) and the same strength of selection on males and females ( $S_{nf} = S_{nm}$  or equivalently  $w_f^2 = w_m^2$ ), the evolutionary optimum under random mating is equidistant from the optimal phenotypes that maximize the fitness of males and females (hereafter called male and female optima; fig 3.1a and eqn 3.22). The selection gradient on males is opposite to the selection gradient on females, thus signaling a sexual conflict (Chapman 2003;

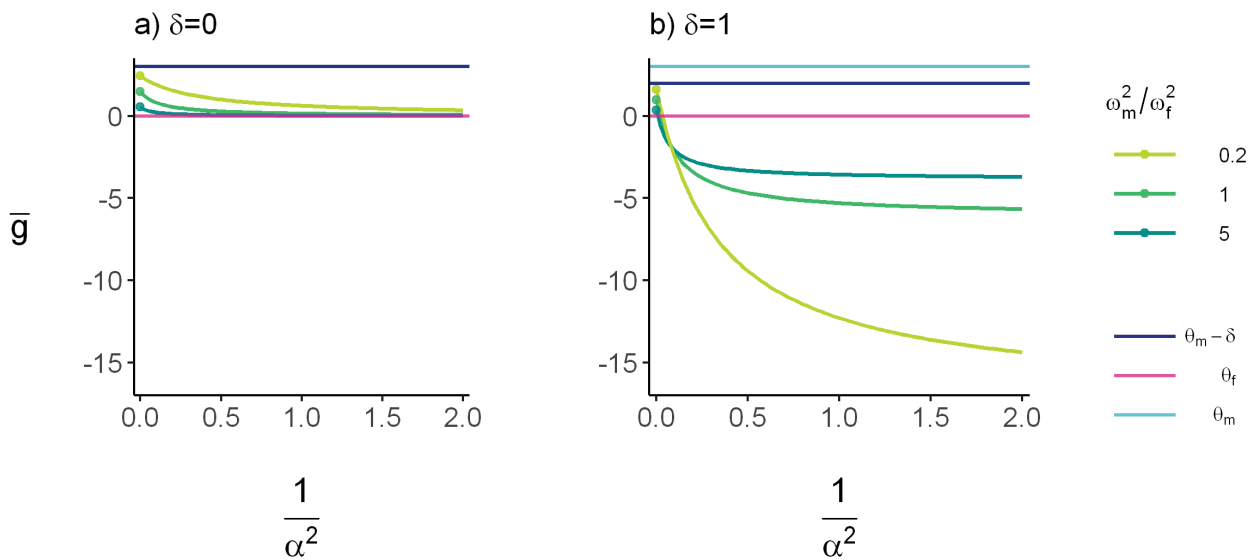


**Figure 3.2:** Effect of a changing environment on the mismatch to the female optimum in several scenarios. This is a schematic representation to aid interpretation of eqn 3.18 (not actual numerical results from the model). The male optimal phenotype (blue line) is larger (first column) or smaller (second column) than the female optimal phenotype (pink line). Optima are first constant and then decrease at the same and constant speed over years. The evolutionary optimum for breeding values (solid black line) depends on the mating pattern, the sex-specific trait expression, the relative position of female and male optima, and the strength of selection on the two sexes. The shift of the evolutionary optimum from the female optimum in a constant environment is indicated by arrow 1. Environmental change results in the population mean breeding value lagging behind this shifting optimum (indicated by arrow 2). Rows illustrate scenarios for which (a and b) the evolutionary optimum is located at equal distance to the female and male optimal phenotypes, for example under random mating ( $S_s = 0$ ), null sex-specific trait expression ( $\delta = 0$ ) and the same strength of selection on both sexes ( $S_{n,f} = S_{n,m}$ ; see fig 3.1a for illustration), (c and d) the evolutionary optimum is closer to the female than the male optimum, for example under assortative mating ( $S_s \neq 0$ ), and a small sexual dimorphism (see fig 3.1 c and d for illustration), or (e and f) the evolutionary optimum overshoots the female optimal phenotype, for example under assortative mating ( $S_s \neq 0$ ) and with a large sex-specific trait expression (see fig 3.1f for illustration).

Cox and Calsbeek, 2009; Connallon and Hall, 2018). The evolutionary optimum under assortative mating is displaced towards the female optimum (fig 3.1b and 3.3a), as  $\bar{\theta}_A = \frac{(1+S_s)\theta_f+(1-S_s)\theta_m}{2}$ . Sexual selection generated by assortative mating weakens the effect of selection on males while it strengthens the effect of selection on females (eqn 3.19). The resulting mismatch under assortative mating is  $M_{A0} = (1 - S_s)M_{R0}$ , and thus the size of the mismatch is smaller under assortative than under random mating ( $|M_{A0}| < |M_{R0}|$ ); in other words, the evolutionary optimum is always closer to the female optimum under assortative than under random mating in this scenario.

### 3.3.1.B Sex-specific selection shifts the evolutionary optimum

With no sex-specific trait expression ( $\delta = 0$ ), stronger natural selection on one sex ( $S_{nm} \neq S_{nf}$ ) shifts the evolutionary optimum under random mating towards the optimum for this sex, as  $M_{R0} = \frac{S_{nm}}{(S_{nm}+S_{nf})}(\theta_f - \theta_m)$  (eqn 3.24). When natural selection is stronger on males than on females, the displacement of the evolutionary optimum towards the male optimum is constrained under assortative mating by sexual selection on males. In contrast, when natural selection is stronger on females than on males, the displacement of the evolutionary optimum towards the female optimum is amplified by sexual selection under assortative mating. The mismatch under assortative mating when natural selection on females differs from that on males is  $M_{A0} = \frac{(S_{nf}+S_{nf})(1-S_s)}{S_{nf}(1+S_s)+S_{nm}(1-S_s)}M_{R0}$ , and thus always smaller, in size, than that under random mating.



**Figure 3.3:** Position at equilibrium of the population mean breeding value compared to male and female optima (respectively light blue and light pink) under a constant environment ( $k = 0$ ) as a function of the strength of assortment ( $\frac{1}{\alpha^2}$ ) and for different ratios of  $\omega_m^2/\omega_f^2$  without (a) or with (b) sex-specific trait expression  $\delta$ . In a constant environment, the population mean breeding value is equal to the evolutionary optimum ( $\bar{g} = \bar{\theta}$ ). Points are predictions under random mating ( $1/\alpha^2 \rightarrow 0$ ; eqn 3.22), while lines are predictions under assortative mating (eqn 3.19). Parameter values are:  $V_g = 5$ ,  $V_e = 4$ ,  $\frac{\omega_f^2\omega_m^2}{\omega_f^2+\omega_m^2} = 100$ .

### 3.3.1.C Sex-specific trait expression modifies the effect of sexual selection

Independently of mating pattern, sex-specific trait expression ( $\delta = z_m - \bar{g} \neq 0$ ) moves the optimal breeding value for males ( $\theta_m - \delta$ ) away from their optimal phenotype ( $\theta_m$ ), and in the direction of the female optimal phenotype (compare dark blue for  $\theta_m - \delta$  and light blue for the  $\theta_m$  in fig 3.3b; eqn 3.19 and 3.22). The position of the evolutionary optimum is affected by the positions of the optimal breeding value for females  $\theta_f$  and males  $\theta_m - \delta$ ; again it depends on the relative strength of natural selection on males and females, and under assortative mating on the effects of sexual selection (eqn 3.19 and 3.22).

Under assortative mating a runaway process emerges from the interaction between sex-specific trait expression and sexual selection. For example when  $\theta_f < \theta_m$  and  $\delta > 0$ , males accessing the fittest females have a smaller breeding value than these females, but their phenotypes match; their offspring have therefore a smaller breeding value than the females, and as a result the mean breeding value in the next generation declines. This runaway process mediated by sexual selection can lead the mean breeding value to overshoot the female optimum. This runaway is stopped by natural selection when both the mean phenotype of females and males depart from their optimum sufficiently to oppose the effect of sexual selection.

There is an optimal value of the sex-specific trait expression for which the mismatch in a constant environment can be null. Under random mating, the optimal value for the sex-specific trait expression is

$$\tilde{\delta}_R = \theta_m - \theta_f \quad (3.25)$$

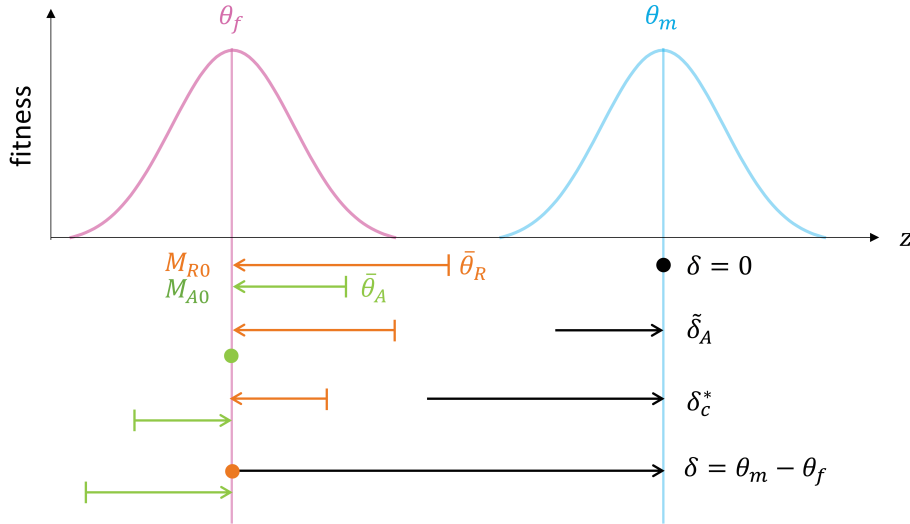
the sign of which depends on the position of  $\theta_m$  compared to  $\theta_f$ .

Note that  $\tilde{\delta}_R$  corresponds actually to the largest possible value of  $\delta$  in our model, such that any deviation of  $\delta$  from this value moves the evolutionary optimum under random mating away from the female optimum (fig 3.4). In other words, the sexual conflict is solved under random mating at  $\delta = \tilde{\delta}_R$  and the evolutionary optimum thus equals the female optimal trait (fig 3.4). When  $|\delta| < |\tilde{\delta}_R|$ , the position of the evolutionary optimum between the optimal breeding values for females  $\theta_f$  and males ( $\theta_m - \delta$ ) depends on the relative strength of natural selection on males and females.

Under assortative mating, the value of  $\delta$  that cancels out the mismatch is :

$$\tilde{\delta}_A = \frac{S_{nm}(1 - S_s)(\theta_m - \theta_f)}{S_{nm}(1 - S_s) + S_s} \quad (3.26)$$

Independently of the relative position of  $\theta_m$  to  $\theta_f$ ,  $\tilde{\delta}_A$  is always smaller than the maximal value of  $\delta$ , *i.e.* the difference between the male and female optima. Therefore, the mismatch under assortative can be either negative or positive depending on the value of  $\delta$  compared to  $\tilde{\delta}_A$ . For example, if the optimal flowering time of females is earlier than for males, the mean breeding value of the population is later than the female optimum but earlier than the male



**Figure 3.4:** Mismatch in a constant environment under assortative (green arrows) or random (orange arrows) mating for several values of the sex-specific trait expression (black arrows). The initial point of black arrows indicates the position of the optimal breeding value for males ( $\theta_m - \delta$ ), while the initial point of the orange and green arrows shows the position of the respective evolutionary optima. Null values are indicated by a point. Gaussian distributions correspond to the selection functions on males (blue) and females (pink) with equal widths ( $\omega_f^2 = \omega_m^2$ ).

optimum when  $\delta < \tilde{\delta}_A$  (fig 3.4). The mean breeding value of the population is earlier than the male and female optima when  $\delta > \tilde{\delta}_A$ . Whenever  $|\delta| > |\tilde{\delta}_A|$  the runaway process emerging under assortative mating makes the evolutionary optimum for the population mean breeding value go beyond the range defined by  $\theta_m$  and  $\theta_f$  and in the direction of the female optimum.

We can also define a critical value for the sex-specific trait expression for which the size of the mismatch under assortative mating equals that under random mating in a constant environment,  $|M_{A0}| = |M_{R0}|$  (fig 3.4):

$$\delta_c^* = \frac{2S_{nm}(S_{nf} + S_{nm}(1 - S_s))(\theta_m - \theta_f)}{2S_{nm}(S_{nf} + S_{nm}(1 - S_s)) + S_s(S_{nf} + S_{nm})} \quad (3.27)$$

with  $|\tilde{\delta}_A| < |\delta_c^*| < |\delta|$ .

When  $|\delta| > |\tilde{\delta}_c|$ , the mismatch under assortative mating in a constant environment is in size smaller than that under random mating, *i.e.* the evolutionary optimum is closer to the female optimum, independently of its position relative to the female optimum  $\theta_f$  (fig 3.4).

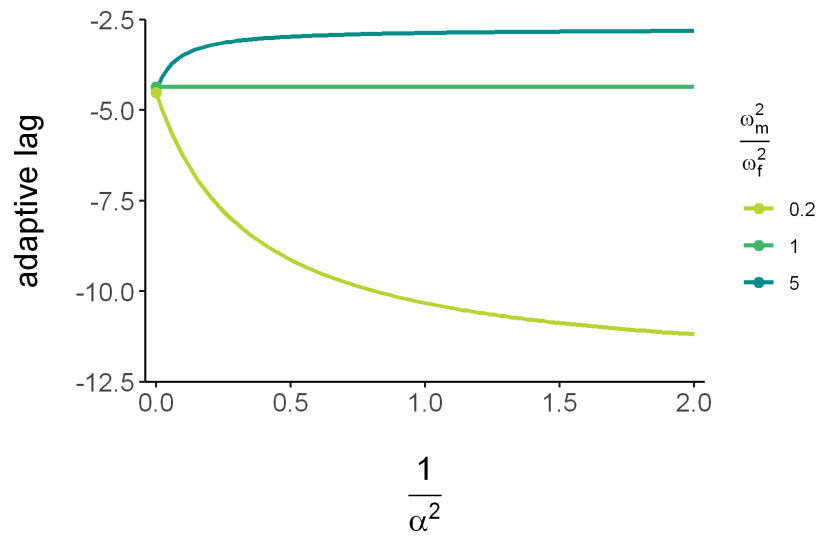
### 3.3.1.D Sexual selection modifies the effect of the sex-specific natural selection on the adaptive lag

Recall that the evolutionary optimum shifts at the same speed than male and female optima. The population mean breeding value lags behind the evolutionary optimum with an asymptotic regime (Lande and Shannon, 1996; Lynch et al., 1991); it is under assortative mating



$M_{Ak} = \frac{k}{h^2 \bar{s}_A}$  while under random mating it is  $M_{Rk} = \frac{k}{h^2 \bar{s}_R}$  (eqn 3.18). With no sex-specific selection or sex-specific trait expression, note that the expression of the adaptive lag under random mating equals that of Lande and Shannon (1996).

The adaptive lag is only affected by  $k$ , and the strength of sexual and/or natural selection on males and females, but not by sex-specific trait expression or the difference between the female and male optima. The adaptive lag under both assortative and random mating decreases with increased mean strength of natural selection on female and male fitness (fig 3.5). Adaptive lags reported in fig 3.5 for random mating are almost identical because of the constant harmonic mean of the widths of the selection functions leading to very similar mean strength of natural selection. The adaptive lag under assortative mating becomes independent of the strength of sexual selection when natural selection is as strong on males as on females. When the strength of natural selection is sex-specific, sexual selection generated by assortative mating again amplifies the effect of natural selection on females and weakens the effect of natural selection on males on  $\bar{s}_A$  (eqn 3.20). One can note that  $\bar{s}_A = \bar{s}_R + \frac{S_s}{2}(S_{nf} - S_{nm})$ , and  $\bar{s}_A > \bar{s}_R$  if  $S_{nf} > S_{nm}$ . The adaptive lag, all else being equal (*i.e.*  $h^2$  and  $k$ ) is thus smaller (*resp.* larger) under assortative than under random mating when natural selection is stronger (*resp.* weaker) on females (*resp.* males) than on males (*resp.* females; fig 3.5). As we model  $k < 0$ , the adaptive lag is negative and can make the mismatch under a changing environment smaller than the mismatch under constant environment (fig 3.2).



**Figure 3.5:** Adaptive lag as a function of the strength of the assortment for three ratios of  $\frac{\omega_m^2}{\omega_f^2}$ , and thus three strengths of natural selection on females compared to males. The evolutionary optimum is assumed to be at the female optimum such that the mismatch to the female optimum is only caused by the shift through years of the optima. Parameter values are:  $V_g = 5$ ,  $V_e = 4$ ,  $\frac{\omega_f^2 \omega_m^2}{\omega_f^2 + \omega_m^2} = 100$ ,  $k = -0.2$ .

### 3.3.2 Total mismatch to the female optimum

#### 3.3.2.A Environmental change can decrease the mismatch to the female optimum

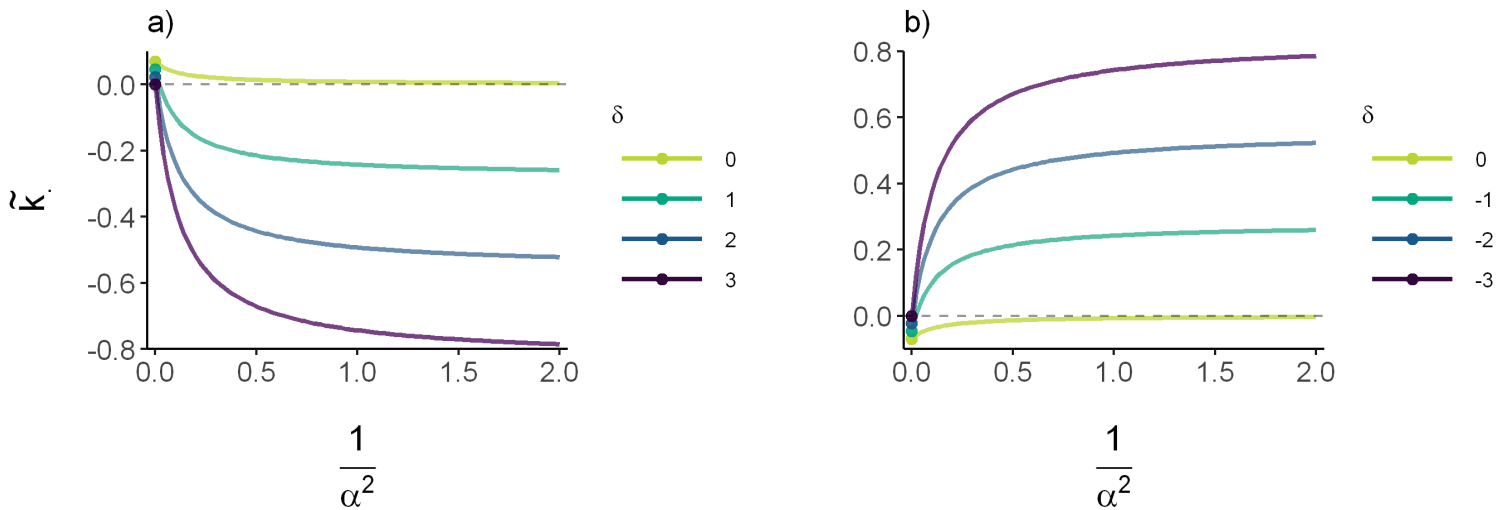
Environmental change defined by  $k < 0$  can decrease the mismatch compared to that under constant environment when the latter is positive. In that case, there is an optimal speed of the environmental change  $\tilde{k}$  for which the mismatch to the female optimum is zero; it is under assortative mating:

$$\tilde{k}_A = h^2 \bar{s}_A (\bar{\theta}_A - \theta_f) = -\frac{1}{2} h^2 (S_{nm} (1 - S_s) (\theta_f - (\theta_m - \delta)) + S_s \delta) \quad (3.28)$$

and under random mating (for which  $\alpha^2 \rightarrow \infty$  and thus  $S_s \rightarrow 0$ ):

$$\tilde{k}_R = h^2 \bar{s}_R (\bar{\theta}_R - \theta_f) = -\frac{1}{2} h^2 S_{nm} (\theta_f - (\theta_m - \delta)) \quad (3.29)$$

Note that for both mating patterns,  $\tilde{k}$  is a linear function of the sex-specific trait expression  $\delta$ , and that  $\tilde{k}_A$  can be expressed as  $\tilde{k}_A = (1 - S_s) \tilde{k}_R - \frac{1}{2} h^2 S_s \delta$ .



**Figure 3.6:** Optimal speed of the environmental change as a function of the strength of preference (measured as  $1/\alpha^2$ ) for different strengths of the sex-specific trait expression when  $\theta_m > \theta_f$  (a) or  $\theta_m < \theta_f$  (b). The optimal speed of the environmental change is indicated by points under random mating (eqn 3.29) and lines under assortative mating (eqn 3.28). Note the differences in scales for the y-axes among plots. Parameter values are:  $\theta_f = 0$ ,  $\theta_m = 3$  for (a) or  $\theta_m = -3$  for (b),  $V_g = 5$ ,  $V_e = 4$ , and  $\omega_m^2 = 100$ .

The effects of sex-specific trait expression, natural selection on males, and sexual selection on the optimal speed depend on whether the male optimum is larger or smaller than the female optimum. With no sex-specific trait expression ( $\delta = 0$ ), the optimal speed of the environmental change is negative for both random and assortative mating when  $\theta_m < \theta_f$  (fig 3.6b), but positive, and thus impossible according to our assumption when  $\theta_m > \theta_f$  (fig 3.6a). Increased sexual selection (through  $S_s$  or  $1/\alpha^2$ ) cancels out the mismatch under assortative mating for slower environmental change than for random mating (fig 3.6). Sex-specific trait expression expands the possible (negative) values for the optimal speed of the environmental change for assortative

mating only and only when  $\theta_m > \theta_f$ . In situations where assortative mating causes large mismatch and female maladaptation already in a constant environment (due to the runaway process described above), there is a larger potential for environmental change to mitigate this mismatch. In particular it can happen in a warming climate, in which earlier flowering is favored ( $k < 0$ ), when female flowering period is too early in a constant environment, and males flower later than do females.

