



HAL
open science

Co-évolution des traits et du génome sous l'effet de la sélection sexuelle : la démo-génétique rencontre l'écologie comportementale.

Louise Chevalier

► **To cite this version:**

Louise Chevalier. Co-évolution des traits et du génome sous l'effet de la sélection sexuelle : la démo-génétique rencontre l'écologie comportementale.. Sciences agricoles. Université de Pau et des Pays de l'Adour, 2021. Français. NNT : 2021PAUU3072 . tel-04631649

HAL Id: tel-04631649

<https://theses.hal.science/tel-04631649>

Submitted on 2 Jul 2024

HAL is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.

UNIVERSITY OF PAU and PAYS ADOUR
DOCTORALE SCHOOL ED 211
SCIENCES EXACTES ET LEURS APPLICATIONS

PHD THESIS

to obtain the title of

PhD of Science

Speciality : BIOLOGY

Defended by

Louise CHEVALIER

Coevolution between mating behaviours and genetic architecture under sexual selection

prepared at UMR ECOBIOP, INRAE

Reviewers : Erik SVENSSON (Professor) - Faculty of science Lund University
Maria SERVEDIO (Professor) - University of North Carolina
President : Emmanuel DESOUHANT (Professor) - Université Claude Bernard Lyon 1
Examiner: Claire DOUTRELANT (Doctor) - CEFE Montpellier
Thesis director: Jacques LABONNE (Doctor) - INRAE/UPPA

Avant-Propos:

L'idée de cette thèse vient de mon stage de master qui fut également encadré par Jacques, et que j'avais contacté car j'étais intéressée par la modélisation des comportements. Suite à nos échanges, il m'a proposé de travailler sur son petit joujou, un modèle un peu fou qu'il avait créé. J'ai immédiatement eu envie de continuer en thèse avec lui. Il a toujours été à l'écoute, a eu confiance en moi, et m'a encouragée à aller plus loin. Merci à toi pour ta pédagogie, ta patience et ta disponibilité. J'ai tellement souvent été toquer à ta porte avec un confus mélange de doutes et de questions en tête, menant inmanquablement à une longue discussion de laquelle je sortais rassurée et avec un grand besoin de noter pleins d'idées sur un papier !

Merci de m'avoir permise de faire cette thèse et merci pour tout le temps que tu m'as consacré !

Je voudrais également remercier FX Dechaume-Moncharmont avec qui j'ai aussi eu la chance de travailler, mon travail sous son encadrement ayant d'ailleurs été à l'origine de mon premier papier. Il m'a ainsi convertie à Latex et donné goût aux figures R. Passionné et perfectionniste, j'ai beaucoup appris en échangeant avec toi. Merci FX pour ton accueil, ta générosité et ton enthousiasme !

Je voudrais ensuite remercier Alexandre Courtiol qui, dès que je l'ai contacté, s'est intéressé à mon travail. Je suis heureuse d'avoir pu venir à Berlin au Leibniz institute of Zoo and WildLife et d'avoir fait partie de la team « Hyena project » le temps de quelques mois. Merci de m'avoir accueillie, d'avoir été disponible autant que possible et merci pour tes critiques constructives concernant mon travail. Une petite pensée en passant pour Colin et Alexandra, avec qui j'ai vraiment apprécié passer du temps et boire du glühwein.

Merci aussi Mathias Gallipaud, pour ton intérêt et pour tes conseils avisés con-

cernant la rédaction d'article.

Merci à Cyril Piou, Sylvie Oddou, François Lefèvre, Mathieu Buoro et les autres personnes du réseau démogénétique avec qui j'ai pu échanger sur mon modèle et sur mon sujet.

Merci aussi aux développeurs de capsis, François de Colligny et Nicolas Beudez toujours là pour répondre à nos questions et pour signaler gentiment quand je remontais du code qui faisait bugger toute la plateforme.

Merci donc à vous tous, avec qui j'ai vraiment apprécié travailler et grâce à qui j'ai beaucoup appris pendant ma thèse.

Etienne Prevost et Valérie Bolliet, toujours bienveillant et à l'écoute, merci de votre accueil dans le laboratoire ECOBIOP. Et plus généralement, je remercie toutes les autres personnes du labo : Cédric, qui m'a fait découvrir l'enseignement; Mathieu, encore, qui m'a remotivée à faire de l'escalade pendant un temps, Pantxica toujours prête à aider, Aurélie, Pascal et tous les autres membres de l'unité, pour leur gentillesse.

Un remerciement spécial pour Dorinda, avec qui j'ai partagé un bureau, mon directeur de thèse et beaucoup de fou rire. Merci de m'avoir apprise à faire du puzzle avec mes phrases. Et merci d'avoir été là pour me nourrir, égayer mes journées et me donner envie de venir au bureau le matin. Sans oublier Hervé qui m'a convaincue de l'importance du détail et des belles figures. Merci aussi à vous deux pour vos conseils en esthétique de manière général d'ailleurs.

Merci à tous les amis que j'ai pu rencontrer ici: Lucie "*la maman des thésards*" toujours là pour conseiller et aider, Clément "*le président du comité des fêtes des thésards*" toujours avec le sourire et à organiser des soirées chez lui, Floren avec qui je partage une belle complicité, Amaia la petite dernière de la team démogénétique, toujours positive et qui apporte de la bonne humeur, Marius, Laura, Stella, et Léa,

toujours là pour papoter autour d'un café. Merci enfin à ma super colloque Florence qui m'a soutenue pendant ces derniers mois de thèse à coup de gateaux vegans et de cours de yoga.

Pour finir, merci à mes parents et à mes sœurs qui sont toujours là pour me soutenir et m'aider à réaliser mes projets.

Abstract:

Sexual selection is one of the main drivers of evolution and biodiversity. It results from differential access to reproduction and controls the transmission of genes to the next generation. It has thus rippling consequences on the maintenance of genetic diversity and the speed of evolution in populations, acting either in synergy or in antagonism with natural selection. Mating behaviours (mate choice, intra-sexual competition) and mating systems characteristics (pairing processes, sex-ratio, mate availability) affect the strength and direction of sexual selection. Interactions between individuals being of main importance. Sexual reproduction is inherently a highly context-dependent process, because social environment is usually very variable, making of mating outcome a pivotal element to predict the direction of evolution.

On the other hand, traits and behaviours may have complex genetic architectures, influencing their expression and their transmission. Genetic variance and covariance between traits in populations can play a crucial role in determining the evolutionary trajectory and equilibrium. Loci's number and location, on either autosomes or sex chromosomes, as well as levels of genetic linkage and pleiotropy among them determine the combination and variance of traits values that are transmitted to offspring through recombination. Yet, genetic architecture itself can also evolve through sexual selection. However, evolution of mating behaviours and of genetic architecture have long been addressed by separate and diverging modelling approaches. More recently, the significance of feedback loops between genetic changes, behaviours, and demography in shaping sexual selection has been highlighted.

During my PhD, I demonstrated and quantified the coevolution of genetic architecture and traits and behaviours evolution under sexual selection, accounting for its complex behavioral processes. The integrative framework I propose allow to represent together the several components of sexual selection and to better understand the evolutionary mechanisms at work. This form of exploratory work is needed to later develop predictive models applied on wild populations and to provide some theoretical predictions in fields where most of the knowledge originates from empirical research (genetic basis of traits under sexual selection for instance).

Keywords: demogenetics, behavioral ecology, genetic architecture, sexual selection, mate preference, mating systems.

Résumé:

La sélection sexuelle constitue l'un des principaux mécanismes d'évolution et de biodiversité. Elle résulte d'un accès différentiel à la reproduction, ce qui contrôle la transmission des gènes à la génération suivante. La sélection sexuelle a des conséquences sur le maintien de la diversité génétique, et sur la vitesse d'évolution des populations naturelles, agissant en synergie ou en antagonisme avec la sélection naturelle. Les comportements reproducteurs (choix du partenaire, compétition intrasexuelle) et les caractéristiques du système d'accouplement (processus d'appariement, sex-ratio, disponibilité des partenaires) vont influencer la force et la direction de la sélection sexuelle. Les interactions entre les individus étant centrales. La reproduction sexuelle est ainsi un processus très dépendant du contexte, car l'environnement social est généralement très variable, or l'issue de la reproduction représente un élément clé pour prédire la direction de l'évolution.

D'autre part, les traits et comportements peuvent avoir des architectures génétiques complexes, influençant leur expression et leur transmission. La variance génétique et la covariance entre les traits dans les populations peuvent jouer un rôle crucial dans la détermination des trajectoires et des équilibres évolutifs. Le nombre de loci, leur localisation sur les autosomes et les chromosomes sexuels ainsi que leur degré de liaison génétique et de pléiotropie déterminent la combinaison et la variance des valeurs de traits transmises à la descendance via recombinaison. En outre, cette architecture génétique, peut elle-même évoluer par le biais de la sélection sexuelle. L'évolution des comportements d'accouplement et l'évolution de l'architecture génétique ont depuis longtemps été abordées par des approches de modélisation séparées et divergentes. Plus récemment, l'importance particulière de l'interaction entre les changements génétiques, les comportements et la démographie pour comprendre la sélection sexuelle, a été mise en évidence.

Ma thèse démontre et quantifie la coévolution entre l'architecture génétique et l'évolution des traits et des comportements dans un cadre de sélection sexuelle, en tenant compte de ses processus comportementaux complexes. Le modèle intégratif que je propose permet de représenter les différentes composantes de la sélection sexuelle afin de mieux comprendre les mécanismes évolutifs à l'œuvre. Ce travail exploratoire est nécessaire pour le développement ultérieure de modèles appliqués aux populations naturelles, et peut permettre de fournir des prédictions théoriques dans des domaines où la plupart des connaissances proviennent de la recherche empirique (concernant par exemple les bases génétiques des traits sous sélection sexuelle).

Mots clés: démogénétique, écologie comportementale, architecture génétique, sélection sexuelle, préférence sexuelle, systèmes d'accouplement.

Contents

1	Introduction	1
1.1	Definition of sexual selection	3
1.2	Drivers of sexual selection	3
1.2.1	Intra-sexual selection: competition	4
1.2.2	Inter-sexual selection: mating preference	5
1.3	Trade-off between reproduction and survival	8
1.4	Effects of sexual selection on traits and mating behaviors	9
1.4.1	The evolution of mating preference	9
1.4.2	The emergence of sex roles	11
1.5	Effect of sexual selection on population genetics and on genetic architecture	13
1.5.1	Effect on population genetics	13
1.5.2	Effect on genetic architecture	14
1.6	Effect of sexual selection on population dynamics	18
1.7	The strong context-dependency of sexual selection and its evolutionary implications	19
1.8	An integrative framework to represent the different aspects of sexual selection and their feedbacks	20
2	Several modelling approaches to study sexual selection	23
2.1	Phenotypic models	24
2.1.1	Fisherian optimality analysis	25
2.1.2	Game theory	27

2.1.3	A case study: fluctuations of the mating market affect the evolution of flexible choosiness	30
2.2	Genetic models	36
2.2.1	Invasibility analysis	36
2.2.2	Quantitative genetics	38
2.2.3	Population genetic models	42
2.2.4	Demogenetic individual based models	43
2.3	Discussion	45
3	Modelling mating behaviours and genetic architecture	51
3.1	Conception of a demogenetic simulation model	53
3.1.1	An overview of the demogenetic agent based model	53
3.1.2	Detailed model description	55
3.1.3	General concepts underlying the design of the model	65
3.2	Exploring the evolutionary dynamic of a particular simulation: co-evolution between Gametic investment and Preference	67
3.2.1	Evolution of reproductive traits:	68
3.2.2	Demographic consequences	71
3.2.3	Evolution of genetic architecture	73
3.3	Effect of mating systems on traits and genetic architecture evolution	77
3.3.1	Evolution of the reproductive traits:	77
3.3.2	Genetic architecture evolution:	82
3.4	The sensibility of parameters change	85
3.4.1	Sensibility to reproductive trade-off:	85
3.4.2	Genetic parameters:	86
3.4.3	Shape of the preference function:	87