### 3.3.2.B Parameter range for which assortative mating helps to track the female optimum

We can define conditions, *i.e.* in terms of speed of the environmental change for a given sex-specific trait expression or vice versa, for which the size of the mismatch under assortative mating equals the size of the mismatch under random mating. For both ranking of  $\theta_m$  to  $\theta_f$ , the critical speed of the environmental change allowing  $|M_A| = |M_R|$  is :

$$k_{c1}^* = -\frac{h^2}{2(S_{nf}(2 + S_s) + S_{nm}(2 - S_s))} \left( 2S_{nm}(S_{nf} + S_{nm}(1 - S_s))(\theta_f - (\theta_m - \delta)) + \delta S_s(S_{nf} + S_{nm}) \right) \quad (3.30)$$

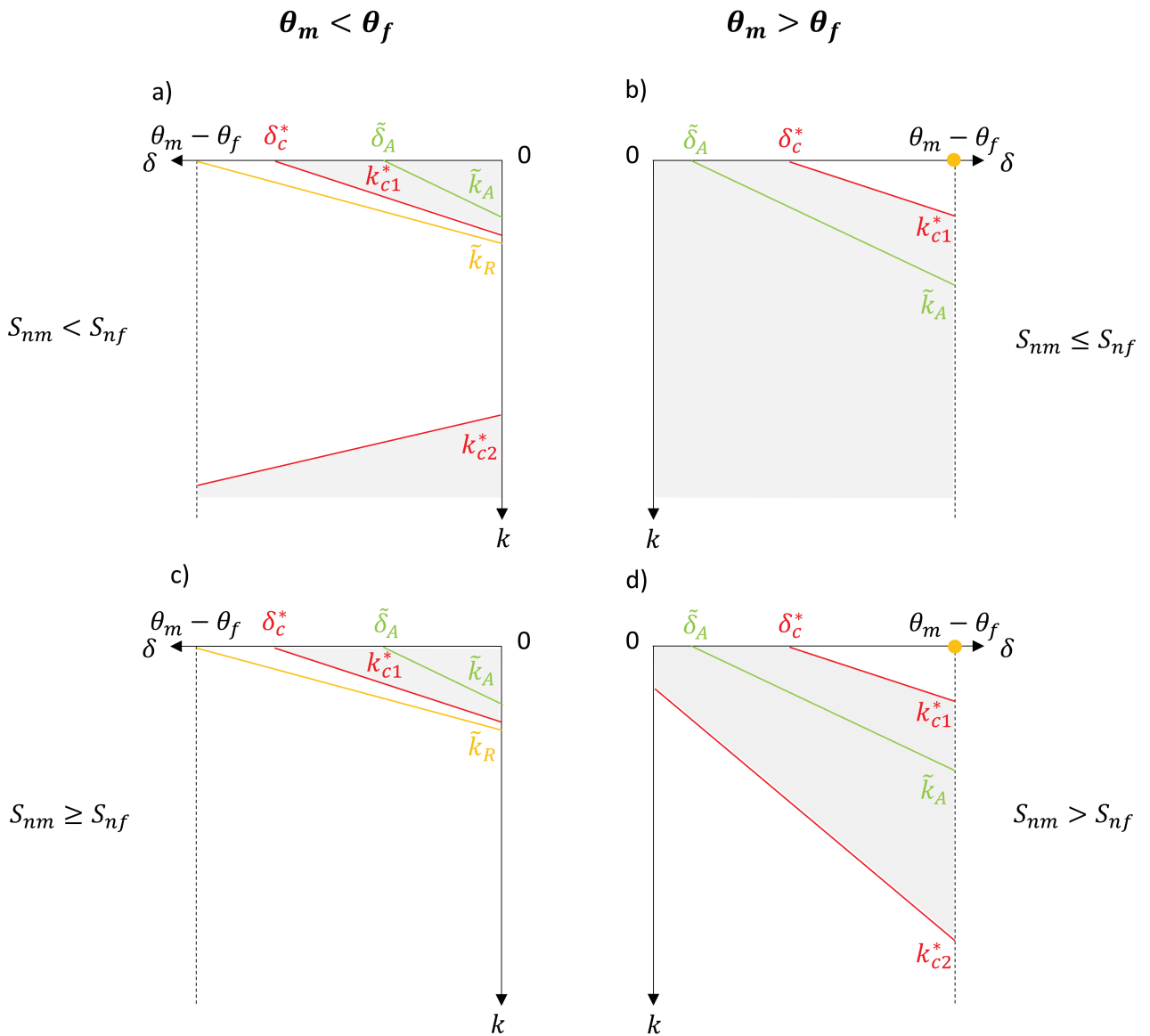
and when  $\theta_m > \theta_f$  and  $S_{nf} < S_{nm}$ , or, when  $\theta_m < \theta_f$  and  $S_{nf} > S_{nm}$ , there is a second critical speed :

$$k_{c2}^* = -\frac{h^2}{2(S_{nf} - S_{nm})} \left( 2S_{nf}S_{nm}(\theta_f - (\theta_m - \delta)) - (S_{nf} + S_{nm})\delta \right) \quad (3.31)$$

These critical speeds of the environmental change help to define combinations of parameters for which  $|M_A| < |M_R|$ ; those conditions strongly depend on whether the optimal value is greater for males or females and the relative strength of natural selection on males and females. With no sex-specific trait expression, and regardless of the ranking of optima, stronger selection on females than males expands the range of possible speeds of the environmental change for which the mismatch under assortative mating is smaller than that under random mating.

The same patterns hold whenever  $\delta \neq 0$ : stronger selection on females than on males, and  $k$  of the opposite sign as  $(\theta_m - \theta_f)$  both allow the mismatch to the female optimum to be smaller for assortative mating compared to random mating for a large range of speeds of environmental change (fig 3.7 a and b). Increased sex-specific trait expression increases the range of speeds of the environmental change for which the mismatch under assortative mating is smaller than that random mating when  $\theta_m > \theta_f$  and  $S_{nm} > S_{nf}$  (fig 3.7d). Otherwise the sex-specific trait expression (in absolute value) decreases the range of speeds of the environmental change for which the mismatch under assortative mating is smaller than that random mating (fig 3.7a, b and d).

Note that the mismatch under assortative mating can be either positive or negative, and that above a critical value for the sex-specific trait expression,  $|\delta| > |\delta_c^*|$  (eqn 3.27), the runaway evolution of the population mean breeding values under assortative mating generated by sexual selection makes the size of the mismatch larger than that under random mating. When selection

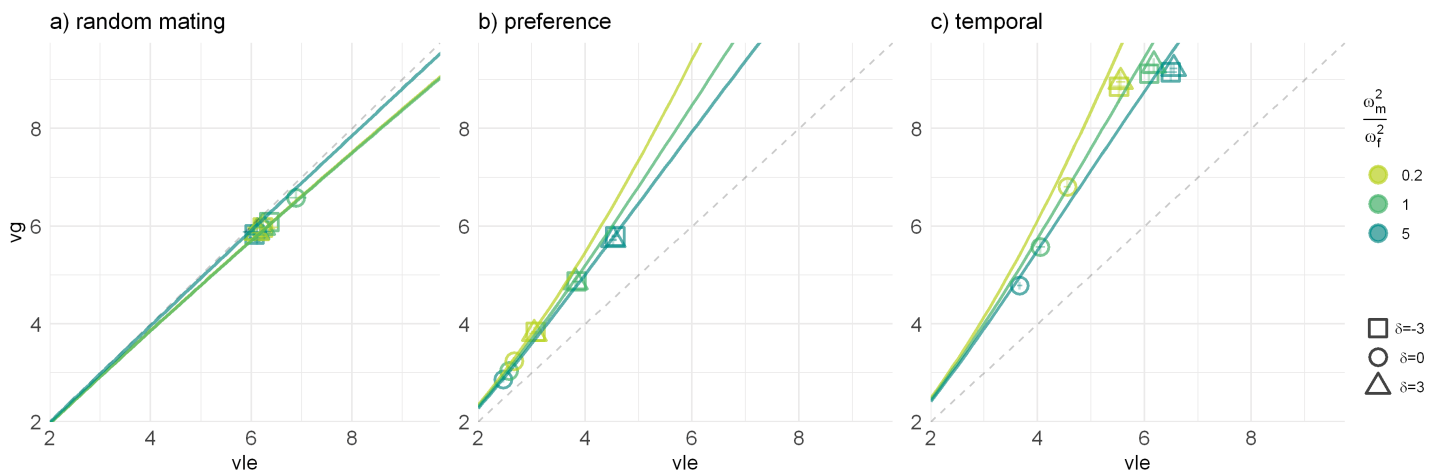


**Figure 3.7:** Range of speed of the environmental change  $k < 0$  and sex-specific trait expression  $\delta$  for which the size of the mismatch under assortative mating is smaller than that under random mating,  $|M_A| < |M_R|$  (shaded area). This is a schematic representation to aid interpretation (not actual numerical results from the model). Green or orange lines correspond to optimal speeds of the environmental change canceling the mismatch under assortative ( $\tilde{k}_A$  in eqn 3.28) and random mating ( $\tilde{k}_R$  in eqn 3.29), respectively. Note that when  $\theta_m > \theta_f$ ,  $\tilde{k}_R = 0$  only when  $\delta = \theta_m - \theta_f$ . The red lines show critical speeds of the environmental change  $k_{c1}^*$  and  $k_{c2}^*$ , respectively, for which  $|M_A| = |M_R|$  (eqn 3.30 and eqn 3.31). The slope of green, orange and red lines can change quantitatively but their relative ranking remains the same for all parameter values. Columns and rows are defined for several combination of strength of natural selection and ranking of female to male optimum.

on females is stronger than that on males and  $\theta_m > \theta_f$ , the beneficial effect of assortative mating on the adaptive lag dominates its detrimental effects on the mismatch in a constant environment when the mismatch due to runaway in a constant environment is not too large ( $\delta$  not large) and the adaptive lag is very large ( $k \ll 0$ ; fig 3.7b). Conversely if selection on males is stronger than on females and  $\theta_m < \theta_f$ , assortative mating makes the adaptive lag larger and helps adaptation to the female optimum only when the mismatch due to runaway in a constant environment is not too large ( $\delta$  not large in absolute value) and the adaptive lag is small ( $k < 0$ ; fig 3.7c).

### 3.3.3 Genetic variance

Recall that the analytical model only predicts genetic variance that scales to constant genetic variance. Analytical predictions for the genetic variance differs between mating patterns (eqn 3.9 and table 3.2). For all mating patterns, the genetic variance is expected to increase with increased genetic variance but to not be affected by sex-specific trait expression or the difference between female and male optima. Below we present how the genetic variance is affected by selection and mating patterns, and compare its prediction, for a fixed genetic variance, to its value obtained with an individual-based model in which genetic and genetic variance can both evolve.



**Figure 3.8:** Genetic variance as a function of the genetic variance for random mating (a), female preference (b) and temporal assortative mating (c). Lines are analytical predictions, assuming constant genetic variance, and points are simulated values with evolving genetic variance. The scale color indicates the ratio of the widths of the selection function. Symbols indicate the value for the sex-specific trait expression. Predictions above (resp. below) the grey line  $V_g = V_{LE}$  helps to visualize when associations among allelic effects are positive (resp. negative). Parameters values are:  $k = 0$ ,  $\theta_f = 0$ ,  $\theta_m = 10$  for  $\delta = 0$  and  $\delta = 3$  or  $\theta_m = -10$  for  $\delta = -3$ ,  $\frac{\omega_f^2 \omega_m^2}{\omega_f^2 + \omega_m^2} = 100$ ,  $\alpha^2 = 4.5$ .

#### 3.3.3.A Expected effects on genetic variance

The structure of the genetic variance under random mating is only affected by the mean strength of natural selection on females and males (eqn 3.9). As we choose parameter values to get  $\frac{\omega_f^2 \omega_m^2}{\omega_f^2 + \omega_m^2}$  exactly constant, the mean strength of selection under random mating is almost constant and the genetic variance is only very slightly affected by the ratios of the selection widths tested here (compare lines on fig 3.8a). The predicted genetic variance is lower than the

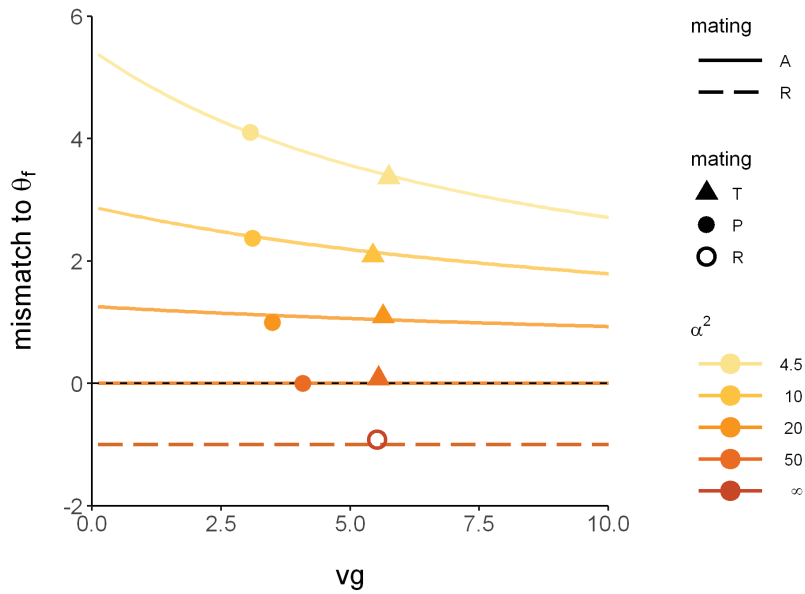
genic variance indicating negative associations among allelic effects selected by the stabilizing selection.

The genetic variance at equilibrium under assortative mating increases as  $S_P^*$  and  $S_T^*$  decrease (eqn 3.9, 3.12 and 3.13). For a fixed genic variance, the genetic variance at equilibrium under temporal assortative mating is always expected to be higher than under female preference as  $S_T^* = S_P^* - \frac{S_s(1-S_s)(1-S_{nm})}{2}$ . As a result, for a fixed genic variance, even if the mismatch of the population mean breeding value to the female optimum has the same expression for both types of assortative mating, this mismatch is expected to be smaller under temporal assortative mating than under female preference. For a fixed genic variance, the genetic variance under random mating is smaller than that under temporal assortative mating if  $S_{nf} < \frac{S_{nm}S_s+2}{S_s+2}$  and under female preference if  $S_{nf} < \frac{S_{nm}+S_s+1}{S_s+2}$ . Note that when the strength of natural selection is the same on males and females, the genetic variance is expected to be the smallest under random mating.

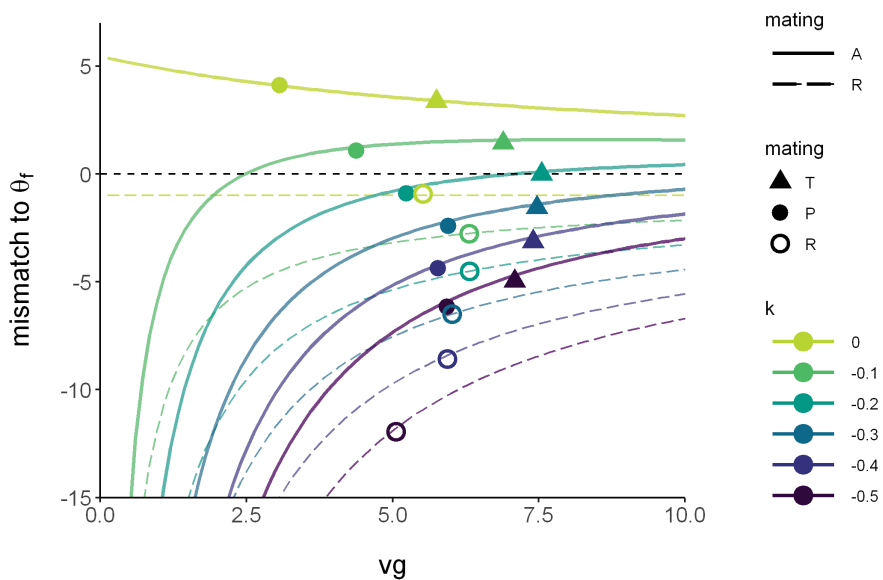
For a fixed genic variance, the genetic variance under assortative mating is expected smaller when natural selection on females is stronger than on males (eqn 3.12, 3.13 and fig 3.8b and c). The effect of sexual selection on  $S_P^*$  and  $S_T^*$  depends on the type of assortative mating (eqn 3.12 and 3.13). The strength of sexual selection amplifies the effect of the strength of natural selection on females on  $S_P^*$  and  $S_T^*$ . It also lowers the effect of the strength of natural selection on males on  $S_P^*$  and  $S_T^*$  but to a lesser extent for female preference than for temporal assortative mating (compare the second term in the numerator of eqn 3.12 with 3.13). However,  $S_T^*$  is also more decreased by sexual selection than  $S_P^*$  (compare the last term in the numerator of eqn 3.12 with 3.13 and fig 3.8b with c). The genetic variance under female preference is expected lower than under temporal assortative mating (eqn 3.12 and 3.13). The genetic variance is not always adaptive as it can increase or decrease the absolute value of the mismatch to the female optimum (fig 3.9 and 3.10).

### 3.3.3.B Genetic and genic variance in the simulations

For the explored range of parameters, simulations fit well the predictions for the mismatch to the female optimum (fig 3.9 and 3.10). Simulations deviate slightly from predictions for genetic variance, especially for temporal assortative mating (fig 3.8). As expected, the genetic variance increases with the strength of the assortment (*i.e.*  $1/\alpha^2$ ; fig 3.9). Female preference and temporal assortative mating can decrease the genic variance in the simulated populations compared to random mating (compare points in fig 3.8) but these mating patterns also generate positive associations among similar allelic effects, and more for temporal assortative mating than female preference (fig 3.8b and c). The positive effect on associations does not always compensate for the loss of genic variance for the female assortative preference model; in most cases, positive effects of associations however exceeds the loss of genic variance in the case of temporal assortative mating, resulting in higher variance than with random mating



**Figure 3.9:** Mismatch of the population mean breeding value as a function of the genetic variance for several strengths of the assortment ( $\frac{1}{\alpha^2}$ ; colour gradient). Lines are analytical predictions under assortative (solid) or random (dashed) mating, while symbols show values obtained in the simulated populations under female preference (filled circle), temporal assortative mating (filled triangle) and random mating (empty circle). Parameter values are  $\delta = 1$ ,  $\theta_m = 3$ ,  $\theta_f = 0$ ,  $k = 0$ ,  $\omega_f^2 = \omega_m^2 = 100$ .



**Figure 3.10:** Mismatch of the population mean breeding value as a function of the genetic variance for different speeds of the environmental change,  $k$  (colour gradient). See fig 3.9 for symbols.

Also contrary to predictions, genetic variance, as well as genic variance, are both increased by increased sex-specific trait expression (fig 3.8b and c). As previously found (Bürger and Lynch, 1995 for random mating; and Godineau et al., 2021 for temporal assortative mating with no sex-specific trait expression), both genic and genetic variances are bell-shaped with increased speed of the environmental change (fig 3.10).

### 3.3.3.C Changes in the genic variance do not affect qualitatively the predictions for the mismatch to the female optimum

Despite the effects of the mating pattern on the genic variance in the simulations, predictions for the mismatch to the female optimum are qualitatively correct (fig 3.9 and 3.10). Indeed, the difference, for a given genic variance, between the mismatch to the female optimum under the two types of assortative mating is maintained when the genic variance evolves in simulations. For example, when the mismatch under assortative mating is predicted smaller in absolute value than under random mating, the mismatch simulated under assortative mating is still smaller than under random mating despite the evolution of the genic variance.

## 3.4 Discussion

### 3.4.1 Main results

Evolutionary responses under assortative mating have been mostly studied with no sex-specific natural selection (Crosby, 1970; Devaux and Lande, 2008; Fox, 2003; Godineau et al., 2021; Kirkpatrick and Nuismer, 2004; Lande, 1977; Sachdeva and Barton, 2017; Weis et al., 2005), although sex-specific natural selection is frequent in natural populations (Cox and Calsbeek, 2009; de Lisle et al., 2018). For example, assortment for flowering time is assortative and male and female flowering are often distinct (Lloyd and Webb, 1986). Sex-specific natural selection being a common cause of sexual dimorphism (Cox and Calsbeek, 2009) it could explain the sexual dimorphism for flowering time. We investigated here how sex-specific natural selection affects both the mismatch of the population mean breeding value to the female optimum, and the genetic variance of a quantitative trait under random or assortative mating, in a changing environment and under different levels of sex-specific trait expression. This mismatch depends on the compensation between the mismatch caused by sex-specific natural selection, sex-specific trait expression and sexual selection in a constant environment, and an adaptive lag, the size of which varies with the strength of both natural and sexual selection. Assortative mating generates sexual selection that weakens the effect of natural selection on males and strengthens it on females, such that the population mean breeding value is more shifted towards the female optimum, compared to random mating. Combined with sex-specific trait expression, assortative mating induces a runaway process, constrained by natural selection, which can lead to the evolution of trait values overshooting the interval between the male and female optima. Female maladaptation can be smaller in a changing environment than in a constant environment regardless of the mating pattern. Stronger selection on females than that on males, small sexual dimorphism and fast



climate change help populations under assortative mating to better track climate change than random mating when the female optimal flowering time is earlier than the male optimal flowering time. We discuss below the separate and combined effects of sex-specific natural selection, sexual selection, sex-specific trait expression on the mismatch to the female optimum, and on the genetic variance. We also discuss the effect of the type of assortative mating on the mismatch to the female optimum, and on the genetic variance and the demographic consequences of these results.

### 3.4.2 Effects of sex-specific natural selection on maladaptation

#### 3.4.2.A Assortative mating decreases the maladaptation under constant environment

With no sex-specific trait expression, sex-specific natural selection induces maladaptation of both sexes in a constant environment, as the population mean fitness results from a trade-off between natural selection on females and natural selection on males, and males and females cannot be simultaneously at their optimum. This process occurs for all mating patterns but is quantitatively affected by mating pattern. The optimal phenotype for males under sex-specific natural selection and assortative mating is a trade-off between natural selection and sexual selection. Indeed, the optimal phenotype for males is a trade-off between the male optimum from natural selection and the phenotype that should allow access to the selected females. Sexual selection generated by assortative mating therefore impedes males to reach their optimum from natural selection (Lande, 1980). Sexual selection generated by assortative mating affects the evolutionary optimum for the population mean breeding value because it increases the effect of the strength of natural selection on females and decreases it on males. Sexual selection thus shifts the evolutionary optimum away from that under random mating and in the direction of the female optimum. This shift is larger when natural selection is stronger on females than males. As we measure adaptation only through the mismatch of the population mean breeding value to the female optimum, combined effects of assortative mating and sex-specific natural selection in a constant environment allows better adaptation of populations compared to random mating when sex-specific trait expression is null.

As adaptation critically depends on the relative strength of selection on sexes, measuring only female fitness components, as usually done in animals or plants (but see, Austen and Weis, 2016 and Tonnabel et al., 2019 for estimates of male fitness components), most probably do not estimate accurately maladaptation of populations. The same claim was made by Cotto et al. (2019), who found that fecundity and survival affect the evolutionary optimum of populations structured in life stages, and therefore that estimates of all fitness components could improve predictions of maladaptation.

#### 3.4.2.B Beneficial effects of a changing environment

We found here, as in the classical theory on adaptation to environmental change under random mating (Lande and Shannon, 1996; Lynch et al., 1991), that the population mean breeding value reaches an asymptotic regime at equilibrium, lagging behind the value that

maximizes population fitness. Environmental change, *i.e.* a shift at the same speed of the male and female optima through years, combined with sex-specific selection and when sex-specific trait expression is null, inevitably brings the population mean breeding value closer to one optimum but further from the other one. When environmental change goes in the opposite direction as the mismatch under a constant environment, there should be an optimal speed of the environmental change  $k \neq 0$  that can make the population mean breeding value be at the female optimum. Those optimal speeds actually depend linearly on the sex-specific trait expression.

The adaptive lag induced by environmental change under random mating is proportional to the mean strength selection on the two sexes. Sexual selection generated by assortative mating modifies the effect of sex-specific natural selection on the adaptive lag induced by environmental change. The effect of assortative mating on the adaptive lag can compensate or add to its effect on the mismatch in a constant environment. Populations under assortative mating can sustain a faster environmental change than can populations under random mating (fig 3.7).

### 3.4.3 In most cases, sex-specific trait expression does not help adaptation

Recall that sex-specific trait expression can not evolve here, and cannot exceed the difference between the female and male optima for fitness. We restricted our analysis to perfect genetic correlations between female and male phenotypes, although under sex-specific selection it should decrease and each sex should evolve towards its optimum (Bonduriansky and Chenoweth, 2009). Note also that in plants, sex-specific trait expression can evolve for other reasons than sex-specific natural selection, such as to avoid selfing (Lloyd and Webb, 1986). For all mating patterns, sex-specific trait expression moves the male breeding values away from the male phenotypes but in the direction of the female optimal value for the trait. For populations under random mating, the breeding value of the males never overshoots the breeding values of the females. For populations under assortative mating, and because of competition for access to females, the male breeding values can overshoot the female breeding values. Combination of sex-specific trait expression and the sexual selection generates a runaway of the population mean breeding value, which is constrained by the strength of natural selection. Sex-specific trait expression therefore can decrease the adaptation of populations under assortative mating compared to random mating in a constant environment. Increased sex-specific trait expression increases the range of speeds of the environmental change for which the mismatch under assortative mating is smaller than that random mating only when the optimal flowering date for males is later than for females and natural selection on males is stronger than on females.

### 3.4.4 Combined effects of sexual and sex-specific natural selection on genetic variance

Classical theory of adaptation to directional selection with no sex-specific selection (Kopp and Matuszewski, 2014; Lande and Shannon, 1996) predicts that increased genetic variance decreases the lag of the population mean breeding value to the optimum. In contrast, under sex-specific selection in a changing environment the genetic variance is not always adaptive

as it can increase or decrease the absolute value of the mismatch to the female optimum (fig 3.10). The analytical model suggests that, sexual selection generated by assortative mating increases the effect of selection on females and decreases the effect of selection on males. As a result, for a given genic variance, the increase of genetic variance through positive associations among allelic effects under assortative mating is larger when selection on males is stronger than that on females. In line with previous studies (Godineau et al., 2021; Kirkpatrick and Nuismer, 2004), simulations show that sexual selection generated by assortative mating decreases the genic variance compared to random mating. The genetic variance under assortative mating in simulations performed can be higher or lower than under random mating in a constant or changing environment. This suggests that the positive effect of assortative mating on genetic associations does not always compensate its negative effect on the genic variance.