3.5	Discussion	92
4	The effect of social environment on the evolution of mating behaviours and genetic architecture	99
4.1	Introduction	100
4.2	Increasing the fluctuations in mate availability	102
4.3	The effect of mating group size on the mating dynamics	103
4.4	Effect on trait evolution	106
4.5	Effect on genetic architecture	109
4.6	Discussion	116
5	How physical constraints on the genome architecture affect the evolution of traits and genetic architecture	119
5.1	Introduction	120
5.2	The effects of genome size and linkage disequilibrium	123
5.3	The effects of sexual chromosomes:	127
5.4	Discussion	133
6	Discussion	137
6.1	Synthesis of the main results	138
6.2	Some remarks concerning our choices of modeling and future development	142
6.3	A short reflection on my work	147
	Bibliography	151

Appendices	189
A Article 1: Fluctuating Dynamics of Mate Availability Promote the Evolution of Flexible Choosiness in Both Sexes	191
B Article 2: A demogenetic agent based model for the evolution of traits and genome architecture under sexual selection	207
C Additional method	245

Introduction

When a biologist is asked to define "life", said biologist often proceeds by breaking down the concept into mandatory functions. Ability for organisms to reproduce is certainly one of them, if not the main one. For as long as death occurs, reproduction is the only way for organisms to perpetuate life, notably by transmitting their genes. These organisms devote considerable time, effort and energy to achieve reproduction. It is enough to be convinced to examine a sea urchin whose gonads exceed in volume all other organs combined, or the complexity of the courtship rituals of birds. Sexual reproduction is old (more than a billion year actually on earth) and is widespread on the phylogenetic tree; many plants, and most animals, reproduce sexually. It entails costs that have long puzzled evolutionary biologists. One of the constraints for separate sexes species is to find a mate in order to fuse gametes and convey life.

It is where reproduction becomes a complex and hazardous quest for mate, since individuals compete for the same goal, and since the partner perceived as ideal will not necessarily have a reciprocal opinion. Sexual reproduction therefore introduces new rules into the game of life, with new challenges, and new consequences.

Questions of the origin, evolution and maintenance of sexual reproduction over asexual reproduction have thus long captured the attention of biologists, from early theories and experiments starting in in the 80's [Smith & Maynard-Smith 1978, Hamilton 1980] to the onset of genomics nowadays, opening new fertile grounds.

Sexual reproduction is an ancient and major event in the evolution of life. It appears in early prokaryotes around two billions years ago and has marked the evolution of traits, behaviours and genomes ever since [Fontaneto *et al.* 2012]. As noted previously, sexual reproduction requires the acquisition of a mate, for what sexual organisms have to spend energy and time, searching and possibly courting. Meanwhile, they undergo risks of predation, disease, and injury while courting and mating [Partridge & Hurst 1998]. To this, is added the “twofold cost” of producing males [Smith 1978, Gibson *et al.* 2017] that is a twofold reduction in a sexual population growth rate compared to an asexual population, as males cannot themselves make offspring. The twofold cost also refers to a ‘cost of meiosis’ in sexually reproducing organisms [Williams 1975, Lively & Lloyd 1990], where each sexual parent only contributes half its genes to its offspring, decreasing its genetic contribution and thus the relatedness between parent and offspring. Lastly, sexual reproduction may break apart favorable combinations of genes built by past selection.

On the other hand, sexual species are expected to adapt faster than asexual one: Meiosis during sexual reproduction has traditionally been regarded as crucial to the evolution of living organisms by creating a diversity of genotypes upon which natural selection can act ([Weismann 1889]p. 279 but see [Otto 2009]). Additionally, recombination of DNA during meiosis could join beneficial mutations together to make a more optimal genome, and prevent the buildup of deleterious mutations. Sex is also a powerful defense against parasites as sexual organisms would have an advantage in the arms race since they shuffle their genes into new combinations that are harder for parasites to adapt to.

Lastly, sexual reproduction gives rise to sexual selection, since variance in reproductive success between individuals depends then on their traits and behaviours.

In this first Chapter, which serves as a general introduction, I will go over over the mechanisms that condition sexual selection, and the pervasive effects that sexual selection in return likely produces on behaviour, traits, population structure, and genomes evolution.

1.1 Definition of sexual selection

Sexual selection is generated by differential access to opposite-sex mates or gametes [Darwin 1871, Andersson 1994]. Indeed, during reproduction, individuals compete for access to mating partners, whose quality may be variable.

By defining sexual selection as “*the advantage which certain individuals have over other individuals of the same sex and species solely in respect of reproduction*”, [Darwin 1871] made a clear distinction between natural and sexual selection. Sexual selection arises from differences in mating success, whereas natural selection is due to variance in all other fitness components. While it is generally accepted that natural selection should improve the fitness of organisms with respect to their local environment, potentially reaching evolutionary optima, sexual selection can lead to non-adaptive situations, promoting trait values beyond their naturally selected optima. Therefore, natural and sexual selection can act either in synergy or in antagonism.

1.2 Drivers of sexual selection

Sexual selection can operate at different stages and through different mechanisms:

Precopulatory sexual selection refers to all the processes involved in the acquisition of mating opportunities (i.e., excluding competitors, attracting, selecting and/or retaining mates). Post-copulatory sexual selection (after gametes have been

released) occurs through competition of the ejaculates of different males over fertilization (sperm competition) and choice of sperm by the female (cryptic female choice).