The effect of the sex-specific trait expression on the genetic variance is unclear. The analytical model predicts it has no effect, while it has effects on both the genetic and genic variance in the simulations (fig 3.8). Also, in the simulations the speed of the environmental change affects the genetic variance through its effect on the genic variance, as previously found for population with no sex-specific selection under random mating (Bürger and Lynch, 1995) or assortative mating (Godineau et al., 2021).

#### 3.4.5 Comparison of the types of assortative mating

We modelled assortative mating through female assortative preference, often assumed in models (*e.g.* Sachdeva and Barton, 2017) or through temporal assortative mating typical for flowering time. Indeed, there is an expected correlation for flowering time because mates must have overlapping flowering periods (Devaux and Lande, 2008; Fox, 2003; Godineau et al., 2021; Weis et al., 2005). We thus decided to compare and discuss the effect of the mechanisms of mating on genetic responses to environmental change under commonly observed sex-specific selection.

We found that the genetic response to selection for temporal assortative mating is the same than that under female preference, for a given genetic variance. Under female preference, the siring success of a male with a given female depends only on how much its phenotype match the phenotype of the female relative to the other males. Under temporal assortative mating, the siring success of a male with a given female depends on how much, at a given time, its reproductive phenology overlaps with that of the female relative to the other males. For both type of assortative mating, all males compete for the access to females and their siring success depends on that of the other males in the population. The siring success of males is thus under soft selection (Wallace 1975; Bell et al. 2021), even if under temporal assortative mating there are different "effective" pollen pools for each female, but also for a given female, different effective pollen pools for each time interval.

The variation of the competition over time could contribute to maintain higher genetic variance under temporal assortative mating than female preference. Indeed, we found that

temporal assortative mating maintains higher genetic variance than female preference, and thus the genetic response under temporal assortative mating is higher than that under female preference. Fox (2003) previously discussed the ability of temporal assortative to maintain higher genetic variance than other types of assortative mating. The analytical model predicts that temporal assortative mating produces more positive genetic associations than female preference (eqn 3.8 and table 3.2). The genic variance in the simulated populations is in addition less decreased under temporal assortative mating than it is under the female preference. The negative effects of the female preference on the genic variance often exceeds its positive effect on genetic associations such that genetic variance can be smaller than that under random mating, but it is less common for temporal assortative mating in a changing environment.

Taken together these results suggest that the mechanism of assortative mating affects the genetic responses and the genetic variance at equilibrium in population under a constant or changing environment. Knowing and understanding what drives assortative mating can help to predict population fate to changing environments.

#### 3.4.6 Potential demographic consequences

We studied here the mismatch of the population mean breeding value to the female optimum because we assumed that the contribution of females to the growth rate is larger than that of males. This assumption is common (Harts et al., 2014) while males are sometimes found to influence the growth rate (Rankin and Kokko, 2007), such for Allee effects (Berec et al., 2001) or biparental care (Cockburn, 2006). Connallon and Hall (2016) similarly suggested that the persistence of populations can be increased by the genetic correlations that facilitate track a directional change in the female optimum. We showed here that sexual selection, generated by assortative mating, combined with sex-specific natural selection can help adaptation to environmental change in populations in which only females limit the population growth rate. Whether environmental changes favor phenotypes that are more female-like or male-like should influence the time to extinction or the time to evolutionary rescue.

### 3.5 Conclusion

Using a theoretical approach, we showed that assortative mating, by generating sexual selection, weakens the effect of natural selection on males and strengthens the effect of natural selection on females on the mismatch to the female optimum. Combination of assortative mating and sex-specific selection can facilitate, compared to random mating, adaptation to a changing environment depending on the speed of the environmental change, sex-specific trait expression, whenever selection on females is stronger than selection on males and whether the male optimum is higher or lower than the female optimum. Results suggest that we need to estimate male and female components of fitness and the mechanisms that drive assortative mating to provide sound predictions of adaptation to environmental change for many populations. Empirical measures of genetic responses of traits under assortative mating combined with estimates of sex-specific

natural selection are still scarce but would strongly help to understand and then quantify of adaptation in a changing environment.

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## 3.1 Appendix 2

### 3.1.1 Joint distribution of breeding and phenotypic values under assortative female preference

We derive predictions for the deterministic changes across generations in the mean and variance of a trait for individuals with separate sexes, assuming a Gaussian distribution of breeding and phenotypic values, and an infinitesimal model of trait inheritance. Note that the model should also apply to hermaphroditic individuals in which a female trait (*e.g.* the date of stigmat receptivity) and the male trait (*e.g.* the date of pollen release) are perfectly genetically correlated and affect respectively the male and female fitness components. The model is an extension of the model in the Appendix S1 of Godineau et al., 2021: it here includes sex-specific trait expression and sex-specific selection for three types of mating patterns. From the predictions of the model, we obtain the mismatch at equilibrium of the population mean breeding value to the female optimum, given the genetic variance, in a constant or a changing environment. The model is an extension of the model in the Appendix S1 of Godineau et al., 2021, which here includes sex-specific trait expression and sex-specific selection. We describe below the equations for changes along the life cycle in the joint distribution of breeding and phenotypic values under female preference, given the genic variance for the evolving trait (the genetic variance at linkage and Hardy Weinberg equilibrium). We compare in the next sections these changes for traits under random mating and temporal assortative mating.

If we assume that the joint distribution of breeding and phenotypic values is a bivariate Gaussian, under the assumptions of our model with Gaussian fitness functions and the infinitesimal model of trait inheritance, we show that these distributions remain Gaussian through the life cycle and in the next generation. The infinitesimal model assumes that, conditional on the breeding value of the father and mother, the distribution of breeding values in the full-sib family is distributed as a Gaussian, with a mean equal to the average breeding values of the two parents, and a fixed variance equal to half the genic variance in the population. The infinitesimal model approximates well the distribution of breeding values within a family when the evolving trait is controlled by a large number of loci, each of small additive effects (Bulmer, 1980; Tufto, 2000 and see a review in Barton et al., 2017).

Each generation is completed within a single year. Climatic conditions within that year determine the optimal phenotypes for the male and female traits, which are either constant or change linearly across years. The mean phenotype of males can be different from that of females because of sex-specific trait expression. This sex-specific trait expression is constant over generations (the degree of sexual dimorphism does not evolve and is the same for all individuals). In the following, we derive the changes from one generation to the next in the distribution of the breeding and phenotypic values due to (i) natural selection on females and males, (ii) sexual selection generated by assortative mating between mates, and (iii) recombination. Changes in the distribution of breeding and phenotypic values are derived separately in males and females.



We use the indices  $f$  and  $m$  throughout the derivations to denote females and males, respectively (note that this notation is different from that in the appendix of Godineau et al. (2021) in which indices  $f$  and  $m$  denote fathers and mothers).

### 3.1.1.A Initial conditions

We assume a single quantitative trait determined by a large number of loci. Breeding values are distributed, before natural selection and reproduction, as a Gaussian with mean  $\bar{g}$  and variance  $V_g$  in each sex. Environmental effects are distributed as a Gaussian with mean  $\bar{e} = 0$  and variance  $V_e$ , regardless of the sex.

At the beginning of a generation, the joint Gaussian distribution of breeding and phenotypic values for females has a probability density function  $p_f(g, z)$ , with means, variances and covariance:

$$\boldsymbol{\mu}_f = \begin{pmatrix} \bar{g} \\ \bar{g} \end{pmatrix} \quad (\text{S1})$$

$$\boldsymbol{\Sigma}_f = \begin{pmatrix} V_g & V_g \\ V_g & V_z \end{pmatrix} \quad (\text{S2})$$

The phenotype of a male depends on sex-specific trait expression (a special form of phenotypic plasticity) such that a male with a breeding value  $g$  has phenotype  $z_m = g + e + \delta$ . The joint Gaussian distribution of breeding and phenotypic values for males is a probability density function  $p_m(g, z)$ , with means, variances and covariance:

$$\boldsymbol{\mu}_m = \begin{pmatrix} \bar{g} \\ \bar{g} + \delta \end{pmatrix} \quad (\text{S3})$$

$$\boldsymbol{\Sigma}_m = \boldsymbol{\Sigma}_f \quad (\text{S4})$$

### 3.1.1.B Joint distribution of breeding and phenotypic values in the offspring generation

Natural selection is sex-specific, and the optimal phenotypes for males and females shift over years at the same speed. The joint distribution of breeding and phenotypic values changes over years because of natural selection. Given the joint distribution of breeding and phenotypic values in a given generation  $p(z, g)$ , we derive the joint distribution of breeding and phenotypic values, after natural selection and reproduction, in the offspring generation for females  $p_f''(z, g)$  and males  $p_m''(z, g)$ . We below ignore the time dependency in equations to simplify the notation, but one should keep in mind that male and female fitness are year-specific. The general derivation for the joint distribution of breeding and phenotypic values in the offspring generation is presented here and then more details are presented in the next sections. Also for simplicity and in the following we call the potential fathers and mothers of the new generation males and females.

The joint distribution of phenotypic and breeding values of females in the offspring generation is:

$$p''_f(z, g) = \eta_f(z|g)p''(g) \quad (\text{S5})$$

with  $\eta_f(z|g)$  the probability that a female offspring has phenotype  $z$  given its breeding value  $g$ , which is distributed as a Gaussian centered on  $g$  with variance  $V_e$ , and  $p''(g)$  the distribution of breeding values  $g$  in the offspring generation.

Similarly, the distribution of phenotypic and breeding values of males in the offspring generation is:

$$p''_m(z, g) = \eta_m(z|g)p''(g) \quad (\text{S6})$$

with  $\eta_m(z|g)$  the probability that a male offspring has phenotype  $z$  given its breeding value  $g$ , which is distributed as a Gaussian centered on  $g + \delta$  with variance  $V_e$ .

The distribution of breeding values  $g$  in the offspring generation is the same for the two sexes:

$$p''(g) = \iint R(g|g_f, g_m)\varphi(g_f, g_m)dg_m dg_f \quad (\text{S7})$$

where  $R(g|g_f, g_m)$  the probability that an offspring has breeding value  $g$  given the breeding values  $g_m$  and  $g_f$  of its father and mother, respectively, and according to the infinitesimal model of trait inheritance, is:

$$R(g|g_f, g_m) = \frac{1}{\sqrt{\pi V_{LE}}} e^{-\frac{(g - \frac{g_m + g_f}{2})^2}{V_{LE}}} \quad (\text{S8})$$

with  $V_{LE}$  the genic variance of the population.

and where  $\varphi(g_f, g_m)$  the probability that a male has breeding value  $g_m$  and that a female has breeding value  $g_f$  in a pair of parents is:

$$\varphi(g_f, g_m) = \int p_f(g_f, x)\tilde{w}_f(x) \int p_m(g_m, y)\tilde{w}_m(y|x)dydx \quad (\text{S9})$$

with  $\tilde{w}_f(x)$  the relative fitness of a female with phenotype  $x$ , and  $\tilde{w}_m(y|x)$  the relative siring success of a male with phenotype  $y$  that mates with a female with phenotype  $x$ .

### 3.1.1.C Natural selection

Natural selection on phenotypes is sex-specific and affects a fitness component such as fertility (*i.e.* the production of gametes) or survival. For a given year, the trait maximizing the female (respectively male) fitness is  $\theta_f$  (resp.  $\theta_m$ ) and the female (resp. male) fitness declines with distance to  $\theta_f$  (resp.  $\theta_m$ ) as a Gaussian function of width  $\omega_f^2$  (resp.  $\omega_m^2$ ).

The fitness of a female with phenotype  $z$  is:

$$w_f(z) = \exp\left(\frac{-(z - \theta_f)^2}{2\omega_f^2}\right) \quad (\text{S10})$$

The fitness of a male with phenotype  $z$  is:

$$w_m(z) = \exp\left(\frac{-(z - \theta_m)^2}{2\omega_m^2}\right) \quad (\text{S11})$$

Note that sex-specific selection induces a sexual conflict between the sexes to reach their own optimum trait value (Bonduriansky and Chenoweth, 2009). Sex-specific expression can decrease this conflict by moving the male phenotypes closer to their optimum; in mathematical terms it means that we explore values for the constant  $\delta$  such as  $\theta_m - \theta_f \geq \delta \geq 0$  if  $\theta_m > \theta_f$ , or,  $\theta_m - \theta_f \leq \delta \leq 0$  if  $\theta_m < \theta_f$ .

### 3.1.1.D Sexual selection

Females have a preference for males with a similar phenotype to their own. The preference function of a female with phenotype  $x$  for males is:

$$A(y|x) = \exp\left(\frac{-(x - y)^2}{2\alpha^2}\right) \quad (\text{S12})$$

with  $\alpha^2$  the preference range of females for male phenotypes.  $1/\alpha^2$  measure the strength of the assortment.

A given female competes with other females of the population to mate. Its relative fitness then is:

$$\tilde{w}_f(x) = \frac{w_f(x)}{\iint p_f(z, g_f) w_f(z) dg_f dz} \quad (\text{S13})$$

A given male competes with other males of the population to mate with a given female. Its relative siring success depends on the preference function of this female such as:

$$\tilde{w}_m(y|x) = \frac{w_m(y) A(y|x)}{\iint p_m(z, g_m) w_m(z) A(z|x) dg_m dz} \quad (\text{S14})$$

### 3.1.1.E Joint distribution of breeding and phenotypic values after natural selection

The joint distribution of breeding and phenotypic values for females after natural selection (indicated in the following by a prime) is computed by using mathematical properties of the product of a bivariate Gaussian distribution (*i.e.*  $p_f(g, x)$ ) with a univariate Gaussian distribution (*i.e.*  $w_f(x)$ ), such that:

$$p'_f(g, x) = \frac{p_f(g, x) \times w_f(x)}{\bar{w}_f} \quad (\text{S15})$$

with  $\bar{w}_f$  constant and equal to:

$$\bar{w}_f = \frac{\omega_f^2}{\sqrt{(V_z + \omega_f^2)}} \exp\left(\frac{-(\bar{z}_f - \theta_f)^2}{2(V_z + \omega_f^2)}\right) \quad (\text{S16})$$

$p'_f(g, x)$  is a bivariate Gaussian with means, variances and covariance:

$$\bar{g}'_f = \bar{g}_f + h^2 S_{nf}(\theta_f - \bar{z}_f) = \bar{g} + h^2 S_{nf}(\theta_f - \bar{g}) \quad (\text{S17})$$

$$V'_{gf} = V_g(1 - h^2 S_{nf}) \quad (\text{S18})$$

$$\bar{z}'_f = \bar{z}_f + S_{nf}(\theta_f - \bar{z}_f) = \bar{g} + S_{nf}(\theta_f - \bar{g}) \quad (\text{S19})$$

$$V'_{zf} = V_z(1 - S_{nf}) \quad (\text{S20})$$

$$\text{Cov}'_f(g, z) = V_g(1 - S_{nf}) \quad (\text{S21})$$

with  $S_{nf}$  a coefficient that measures the strength of the natural selection on females:

$$S_{nf} = \frac{V_z}{V_z + \omega_f^2} \quad (\text{S22})$$

Similarly for males, the joint distribution of breeding and phenotypic values after natural selection (indicated in the following by a prime) is computed by using the same result on the product of a bivariate Gaussian distribution with a univariate Gaussian distribution such that:

$$p'_m(g, y) = \frac{p_m(g, y) \times w_m(y)}{\bar{w}_m} \quad (\text{S23})$$

with  $\bar{w}_m$  a constant equal to:

$$\bar{w}_m = \frac{\omega_m^2}{\sqrt{(V_z + \omega_m^2)}} \exp \frac{-(\bar{z}_m - \theta_m)^2}{2(V_z + \omega_m^2)} \quad (\text{S24})$$

$p'_m(g, y)$  is a bivariate Gaussian with means, variances and covariance:

$$\bar{g}'_m = \bar{g}_m + h^2 S_{nm}(\theta_{zm} - \bar{z}_m) = \bar{g} + h^2 S_{nm}((\theta_m - \delta) - \bar{g}) \quad (\text{S25})$$

$$V'_{gm} = V_g(1 - h^2 S_{nm}) \quad (\text{S26})$$

$$\bar{z}'_m = \bar{z}_m + S_{nm}(\theta_{zm} - \bar{z}_m) = \bar{g} + \delta + S_{nm}((\theta_m - \delta) - \bar{g}) \quad (\text{S27})$$

$$V'_{zm} = V_z(1 - S_{nm}) \quad (\text{S28})$$

$$\text{Cov}'_m(g, z) = V_g(1 - S_{nm}) \quad (\text{S29})$$

with  $S_{nm}$  a coefficient that measures the strength of the natural selection on males:

$$S_{nm} = \frac{V_z}{V_z + \omega_m^2} \quad (\text{S30})$$

and  $(\theta_m - \delta)$  the optimal breeding value of males, which is shifted by sex-specific trait expression from the optimal phenotype  $\theta_m$ .

The strength of the natural stabilizing selection on females (or males) reduces the deviation of both the population mean breeding value and the mean phenotype of females to the female optimum (or the male optimum for the breeding value  $(\theta_m - \delta)$ ). The strength of the natural stabilizing selection on females (or males) also reduces their genetic and phenotypic variances, as well as the covariances between them.

### 3.1.1.F Selection function on males to mate with a given female with phenotype $x$

We define the numerator of the relative siring success of males *i.e.* such as  $w_m^*(z|x) = w_m(y)A(y|x)$ . One can also write  $w_m^*(z|x)$  as:

$$w_m^*(z|x) = W_m^*(x) * \exp\left(\frac{-(z - \theta_m^*(x))^2}{2\omega_m^*}\right) \quad (\text{S31})$$

with  $W_m^*(x)$  a constant and

$$\theta_m^* = \theta_m \gamma + x(1 - \gamma) \quad (\text{S32})$$

with  $\gamma = \frac{\alpha^2}{\alpha^2 + \omega_m^2}$ , and

$$\omega_m^{2*} = \omega_m^2 \gamma \quad (\text{S33})$$

The optimum phenotype for males is close to the one favored by natural selection when the female preference is weak (and  $\alpha^2$  is large); conversely the success of a male depends mostly on its resemblances to the female when natural selection on male fitness is weak (and  $\omega_m^2$  is large). Selection on male siring success is less intense when the natural selection on males and the female preference are weak.

### 3.1.1.G Probability that a male has breeding value $g_m$ given the phenotype $x$ of a female in a pair of parents

To compute  $\varphi(g_f, g_m)$  (eqn S9), we proceed in several steps. We first define the probability that a male has breeding value  $g_m$  given the phenotype  $x$  of a female in a pair of parents (second integral in eqn S9):

$$\chi(g_m|x) = \int p_m(y, g_m) \tilde{w}(y|x) dy \quad (\text{S34})$$

By using the results on the product of Gaussian distributions, one can find that this distribution has a Gaussian form with expectation and variance:

$$E_{\chi_P}(g_m|x) = \bar{g}_m + h^2 S_m^* (\theta_m^*(z|x) - \bar{z}_m) \quad (\text{S35})$$

$$V_\chi(g_m|x) = V_g(1 - h^2 S_m^*) \quad (\text{S36})$$

with  $S_m^* = \frac{V_z}{V_z + \alpha^2(1-\gamma)} = \frac{V_z}{V_z + \omega_m^2}$  the strength of the selection on males around phenotype  $x$  of a given female. This strength of the selection is caused by successive natural and sexual selection.  $S_m^*$  is always stronger than natural selection on males  $S_{nm}$ .

### 3.1.1.H Probability that a male has breeding value $g_m$ and that a female has breeding value $g_f$ in a pair of parents

The probability that a male has the breeding value  $g_m$  given the breeding value  $g_f$  of a female in a pair of parents, denoted  $\varphi(g_f, g_m)$  (eqn S9), has a Gaussian form with expectations, variances and covariance:

$$E_\varphi(g_f) = \bar{g}'_f \quad (\text{S37})$$

$$E_\varphi(g_m) = \bar{g} + h^2 S_m^* (\theta_m \gamma + \bar{z}'_f (1 - \gamma) - \bar{z}_m) \quad (\text{S38})$$

$$V_\varphi(g_f) = V'_{gf} \quad (\text{S39})$$

$$V_\varphi(g_m) = V_g(1 - h^2 \tilde{S}_m) \quad (\text{S40})$$

$$Cov_\varphi(g_f, g_m) = h^2 V_g S_m^* (1 - \gamma)(1 - S_{nf}) \quad (\text{S41})$$

with the variance of the breeding values of potential males in a pair of parents  $\tilde{S}_m$  is:

$$\tilde{S}_m = S_m^* (1 - S_m^* (1 - \gamma)^2 (1 - S_{nf})) \quad (\text{S42})$$

#### 3.1.1.I Distribution of breeding and phenotypic values in the offspring generation

By computing  $p''(z, g)$ , in eqn S6, with the intermediate results from eqn S7 and S9, one finds that the distribution of breeding values in the offspring generation is a bivariate Gaussian with expectation and variance:

$$\begin{aligned} \bar{g}'' &= \frac{E_\varphi(g_f)}{2} + \frac{E_\varphi(g_m)}{2} \\ &= \bar{g} + \frac{h^2}{2} \left( S_{nf}(\theta_f - \bar{g}) + S_m^* \theta_m \gamma + S_m^* \bar{z}'_f (1 - \gamma) - S_m^* \bar{z}_m (1 - \gamma) - S_m^* \bar{z}_m \gamma \right) \end{aligned} \quad (\text{S43})$$

$$\begin{aligned} V_g'' &= \frac{V_{LE}}{2} + \frac{V_\varphi(g_f)}{4} + \frac{V_\varphi(g_m)}{4} + \frac{Cov_\varphi(g_f, g_m)}{2} \\ &= \frac{V_{LE}}{2} + \frac{V_g}{2} \left( 1 - h^2 \frac{S_{nf} + S_m^* (1 - \gamma) + S_m^* \gamma - S_m^* 2(1 - \gamma)^2 (1 - S_{nf}) - 2S_m^* (1 - \gamma)(1 - S_{nf})}{2} \right) \end{aligned} \quad (\text{S44})$$

Stabilizing selection decreases the genetic variance in the offspring generation through the second and third terms in eqn S44, whereas positive associations among allelic effects among and within loci increases the genetic variance in the offspring generation through the last positive term.

One can note that  $S_m^*(1 - \gamma) = \frac{V'_{zm}}{V'_{zm} + \alpha^2}$ , which is a measure of the intensity of stabilizing sexual selection acting on males, denoted  $S_s$ . When also noting that  $S_m^*\gamma = S_{nm}(1 - S_s)$ , eqn 11 and S44 simplify to :

$$\bar{g}'' = \bar{g} + \frac{h^2}{2} [S_{nf}(\theta_f - \bar{g})(1 + S_s) + S_{nm}(\theta_m - \bar{g} - \delta)(1 - S_s) - \delta S_s] \quad (\text{S45})$$

$$V_g'' = \frac{VLE}{2} + \frac{V_g}{2}(1 - h^2 S^*) \quad (\text{S46})$$

with

$$S^* = \frac{S_{nf}(1 + S_s)^2 + S_{nm}(1 - S_s) - S_s(1 + S_s)}{2} \quad (\text{S47})$$

The strength of sexual selection amplifies the effect of the strength of natural selection on females (first term in  $S^*$ ), and weakens the strength of natural selection on males on the genetic variance at equilibrium (second term in eqn S47). The last term in  $S^*$ , caused by sexual selection, decreases  $S^*$ .

### 3.1.1.J Mismatch to the female optimum at equilibrium

At equilibrium, the mean breeding value (eqn S45) in a constant environment is constant ( $\bar{g} - \bar{g}'' = 0$ ) and is:

$$\bar{g} = \frac{1}{2\bar{s}}(S_{nf}(1 + S_s)\theta_f + S_{nm}(1 - S_s)(\theta_m - \delta) - \delta S_s) \quad (\text{S48})$$

with:

$$\bar{s} = \frac{S_{nf}(1 + S_s) + S_{nm}(1 - S_s)}{2} \quad (\text{S49})$$

In a constant environment, the population mean breeding value is at its evolutionary optimum, *i.e.* the phenotype that maximizes the mean population fitness:

$$\bar{\theta} = \bar{g} = \frac{1}{2\bar{s}}(S_{nf}(1 + S_s)\theta_f + S_{nm}(1 - S_s)(\theta_m - \delta) - \delta S_s) \quad (\text{S50})$$

The evolutionary optimum for the population mean breeding value depends on the trade-off between males and females caused by sex-specific selection, and sex-specific trait expression. Sexual selection selects males that are most similar to females. The strength of sexual selection thus increases the contribution of natural selection on females (first term in the brackets in eqn S50), and decreases the contribution of natural selection on males (second term in the brackets in eqn S50). With no sex-specific trait expression ( $\delta = 0$ ) and equal strength of natural selection on males and females, the strength of sexual selection therefore shifts the evolutionary optimum in the direction of the female optimum. The sex-specific trait expression shifts the optimal breeding

value of males ( $\theta_m - \delta$ ) in the direction of females because here we assume that  $\theta_m - \theta_f \geq \delta \geq 0$  if  $\theta_m > \theta_f$ , or,  $\theta_m - \theta_f \leq \delta \leq 0$  if  $\theta_m < \theta_f$ . The last term in brackets in eqn S50 indicates that the strength of sexual selection shifts the evolutionary in the direction of the female optimum.  $\delta > \bar{\theta}_m - \theta_f$  if  $\theta_f < \theta_m$  (or when  $\delta < \bar{\theta}_m - \theta_f$  if  $\theta_f > \theta_m$ ) if sexual selection is strong and sex-specific trait expression is large. The mismatch of the evolutionary optimum to the female optimum therefore changes sign because the evolutionary optimum shifts beyond the female optimum.

The evolutionary optimum and the population mean breeding value in a constant environment are distinct from the male and female optima in most cases (S50). One can define the mismatch of the population to the female or the male optimum. We here focus on the mismatch to the female optimum because in nature it is common that males do not limit reproduction, and that the contribution of females to the population growth rate is higher than that of males (“female demographic dominance”; Crowley 2000; Harts et al. 2014). Conditions decreasing the mismatch of the population mean breeding value to the female optimum can thus increase the probability of persistence of the population. This mismatch in a constant environment is:

$$\theta_f - \bar{g} = \theta_f - \bar{\theta} \quad (\text{S51})$$

In a changing environment with both male and female optima shifting over time at the same speed  $k$ , the population mean breeding value progressively lags behind the evolutionary optimum until it reaches an asymptotic regime (Lynch and Lande 1993, Lande and Shannon, 1996). In this asymptotic regime,  $\bar{g} - \bar{g}' = k$ . From eqn 11 the population mean breeding value is:

$$\bar{g} = \bar{\theta} - \frac{k}{h^2 \bar{s}} \quad (\text{S52})$$

The mismatch of the mean breeding value to the female optimum in the asymptotic regime thus is:

$$\theta_f - \bar{g} = (\theta_f - \bar{\theta}) + \frac{k}{h^2 \bar{s}} \quad (\text{S53})$$

The mismatch to the female optimum is composed of two parts: the first part is caused by the shift of the evolutionary optimum to the female optimum ( $\theta_f - \bar{\theta}$ ) in a constant environment because of sexual selection and sex-specific natural selection; the second part is caused by the shift at the same speed through time of the male and female optima, and is called the adaptive lag ( $\frac{k}{h^2 \bar{s}}$ ). Note that the adaptive lag has the same form than in the classical theory of adaptation to environmental change with no sex-specific trait expression and under random mating if one substitutes  $\bar{s}$  by  $s = \frac{V_z}{V_z + \omega^2}$  (Lande and Shannon, 1996) and is caused by the deviation of the mean trait to the evolutionary optimum.



### 3.1.2 Joint distribution of breeding and phenotypic values under random mating

The relative siring success of a male with phenotype  $y$  is independent of the phenotype  $x$  of the female under random mating. The distribution of offspring breeding and phenotypic values after natural selection and reproduction under random mating has a Gaussian form with mean and variance:

$$\bar{g}'' = \bar{z}'_f = \bar{g} + \frac{h^2}{2}[S_{nf}(\theta_f - \bar{g}) + S_{nm}(\theta_m - \bar{g} - \delta)] \quad (\text{S54})$$

$$V_g'' = \frac{VLE}{2} + \frac{V_g}{2}(1 - h^2 S^*) \quad (\text{S55})$$

with

$$S^* = \frac{S_{nf} + S_{nm}}{2} \quad (\text{S56})$$

The mismatch of the mean breeding value to the female optimum in a constant environment and at equilibrium is:

$$\theta_f - \bar{g} = \theta_f - \bar{\theta} \quad (\text{S57})$$

with the evolutionary optimum for the breeding value:

$$\bar{\theta} = \frac{1}{2\bar{s}}(S_{nf}\theta_f + S_{nm}(\theta_m - \delta)) \quad (\text{S58})$$

with:

$$\bar{s} = \frac{S_{nf} + S_{nm}}{2} \quad (\text{S59})$$

Note that the position of the evolutionary optimum depends on the mean of strength of natural selection on females and males. Sex-specific trait expression shifts the optimal breeding value of males ( $\theta_m - \delta$ ) in the direction of the female optimum. The evolutionary optimum is equidistant of the optimal breeding value (phenotype) of females and the optimal breeding value of males when the strength of natural selection is the same on both sexes. The mean breeding value is at the female optimum when  $\delta = \theta_m - \theta_f$ .

In a changing environment, similarly to assortative mating by female preference, the mismatch to the female optimum in a constant environment and in the asymptotic regime is:

$$\theta_f - \bar{g} = \theta_f - \bar{\theta} + \frac{k}{h^2 \bar{s}} \quad (\text{S60})$$

### 3.1.3 Joint distribution of breeding and phenotypic values under temporal assortative mating

We also study a scenario with an individual distribution of the fertility. For example in plants, the individual distribution of male and female gametes is defined by the individual

distribution of open flowers through days within a year. In birds the individual distribution of gametes is defined by the individual distribution of migration dates over years. The distributions of the number of gametes produced over time for females and males, respectively denoted  $\phi_m(t|x)$  and  $\phi_f(t|y)$ , decline as Gaussian functions with mean  $x$  or  $y$  and width  $\alpha^2$ . Individuals can mate only if their gamete distribution overlap.

In the following we study two extreme scenarios: (i) a given male competes for the access to the fittest females with males having different fitness, and (ii) a given male competes for the access to the fittest females with males that have the same fitness. This last scenario connects to that in Godineau et al., 2021; in which we show in a model with no sex-specific selection and sex-specific trait expression, that competition between males within a time window cancels natural selection on males because males that compete for access to the fittest females have same fitness. We also found that, given a constant genetic variance, the adaptive lag is the same under temporal assortative mating and random mating.

### 3.1.3.A Competition between males with different fitness

The relative mating success between a male with phenotype  $y$  and a female with phenotype  $x$  is:

$$\tilde{w}_m(y|x) = \int \phi_f(t|x) \frac{w_m(y)\phi_m(t|y)}{\iint p_m(g_m, z)w_m(z)\phi_m(t|z)dg_m dz} dt \quad (\text{S61})$$

The distribution of offspring breeding values after natural selection and reproduction is obtained by replacing eqn S61 in eqn S6. The distribution of offspring breeding values has a Gaussian form with mean and variance:

$$\bar{g}'' = \bar{g}' = \bar{g} + \frac{h^2}{2} [S_{nf}(\theta_f - \bar{g})(1 + S_s) + S_{nm}(\theta_m - \bar{g} - \delta)(1 - S_s) - \delta S_s] \quad (\text{S62})$$

$$V_g'' = \frac{VLE}{2} + \frac{V_g}{2} [1 - h^2 S^*] \quad (\text{S63})$$

with:

$$S^* = \frac{S_{nf}(1 + S_s)^2 + S_{nm}(1 - S_s^2) - 2S_s}{2} \quad (\text{S64})$$

Note that the population mean breeding value after natural selection and reproduction is the same than under female preference (eqn S45 and S62). As a result, the mismatch of the population mean breeding value to the female optimum at equilibrium and for a given genetic variance, is the same than under female preference (eqn S51 and S53).

### 3.1.3.B Competition between males with the same fitness

When the quality of gametes depends on time regardless of  $x$  or  $y$ , the distribution of the quality of gametes at time  $t$  is:

$$v_f(t) = \exp\left(\frac{-(t - \theta_f)^2}{2\sigma_f^2}\right) \quad (\text{S65})$$

$$v_m(t) = \exp\left(\frac{-(t - \theta_m)^2}{2\sigma_m^2}\right) \quad (\text{S66})$$

with  $\sigma_f^2$  and  $\sigma_m^2$  the width of the fitness function on the female and male fertility over time.

The individual fitness of females and males is then:

$$w_f(x_f) = \int v_f(t)\phi_f(t|x_f)dt = \sqrt{\frac{\sigma_f^2}{\alpha^2 + \sigma_f^2}} \exp\left(\frac{-(x_f - \theta_f)^2}{2(\alpha^2 + \sigma_f^2)}\right) \quad (\text{S67})$$

$$w_m(y_m) = \int v_m(t)\phi_m(t|y_m)dt = \sqrt{\frac{\sigma_m^2}{\alpha^2 + \sigma_m^2}} \exp\left(\frac{-(y_m - \theta_m)^2}{2(\alpha^2 + \sigma_m^2)}\right) \quad (\text{S68})$$

In order to compare the strength of selection on females and males among types of temporal assortative mating, we assume that  $\omega_f^2 = \alpha^2 + \sigma_f^2$  and  $\omega_m^2 = \alpha^2 + \sigma_m^2$  such that when the quality of gametes is variable through time within year, the individual fitness of females and males can be written as:

$$w_f(x_f) = \sqrt{\frac{\sigma_f^2}{\omega_f^2}} \exp\left(\frac{-(x_f - \theta_f)^2}{2\omega_f^2}\right) \quad (\text{S69})$$

$$w_m(y_m) = \sqrt{\frac{\sigma_m^2}{\omega_m^2}} \exp\left(\frac{-(y_m - \theta_m)^2}{2\omega_m^2}\right) \quad (\text{S70})$$

The relative mating success between a male with phenotype  $y$  and a female with phenotype  $x$  is:

$$\tilde{w}_m(y|x) = \int \tilde{\phi}_f(t|x) \frac{\phi_m(t|y)}{\iint p_m(g_m, z)\phi_m(t|z)dg_m dz} dt \quad (\text{S71})$$

with the proportion of offspring of a female with phenotype  $x$  conceived at time  $t$ :

$$\tilde{\phi}_f(t|x) = \frac{\phi_f(t|x)v_f(t, x)}{w_f(x)} \quad (\text{S72})$$

The distribution of offspring breeding values after natural selection and reproduction is obtained by replacing eqn S71 in eqn S6. The distribution of offspring breeding values has a Gaussian form with mean and variance:

$$\bar{g}'' = \bar{g} + \frac{h^2}{2} [2S_{n,f}(\theta_f - \bar{g}) - \delta S_s] \quad (\text{S73})$$

$$V_g'' = \frac{VLE}{2} + \frac{V_g}{2}(1 - h^2 S^*) \quad (S74)$$

with:

$$S^* = \frac{1}{2} \left( S_{nf} (2 + h^2 + h^4) + S_{nm} (1 - S_s(\gamma(1 - S_s) + S_s)) + 2S_s(\gamma - 1) \right. \\ \left. + h^2 (S_s \gamma (S_s(1 - \gamma) - 1) - h^2 S_s \gamma (S_s(1 - \gamma) + 1)) \right) \quad (S75)$$

With no sex-specific trait expression, the genetic response depends only on natural selection on females as previously found in Godineau et al. (2021) in a model with no sex-specific selection or sex-specific trait expression. The genetic response is different from that under random mating because of the sex-specific selection.

The mismatch to the female optimum at equilibrium and in a constant environment is:

$$\theta_f - \bar{g} = \theta_f - \bar{\theta} \quad (S76)$$

with the evolutionary optimum for the breeding value:

$$\bar{\theta} = \theta_f - \frac{\delta S_s}{2S_{nf}} \quad (S77)$$

In a changing environment, the mismatch to the female optimum at equilibrium is:

$$\theta_f - \bar{g} = \theta_f - \bar{\theta} + \frac{k}{h^2 S_{nf}} \quad (S78)$$



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## Chapitre 3 : Fluctuations in selection strength affect the long-term response to climate change

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### Abstract

There is plenty of empirical evidence of fluctuating selection and fluctuations of the strength of selection in natural populations. Theory has historically been used to understand the effect of fluctuating selection on adaptation, but not the effects of variation in the strength of selection. For example variation in the width of a Gaussian selection function has not yet been studied, despite predicted enhanced variation in the width of the fitness function due to human activities. Here, we derived analytical approximations and used a numerical investigation to evaluate the effects of the variance in the width of a Gaussian fitness function on the long-run responses of populations to climate change. The variation in the width of the fitness function increases the mean strength of selection, and thus decreases both the adaptive lag and the genetic variance of populations. The evolutionary and variance loads are also affected, positively or negatively, by the variation in the width of the fitness function through direct and indirect pathways through the genetic variance and the mean and variance of lag. The variance in the width of the fitness function has mostly negative effects on the long-run growth rate. Our study highlights that the positive effect of the variation in the width of the fitness function on rapid evolutionary responses to climate change does not necessarily mitigate its negative consequences on demography.



## 4.1 Introduction

### 4.1.1 Fluctuating selection and its impacts on evolution

There is empirical evidence of fluctuating selection in natural populations (Ehrlén and Valdés, 2020; Grant and Grant, 2002; McAdam et al., 2018; Nosil et al., 2018; Reimchen and Nosil, 2002; van Buskirk and Smith, 2021), some of which has become textbook example such as fluctuating selection on body size or beak shape of Darwin's Finches (Grant and Grant, 2002). Based on a 30 years study, Grant and Grant (2002) highlights that body size and beak shape of Darwin's Finches respond to changes in the direction of selection, which is caused by variation in their food resource due to rain and drought. Another long-term survey shows that fluctuations in the strength of directional selection on flowering time (Ehrlén and Valdés, 2020) are linked to climate: increased minimum spring temperature and decreased spring precipitation both increase the strength of selection for early flowering. Climate also induces variations in the density of predators (Reimchen and Nosil, 2002) or variations in the colour of plants for cryptic insects (Nosil et al., 2018). Those variations can cause fluctuating selection on phenotypes of prey and cryptic insects. Climate therefore is an important source of fluctuating selection in nature. Fluctuating selection has strong consequences on the evolution of populations: it can maintain phenotypic diversity (Bull, 1987; Lande and Shannon, 1996; Tufto, 2015) and generate phenotypic variation by favouring the evolution of mechanisms such as bet hedging and/or phenotypic plasticity (Bull, 1987; Gavrillets and Scheiner, 1993; Gillespie, 1974; Tufto, 2015). Fluctuating selection can also connect micro- and macro-evolution by explaining how populations can rapidly respond to environmental change despite evolutionary stasis at geological time scale (Uyeda et al., 2011). Fluctuating selection received also attention because of its demographic consequences (Lande, 1993; Lande et al., 2003; Sæther and Engen, 2015). Indeed fluctuating selection, in combination with demographic stochasticity, affects the long-run growth rate of populations, which is an indicator of the extinction risk of populations (Lande, 1993). Theoretical studies of the effects of fluctuating selection on evolution commonly assume a fitness function with a Gaussian form and an optimal phenotype that maximises fitness and that varies over time but a constant width for such fitness function (Bürger and Lynch, 1995; Chevin et al., 2015; Lande and Shannon, 1996; Lynch et al., 1991; Lynch et al., 1993, but see Revell, 2007 for variation of the selection strength). Models therefore assume that the strength of stabilising selection does not vary with time, only the position of the optimum does. There are many reasons to expect the strength of stabilising selection to vary with climate, for instance phenology should be under weaker selection in years where the favourable season is long compared to when it is short. Increased temperature increases the duration of seasons by advancing the beginning of the growing season of plants and delaying its end, except when an extreme drought shortens the season (Anderson et al., 2012; Bradshaw and Holzapfel, 2008; Hamann et al., 2018; Parmesan, 2006). Anthropogenic activities can increase variation in the duration of seasons through their effects on temperature and precipitation regimes (IPCC, 2014). We here study the effects of

the variation of the width of the fitness function on the evolution of populations adapting to a fluctuating and warming climate.

#### 4.1.2 Measuring fluctuating selection

Selection is frequently quantified by selection coefficients (see Bell, 2010 for a review) or selection gradients (Lande and Arnold, 1983, see reviews in Kingsolver et al., 2012; Kingsolver and Pfennig, 2007; Morrissey and Hadfield, 2012; Siepielski et al., 2009). Linear coefficients in gradients of selection measure how selection relates the population mean phenotype to the population mean fitness, whereas quadratic coefficients in the empirical relationship between phenotypes and fitness measures how variance of the trait and covariance with others measured translate into population mean fitness (Brodie et al., 1995). The quadratic gradient of selection indicates if the relationship between the fitness and phenotypes is concave or convex (Lande and Arnold, 1983). Sufficient phenotypic variation is thus required to estimate selection gradients in natural populations. Meta-analyses highlight the difficulties to detect significant fluctuating selection in linear and quadratic terms of selection because of large sampling errors in estimates (Kingsolver et al., 2012; Kingsolver and Pfennig, 2007; Morrissey and Hadfield, 2012; Siepielski et al., 2009). This suggests that, in most cases, we probably need more data of better quality (*i.e.* with smaller sampling error) to clearly detect fluctuating selection (de Villemereuil et al., 2020; Haller and Hendry, 2014; Kingsolver and Pfennig, 2007; Morrissey and Hadfield, 2012). When fluctuating selection is detected, it is difficult to define whether fluctuations are caused by variation in the optimum and/or variation in the width of the selection function (Chevin et al., 2015). A recent study however shows that fluctuations among years in the severity of disturbance and the intensity of biotic interactions change the curvature of selection on pool location and hatching date of a frog species (van Buskirk and Smith, 2021).

#### 4.1.3 How can theory clarify the role of the fluctuating selection ?

Theory can complement empirical data to understand the effects of fluctuating selection on the fate of populations. A Gaussian function of selection with a given optimal phenotype and a given width is classically assumed (see a review in Kopp and Matuszewski, 2014). This hypothesis is consistent with empirical data (de Villemereuil et al., 2020). The optimum can change linearly and/or fluctuate over time whereas the fitness width is always assumed constant (Kopp and Matuszewski, 2014 but see Revell, 2007). In a constant environment, even when the mean phenotype in the population matches the optimum, in the presence of phenotypic variance around this mean, the deviation of individual phenotypes from the optimum induces a loss in the population mean fitness, which is called a variance load (Lande and Shannon, 1996). The variance load increases with the phenotypic variance and the inverse of the fitness width, *i.e.* the strength of stabilising selection. In a changing environment, the deviation of the mean phenotype to the optimum also induces a loss in the population mean fitness called the evolutionary load (Lande and Shannon, 1996). A narrow fitness function limits the deviation of the mean phenotype to the moving optimum. When the optimum fluctuates with little temporal autocorrelation or in complete unpredictable manner, genetic response to selection a given generation can be

maladaptive at the next generation (Lande and Shannon, 1996). A fluctuating optimum impacts both the variance and evolutionary loads and the resulting population mean fitness. Theoretical studies including variation in the width of the fitness function however are scarce (but see Revell, 2007). In a study with several selection episodes over a generation, a recent study showed that the total strength of selection over this generation is similar to a harmonic mean of the width of the fitness function (Cotto and Chevin, 2020).

#### 4.1.4 Demographic consequences of fluctuating selection

Population mean fitness  $\bar{w}(t)$  at time  $t$  defines the instantaneous growth rate of the population  $r(t) = \ln(\bar{w}(t))$ . The population size in the next time step increases if the growth rate is positive, is constant if the growth rate is null, and decreases if the growth rate is negative (Lande, 1993). However demographic and environmental stochasticity induce temporal variations in the population mean fitness (Lande, 1993). Beyond having the best population growth rate at a given time, the most important for population persistence is to have the best population growth rate over a long time. For example, a population with a high population growth rate in good years and a low population growth rate in bad years would be more prone to extinction in bad years than a population with an almost constant population growth rate. Evolution therefore does not maximise the arithmetic population mean fitness, but instead, the population geometric mean fitness (*i.e.* the geometric mean of growth rates; Sæther and Engen, 2015), which measures the long-run growth rate of the population (Lewontin and Cohen, 1969). For a given population, the geometric mean fitness depends on the temporal expectation of the logarithm of its mean fitness  $\exp(E_t[\ln(\bar{w}(t))])$ . Strategies reducing the temporal variance of the population mean fitness over time decrease the negative effect of fluctuating selection in the population mean fitness and can thus be selected; such strategies include phenotypic plasticity and bet-hedging (Bull, 1987; Gavrilets and Scheiner, 1993; Gillespie, 1974; Tufto, 2015).

#### 4.1.5 Questions, methods and main results

We here investigate how variation in the width of the fitness function affects the strength of selection, the genetic variance at equilibrium, the evolutionary load, the variance load, and the geometric mean fitness of populations. Based on the classical literature of adaptation to a changing environment, we use a selection function with a Gaussian form. We here assume that the width of this function fluctuates across years/generations. In years with strong selection, we expect based on previous theory that the genetic variance decreases, but the population better tracks its optimum. More selective deaths should also occur in years with strong selection. Conversely, in years with weak selection, the population mean phenotype may lag further from the optimum, the genetic variance is less depleted by selection and the mean fitness should be higher. Whether these effects just compensate each other over time when the strength of stabilising selection varies randomly across years is however hard to predict a priori. We therefore use a model to clarify our expectations in this regard. We develop analytical approximations, assuming a constant genetic variance across years, for the strength of selection, the genetic variance at equilibrium, the evolutionary load, the variance load, and the geometric mean fitness

of populations. We compare those analytical approximations with a numerical investigation of an infinitesimal model of trait inheritance where the genetic variance evolves and fluctuates across years. We find that the variation in the width of the fitness function increases the mean strength of selection and thus facilitates evolutionary responses. It also increases or decreases the evolutionary and variance loads through direct and indirect pathways. In most cases, the variance in the width of the fitness function has negative effects on the long-run growth rate.

## 4.2 Methods

### 4.2.1 Genetic architecture

We assume a single quantitative trait determined by a large number of loci. In generation  $t$ , the distribution of breeding values for the trait in the population is a Gaussian with mean  $\bar{g}(t)$  and variance  $V_g(t)$ . At birth, environmental effects are distributed as a Gaussian with mean 0 and variance  $V_e$ . The phenotypic value of an individual is the sum of its breeding value  $g$  and a random environmental effect  $e$ :

$$z = g + e \quad (1)$$

Under these assumptions, the distribution of the trait in the population in generation  $t$ , denoted  $p_z(t)$ , is also a Gaussian with mean  $\bar{z}(t) = \bar{g}(t)$  and variance  $V_z(t) = V_g(t) + V_e$ .

### 4.2.2 Fluctuating selection

Selection acts on the survival or fecundity of individuals. Fitness varies over years and has a Gaussian form with optimum  $\theta(t)$  and width  $\omega^2(t)$ , hereafter called fitness width. The fitness of an individual with phenotype  $z$  in generation  $t$  is:

$$w_z(t) = \exp\left(-\frac{(z - \theta(t))^2}{2\omega^2(t)}\right) \quad (2)$$

The fitness of an individual increases with the fitness width  $\omega^2(t)$  and the match between its phenotype  $z$  and the optimum  $\theta(t)$ . The optimum can randomly fluctuates over years and can increase with a linear trend. The fitness width randomly fluctuates over years (*i.e.* white noise) and independently from the optimum.