Sexual selection can operate both intra-sexually - by means of competition - and inter-sexually, by means of inter-sexual preference or mate choice. These two mechanisms are usually referred as to "agents of sexual selection".

1.2.1 Intra-sexual selection: competition

Individuals of the same sex compete for access to mating partners. The most competitive individuals will be able to limit or even completely prevent reproduction for less competitive individuals of the same sex. Two types of competition can be distinguished:

- "Scramble competition" is inherent of all mating systems and results from the consumption of a resource (here, the mates) by other competitors. Its intensity varies with the availability of potential mates (sex-ratio, density etc., [Emlen & Oring 1977, Dechaume-Moncharmont *et al.* 2016, Chevalier *et al.* 2020]).
- "Active competition" occurs when members of the same sex attempt to out-compete rivals during direct encounters [Fawcett *et al.* 2012]. Active competition is typically thought to be responsible for the evolution of male armaments such as deer antlers, beetle horns, and large body size, that provide individuals with an advantage when fighting off potential competitors.

1.2.2 Inter-sexual selection: mating preference

By mating with a particular partner, individuals can gain direct or indirect benefits [Andersson 1994, Kokko *et al.* 2002]. Direct benefits result in a direct gain in terms of fecundity or survival for the chooser (i.e. direct selection, because of a partner who is more fertile or who offers protection for instance). Indirect benefits result in gains for the offspring (e.g. “good genes” or “sexy sons” [Fisher 1930, Kokko *et al.* 2002]). Different properties of mating preferences can be distinguished [Jennions & Petrie 1997, Kilmer *et al.* 2017, Neelon *et al.* 2019]:

The preference function: The preference function plots the probability that an individual will accept a mate along a continuum of trait values. In the field, acceptance likelihood can be measured as responsiveness to mate signal. Preference function shapes can be unimodal, and favor intermediate trait values (Fig.1.1 left panels) or open-ended and favor extreme trait values (Fig.1.1 right panels). Preference functions can be described by many components (which could themselves be considered as traits). The ‘peak’ indicates the most preferred trait value, the ‘tolerance’ describes the acceptance of trait values that deviate from the peak, the ‘strength’ measures the difference in mate response to more preferred mates versus less-preferred mates, and ‘responsiveness’ indicates the mean response levels across all trait values. In general however, empirical and theoretical studies deliberately focus on only one of these aspects, thus assuming the others to be constant. An important concern when measuring preference in the field is also whether preferences for different stimuli should be recorded sequentially or in a competitive situation [Wagner 1998], as it implies different cognitive mechanisms (memory, experience, sampling strategy) [Dougherty & Shuker 2015].

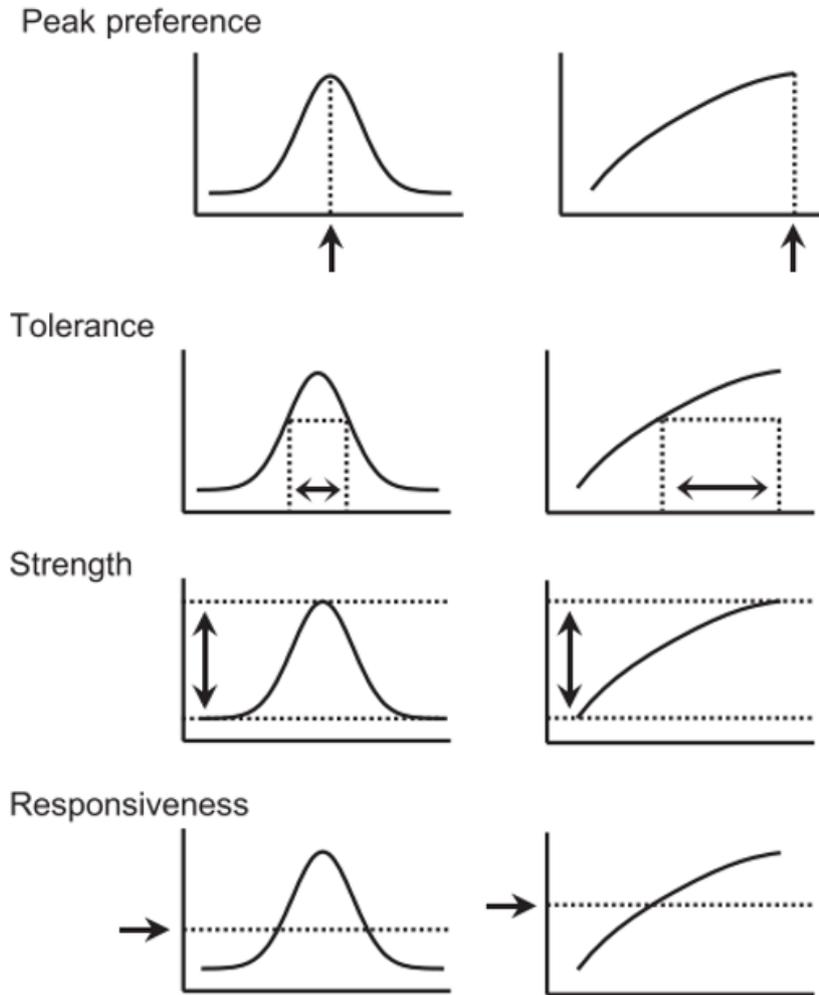


Figure 1.1: : The different descriptors of the preference function. Left panels show an unimodal shape of the preference function (intermediaire traits values being the most attractive). Right panels show a directional shape of the preference function (maximum trait values being the most attractive). The tolerance, strength and responsiveness are sometimes called "choosiness". From [Kilmer *et al.* 2017]

The choosiness: [Jennions & Petrie 1997] proposed that preference functions and choosiness are distinct traits that can evolve independently, and defined the choosiness as the effort or energy that an individual is prepared to invest in assessing

mates, both in terms of the number of mates sampled and the amount of time spent examining each mate (e.g. [Lindström & Lehtonen 2013, Judge *et al.* 2014]). But [Reinhold & Schielzeth 2015] argued that this would be better called cost of mate choice and advocated that linking choosiness to costs confounds the analysis of preference functions with the costs that, together with their benefits, determine how preference functions are shaped by selection. They defined as choosiness the specificity of a mating preference and hence the tolerance of individuals to deviations from the most preferred stimulus (such definition is also used by [Kokko & Monaghan 2001, Kokko & Johnstone 2002, Courtiol *et al.* 2016]). So they saw choosiness as one aspect of the preference function. Choosiness is also sometimes described as the mating threshold with regard to trait values (i.e. responsiveness, e.g; [Johnstone *et al.* 1996, Johnstone 1997, Etienne *et al.* 2014]).