### 4.2.3 Adaptive lag

The mal-adaptation of the population is measured by the lag of the population mean phenotype to the optimal phenotype (Lande and Shannon, 1996) in generation  $t$  as:

$$l(t) = \bar{z}(t) - \theta(t) \quad (3)$$

#### 4.2.4 Population mean fitness and fitness loads

Using results on the product of two Gaussian functions and our assumption of a Gaussian distribution of phenotypes, the population mean fitness in generation  $t$  is:

$$\bar{w}(t) = \int w_z(t)p_z(t)dz = \sqrt{\frac{\omega^2(t)}{\omega^2(t) + V_z(t)}} \exp\left(-\frac{(\bar{z}(t) - \theta(t))^2}{2(\omega^2(t) + V_z(t))}\right) \quad (4)$$

When the mean phenotype is at the optimum and that there is no phenotypic variance, the maximal fitness is equal to one. The fitness load is the expected loss of mean Malthusian fitness due to genetic and evolutionary factors (Lande and Shannon, 1996) such as:

$$L(t) = \log(w_{max}) - \log(\bar{w}(t)) = \log(w_{max}) - \frac{1}{2} \log\left(\frac{\omega^2(t)}{\omega^2(t) + V_z(t)}\right) + \frac{(\bar{z}(t) - \theta(t))^2}{2(\omega^2(t) + V_z(t))} \quad (5)$$

The loss of fitness caused by the phenotypic variance is measured by the variance load (Lande and Shannon, 1996) such as:

$$L_v(t) = -\frac{1}{2} \log\left(\frac{\omega^2(t)}{\omega^2(t) + V_z(t)}\right) = -\frac{1}{2} \log(\omega^2(t)\gamma(t)) \quad (6)$$

with the strength of selection measured as:

$$\gamma(t) = \frac{1}{V_z(t) + \omega^2(t)} \quad (7)$$

The loss of fitness caused by the deviation of the mean phenotype to the optimal phenotype is measured by the evolutionary load (Lande and Shannon, 1996) such as:

$$L_e(t) = \frac{(\bar{z}(t) - \theta(t))^2}{2(\omega^2(t) + V_z(t))} = \frac{1}{2}l(t)^2\gamma(t) \quad (8)$$

The geometric mean fitness is a measure of the long-run growth rate of the population (Lewontin and Cohen, 1969) and is:

$$\bar{w}^* = \exp(E_t[\log(\bar{w}(t))]) \quad (9)$$

#### 4.2.5 Distribution of breeding values in the offspring generation

We use an infinitesimal model of trait inheritance to derive predictions for the changes over generations in the mean breeding value and in the genetic variance. Given the breeding values of parents, the infinitesimal model assumes that the distribution of breeding values in the family is distributed as a Gaussian, with mean equal to the average breeding value of the two parents. The genetic variance of the family is constant and equal to half the genic variance in the population  $V_{LE}$  (i.e. the genetic variance at linkage and Hardy Weinberg equilibrium). The probability that

an offspring has breeding value  $g$  given the breeding value  $g_f$  and  $g_m$  of its father and its mother respectively is:

$$R(g|g_f, g_m) = \frac{1}{\sqrt{\pi V_{LE}}} \exp\left(-\frac{(g - \frac{g_f + g_m}{2})^2}{V_{LE}}\right) \quad (10)$$

The distribution of the breeding values in the offspring generation after natural selection and reproduction (Barton et al., 2017) is a Gaussian with mean and variance:

$$\bar{g}(t+1) = \bar{g}(t) + V_g(t)\gamma(t)(\theta(t) - \bar{g}(t)) \quad (11)$$

$$V_g(t+1) = \frac{V_{LE}}{2} + \frac{V_g(t)}{2}(1 - V_g(t)\gamma(t)) \quad (12)$$

#### 4.2.6 Analytical approximations

We derive approximations for the temporal expectation of (i) the genetic variance, (ii) the strength of selection, (iii) the adaptive lag, (iv) the fitness loads and (v) the geometric mean fitness (eqn 12, 7, 3, 6, 8, 9). The genetic variance is assumed to have reached an equilibrium and to slightly fluctuate around its equilibrium such that  $V_g(t) = V_g = E_t[V_g(t)]$ . The population is also assumed to be in a stationary equilibrium such that the average change in the phenotypes over time is equal to the average speed of the environmental change.

The deviation of the fitness width in generation  $t$  to its expectation, denoted  $\delta(t)$ , is small such that  $\delta(t) = \omega^2(t) - E[\omega^2(t)] \approx 0$ . We use a Taylor series around  $\delta(t) = 0$  to the order 2 for the strength of selection, the evolutionary load and the variance load (eqn 7, 6, 8).

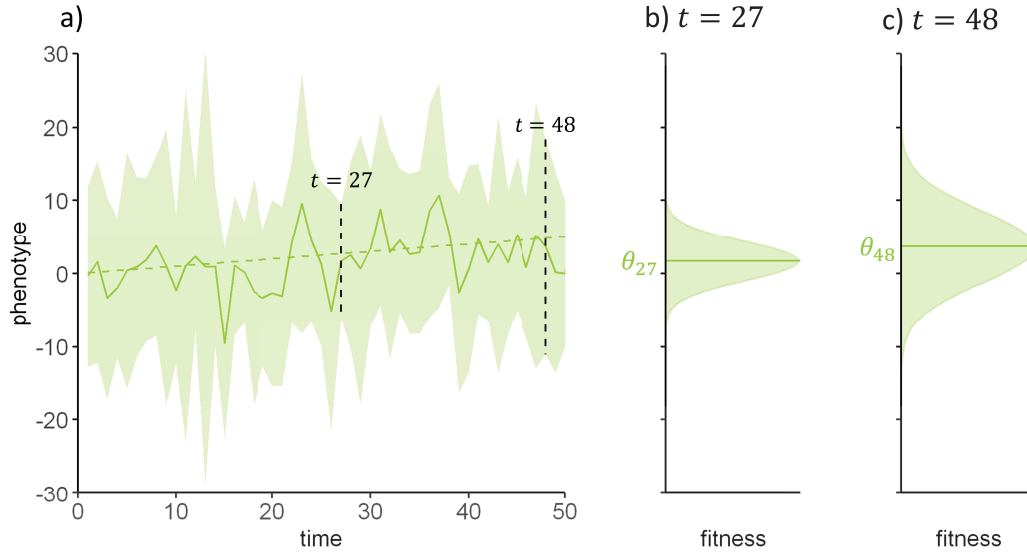
#### 4.2.7 Numerical investigation

We then test the robustness of the analytical approximations to an evolving genetic variance by comparing approximations with simulations by a recurrence over time on the mean breeding value and the genetic variance (eqn 11, 12). The optimum increases with a linear trend and randomly fluctuates over years:

$$\theta(t+1) = \theta(0) + k \times t + \epsilon(t) \quad (13)$$

with  $\theta(0)$  the initial optimum,  $k$  the speed of change of the optimum (hereafter called speed of the environmental change) and set to 0.05, 0.1, 0.15 or 0.2, and  $\epsilon(t)$  the random deviation of the optimum to the linear trend in generation  $t$  sampled in a Normal distribution with mean 0 and variance  $V_\theta = 0, 3$  or 10 (*i.e.* white noise). The fitness width  $\omega^2(t)$  randomly fluctuates over years (*i.e.* white noise) and independently from the optimum. We ensure that the sampled fitness width are strictly positive by sampling in a LogNormal distribution with mean  $E_t[\omega^2(t)]$  and variance  $V_t[\omega^2(t)]$ . The temporal expectation of the fitness width  $E_t[\omega^2(t)]$  is 20, 50 or 100. The variance of the fitness width  $V_t[\omega^2(t)]$  takes values in range of integers from 0 to 500 (see

fig 1 for an example of environmental change). Simulations last for 10 000 generations. The strength of selection, the adaptive lag, the geometric mean fitness, fitness loads and the genetic variance are averaged over the last 9 000 generations. We thus ensure to measure metrics when the population has reached a stationary equilibrium such that the slope of the genetic variance over time is quasi null (from  $-8 \times 10^{-5}$  to  $7.9 \times 10^{-5}$ ).



**Figure 1:** Example of variation of the optimum (solid green line) and the fitness width (green shaded area) over 50 generations. The dashed green line indicates the linear trend of the optimum. The middle panel represents the Gaussian fitness function at the 27<sup>th</sup> generation indicated by the first black dashed line on the left panel. The right panel represents the Gaussian fitness function at the 48<sup>th</sup> generation indicated by the second black dashed line on the left panel. Parameter values are:  $V[\omega^2(t)] = 200$ ,  $E[\omega^2(t)] = 20$ ,  $k = 0.1$ ,  $V_\theta = 3$ .

## 4.3 Results

### 4.3.1 Reference case with a constant fitness width

We first recall the predictions for the strength of selection, the adaptive lag, the evolutionary load and the variance load with a constant fitness width such as  $\omega^2 = E_t[\omega^2(t)]$  (derivations in the Appendix 3). The genetic variance is assumed to be at equilibrium such that  $V_g(t) = V_g = E_t[V_g(t)]$ . To simplify the expressions below,  $E_t[V_g(t)]$  is denoted as  $V_g$ . In a such scenario, the strength of selection is:

$$\gamma_c = \frac{1}{V_g + V_e + \omega^2} \quad (14)$$

Increased genetic variance at equilibrium and increased fitness width decreases the strength of stabilising selection.

The temporal expectation of the adaptive lag is:

$$E_t[l_c(t)] = \frac{k}{V_g \gamma_c} = \frac{k(V_g + V_e + \omega^2)}{V_g} \quad (15)$$

Increased speed of the environmental change  $k$  and increased fitness width both increase the adaptive lag, while increased strength of selection and increased genetic variance decrease the adaptive lag.

The temporal expectation of the evolutionary load, as defined by Lande and Shannon (1996), is:

$$E_t[L_{ec}(t)] = \frac{1}{2}(V_t[l_c(t)] + E_t[l_c(t)]^2)\gamma_c \quad (16)$$

The strength of selection, the temporal mean and variance of the adaptive lag increase the evolutionary load.

The temporal expectation of the variance load, as defined by Lande and Shannon (1996), is:

$$E_t[L_{vc}(t)] = \frac{1}{2} \log\left(\frac{1}{\omega^2 \gamma_c}\right) = \frac{1}{2} \log\left(\frac{V_g + V_e + \omega^2}{\omega^2}\right) \quad (17)$$

Increased strength of selection decreases the variance load. The genetic variance increases the variance load whereas the fitness width decreases the variance load.

Interestingly, the strength of selection  $\gamma_c$  is a key factor in the prediction of the adaptive lag and the two fitness loads.

#### 4.3.2 Results with variation in the fitness width

##### 4.3.2.A Strength of selection

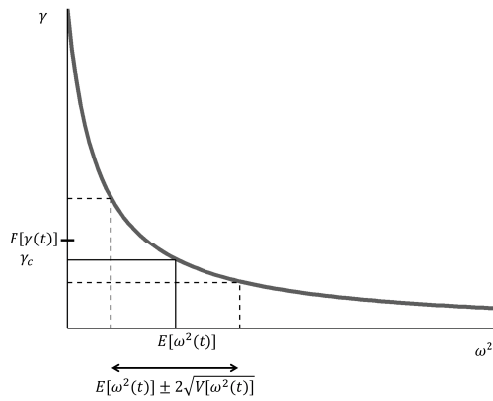
The temporal expectation for the strength of selection for a constant genetic variance (eqn 7; see Appendix 3 for the derivation) is:

$$E_t[\gamma(t)] \approx \frac{1}{E_t[\omega^2(t)] + V_g + V_e} + \frac{V_t[\omega^2(t)]}{(E_t[\omega^2(t)] + V_g + V_e)^3} \approx \gamma_c(1 + V_t[\omega^2(t)]\gamma_c^2) \quad (18)$$

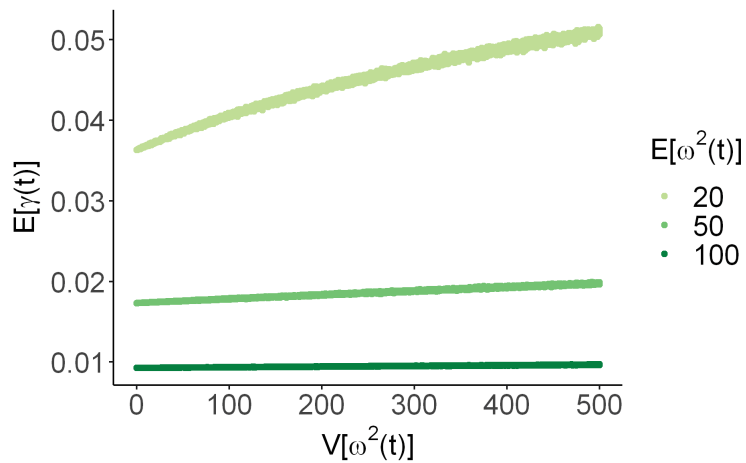
The relation between the strength of selection and the fitness width is concave (fig 2). As a consequence, the temporal expectation for the strength of selection with variation of the fitness width is higher than the strength of selection with a constant fitness width (eqn 14). This indicates that years of strong selection have a stronger impact on the strength of selection than do years of low selection.

The variation in the fitness width  $V_t[\omega^2(t)]$  increases the expectation for the strength of selection compared to a constant fitness width (see the second term in brackets on the right hand side of eqn 18 and fig 2). When selection is weak on average, the variation in the fitness width has little effect on the strength of selection (see the denominator of the second term on the middle equation in eqn 18). The predicted pattern is robust to evolving genetic variance (fig 3). The quality of the prediction however decreases with the variation in the fitness width (fig S2). The deviation of the analytical investigation from its simulated value increases with decreased





**Figure 2:** Strength of selection as a function of the fitness width. The temporal expectation for the strength of the selection with variation in the fitness width  $E_t[\gamma(t)]$  is higher than the strength of the selection with a constant fitness width  $\gamma_c$ . This effect is caused by the curvature of the relation between the strength of selection and the fitness width.



**Figure 3:** Strength of selection averaged over time in simulations as a function of the variation in the fitness width. The colour of symbols indicates the average fitness width.

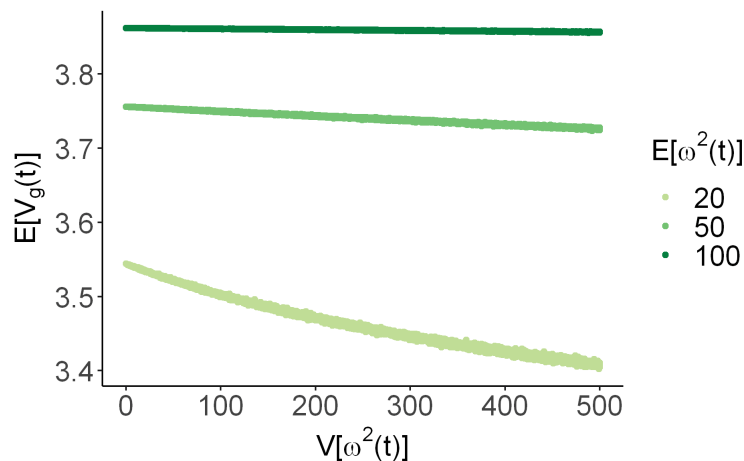
average fitness width, and is probably caused by the violation of the hypothesis that the variation in fitness width is small.

#### 4.3.2.B Genetic variance at equilibrium

When genetic variance slightly fluctuates around its equilibrium, we find that the temporal expectation of the genetic variance (eqn 12; see Appendix for derivation) is approximated by:

$$E_t[V_g(t)] \approx \frac{-1 + \sqrt{1 + 4V_{LE}E[\gamma(t)]}}{2E[\gamma(t)]} \quad (19)$$

The average strength of selection  $E_t[\gamma(t)]$  decreases the average genetic variance. The variation in the fitness width thus decreases the genetic variance through its positive effect on the strength of selection. When selection is weak on average, the variation in the fitness width has little effect on the genetic variance. We have also showed previously that the genetic variance decreases the average strength of selection  $E_t[\gamma(t)]$ . Those results suggest a negative feedback between the genetic variance and the strength of selection.



**Figure 4:** Genetic variance averaged over time in simulations at equilibrium as a function of the variation in the fitness width. The colour of symbols indicates the average fitness width.

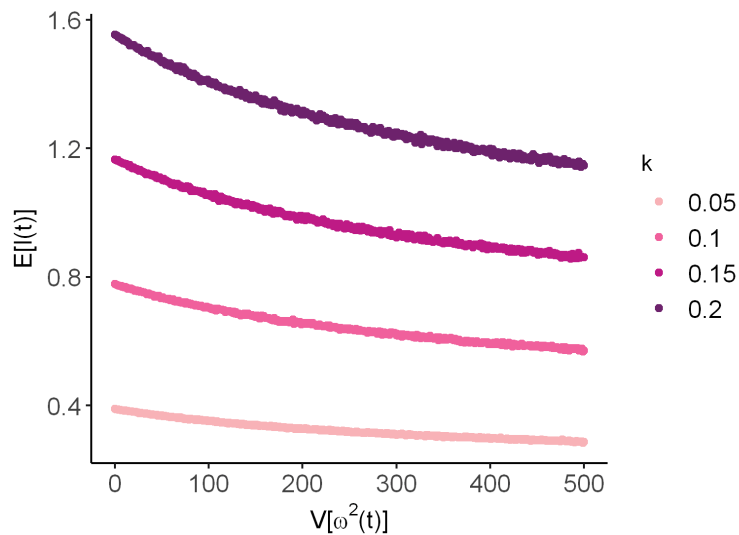
In simulations, the coefficient of variation of the genetic variance around its equilibrium value is small ( $< 0.72\%$ ), indicating that the fluctuations of the genetic variance around its equilibrium are small (see also fig S1 for an example of temporal dynamic of the genetic variance). The predicted pattern is robust to evolving genetic variance (fig 4). Simulated genetic variances are very well predicted by approximations (prediction error ranging from  $-0.32$  to  $0.008\%$ ; fig S3). The quality of the prediction slightly decreases with the variation in the fitness width, and with decreased average width of fitness. This is probably caused by the deviation of the hypothesis that the genetic variance slightly fluctuates.

### 4.3.2.C Adaptive lag

Assuming that the mean phenotype slightly fluctuates, the temporal expectation for the adaptive lag (eqn 3; see Appendix for derivation) is:

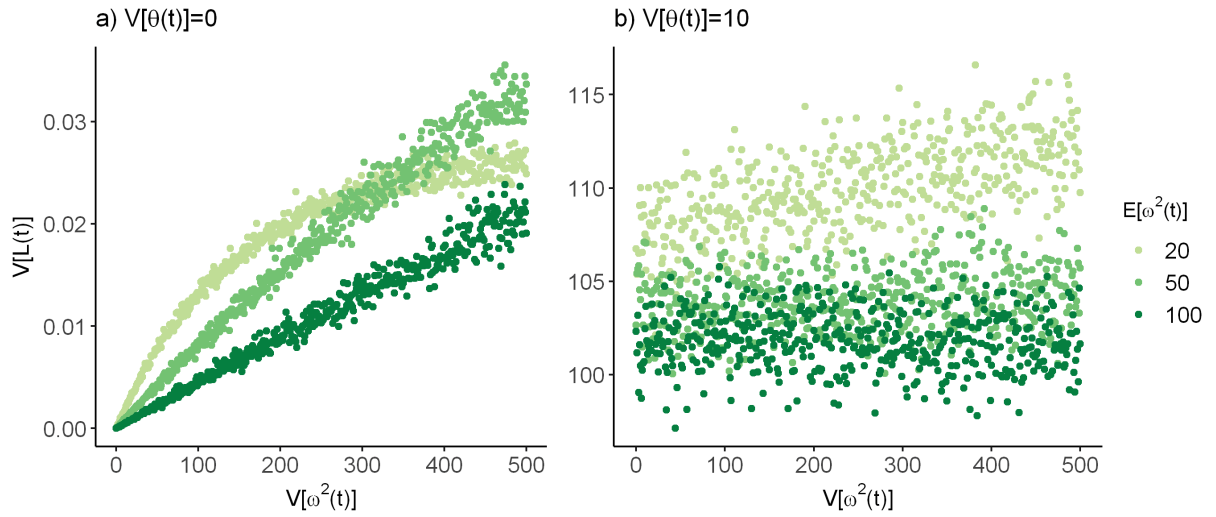
$$E_t[l(t)] \approx \frac{k}{V_g E[\gamma(t)]} \quad (20)$$

Interestingly, the expectation for the adaptive lag  $E_t[l(t)]$  is explained by the expectation of the strength of selection (eqn 18 and fig 5). The variation in the fitness width decreases the adaptive lag through its positive effect on the strength of selection. The speed of the environmental change increases the adaptive lag. With no fluctuation of the optimum, the prediction fits well the simulated values (error prediction ranging from  $-0.55$  to  $0.03\%$ ; fig S4). The quality of the prediction however decreases with increased fluctuations of the optimum (error prediction ranging from  $-55.7$  to  $23.3\%$ ; fig S4). This may be caused by the violation of the hypothesis that the mean phenotype slightly fluctuates.



**Figure 5:** Adaptive lag averaged over time in simulations as a function of the variation in the fitness width. The colour of symbols indicates the speed of the environmental change. The optimum does not fluctuate around its trend. Results presented for  $E[\omega^2(t)] = 20$ .

As the evolutionary load (eqn 16) depends on the variance of the adaptive lag, we also investigated the effect of the variation in the fitness width on the variance of the adaptive lag. This effect depends on the variance of the optimum of the selection function and the average width of the fitness function (fig 6). When the optimum shifts at a constant speed with no fluctuation, the variation in the fitness width slightly increases the variance of the adaptive lag (fig 6a). When the optimum fluctuates around a trend, the effect of the variation in the fitness width on the variance of the adaptive lag depends on the average fitness width (fig 6b). Indeed, the variance of the adaptive lag increases (respectively decreases) with increased variation in the fitness width when the average fitness width is low (respectively high).



**Figure 6:** Variance over time in the simulations for the adaptive lag as a function of the variation in the fitness width. The colour of symbols indicates the average fitness width. The optimum is constant (a) or fluctuating (b). Note the difference in scales for y-axes.

#### 4.3.2.D Evolutionary load

Assuming slight variation in the fitness width, the temporal expectation for the evolutionary load (eqn 8; see Appendix for derivation) is:

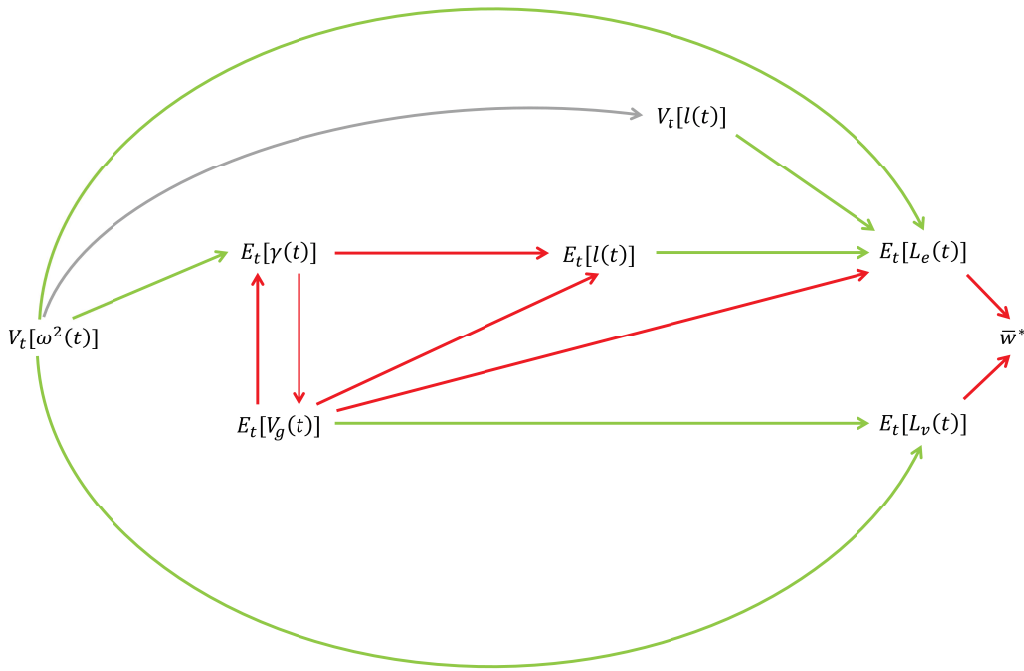
$$E_t[L_e(t)] \approx L_{ec}(1 + V_t[\omega^2(t)]\gamma_c^2) \quad (21)$$

Variation in the fitness width increases the evolutionary load compared to a constant fitness width (eqn 21). The strength of selection with a constant fitness width increases the positive effect of the variation in the fitness width on the evolutionary load. One can also write the evolutionary load as:

$$E_t[L_e(t)] \approx \frac{V_t[l(t)] + E_t[l(t)]^2}{2(E_t[\omega^2(t)] + V_g + V_e)} \left( 1 + \frac{V_t[\omega^2(t)]}{(E_t[\omega^2(t)] + V_g + V_e)^2} \right) \quad (22)$$

The average and the variance of the adaptive lag increase the evolutionary load (eqn 22). The variation in the fitness width has therefore an indirect negative effect through the average adaptive lag and an indirect positive or negative effect through the variance of the adaptive lag (fig 7). The genetic variance decreases the evolutionary load and the variation in the fitness width has therefore an indirect positive effect on the evolutionary load through the genetic variance. The effect of  $V_t[\omega^2(t)]$  on the expectation of the evolutionary load thus depends on which of its positive or negative effect dominates the other.