Choosiness, together with the existing variation in phenotypic traits and the mate sampling strategy, is directly connected to the strength of sexual selection on the signalling trait and hence to the evolution of the traits and preference [Arnold 1983]. This is why an exclusive focus on the peak preference of individuals, without relating it to the choosiness, might lead to an overestimation of the role of preference in sexual selection.

The cost of preference and choosiness: Mating preference incurs several costs [Pomiankowski 1987, Andersson 1994, Jennions & Petrie 1997]: First, there is an inherent developmental cost because certain cognitive abilities (visual discrimination, etc.) had to be developed to allow the existence of such preferences. Second, there can also be an energetic cost devoted to sampling mates - as direct loss of energy or loss of time that could be used to another activity such as feeding [Milinski & Bakker 1992, Booksmythe *et al.* 2008, Fawcett *et al.* 2012],

or escaping predators [Sih 1994, Booksmythe *et al.* 2008]. Lastly, another possible cost lies in the risk that the current mating opportunities may become unavailable (named as a “collection risk” ;[Houston & McNamara 1982, Fawcett *et al.* 2012] or an “opportunity cost“,[Dechaume-Moncharmont *et al.* 2016]). It emerges from the pairing dynamics [Johnstone 1997, Chevalier *et al.* 2020] as available partners at a given time may become unavailable in the future. And its intensity varies depending on the social context (i.e. the number of available partners and the intensity of competition for mates).

1.3 Trade-off between reproduction and survival

As suggested at the beginning of this introduction, reproduction is possibly the most costly function for organisms. The reproductive investment or reproductive effort is defined as the fraction of resources invested in reproduction. This definition is based on the theory of energy allocation, which states that the total investment of reproductive individuals consists of somatic investment and reproductive investment [Williams 1966, Gadgil & Bossert 1970]. As the energy invested in reproduction is not put into somatic investment (i.e. growth, immune function, tissue repair, etc; [Nordling *et al.* 1998]), it has a cost to survivorship or future fertility [Stearns 2000]. The allocation of reproductive effort is thus a key aspect of life history strategy and has become a central concept in theories of life history evolution. This led to the formulation of r and K selection theory, which predicts an association of life-history traits into two groups depending on environmental and social conditions [Wilson & MacArthur 1967, Parry 1981]:

r-selection is selection for maximum population growth in uncrowded populations, leading to species characterised by an early age of maturity, a large fecundity,

semelparity, no parental care.

K-selection is selection for competitive ability in crowded populations, leading to species characterized by a delayed reproduction, a smaller fecundity, iteroparity, parental care.

1.4 Effects of sexual selection on traits and mating behaviors

The reproductive investment can be allocated in various tasks, and individuals can potentially adopt different strategies/ behaviours to reproduce.

I here distinguish *traits* that are morphological characters determined at some point in life (e.g. ornaments) from *behaviours* which are much more plastic and context dependent.

The evolution of some traits and behaviors is complex to understand because it implies both direct and indirect selection, and presents multiple and context-dependent benefits and costs. For instance, sexual selection can sometimes lead to the evolution of “extreme” and “costly” traits as a result of an “arm race” in the population : competition between males may favor traits that allow males to compete with each other, even when the trait also reduces survival (e.g. [Arnqvist & Rowe 1995, Chapman *et al.* 2003]). The female choice may also favor male traits that reduce male life expectancy [Zahavi 1975] and/or the survival or fertility of offspring [Brooks 2002].

1.4.1 The evolution of mating preference

Traditionally, theories address the evolution of female preference for males traits; yet, it is now quite clear that males too can express preference [Gowaty 2003,

Tang-Martinez & Ryder 2005]. This observation indicates that one should always envision all possibilities regarding the evolution of mating systems and, here evolution of preference. This is why I will try to keep my discourse general in this manuscript, and avoid making a distinction between the sexes when it is not necessary.

How and why mating preference evolves has been vigorously debated and several mechanisms have been proposed (reviewed in [Andersson 1994]). Some of the alternatives mechanisms, which are all compatible and may apply in combination (depending on species and on their mating systems), are direct material advantage, avoidance of hybrids mating, Fisherian selection, genetic indicator (handicap) mechanism. I propose here a quick classification and explanation of some of the most supported mechanisms:

Mechanisms involving direct selection on preference: Preference can simply confer a direct benefit for the chooser in term of increased fecundity, protection, or parental care from the partner [Kirkpatrick 1996, Iwasa & Pomiankowski 1999], or through the avoidance of risky breeding (hybrid mating).

Mechanisms involving indirect selection on preference: An indirect benefit is an advantage given for the offspring through inheritance of genes carried by the parents. Two kinds of advantages were identified: (1) The inheritance of “good genes” which confer higher viability or fecundity [Smith 1991]; (2) The inheritance of the preferred character which confers higher mating success for the offspring (i.e “sexy sons hypothesis” [Fisher 1915])¹.