The numerical investigation suggests that the indirect negative effect of the variation in the fitness width on the evolutionary load dominates when the optimum does not fluctuate around its trend (fig 8a). In contrast, the positive effect of the variation in the fitness width on



**Figure 7:** Direct and indirect pathways through which the variation in the fitness width affects the temporal expectation of the components of the model. Positive effects are indicated by green arrows and negative effects are indicated by red arrows. The grey arrow indicates an effect that can be positive or negative depending on the parameter range.

the evolutionary load dominates when the optimum fluctuates (fig 8b). The quality of predictions however decreases with the variation in the strength of selection (prediction error ranging from  $-22.1$  to  $1.4\%$ ; fig S5). The deviation of the analytical approximation from its simulated value increases with decreased average fitness width, and is probably caused by the violation of the hypothesis that the variation in fitness width is slight.

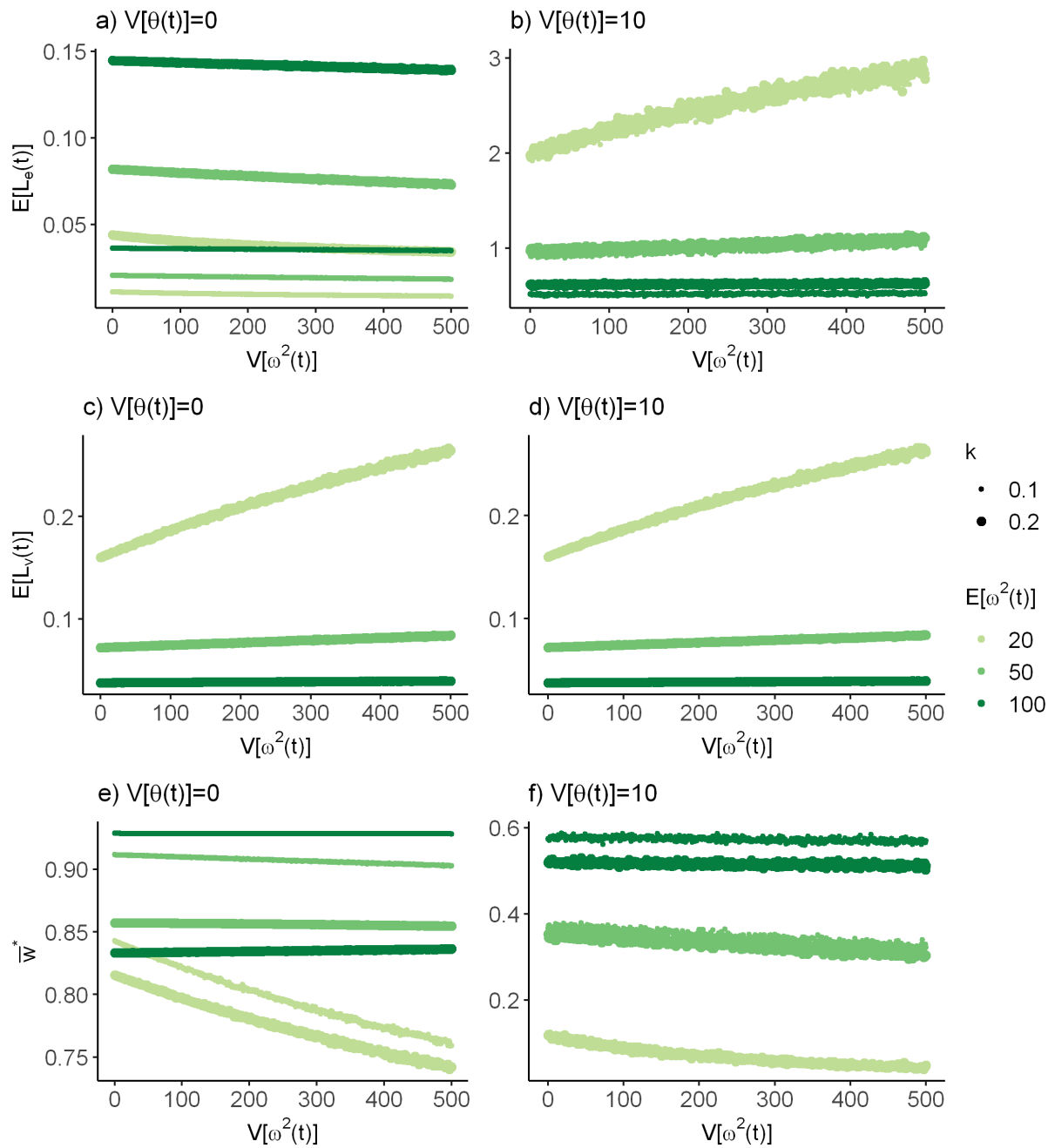
#### 4.3.2.E Variance load

Assuming slight variation in the fitness width, the temporal expectation for the variance load (eqn 6; see Appendix for derivation) is:

$$E_t[L_v(t)] \approx L_{vc} + V_t[\omega^2(t)]\gamma_c^2 \frac{V_z(2E_t[\omega^2(t)] + V_z)}{4E_t[\omega^2(t)]^2} \quad (23)$$

The variation in the fitness width increases the expectation for the variance load  $E_t[L_v(t)]$  compared to a constant fitness width (eqn 23). The strength of selection with a constant fitness width increases the positive effect of the variation in the fitness width on the variance load (see the second term in eqn 23). One can also write the variance load as:

$$E_t[L_v(t)] \approx \frac{1}{2} \log \left( \frac{E_t[\omega^2(t)] + V_z}{E_t[\omega^2(t)]} \right) + V_t[\omega^2(t)] \frac{V_z(2E_t[\omega^2(t)] + V_z)}{4E_t[\omega^2(t)]^2(E_t[\omega^2(t)] + V_z)^2} \quad (24)$$



**Figure 8:** Evolutionary load (a and b), variance load (c and d), and geometric mean fitness (e and f) averaged over time in the simulations as a function of the variation in the fitness width. Left panels correspond to a constant optimum while right panels correspond to a fluctuating optimum such as  $V_\theta = 10$ . The colour of symbols indicates the average fitness width. The size of symbols indicates the speed of the environmental change. Note the difference in scales for y-axes.

Genetic variance increases the variance load (see the numerator of the last term in eqn 24). We previously found that the variation in the fitness width decreases the genetic variance. As a result, the variation in the fitness width indirectly decreases the variance load through the genetic variance (eqn 19). The effect of  $V_t[\omega^2(t)]$  on the expectation of the variance load thus depends on which of its positive or negative effect dominates the other.

The numerical investigation suggests that the direct positive effect of the variation in the fitness width on the variance load dominates its indirect negative effect (fig 8c and d). The quality of the prediction however decreases with the variation in the strength of selection (prediction error ranging from  $-16.9$  to  $1.1\%$ ; fig S6). The deviation of the analytical approximation from its simulated value increases with decreased average fitness width, and is probably caused by the violation of the hypothesis that the variation in fitness width is slight.

#### 4.3.2.F Geometric mean fitness

The geometric mean fitness (eqn 9) is:

$$\bar{w}^* = \exp(-E[L_v(t)] - E[L_e(t)]) \quad (25)$$

The expectation of both fitness loads decreases the geometric mean fitness. The variation in the fitness width can have positive or negative effect on the fitness loads (eqn 24 and 22). The effect of the variation in the fitness width depends thus on which load dominates the other.

In our numerical investigation, the variation in the fitness width decreases the geometric mean fitness in most cases (fig 8e and f). Indeed, with a constant optimum, the variance load is larger than the evolutionary load and the positive effect of the variation in the fitness width on the variance load therefore dominates its negative effect on the evolutionary load. As a result, the variation in the fitness width decreases the geometric mean fitness (fig 8e), except when the environmental change is fast and the average fitness width is low. In that case, the negative effect of the variation in the fitness width on the evolutionary load dominates its positive effect on the variance load. The variation in the fitness width therefore increases the geometric mean fitness (fig 8e). With fluctuations in the optimum, the positive effect of the variation in the fitness width on the two fitness loads decreases the geometric mean fitness (fig 8f).

## 4.4 Discussion

### 4.4.1 Main results

Despite an historical strong interest in understanding the effect of fluctuating selection on adaptation, the effect of the variation in the fitness width remains undetermined. However, empirical data suggest that the fitness width fluctuates over time (van Buskirk and Smith, 2021)

and its variation could increase with human activities (IPCC, 2014). Here, we derived analytical approximations and used a model of evolution of breeding values to investigate the effects of the variation in the fitness width on the evolutionary responses and the long-run growth rate of populations. We found that variation in the fitness width increases the strength of selection and decreases the genetic variance. Evolutionary and variance loads are both affected by the variation in the fitness width through direct and indirect pathways through the genetic variance and the mean and variance of lag. The strength of selection is a key element to understand the indirect effect of the variation in the fitness width on the evolutionary and variance loads as it defines the genetic variance and the mean lag. The effect of the variation in the fitness width on the geometric mean fitness is negative, except when the environmental change is fast and the average fitness width is high.

#### 4.4.2 The geometric mean fitness is affected directly and indirectly by the variation in the fitness width

We found that the variation in the fitness width has direct and indirect effects on the geometric mean fitness. The variation in the fitness width increases directly the evolutionary and variance fitness loads compared to a constant width for the selection function (Lande and Shannon, 1996). Fitness loads are also indirectly affected by the variation in the fitness width through the strength of selection. The variation in the fitness width, through the mean adaptive lag, has a negative effect on the evolutionary load. It also has positive or negative effect on the evolutionary load through the variance of the adaptive lag. Through its effect on the genetic variance, the variation in the fitness width has a positive effect on the evolutionary load and a negative effect on the variance load. The mean and the variation in the fitness width, the genetic variance, the variance of the adaptive lag and the variance of the optimum define if the variation in the fitness width has a positive or a negative effect on the evolutionary load. The mean and the variation in the fitness width, as well as the genetic variance, define which of the positive or the negative effect on the variance load of the variation in the fitness width dominates the others. The effects of the variation in the fitness width on the fitness loads, and consequently on the long-run growth rate are therefore non-trivial and deserve to be included in future studies to improve the quality of predictions.

#### 4.4.3 Interaction between the mean and the variation in the fitness width

The mean width of the selection function modulates the effect of the variation in the fitness width on the geometric mean fitness. Studies on the thermal tolerance of species show similar interaction between the mean and variance of optimal temperature in responses to climate change (Kingsolver et al., 2013; Vasseur et al., 2014). Increased mean temperature could decrease the fitness of tropical species already experiencing temperature close to their optimum (Kingsolver et al., 2013). In mid-latitudes, increased mean temperature could have positive effect on fitness (Kingsolver et al., 2013; Vasseur et al., 2014). The relationship between temperature and performance, steeply falling once the optimal temperature is surpassed, induces that extreme events have strong negative effects on the performance of species and can have greater effects



than does the mean temperature (Vasseur et al., 2014). We found that for most interactions between the mean and the variation in the fitness width, the average fitness width decreases the effect of the variation in the fitness width. Climate change would therefore amplify the effects of the variation in the fitness width when it decreases the growing season.

#### 4.4.4 Measuring the position of the population in the fitness landscape

Fitness loads depend on the genetic variance and their interaction with the mean and the variation in the fitness width. Measuring the genetic variance is thus essential to predict the long-run growth rate of natural populations. The evolutionary load also depends on the linear trend of the optimum and its fluctuations. Predicting the fate of populations requires therefore to have a deep knowledge of the fitness landscape (optimum and width), its temporal changes (linear trend of the optimum and its fluctuations and the variation in the fitness width), and the position of the population in this fitness landscape.

However, previous studies have highlighted the difficulties in measuring selection and its fluctuations (Chevin and Haller, 2014; Haller and Hendry, 2014; Kingsolver and Pfennig, 2007). In most cases, sampling error does not allow precisely measuring the fluctuations of the selection (de Villemereuil et al., 2020; Morrissey and Hadfield, 2012). Those difficulties in measuring fluctuating selection can explain the current gap between empirical and theoretical results. For example, Chevin and Haller (2014) shows that measuring the directional selection gradient is not enough to estimate the fluctuations of the optimum.

Even if currently all empirical measures are not available to predict the long-run growth rate of populations, relationships that are theoretically identified could be used to explore a range of values for the unknown variables. In some cases, favourable conditions for the long-run growth rate could be identified. Laboratory experiments also constitute a promising possibility to connect theoretical and empirical results, because they allow replication in controlled conditions and a large number of generations when using short lived organisms (Rescan et al., 2020).

#### 4.4.5 Phenotypic plasticity and bet-hedging as strategies to cope with fluctuations of the selection strength?

The mean and the variance of the adaptive lag are the two ways by which the variation in the fitness width can decrease the evolutionary load and increase the long-run growth rate. Fluctuating selection can favour the evolution of phenotypic plasticity and/or bet-hedging (Bull, 1987; Gavrilets and Scheiner, 1993; Gillespie, 1974; Tufto, 2015). Phenotypic plasticity is expected to evolve when the environment is partially predictable (*i.e.* auto-correlated fluctuations of the optimum; Chevin, 2013; Tufto, 2015). Adaptive phenotypic plasticity can facilitate the track of the optimum and thus decrease the mean and the variance of the adaptive lag compared to genetic responses alone. Phenotypic plasticity could thus increase negative effects of the variation in the fitness width on the geometric mean fitness. However, the variation in the fitness width could also decrease the genetic variance for phenotypic plasticity. How phenotypic

plasticity could affect the long-run growth rate in an environment with variance in the width of the selection function is thus an open question.

Bet-hedging allows decreasing the environmental variance of the fitness. Two main forms of bet-hedging are traditionally explored even if intermediate forms probably exist. Diversifying bet-hedging, also called "don't put all your eggs in one basket", is a strategy by which a genotype produces offspring with a variety of specialised phenotypes, for example to good or bad years. In this way, even if the fluctuations of the environment are unpredictable, a part of offspring will perform well the next year. Diversifying bet-hedging is expected to evolve when the auto-correlation of the environment is low and that the variance of the adaptive lag is larger than that of the optimum (Tufto, 2015). We found that strong fluctuations of the optimum can lead to a variance of the adaptive lag larger than that of the optimum, in which case diversifying bet-hedging could eventually emerge in an environment with variance in the width of the selection function. An alternative strategy is conservative bet-hedging, also called "always play it safe". This is a strategy by which a genotype produces a mostly constant phenotype instead of producing very good phenotypes in good years and very bad phenotypes in bad years. This strategy would decrease or increase the mean and the variance of the adaptive lag, and thus have positive or negative effects on the long-run growth rate.

#### 4.4.6 Usefulness of an individual-based model

We here ignored demographic stochasticity and random genetic drift by using an analytical model with an infinite population size, while an individual-based model with a finite population size would allow to take into account of both. Demographic stochasticity could enhance the probability of extinction by increasing the effect of the environmental stochasticity during extreme selection events (Lande, 1993; Melbourne and Hastings, 2008).

The infinitesimal model assumes that the genic variance, a measure of the genetic polymorphism, is constant over time. Our numerical investigation, based on the infinitesimal model, showed that the evolution of genetic associations among allelic effects does not affect the patterns predicted by the analytical predictions. The robustness of the results should be tested by letting evolve the genic variance and genetic associations among allelic effects in an individual-based model.

Matthieu Fontaine during his internship on this project, used an individual-based model based on the same assumptions as those in this study, except for the sampling of temporal series of fitness width. Despite this difference, the average strength of selection increases with the variation in the fitness width. He showed that the adaptive lag decreases with the average strength of selection (fig S7). He also showed that the relationship between the arithmetic mean fitness in a changing environment and the average strength of selection is non-linear (fig S8). Those results are consistent with our predictions, and suggest that our predictions are robust to random genetic drift, evolution of the genic variance and the distribution in which temporal series of fitness width are sampled.

We found here that the fluctuations of the genetic variance around its equilibrium value are very low and slightly increase with the variation in the fitness width. In a model with a bivariate selection, Revell (2007) showed that the **G** matrix is less stable when the correlation coefficient of the fitness surface takes moderate values. The difference between our results and those of Revell (2007) may come from our assumptions of a constant genic variance and/or an infinite population size. The use of an individual-based model relaxing these hypothesis could reconcile these results.

#### 4.4.7 Possible extensions of the model

We here have used a simple approach to explore the effects of the variation in the fitness width on the long-run growth rate. This step was necessary to better understand the complex effects of the variation in the fitness width on the long-run growth rate. One can increase the realism of the model by exploring the effects of a correlation between the variation of the optimum and the width of the selection function. For example, warm years could lead to earlier optimal flowering date because of earlier snow melt (Anderson et al., 2012). It could also advance the beginning of the season and delay the end of the season at high elevation, or advance the end of the season because of increased drought at low elevation (Anderson and Wadgyamar, 2020).

We here studied the effects of a single episode of selection in a given generation. However, Cotto and Chevin (2020) showed that several episodes of fluctuating selection within a generation also induce an additional load. This load could add to or interact with the loads identified in our study.

#### 4.4.8 Conclusion

In the context of the current climate change that increases the magnitude and the frequency of extreme events, studying the effects of the variation in the fitness width on the long-run growth rate is essential. We showed that the variation in the fitness width has non-trivial effects on the long-run growth rate. The variance and the mean of the width of the selection function both interact to define the long-run growth rate. Including the variation in the fitness width could therefore improve future predictions on the fate of populations. Connecting those results with empirical data is not straightforward and probably require time to acquire new data.

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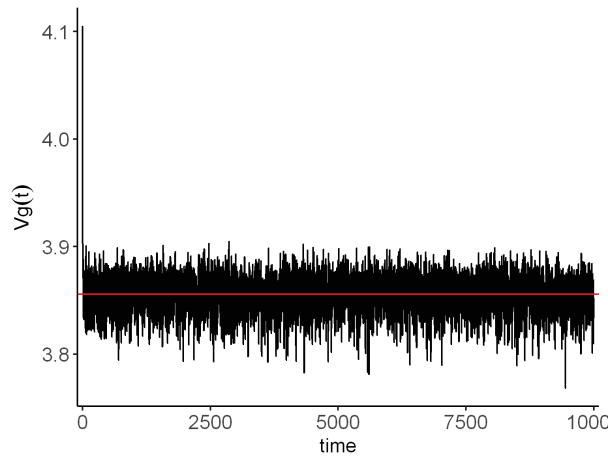
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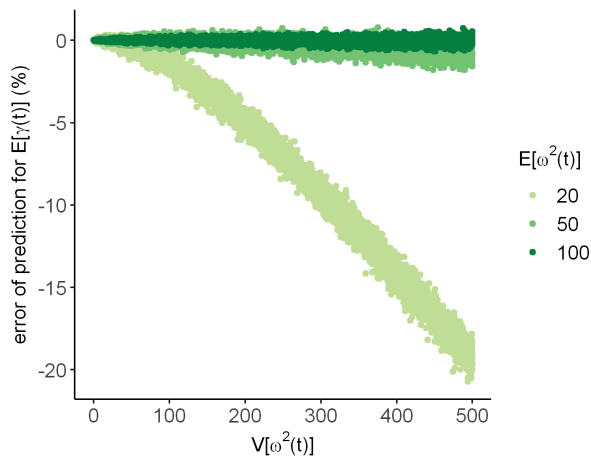
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## 4.1 Appendix 3

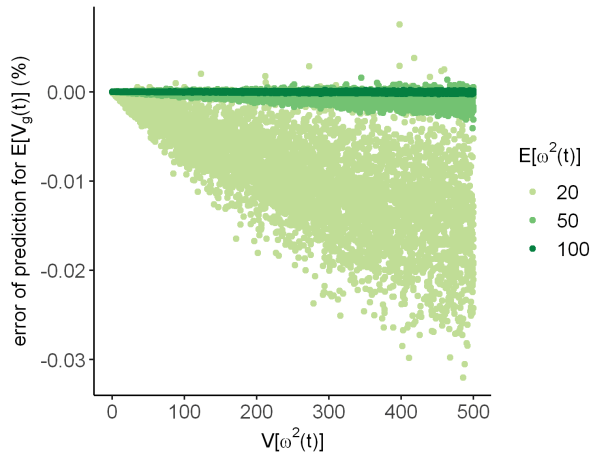
### 4.1.1 Figures



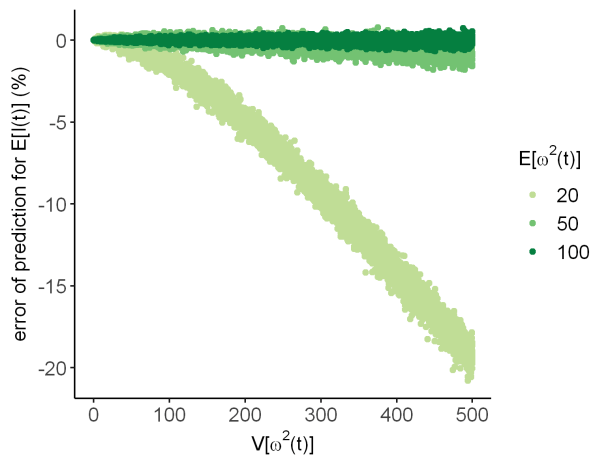
**Figure S1:** Temporal dynamic of the genetic variance for  $V[\omega^2(t)] = 500$ ,  $E[\omega^2(t)] = 100$ ,  $V[\theta(t)] = 10$  and  $k = 0.2$ . The red line indicates the equilibrium value for the genetic variance. The fluctuations of the genetic variance around its equilibrium value are on mean lower than 0.72%.



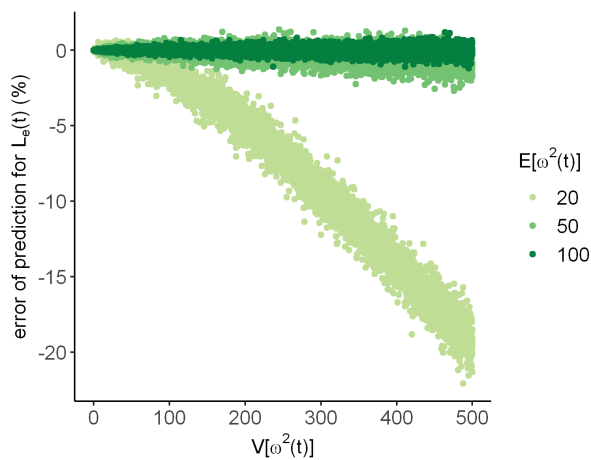
**Figure S2:** Error of the predicted strength of selection, measured in percentage, as a function of the variation in the fitness width. The colour of the symbols indicates the average fitness width.



**Figure S3:** Error of the predicted genetic variance, measured in percentage, as a function of the variation in the fitness width. The colour of the symbols indicates the average fitness width.

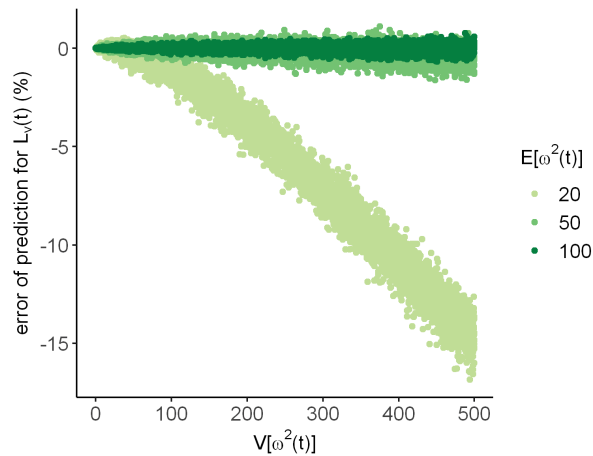


**Figure S4:** Error of the predicted mean adaptive lag, measured in percentage, as a function of the variation in the fitness width. The colour of the symbols indicates the average fitness width.