The "sexy sons" mechanism may trigger a rapid co-evolution between the pref-

¹"sexy sons" can be see as a form “good genes” mechanism wherein the advantage is only related to higher attractiveness [Kokko *et al.* 2002]

erence and the trait subject to preference: as the advantage of the trait (in term of mating success) increases with the intensity of preferences, and the advantage of expressing stronger preferences also increases with the advantage conferred by the trait. The two advantages are therefore mutually reinforcing and the process can be overwhelming (i.e. "runaway selection", [Fisher 1915]) and lead to costly ornamental values in regards to natural selection and preferences for that expensive ornament [Fisher 1915, Darwin 1871]. To be triggered, such a process requires first a certain intensity of preference for the trait in the population. Initially the preference may have evolved in response to an advantageous trait (through direct or indirect benefit). Its increase in frequency in the population leads to the trait becoming more and more sexy, which creates a favorable situation to the implementation of "runaway selection".

Alternatively, preference for costly trait can also be explained by the handicap principle [Zahavi 1975]. According to this theory, costly trait (i.e. handicap) could be a honest indicator of quality because only vigorous individuals would be able to survive with this handicap.

Sensory bias : This theory suggests that mating preferences are by-products of natural selection on sensory systems [Endler & Basolo 1998]. For example, across some populations of guppies, it has been proposed that female preference for males with larger orange spots could be the result of an attraction for orange food items that were diverted by the males as a means of attracting females [Rodd *et al.* 2002].

1.4.2 The emergence of sex roles

So far we did not specifically refer to male and female behaviors. Yet, it is widely known that sexual selection can select for different behaviors/ reproductive strategies

between sexes.

Difference of mating behaviours between sexes was seminal in the discovery of sexual selection, and has been widely acknowledged [Williams 1966, Darwin 1871, Trivers 1972, Williams 1975, Berglund *et al.* 1989, Souroukis & Murray 1994]. Traditionally males are thought to be in competition for partner access and females are expected to express mate choice. The theoretical framework explaining the "classical sexes roles" was laid down by [Bateman 1948]. He established that the relative strength of sexual selection in males and females is determined by the relationship between mating success (number of copulation partners) and reproductive success (offspring production). Bateman found in his studies of *Drosophila melanogaster* that multiple copulations led to a greater increase in the number of offspring produced in males than in females. This would stem from anisogamy that allows higher potential reproductive rate (*PRR*) of males as they produce more gametes than females. As a consequence, the ratio of ready to mate males and females (operational sex ratio, *OSR*) is biased toward an excess of available males [Clutton-Brock & Vincent 1991, Clutton-Brock & Parker 1992]. This unbalanced intensity of mating competition between sexes apparently favors male-male competition and female choice. Anisogamy can generate another crucial difference between males and females in some species, where females control over the internal fertilization process promoting sperm competition (cryptic female choice). In addition, the resulting parentage uncertainty for males would select against male cares [Lehtonen & Kokko 2011]. The evolution of such differences in mating strategies will again increase the unbalance in the sex ratio, potentially reinforcing the divergence of strategies between sexes [Kokko & Jennions 2008]. The sex-ratio can be affected at two levels: higher mortality rates in males (caused by stronger competitive interactions) will bias the adult sex ratio (*ASR*). Higher reproductive rate of males

(as they produce more gametes and they may have shorter latency period after reproduction) will bias the *OSR* [Emlen & Oring 1977, Kvarnemo & Ahnesjo 1996]). The predictive power of *OSR* for the evolution of mating behaviors raised much interest because this variable can be estimated relatively easily in empirical approaches or in the field. In short, an *OSR* biased toward an excess of available males in the population would explain difference between male and female choosiness in many mating systems. Still, this pattern can be questioned, and may have been exaggerated, because relatively few empirical studies addressed the question of male mate choice [Gowaty 2003]. Increasing evidence now shows that males can be choosy too, even in apparently unexpected situations (i.e. others than the "classic" sex role reversal situation, [Trivers 1972]), such as under polygyny or in the absence of male parental care [Edward & Chapman 2011], leading to mutual choice [Amundsen *et al.* 1997, Bonduriansky 2001]. Besides, female competition is potentially widespread [Campbell 2004, Stockley & Bro-Jørgensen 2011, Rosvall 2011]; although females tend to limit aggressive interactions, and generally compete for mates by advertising their qualities and using indirect means to denigrate their rivals [Campbell 2004, Benenson 2013].

1.5 Effect of sexual selection on population genetics and on genetic architecture

1.5.1 Effect on population genetics

Sexual selection acts on genetic variance and covariance in the population: it can either reduce the genetic variance of the trait under selection (eg. selection of a particular trait value of preference and/or competition), or alternatively increase genetic variance through direct or indirect mechanisms. For instance, preference

can directly select genetically dissimilar mates to get complementary alleles for immune genes of the major histo-compatibility complex (MHC) to reach an optimal combination of MHC alleles for the offspring (as a form of “good genes” mechanism, [Wedekind & Penn 2000]). Genetic variance can also be promoted by frequency dependent selection potentially making alternative strategy more successful than the common one. Lastly, the co-selection of reproductive traits and assortative mating can induce genetic correlation between traits (e.g. the building up of genetic covariance between female preference and male trait by nonrandom mating; [Fisher 1915, Lande 1981]).

1.5.2 Effect on genetic architecture

Genetic characteristics of the population stem from change in the genome (e.g. fixation of alleles, linkage disequilibrium, pleiotropy). Genetic architecture describes the underlying genetic basis of phenotypic traits and their variational properties [Hansen 2003, Hansen 2006, Rajon & Plotkin 2013, Springer *et al.* 2011]. Description of genetic architecture can encompass gene and allele numbers, the distribution of allelic effects and mutational rate, and patterns of pleiotropy, dominance, and epistasis. The constant progress in genetics have allowed the gathering of data on the genome of many eukaryotic organisms (karyotype.org, treeofsex.org). In particular, the 'Tree of Sex' project associates sexual systems and sex chromosomes informations, with the idea that it could bolster comparative studies concerning the relation between the evolution of sexual systems (sex determination, separate sexes or not) and sex chromosomes evolution [Ashman *et al.* 2014]. Additionally, the development of gene expression analysis allows to investigate behavior-specific genes expression : QTL (quantitative trait loci) and transcriptomic analyses can be used to identify the genes whose

expression is correlated with behavior [Rittschof & Robinson 2014]. By shaping mating behaviours, sexual selection is likely to reshape the genome. Recent results reveals that mating behaviours have genetic basis usually presenting a polygenic architecture [Valette *et al.* 2020]. There is also ample evidence that sexual selection might reduce mutation load [Whitlock & Agrawal 2009], promote tight genetic linkage between multiple functional loci (i.e. supergenes)[Wellenreuther *et al.* 2014], affect the genetic diversity on sex chromosomes and autosomes [Ellegren 2009, Corl & Ellegren 2012, Wright & Mank 2013] and their rate of evolution [Mank *et al.* 2007, Wright & Mank 2013], and drive the evolution of sex chromosomes [Mank 2009].

Theoretical approaches can also be used to understand how genetic architecture may be shaped by sexual selection. Quantitative genetics describe the overall effect of the whole genome with the genetic variance-covariance matrix [Lande 1981, Lande 1982, Mead & Arnold 2004]. Assuming that the evolutionary effect of an allele could be found by averaging over all the genotype combinations in which it participates [Fisher 1930] and that complex gene interactions can be averaged and treated like statistical noise.

More complex approaches allows to study change in genetic architecture *per se*, and are interested in the evolution of polygeny (i.e. number of genes coding for a trait), pleiotropy (i.e. genes affecting more than one functional trait) or epistatic interactions [Pavlicev *et al.* 2011, Guillaume & Otto 2012, Rajon & Plotkin 2013].

For instance, following heritable change of locus effect, [Rajon & Plotkin 2013] investigate the evolution of polygeny in response to selection strength. [Guillaume & Rougemont 2006] explicitly formulate the link between genes, phenotypes and fitness to investigate the evolution of pleiotropic effect among genes, with a simple genetic map (one or two loci) by following heritable change at modifiers

loci (changing the contribution of genes to different traits).

Importantly, genetic architecture determines the ability of the genetic system to produce and maintain potentially adaptive genetic variants, and can condition the speed and the extent of traits evolution [Wagner & Altenberg 1996, Hansen 2003]. This characteristic is called evolvability and is determined by some aspects of genome organisation:

- The autonomy: describes the genetic possibility for characters to evolve independently [Hansen 2003, Welch & Waxman 2003]. Genetic architecture modularity would allow to maximize the autonomy of phenotypes [Wagner & Altenberg 1996, Pavlicev *et al.* 2011]. Modularity refers to a genotype-phenotype map in which there are few pleiotropic effects among characters serving different functions and with pleiotropic effects among characters that serve the same function.
- The mutability: is determined by mutation rate and effects. Mutation being the source of genetic variation and therefore set an upper limit to evolvability. The mutation rate is primarily influenced by the number and the size (in term of base pairs) of genes affecting a trait [Houle 1998].

Box 1: What is pleiotropy?

Pleiotropy refers to the fact that a gene affects multiple traits. This simple definition is actually imprecise, because the product of a gene can operate at different levels in the organism. [Paaby & Rockman 2013] made a distinction between ‘*molecular pleiotropy*’, ‘*developmental pleiotropy*’, and ‘*selectional pleiotropy*’. Although this classification scheme is more a semantic view, it shows the broad scope of the pleiotropy concept. The basis for this distinction lies in the way in which the traits are defined. The pleiotropic aspect of a mutation will vary depending on whether we look at the level of individual cells, tissues, organs, organisms, or populations. ‘*Molecular pleiotropy*’ arises when a gene product several molecular functions. In this context, the Strong Hypothesis of Universal Pleiotropy (*SHUP*) considers that all molecular genes compete for a common pool of polymerase, ribosomes, and nucleotides, etc. Mutations that alter the activity of genes impact this molecular machinery and thus alter the activities of all other genes. Yet mutations affecting the shared metabolite pool may have no detectable effect on one character even when another character is seriously affected. According to the *SHUP* view, this would only conceal the fact that there is universal pleiotropy. Whereas others point out that such trivial effects are biologically meaningless, and that the question is about the number of traits impacting fitness that are affected by a mutation (i.e. ‘*selectional pleiotropy*’ [Wright 1984]).

Pleiotropy is often perceived as an inevitable consequence of molecular and cellular functioning, and would prevent traits from evolving independently. The rational is that the more pleiotropic the effects of a gene are, the more likely the

gene is to affect the phenotype of some trait unfavourably and hence the more likely it is to inhibit evolutionary change [Fisher 1930, Orr 2000].

Empirically, detectable pleiotropy seems highly structured [Wang *et al.* 2010], and such pattern could support the Hypothesis of Modular Pleiotropy (*HMP*) [Wagner *et al.* 2007]. Under modular pleiotropy, the genetic architecture consists of a set of genes that tends to have pleiotropic effects on the same set of traits, but few and weaker effects on other traits. Such a structuring of pleiotropy leads to think that pleiotropy is a characteristic of the genetic architecture that can evolve.

Despite recent advances on the topic, we are still uncertain about how genetic architecture may be shaped by sexual selection, how many genes generally underlie variation in quantitative sexual traits, what the distributions of the effect sizes of these genes are, and what kinds of pleiotropic effects they may have [Fitzpatrick 2004, Phillips 2005, Albert *et al.* 2008].

1.6 Effect of sexual selection on population dynamics

Through its effect on the evolution of mating behaviors and traits, sexual selection affects the fitness of the population. In particular, it can bring about the evolution of costly traits that lower life time expectancy of individuals [Andersson 1994, Promislow 1992, Kokko & Brooks 2003]. And this certainly has consequences on the size of the population.