**Figure S5:** Error of the predicted mean evolutionary load, measured in percentage, as a function of the variation in the fitness width. The colour of the symbols indicates the average fitness width.



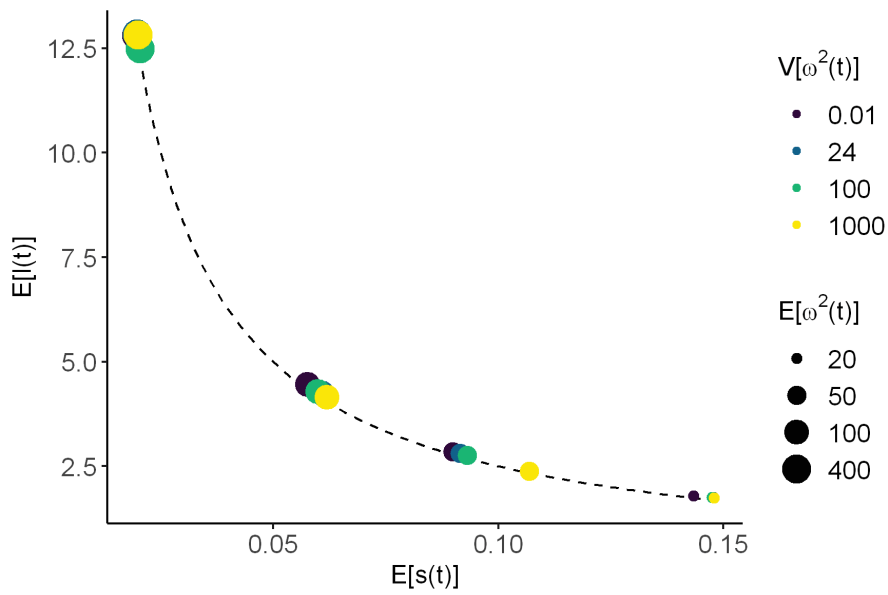


**Figure S6:** Error of the predicted mean variance load in percentage as a function of the variation in the fitness width. The colour of the symbols indicates the average fitness width.

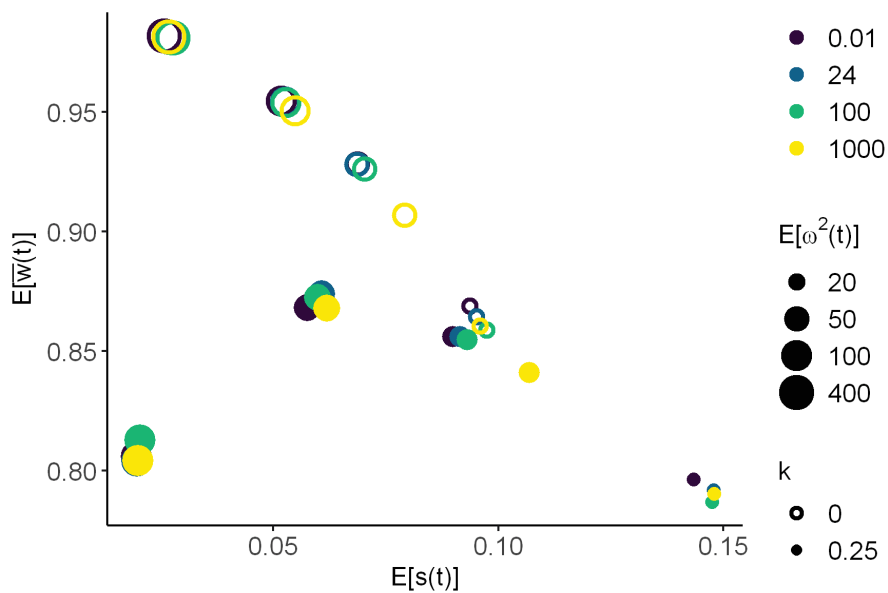
#### 4.1.2 Preliminary results from Matthieu Fontaine

Matthieu Fontaine did a first year master internship on this project. He used an individual-based model developed on the same assumptions as those in this study, except for the definition of the strength of selection and the sampling of temporal series of the fitness width. The strength of selection was defined as  $s(t) = V_g(t)\gamma(t)$ . The temporal series of the fitness width were sampled in a Normal distribution, assuming a minimal limit to avoid null or negative fitness width. Matthieu measured in simulations, among other metrics, the adaptive lag and the arithmetic population mean fitness.

He found a decreasing relation between the adaptive lag and the strength of selection (fig S7). He also found a non-linear relation between the arithmetic mean fitness of the population and the strength of selection, measured as  $s(t)$  (fig S8).



**Figure S7:** Mean adaptive lag as a function of the strength of selection. The colour of symbols indicates the variation of the fitness width. The size of the symbols indicates the mean fitness width. The dashed line corresponds to the prediction  $k/E_t[s(t)]$ . Figure adapted from the master thesis of Matthieu Fontaine.



**Figure S8:** Arithmetic mean fitness of the population as a function of the strength of selection. The colour of the symbols indicates the variation of the fitness width. The size of the symbols indicates the mean fitness width. The shape of the symbols indicates the speed of the environmental change. Figure adapted from the master thesis of Matthieu Fontaine.

### 4.1.3 Analytical approximations

#### 4.1.3.A Approximation for the strength of selection

The strength of selection around the optimum is defined as:

$$\gamma(t) = \frac{1}{V_g(t) + V_e + \omega^2(t)} \quad (\text{S1})$$

where  $V_g(t)$  is the genetic variance at time  $t$ , and  $\omega^2(t)$  is the width of the fitness function at time  $t$ .

The genetic variance is assumed to be at equilibrium such as  $V_g(t) = V_g = E_t[V_g(t)]$ . To simplify the expressions below,  $E_t[V_g(t)]$  is denoted as  $V_g$ .

With a constant fitness width, the fitness width is assumed to be constant such that  $\omega^2(t) = E_t[\omega^2(t)]$ . The strength of selection is then:

$$\gamma_c = \frac{1}{V_g + V_e + E_t[\omega^2(t)]} \quad (\text{S2})$$

Now we assume that the fitness width varies, and that the deviation of the width of the selection function at generation  $t$  to its expectation, denoted  $\delta(t)$ , is small such that  $\delta(t) = \omega^2(t) - E_t[\omega^2(t)] \approx 0$ . We use a Taylor series around  $\delta(t) = 0$  to the order 2 to approximate the strength of selection as:

$$\gamma_{\delta(t)}(t) \approx \frac{1}{E_t[\omega^2(t)] + V_g + V_e} - \frac{\delta(t)}{(E_t[\omega^2(t)] + V_g + V_e)^2} + \frac{\delta(t)^2}{(E_t[\omega^2(t)] + V_g + V_e)^3} + o(\delta(t))^3 \quad (\text{S3})$$

The temporal expectation for the strength of selection is:

$$E_t[\gamma_{\delta(t)}(t)] \approx \frac{1}{E_t[\omega^2(t)] + V_g + V_e} + \frac{E_t[\delta(t)]}{(E_t[\omega^2(t)] + V_g + V_e)^2} + \frac{E_t[\delta(t)^2]}{(E_t[\omega^2(t)] + V_g + V_e)^3} \quad (\text{S4})$$

By replacing  $E_t[\delta(t)^2] = V_t[\omega^2(t)] + E_t[\delta(t)]^2$  and recalling that  $E_t[\delta(t)] = 0$ , one can also write:

$$E_t[\gamma_{\delta(t)}(t)] \approx \gamma_c(1 + V[\omega^2(t)]\gamma_c^2) \quad (\text{S5})$$

#### 4.1.3.B Approximation for the adaptive lag

With a constant fitness width, the mean phenotype in the offspring generation is:

$$\begin{aligned} \bar{z}(t+1) &= \bar{z}(t) + V_g(t)\gamma_c(\theta(t) - \bar{z}(t)) \\ &= \bar{z}(t) + V_g(t)\gamma_c I_c(t) \end{aligned} \quad (\text{S6})$$

$\bar{z}(t)$  reaches an asymptotic regime for which the mean phenotype shifts at the same speed as the optimum, and which is  $E_t[\bar{z}(t+1) - \bar{z}(t)] = k$ . The genetic variance is assumed to be at equilibrium such that  $V_g(t) = V_g = E_t[V_g(t)]$ .

The temporal expectation of the adaptive lag is then:

$$E_t[l_c(t)] \approx \frac{k}{V_g \gamma_c} \quad (\text{S7})$$

Now assuming variation in the fitness width, the mean phenotype in the offspring generation becomes:

$$\begin{aligned} \bar{z}(t+1) &= \bar{z}(t) + V_g(t)\gamma(t)(\theta(t) - \bar{z}(t)) \\ &= \bar{z}(t) + V_g(t)\gamma(t)l(t) \end{aligned} \quad (\text{S8})$$

For a constant genetic variance and at the asymptotic regime, the temporal expectation of the adaptive lag is:

$$E_t[l(t)] = \frac{k - \text{cov}(\gamma(t), l(t))}{V_g E_t[\gamma(t)]} \quad (\text{S9})$$

We assume that the covariance between the strength of selection and the adaptive lag  $\text{cov}(\gamma(t), l(t))$  is null. The temporal expectation for the adaptive lag, for a constant genetic variance, is:

$$E_t[l(t)] \approx \frac{k}{V_g E_t[\gamma(t)]} \quad (\text{S10})$$

#### 4.1.3.C Approximation for the mean fitness

The population mean fitness is:

$$\bar{w}(t) = \int w_z(t)p_z(t)dz = \sqrt{\frac{\omega^2(t)}{\omega^2(t) + V_z(t)}} \exp\left(-\frac{(\bar{z}(t) - \theta(t))^2}{2\omega^2(t)}\right) \quad (\text{S11})$$

The fitness load is the expected loss in the mean Malthusian fitness due to genetic and evolutionary factors (Lande and Shannon 1996) such that:

$$L(t) = \log(w_{max}) - \log(\bar{w}(t)) \quad (\text{S12})$$

with  $w_{max}$  the maximal fitness, which equals one.

The previous equation can also be written as:

$$L(t) = -\frac{1}{2} \log(\omega^2(t)\gamma(t)) + \frac{1}{2} l(t)^2 \gamma(t) \quad (\text{S13})$$

The first term on the right hand side is the loss in fitness caused by genetic variance, called the variance load and denoted  $L_v(t) = -\frac{1}{2} \log(\omega^2(t)\gamma(t))$ , while the second term is the loss in fitness caused by the evolutionary lag, called the evolutionary load and denoted  $L_e(t) = \frac{1}{2}l(t)^2\gamma(t)$ . In the following we present an approximation for these two fitness loads.

#### 4.1.3.3.a Approximation for the variance load

With a constant fitness width and a constant genetic variance, the temporal expectation of the variance load is:

$$E_t[L_{vc}(t)] = -\frac{1}{2} \log(E_t[\omega^2(t)]\gamma_c) \quad (\text{S14})$$

Now assuming variation in the fitness width and that the deviation of the fitness width at generation  $t$  to its expectation, denoted  $\delta(t)$ , is small such that  $\delta(t) = \omega^2(t) - E[\omega^2(t)] \approx 0$ . We use a Taylor series around  $\delta(t) = 0$  to the order 2 to approximate the variance load:

$$L_v(t) \approx \frac{1}{2} \log\left(\frac{E_t[\omega^2(t)] + V_z}{E_t[\omega^2(t)]}\right) - \frac{\delta(t)V_z}{2(E_t[\omega^2(t)](E_t[\omega^2(t)] + V_z))} + \frac{\delta^2(t)V_z(2E_t[\omega^2(t)] + V_z)}{4E_t[\omega^2(t)]^2(E_t[\omega^2(t)] + V_z)^2} + o(\delta(t))^3 \quad (\text{S15})$$

The temporal expectation for the variance load is:

$$E_t[L_v(t)] \approx \frac{1}{2} \log\left(\frac{E_t[\omega^2(t)] + V_z}{E_t[\omega^2(t)]}\right) - \frac{E_t[\delta(t)]V_z}{2(E_t[\omega^2(t)](E_t[\omega^2(t)] + V_z))} + \frac{E_t[\delta^2(t)]V_z(2E_t[\omega^2(t)] + V_z)}{4E_t[\omega^2(t)]^2(E_t[\omega^2(t)] + V_z)^2} \quad (\text{S16})$$

By replacing  $E_t[\delta(t)^2] = V_t[\omega^2(t)] + E_t[\delta(t)]^2$  and recalling that  $E_t[\delta(t)] = 0$ , one can also write:

$$E_t[L_v(t)] \approx \frac{1}{2} \log\left(\frac{E_t[\omega^2(t)] + V_z}{E_t[\omega^2(t)]}\right) + \frac{V_t[\omega^2(t)]V_z(2E_t[\omega^2(t)] + V_z)}{4E_t[\omega^2(t)]^2(E_t[\omega^2(t)] + V_z)^2} \quad (\text{S17})$$

and finally the variance load is:

$$E_t[L_v(t)] \approx L_{vc} + V_t[\omega^2(t)]\gamma_c^2 \frac{V_z(2E_t[\omega^2(t)] + V_z)}{4E_t[\omega^2(t)]^2} \quad (\text{S18})$$

#### 4.1.3.3.b Approximation for the evolutionary load

With a constant fitness width and a constant genetic variance, the temporal expectation of the evolutionary load is:

$$E_t[L_{ec}(t)] = \frac{1}{2}E_t[l_c^2(t)]\gamma_c = \frac{1}{2}(V_t[l_c(t)] + E_t[l_c(t)]^2)\gamma_c \quad (\text{S19})$$

Now assuming variation in the fitness width, and that the deviation of the fitness width at generation  $t$  to its expectation is small, and using a Taylor series around  $\delta(t) = 0$  to the order 2, one can approximate the evolutionary load as:

$$L_e(t) \approx \frac{l^2(t)}{2(E_t[\omega^2(t)] + V_z)} - \frac{l^2(t)\delta(t)}{2(E_t[\omega^2(t)] + V_z)^2} + \frac{l^2(t)\delta(t)^2}{2(E_t[\omega^2(t)] + V_z)^3} + o(\delta(t))^3 \quad (\text{S20})$$

The temporal expectation for the evolutionary load is:

$$E_t[L_e(t)] \approx \frac{E_t[l^2(t)]}{2(E_t[\omega^2(t)] + V_z)} - \frac{E_t[l^2(t)\delta(t)]}{2(E_t[\omega^2(t)] + V_z)^2} + \frac{E_t[l^2(t)\delta(t)^2]}{2(E_t[\omega^2(t)] + V_z)^3} \quad (\text{S21})$$

$$E_t[L_e(t)] \approx \frac{E_t[l^2(t)]}{2(E_t[\omega^2(t)] + V_z)} - \frac{E_t[l^2(t)]E_t[\delta(t)]}{2(E_t[\omega^2(t)] + V_z)^2} + \frac{E_t[l^2(t)]E_t[\delta(t)^2]}{2(E_t[\omega^2(t)] + V_z)^3} \quad (\text{S22})$$

By replacing  $E_t[\delta(t)^2] = V_t[\omega^2(t)] + E_t[\delta(t)]^2$  and recalling that  $E_t[\delta(t)] = 0$ , one can also write:

$$E_t[L_e(t)] \approx \frac{E_t[l^2(t)]}{2(E_t[\omega^2(t)] + V_z)} \left(1 + \frac{V_t[\omega^2(t)]}{(E_t[\omega^2(t)] + V_z)^2}\right) \quad (\text{S23})$$

and thus find the evolutionary load:

$$E_t[L_e(t)] \approx L_{ec}(1 + V_t[\omega^2(t)]\gamma_c^2) \quad (\text{S24})$$

#### 4.1.3.D Approximation for the genetic variance

We first assume a constant fitness width. From the infinitesimal model of trait inheritance, the genetic variance after natural selection and random mating and with a constant fitness width is:

$$V_g(t+1) = \frac{V_{LE}}{2} + \frac{V_g(t)}{2}(1 - V_g(t)\gamma_c(t)) \quad (\text{S25})$$

with  $\gamma_c(t) = \frac{1}{V_g(t) + V_e + E_t[\omega^2(t)]}$ .

The temporal expectation of the genetic variance is:

$$\begin{aligned} E_t[V_g(t+1)] &= \frac{V_{LE}}{2} + \frac{E_t[V_g(t)]}{2} - \frac{E_t[V_g^2(t)\gamma_c(t)]}{2} \\ &= \frac{V_{LE}}{2} + \frac{E_t[V_g(t)]}{2} - \frac{E_t[V_g^2(t)]E_t[\gamma_c(t)]}{2} - \frac{cov(V_g^2(t), \gamma_c(t))}{2} \end{aligned} \quad (S26)$$

We assume that the fluctuations of the genetic variance are small such that  $E[V_g(t+1) - V_g(t)] \approx 0$  and thus  $cov(V_g^2(t), \gamma_c(t)) \approx 0$ . The temporal expectation of the genetic variance simplifies to:

$$E_t[V_g(t)] \approx V_{LE} + E_t[V_g^2(t)]E_t[\gamma_c(t)] \quad (S27)$$

One can derive the roots of the previous equation and find:

$$E_t[V_g(t)] \approx \frac{-1 + \sqrt{1 + 4V_{LE}E[\gamma_c(t)]}}{2E[\gamma_c(t)]} \quad (S28)$$

Now we assume variation in the fitness width. Again from the infinitesimal model of trait inheritance, the genetic variance after natural selection and random mating, assuming variation in the fitness width, is:

$$V_g(t+1) = \frac{V_{LE}}{2} + \frac{V_g(t)}{2}(1 - V_g(t)\gamma(t)) \quad (S29)$$

With the same reasoning than in the previous section, one can find the temporal expectation for the genetic variance:

$$E_t[V_g(t)] \approx \frac{-1 + \sqrt{1 + 4V_{LE}E[\gamma(t)]}}{2E[\gamma(t)]} \quad (S30)$$

## Discussion

La phénologie de floraison est un trait pour lequel des réponses évolutives rapides au changement climatique sont observées empiriquement (Parmesan, 2006 ; Parmesan et Yohe, 2003). Au cours de cette thèse nous avons cherché à identifier les facteurs favorisant ou limitant les réponses évolutives de la phénologie de floraison au changement climatique. Nous avons dans un premier temps cherché à comprendre si l'homogamie pour la date de floraison pouvait expliquer les réponses évolutives rapides observées dans les populations naturelles. Dans un second temps, nous avons pris en compte l'effet de la sélection sexe-spécifique sur les réponses évolutives. La sélection sexe-spécifique est fréquente dans les populations naturelles et pourrait expliquer le dimorphisme sexuel observé pour la date de floraison, mais aussi contraindre les réponses évolutives de la date de floraison. Enfin nous avons étudié les effets des fluctuations de la durée de la saison de floraison, accentuées par le changement climatique, sur les réponses évolutives. Ci-dessous je résume les résultats principaux des différents axes. Je propose ensuite quelques connections possibles avec les données empiriques et quelques perspectives.

### 5.1 Résultats principaux

#### 5.1.1 Effets de l'homogamie sur les réponses évolutives à la sélection

Je rappelle ici les différents patrons d'appariement que nous avons étudiés et les hypothèses sous-jacentes avant de discuter de l'effet de ces patrons d'appariement sur les réponses évolutives (*i.e.* les changements de génotype moyen entre générations). Nous avons comparé les réponses évolutives à la sélection pour des populations homogames ou panmictiques. Nous avons également comparé deux mécanismes d'homogamie : l'homogamie par préférence femelle et l'homogamie temporelle. L'homogamie par préférence femelle suppose que les femelles sélectionnées s'apparient avec des mâles ayant un phénotype qui leur ressemble. Elle est modélisée par une fonction de préférence de forme Gaussienne dont la largeur affecte l'intensité de la corrélation entre les partenaires. Sous homogamie temporelle, la production de gamètes est distribuée au cours de la saison. Par exemple chez les plantes, la distribution des gamètes mâles et femelles est définie par la distribution des fleurs ouvertes au cours de la saison. Nous avons étudié deux scénarios extrêmes d'homogamie temporelle. Dans le premier chapitre, nous avons supposé que la qualité des gamètes d'un individu varie au cours du temps et ce indépendamment du phénotype de l'individu. La fertilité d'un individu varie donc au cours de la saison à la fois



parce que le nombre de gamètes produits change avec le nombre de fleurs ouvertes et parce que la qualité de ces gamètes change. A un instant donné, tous les gamètes de la population ont cependant la même qualité. Dans la suite de la discussion, ce scénario d'homogamie est appelé "homogamie temporelle à qualité variable". Dans le deuxième chapitre, nous avons supposé que la qualité des gamètes d'un individu dépend uniquement de son phénotype et qu'elle était constante au cours de la saison. La fertilité d'un individu varie alors au cours de la saison uniquement parce que le nombre de gamètes produits varie. A un instant donné, les gamètes de la population ont des qualités différentes dépendant du phénotype de l'individu qui les a produits. Dans la suite de la discussion, ce scénario d'homogamie est appelé "homogamie temporelle à qualité constante". Un élément clé pour comprendre les points communs et les différences de réponses évolutives entre patrons d'appariement est la compétition entre les mâles.

Dans le premier chapitre, nous avons montré que la variation du nombre et de la qualité des gamètes au cours de la saison modifie la fertilité efficace des individus. La fertilité efficace maximale d'un individu est déplacée vers la date de floraison maximisant la qualité des gamètes et est donc différente de son pic de production de gamètes. La période de floraison efficace pour un individu est plus petite que la période de floraison. Le succès reproducteur relatif d'un mâle est indépendant de la qualité de ses gamètes car, à un instant donné, tous les mâles en compétition ont la même qualité de gamètes. Le succès reproducteur relatif d'un mâle dépend alors de la proportion de gamètes produits à cet instant par rapport aux autres mâles de la population et de la fertilité efficace de la femelle à cet instant. La réponse des mâles à la sélection sexuelle induite par l'homogamie est identique à la réponse des femelles à la sélection naturelle, suggérant que la sélection naturelle sur les mâles est totalement substituée par la sélection naturelle sur les femelles. Pour une variance génétique donnée, la réponse à la sélection sous homogamie temporelle à qualité variable est donc identique à celle sous panmixie. Rappelons que dans ce modèle l'intensité de la sélection sur les sexes est identique et que le dimorphisme sexuel est nul.

Dans le deuxième chapitre, nous avons comparé la réponse à la sélection sous préférence femelle avec celle sous homogamie temporelle à qualité constante. Bien que la compétition entre mâles se produise à différentes échelles, *i.e.* une femelle pour la préférence, ou un instant donné pour l'homogamie temporelle, le succès reproducteur d'un mâle dépend de celui des autres mâles de la population. Ce point commun pourrait expliquer pourquoi, pour une variance génétique donnée, la réponse à la sélection sous homogamie temporelle à fertilité constante est identique à celle sous préférence femelle. Nous avons également montré que la réponse à la sélection sous préférence femelle (ou homogamie temporelle à qualité constante) peut être plus petite ou plus grande que celle sous panmixie selon l'intensité de la sélection sur les sexes, la vitesse du changement environnemental et le degré de dimorphisme sexuel.

### 5.1.2 Effets de l'homogamie sur la structure de la variance génétique

En combinant l'utilisation de modèles théoriques aux hypothèses complémentaires, nous avons montré les effets antagonistes de l'homogamie sur les composantes de la variance

génétique. Le modèle analytique est basé sur le modèle infinitésimal d'héritabilité du trait qui suppose une variance génique constante, une taille de population infinie et un grand nombre de locus déterminant le trait. Les prédictions de ce modèle ont permis de contraster les effets des patrons d'appariement sur les associations entre effets alléliques. Sous panmixie, l'intensité de la sélection stabilisante génère des associations négatives entre effets alléliques (Bulmer effect; Bulmer, 1971), diminuant ainsi la variance génétique. Sous homogamie, la corrélation entre les phénotypes des partenaires associe des allèles à effets similaires sur le phénotype, et augmente la variance génétique. L'effet positif de l'homogamie sur les associations alléliques est plus fort quand la sélection naturelle stabilisante sur les femelles est moins forte que celle sur les mâles. L'homogamie induit aussi une différence de succès reproducteur entre mâles, produisant de la sélection sexuelle. Cette sélection sexuelle s'ajoute à la sélection naturelle stabilisante et augmente l'intensité de la sélection stabilisante sur le trait. Nous avons utilisé un modèle individu-centré qui laisse évoluer le polymorphisme génétique afin de comprendre les effets de la sélection sexuelle sur celui-ci. Ce modèle suppose une taille finie de population et un nombre limité de locus déterminant le trait. Les simulations montrent que la sélection sexuelle produite par l'homogamie diminue le polymorphisme génétique. En lien avec les travaux de Kirkpatrick et Nuismer (2004), nous avons montré que les effets positifs de l'homogamie sur les associations entre allèles peuvent compenser ou non ses effets négatifs sur le polymorphisme génétique. Dans le premier chapitre, nous avons mis en évidence que l'effet négatif de l'homogamie sur le polymorphisme génétique maintient moins de variance génétique que sous panmixie quand la sélection stabilisante est forte. A l'inverse, quand la sélection stabilisante est faible, l'effet positif de l'homogamie sur les associations positives entre effets alléliques augmente la variance génétique par rapport à celle maintenue sous panmixie. Nos résultats permettent de connecter les attendus théoriques précédemment obtenus en absence de sélection et sous sélection stabilisante. La diminution des effets positifs de l'homogamie sur la variance génétique avec l'intensité de la sélection stabilisante explique pourquoi les études théoriques en absence de sélection prédisent un fort effet de l'homogamie sur la variance génétique (Crow et Felsenstein, 1968; Crow et Kimura, 1970; Devaux et Lande, 2008; S. Wright, 1921) tandis qu'une étude sous sélection stabilisante prédit une absence d'effet de l'homogamie (Lande, 1977). Nos résultats permettent également d'expliquer pourquoi les études empiriques sous sélection directionnelle artificielle trouvent un effet faible, voire nul, de l'homogamie sur les réponses génétiques (Breese, 1956; Campo et Gil, 1994; Garcia et Sanchez, 1992; McBride et Robertson, 1963; Sutherland et al., 1968; Wilson et al., 1965). Les simulations montrent aussi que la vitesse de changement environnemental modifie la variance génétique et le polymorphisme génétique. Cependant, la variance génétique dans les populations homogames est généralement supérieure à celle des populations panmictiques dans notre modèle quand l'environnement change.

Dans le chapitre 2, nous avons montré qu'une plus forte sélection naturelle sur les femelles que sur les mâles diminue l'effet de l'homogamie sur les associations entre effets alléliques. La variance génétique dépend aussi du type d'appariement. La variance génétique sous homogamie temporelle est supérieure à celle sous panmixie, du moins dans la gamme de paramètres simulée. En revanche, la variance génétique sous préférence femelle peut être inférieure à celle sous panmixie suggérant que l'effet négatif de la préférence femelle sur le polymorphisme génétique

peut dépasser son effet positif sur les associations entre effets alléliques. Pour une valeur de polymorphisme génétique donnée, l'homogamie temporelle produit plus d'associations positives que la préférence femelle. L'homogamie temporelle maintient aussi plus de polymorphisme génétique que la préférence femelle. La variance génétique sous homogamie temporelle est donc supérieure à celle sous préférence femelle. La distribution individuelle des gamètes en homogamie temporelle pourrait expliquer cette différence de variance génétique. L'annexe du chapitre 2 définit la variance génétique des mâles accédant à une femelle donnée. Cette variance est plus petite que celle des mâles accédant à la reproduction à l'échelle de la population. En effet la distribution des phénotypes des femelles implique que la variance des mâles qui se reproduisent est supérieure à celle d'une population dans laquelle il y aurait un unique phénotype femelle. De manière similaire, on peut supposer que la distribution individuelle des gamètes au cours de la saison augmente la variance phénotypique des mâles accédant à une femelle donnée. A l'échelle de la population, il est probable que la distribution individuelle des gamètes augmente la variance génétique des pères et donc augmente la variance génétique à la génération suivante. Les données empiriques montrent que les phénologies de floraison répondent au changement climatique par des réponses à la fois évolutives et plastiques (Anderson et al., 2012 ; Franks et al., 2014 ; Hamann et al., 2018) tandis que chez les animaux les réponses des phénologies de reproduction semblent majoritairement plastiques (Charmantier et Gienapp, 2014 ; Gienapp et al., 2008 mais voir aussi Réale et al., 2003). Il pourrait être intéressant d'explorer si la plus grande variance génétique maintenue sous homogamie temporelle pour la date de floraison que sous préférence femelle, commune chez les animaux, peut expliquer les différences de rôles entre réponses plastiques et évolutives chez les plantes et les animaux.

### 5.1.3 Effets de l'homogamie sur l'adaptation

L'homogamie temporelle réduit le retard des populations à l'optimum par rapport à la panmixie. En effet, bien que les réponses à la sélection directionnelle sont attendues équivalentes sous homogamie temporelle à qualité variable et panmixie pour une variance génétique donnée, l'augmentation de la variance génétique sous homogamie diminue le retard adaptatif. L'homogamie diminue donc le fardeau évolutif par rapport à la panmixie. L'augmentation de variance génétique sous homogamie augmente en revanche le fardeau de variance. Dans la plupart des scénarios étudiés dans le chapitre 1, la diminution du fardeau de retard dépasse l'augmentation du fardeau variance, augmentant ainsi la valeur sélective moyenne des populations homogames. L'homogamie peut donc expliquer, au moins en partie, les réponses évolutives rapides des phénologies de floraison au changement climatique.

Dans le deuxième chapitre, la sélection sexe-spécifique empêche les mâles et les femelles d'être simultanément à leur optimum et fait émerger un optimum évolutif maximisant la valeur sélective de la population. Cet optimum évolutif dépend de la position relative des optimums mâles et femelles, de l'intensité de la sélection sur chaque sexe, du dimorphisme sexuel et de la sélection sexuelle. Nous avons choisi de mesurer l'adaptation par rapport aux femelles qui sont souvent plus limitantes pour la reproduction que ne le sont les mâles (Harts et al., 2014). Nous avons décomposé la déviation de la valeur génétique moyenne de la population à l'optimum

femelle en deux parties. La première partie est causée par la déviation de la valeur génétique moyenne de la population à l'optimum femelle en environnement constant. La sélection sexuelle générée par l'homogamie augmente l'effet de la sélection naturelle sur les femelles et diminue l'effet de la sélection naturelle sur les mâles. En absence de dimorphisme sexuel, la sélection sexuelle et une plus forte sélection sur les femelles que sur les mâles diminue la déviation de la valeur génétique moyenne de la population par rapport à l'optimum femelle. La deuxième partie de la déviation à l'optimum femelle correspond à la déviation de la valeur génétique moyenne de la population à l'optimum évolutif en environnement changeant. Cette part de la déviation à l'optimum femelle est ainsi comparable au retard évolutif mesuré dans le premier chapitre. Nous avons montré que le retard évolutif est plus petit (en valeur absolue) sous homogamie qu'en panmixie quand la sélection sur les femelles est plus forte que celle sur les mâles. En revanche le retard est plus grand (en valeur absolue) sous homogamie qu'en panmixie quand la sélection sur les femelles est plus faible que celle sur les mâles. En absence de dimorphisme sexuel, la sélection sexe-spécifique et la vitesse du changement environnemental définissent les conditions pour lesquelles la valeur absolue de la déviation de la valeur génétique moyenne de la population à l'optimum femelle est inférieure en homogamie par rapport à celle en panmixie. La sélection sexe-spécifique limite les conditions pour lesquelles l'homogamie aide l'adaptation à un environnement changeant.

Le dimorphisme sexuel est une réponse très fréquente pour résoudre les conflits entre sexes engendrés par la sélection sexuelle et/ou la sélection naturelle sexe-spécifique (Cox et Calsbeek, 2009 ; de Lisle et al., 2018). De plus, pour les phénologies de floraison, le dimorphisme sexuel est aussi sélectionné pour éviter l'autofécondation (Lloyd et Webb, 1986). Sous les hypothèses de notre modèle, le dimorphisme sexuel déplace le phénotype des mâles de leur valeur génétique. Sous homogamie, le dimorphisme sexuel entraîne l'optimum évolutif de la population dans la direction des femelles. Dans une large gamme de paramètres, l'optimum évolutif peut même dépasser l'optimum femelle. La déviation de la valeur génétique moyenne de la population à l'optimum femelle change alors de signe et peut devenir supérieure à celle sous panmixie. Nous avons montré que le dimorphisme sexuel limite l'adaptation des populations sous homogamie par rapport à celles sous panmixie sauf quand l'optimum de floraison mâle est plus tardif que l'optimum femelle et que la sélection sur les mâles est plus forte que celle sur les femelles.

#### 5.1.4 Effets des fluctuations de l'intensité de la sélection

La plupart des modèles de génétique quantitative d'adaptation au changement environnemental supposent une fonction de sélection de forme Gaussienne avec un optimum mobile et une largeur constante (voir la revue de Kopp et Matuszewski, 2014). Dans le troisième chapitre, nous avons montré que les fluctuations de la largeur de la fonction de sélection augmentent l'intensité de la sélection par rapport à une largeur constante de la fonction de sélection. L'augmentation de l'intensité de la sélection diminue la variance génétique et le retard adaptatif.

En lien avec la littérature classique (Lande et Shannon, 1996, voir aussi la revue de Kopp et Matuszewski, 2014), nous avons décomposé la diminution de la valeur sélective par rapport à la valeur sélective maximale en un fardeau de variance et un fardeau évolutif. Nous avons estimé les effets des fluctuations de la largeur de la sélection sur le taux de croissance à long terme des populations en mesurant la moyenne géométrique des valeurs sélectives au cours du temps. Nous montrons que les fluctuations de la largeur de la sélection augmentent directement le fardeau de variance et le fardeau évolutif. Ces fardeaux sont aussi affectés indirectement par les fluctuations de la largeur de la sélection via différentes voies. Les fluctuations de la largeur de la sélection ont un effet indirect négatif sur le fardeau de variance via la variance génétique. Le fardeau évolutif augmente avec la moyenne et la variance du retard adaptatif et diminue avec la variance génétique. Les fluctuations de la largeur de la sélection ont donc un effet indirect sur le fardeau évolutif via la variance génétique et le retard adaptatif. L'intensité de la sélection est un élément clé pour comprendre les effets indirects des fluctuations de la largeur de la sélection sur les fardeaux de variance et évolutif puisqu'elle détermine la variance génétique et le retard adaptatif. Les simulations mettent en évidence un effet des fluctuations de la largeur de la sélection sur la variance du retard adaptatif. L'effet des fluctuations de la largeur de la sélection sur les fardeaux résultent donc d'effets direct et indirect ayant des signes opposés. L'exploration numérique suggère que les fluctuations de l'intensité de la sélection diminuent la moyenne géométrique des valeurs sélectives sauf quand l'environnement change rapidement et que la force moyenne de la sélection est faible. Les fluctuations de l'intensité de la sélection ont donc un coût démographique qui pourrait aggraver le déclin des populations.

## 5.2 Comment connecter ces résultats théoriques à des résultats empiriques ?

L'objectif de ces travaux de thèse était de fournir des prédictions explicatives aux réponses évolutives au changement climatique. En nous basant sur des observations empiriques, nous avons utilisé une approche théorique pour affiner la compréhension des réponses évolutives au changement climatique. Il serait maintenant intéressant de vérifier si les données empiriques appuient ou réfutent nos prédictions.

Nos résultats suggèrent que l'adaptation des populations naturelles peut être prédite si nous avons accès à des estimations des paysages de valeurs sélectives au cours du temps. Cependant les mesures empiriques de la sélection présentent plusieurs limites. Parmi ces limites, l'erreur d'échantillonnage représente une part importante de la variance des estimations et rend difficile l'interprétation des fluctuations temporelles de la sélection (Morrissey et Hadfield, 2012; Siepielski et al., 2009). Les données actuelles ne sont aussi peut-être pas suffisantes pour estimer la sélection avec suffisamment de précision (de Villemereuil et al., 2020; Haller et Hendry, 2014; Kingsolver et Pfennig, 2007; Morrissey et Hadfield, 2012). Enfin, les études considèrent souvent une seule composante de la valeur sélective plutôt que l'ensemble des composantes de valeur sélective (Kingsolver et Pfennig, 2007). De nouvelles méthodes telles que la méthode

*aster* permettent désormais des estimations plus précises des fonctions de sélection (Shaw et Geyer, 2010 ; Shaw et al., 2008). Ces méthodes pourraient ainsi faciliter les connections entre les résultats théoriques et empiriques.

Nous avons ici souhaité fournir des prédictions généralistes des conditions favorisant ou contraignant l'adaptation des populations au changement climatique. Nous pourrions maintenant envisager de paramétrer les modèles avec des estimations spécifiques à une espèce donnée pour prédire ses réponses au changement climatique et guider les stratégies de gestion. Cependant une difficulté est que l'ensemble des informations nécessaires pour paramétrer les modèles sont rarement disponibles. Une possibilité serait néanmoins d'explorer une gamme de valeurs réalistes pour les paramètres auxquels nous n'avons pas accès et de réaliser une analyse de sensibilité des résultats. Les conditions favorisant l'adaptation de cette espèce en particulier pourraient ainsi être identifiées.

## 5.3 Perspectives

### 5.3.1 Effet de l'autofécondation sur les réponses évolutives au changement climatique

Nous avons vu que l'homogamie associe des individus aux phénotypes similaires associant ainsi des allèles à effets similaires sur les phénotypes. Les associations positives entre allèles augmentent la variance génétique favorisant dans certains cas les réponses évolutives au changement climatique. L'autofécondation crée aussi des associations positives fortes entre allèles, mais sur l'ensemble du génome. Il serait donc intéressant de faire un parallèle entre les effets de l'homogamie et de l'autofécondation sur les réponses évolutives au changement climatique.

#### 5.3.1.A Environnement constant

Les effets de l'autofécondation sur la variance génétique ont été étudiés historiquement pour comprendre les transitions entre l'autofécondation, l'allofécondation et les systèmes mixtes (Lande et Schemske, 1985 ; Schemske et Lande, 1985 ; S. I. Wright et al., 2013). L'autofécondation agit sur la variance génétique avec des mécanismes différents de ceux de l'homogamie. D'une part, en absence de sélection, l'autofécondation augmente l'homozygotie en associant des génotypes identiques. Les allèles délétères sont alors exposés à la sélection et l'effet positif de l'augmentation de l'homozygotie est compensé par la purge des allèles délétères (Lande, 1977). La sélection stabilisante favorise les associations entre effets alléliques opposés sur le phénotype (Bulmer, 1971). La variance génétique est ainsi réduite mais le polymorphisme génétique est maintenu. L'autofécondation favorise ces associations négatives entre allèles en réduisant la recombinaison et diminue l'efficacité de la sélection. Dans son modèle d'évolution de la variance génétique d'un trait polygénique soumis à la mutation et à la sélection stabilisante, Lande (1977) montre que les effets positifs et négatifs de l'autofécondation se compensent. La variance génétique sous

autofécondation n'est donc pas différente de celle sous panmixie. Lande et Porcher (2015) ont étendu la gamme de taux d'autofécondation étudiée et ont identifié un taux d'autofécondation seuil au-dessus duquel, la variance génétique des populations autofécondantes est inférieure à celle des populations panmixiques. Les données empiriques confirment que l'autofécondation diminue la variance génétique par rapport à la panmixie (voir Clo et al., 2019 pour une méta-analyse).

### 5.3.1.B Environnement changeant

Les effets de l'autofécondation sur les réponses évolutives au changement environnemental ont été moins étudiés. L'autofécondation peut accélérer la réponse à la sélection par rapport à l'alofécondation, au moins dans les premières générations suivant un changement de l'environnement (Noël et al., 2017). Dans une étude récente, Clo et al. (2020) utilise des simulations individu-centré supposant une fonction de sélection de forme Gaussienne dont l'optimum change ponctuellement. Les auteurs montrent que les associations négatives entre effets alléliques générées par l'autofécondation permettent de stocker le polymorphisme génétique quand le taux de mutation est élevé. Le polymorphisme génétique peut alors être mobilisé suite à un changement ponctuel de l'environnement. Les populations autofécondantes (taux d'autofécondation  $< 1$ ) peuvent ainsi s'adapter aussi bien que les populations alofécondantes. L'autofécondation est donc un régime de reproduction qui présente des propriétés intéressantes dans le cadre du changement climatique. Il serait pertinent de comparer les effets de l'autofécondation et de l'homogamie sur les réponses évolutives à un changement soutenu de l'environnement. Les conditions dans lesquelles l'homogamie favorise plus ou moins les réponses évolutives par rapport à l'autofécondation pourraient ainsi être déterminées.

En particulier, on peut s'attendre à ce que les épisodes de forte sélection annulent les effets positifs de l'homogamie sur la variance génétique. Suite à des épisodes de forte sélection, l'homogamie, par son effet sur les associations positives en effets alléliques, pourrait favoriser le regain rapide de variance génétique si le polymorphisme génétique est suffisant. En revanche, si les épisodes de forte sélection sont trop fréquents, les effets négatifs de l'homogamie sur le polymorphisme pourraient s'ajouter à ceux des fluctuations de l'environnement et compromettre l'adaptation. Dans cette situation, l'autofécondation protégeant le polymorphisme génétique pourrait être plus avantageuse que l'homogamie.

### 5.3.2 Effet des variations de la taille de population sur les réponses évolutives au changement climatique

Au cours de cette thèse, nous nous sommes intéressées aux conditions dans lesquelles la variance génétique peut faciliter les réponses au changement climatique en supposant une taille de population très grande ou constante. Les méta-analyses des données empiriques identifient la taille de population comme étant l'un des principaux facteurs d'extinction (O'Grady et al., 2004). La théorie s'est ainsi attachée à clarifier l'effet de la stochasticité sur la taille de population sur le risque d'extinction (Lande, 1993 ; Lande et al., 2003 ; Melbourne et Hastings, 2008 ; Sæther

et Engen, 2015). La stochasticité de la taille de population peut être d'origine démographique ou environnementale. La stochasticité environnementale est causée par les fluctuations de la probabilité de mortalité et de reproduction de tous les individus de la population (Lande, 1993). La stochasticité environnementale agit donc sur l'ensemble de la population quelle que soit sa taille et représente un risque important d'extinction pour les populations. La stochasticité démographique est causée par la variation de la probabilité de survie et de reproduction entre individus d'un même génotype (Lande, 1993). Lorsque la taille de population initiale est grande, la stochasticité démographique a peu d'effet sur la taille de population à la génération suivante (Lande 1993). Cependant, quand la taille de population initiale est faible à modérée, les effets de la stochasticité démographique sur le risque d'extinction s'ajoutent à ceux de la stochasticité environnementale. En effet, dans les petites populations la diversité génétique est plus faible que dans les grandes populations, limitant ainsi la réponse évolutive au changement environnemental. De plus, la dérive génétique est plus forte dans les petites populations que dans les grandes populations. Des allèles bénéfiques pour les réponses au changement environnemental sont alors plus facilement perdus. Les stochasticités démographique et environnementale ont donc des effets synergiques sur le risque d'extinction et ne pas prendre en compte les effets de la stochasticité démographique peut conduire à sous-estimer le risque d'extinction (Bürger et Lynch, 1995 ; Melbourne et Hastings, 2008). Une extension possible aux travaux de cette thèse serait de laisser la taille de population varier afin de déterminer si nos conclusions sur les effets de l'homogamie et des fluctuations de l'intensité de la sélection sont robustes à la stochasticité démographique.

### 5.3.3 Effet des stades de vie sur les réponses évolutives au changement climatique

Comme la plupart des études théoriques, nous avons considéré l'adaptation d'une espèce avec un cycle de vie très simplifié. Cependant, la plupart des organismes présentent un cycle de vie à plusieurs stades. Le réalisme de nos modèles pourrait donc être amélioré en prenant en compte des cycles de vie à plusieurs stades. Les effets de la complexité des cycles de vie sur l'adaptation ont été étudiés pour des populations à reproduction panmictique (Cotto et Chevin, 2020 ; Cotto et Ronce, 2014 ; Cotto et al., 2019 ; Marshall et al., 2016). Ces modèles ont pris en compte les variations de la sélection entre stades de vie et montrent qu'un cycle de vie à plusieurs stades peut contraindre ou faciliter les réponses évolutives. Les stades juvéniles sont souvent soumis à de plus fortes pressions de sélection que les stades âgés (Przeslawski et al., 2015). Les réponses des stades juvéniles sont donc attendues différentes de celles des stades adultes (Cotto et Ronce, 2014). De plus, les réponses des stades ne sont pas indépendantes : l'évolution des stades juvéniles influence l'évolution des stades plus âgés (Marshall et al., 2016). Les différences d'intensité de sélection entre stades peuvent faciliter ou contraindre l'adaptation selon notamment les corrélations génétiques entre stades de vie, la variance génétique et la plasticité de chaque stade (Marshall et al., 2016). Cotto et Chevin (2020) modélise une population soumise à plusieurs épisodes de sélection fluctuante au cours d'une génération. A l'aide d'un modèle d'évolution d'un trait polygénique, les auteurs montrent que les dynamiques évolutives dépendent d'un optimum intégratif correspondant à une somme des optimums pondérée par la contribution de chaque épisode de sélection à la sélection totale. L'intensité de la sélection est



quant à elle proportionnelle à une moyenne harmonique des largeurs de fonctions de sélection. Les fluctuations intra-génération de la sélection résultent en l'élimination sélective d'individus. Dans le chapitre 3 nous avons montré que les variations de l'intensité de la sélection entre années peuvent aussi aggraver le déclin des populations. Il serait intéressant de comparer la part de la variation de l'intensité de la sélection entre années (chapitre 3) et entre stades de vie dans les changements de taux de croissance à long terme des populations.

Chez certains organismes comme les plantes ou les coraux, la phase de dispersion se produit uniquement au stade de gamète et au stade juvénile (*i.e.* graine ou larve). Au delà du fait que les phases juvénile et adulte ne subissent pas les mêmes pressions de sélection, la phase fixée peut contraindre les changements rapides d'aire de distribution et les réponses évolutives (Aguilée et al., 2016 ; Cotto et al., 2017). Chez les plantes, la phase de dispersion est étroitement liée à la phénologie de floraison et l'homogamie pour la date de floraison peut agir comme un filtre aux flux de gènes (Soularue et Kremer, 2012). L'homogamie pourrait ainsi avoir des conséquences non négligeables sur les changements d'aire de distribution et les changements de niche écologique dans un climat changeant. Il serait intéressant d'étudier les effets de l'homogamie sur les changements d'aire de distribution des plantes.

## 5.4 Conclusion générale

Au cours de cette thèse, nous avons utilisé une approche théorique pour tenter d'améliorer le réalisme des modèles d'adaptation des phénologies de floraison au changement climatique. Nous avons montré que la sélection sexuelle engendrée par l'homogamie affecte les conflits de sélection entre mâles et femelles. La sélection sexuelle et la sélection naturelle sexe-spécifique, en absence de dimorphisme sexuel, déplacent la valeur génétique moyenne de la population en direction de l'optimum des femelles en environnement constant. Dans un environnement changeant, il existe une gamme de vitesse de changement environnemental, variant avec la sélection sexe-spécifique, pour laquelle l'homogamie facilite l'adaptation à l'optimum femelle par rapport à la panmixie. Le dimorphisme sexuel peut entraîner la valeur génétique moyenne de la population au-delà de l'optimum femelle, et ainsi augmenter ou diminuer la déviation de la valeur génétique moyenne de la population par rapport à l'optimum femelle. Le dimorphisme sexuel diminue souvent la gamme de vitesses de changement environnemental pour laquelle l'homogamie facilite l'adaptation à l'optimum femelle par rapport à la panmixie. Nous avons aussi montré que la sélection sexuelle a des effets antagonistes sur la variance génétique : elle s'ajoute à la sélection stabilisante et diminue le polymorphisme génétique mais elle augmente aussi les associations positives entre effets alléliques. Les associations positives sont moins importantes quand la sélection sur les femelles est plus forte que celle sur les mâles. Dans de nombreux cas, les effets positifs de l'homogamie dominent ses effets négatifs et l'homogamie augmente la variance génétique par rapport à la panmixie. La manière de modéliser l'homogamie affecte la compétition entre les mâles et la variance des mâles accédant la reproduction. Enfin nous avons montré que les fluctuations de la durée de la saison de floraison, accentuées par le changement climatique, diminuent la variance génétique et le retard adaptatif. Les fluctuations de la durée de

la saison de floraison impliquent dans de nombreux cas un coût démographique qui compromet le taux de croissance à long terme des populations.



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