Sexual selection also affects the reproductive rate of the population which can have rippling consequences on population demography too, in particular, situations where females have little or no opportunity to choose a mate (e.g. low density)

can give rise to Allee effect which can affect the persistence of small populations [Møller & Legendre 2001, Gascoigne *et al.* 2009], and may increase the risk of extinction in some situations [Kokko & Brooks 2003].

Sexual selection will also have an impact on exchanges between and within populations: preferences for locally adapted phenotypes can favor premating isolation [Liou & Price 1994, Servedio 2001]; or alternatively, heterogamous preference can promote hybridization between two gene pools [Gil *et al.* 2016]. Within a population, mate searching can bolster individuals movements, increasing connectivity between subpopulations. For instance, female preference for immigrant males would stimulate male dispersion in some species (e.g. Spotted hyenas, [Davidian *et al.* 2016]).

1.7 The strong context-dependency of sexual selection and its evolutionary implications

Several studies observed context-dependence in the expression of sexual traits and behaviors [Gauthey *et al.* 2015]. Arising from these behavioral processes, the strength of sexual selection, is also changing with the environmental and social context [Cotton *et al.* 2006, Gauthey 2014].

The importance of social context and of interactions between individuals on the evolution of mating behaviour has been considered by game theory models that explicitly incorporate individual decision-making (e.g.[Janetos 1980, Parker 1983, Real 1990, Kokko & Johnstone 2002, Kokko & Rankin 2006, Courtiol *et al.* 2016, Dechaume-Moncharmont *et al.* 2016, Chevalier *et al.* 2020]). Such approaches demonstrate that the best choice/ behavior for an individual depends on the environmental/social context and on others' strategies. Behavioural interactions are

particularly important for sexual selection: for instance, the satisfaction of an individual's preference will depend on the distribution of available phenotypes in the population and the success of an individual will depend on the number of competitors and their performance. Being explicit about the process by which individuals select mates can affect the equilibria of preference-trait coevolution by imposing a variable and context-dependent cost to the preference [De Jong & Sabelis 1991, Kokko *et al.* 2015]. Indeed, when assuming constant direct selection against female preferences (i.e. fixed costs), mating preferences for a sexual ornament does not persist at equilibrium, whereas when the cost of mating preference is derived from mate-sampling processes, mating preference can coevolve with a male trait so that both persist in the presence or absence of biased mutation [Kokko *et al.* 2015]. To state another example, context and individual-specific mate choice can promote polymorphism in traits [Kokko *et al.* 2007, Tazzyman & Iwasa 2010]. Besides, correlational selection on traits (which is often generated by frequency dependent selection) generates genetic correlations [Roff & Fairbairn 2012, Sinervo & Svensson 2002, Walsh & Blows 2009] which could, at the scale of genetic architecture, results in the establishment of pleiotropy or linkage disequilibrium.

1.8 An integrative framework to represent the different aspects of sexual selection and their feedbacks

I hope to have shown in this first Chapter that sexual selection is likely to impact populations at different levels, affecting demography, behaviours and traits, and genome architecture. All these elements are rarely handled together in a single framework [Govaert *et al.* 2019, Svensson 2019]. The purpose of my Philosophical Dissertation is to take some steps in that direction : integrating these various compo-

nents, so to make new progresses on the evolutionary importance of sexual selection. I therefore propose, in a Chapter 2, a review of the different modeling approaches that are used to study the evolution of mating behaviors and genetic architecture, and discuss the advantages and shortcomings of each approach. This chapter highlights that the conjoint evolution of mating behaviors and their genetic architecture would gain from being studied in a framework representing eco-evolutionary processes and explicit genetic architecture. Based on this work, I build in Chapter 3 a framework (a *demogenetic* model) that aims at integrating all the components of sexual selection. Using the model I show some simulation examples highlighting feedbacks between mating behaviors, demography and genetic architecture. This form of exploratory work allows i/ to gain a better understanding of the evolutionary mechanisms at work and ii/ to shed some light on the relationship between the evolution of mating behaviors and the evolution of genetic architecture. Using the above mentioned integrative framework, I then proceed to Chapters 4 and 5, that each investigates an important avenue for understanding evolution of reproductive traits and behaviours. In the Chapter 4, I ask how the social environment can influence the evolution of traits and their genetic architecture. In the Chapter 5, I look at the effects of physical constraints in the genome (maximum number of genes, number and nature of chromosomes, genetic linkage) on traits and genetic architecture evolution. Finally, the Chapter 6 discusses my approach and results and proposes some perspectives for future work on the subject.

Several modelling approaches to study sexual selection

“If models reach different conclusions, they must be making different assumptions. Differences should therefore be considered illuminating ” (Kokko et al., 2006).

Contents

2.1 Phenotypic models	24
2.1.1 Fisherian optimality analysis	25
2.1.2 Game theory	27
2.1.3 A case study: fluctuations of the mating market affect the evolution of flexible choosiness	30
2.2 Genetic models	36
2.2.1 Invasibility analysis	36
2.2.2 Quantitative genetics	38
2.2.3 Population genetic models	42
2.2.4 Demogenetic individual based models	43
2.3 Discussion	45

24 Chapter 2. Several modelling approaches to study sexual selection

In this second chapter, I give a description of the various modelling and theoretical approaches to sexual selection in the literature. I endeavour at analyzing their assumptions, their differences, their respective strengths and weaknesses. This chapter will therefore relate to the different aspects of sexual selection and the specific approaches used to address them (behavioral and phenotypic evolution, genetic constraint).

The diversity of theoretical approaches to study the evolution of traits and behaviors under sexual selection can be divided into three main types of modelling: phenotypic approaches, genetic approaches and demogenetic or eco-evolutionary approaches. These three types of approaches each have their advantages and shortcomings: For example, the phenotypic approach allows to understand the effect of variations in the environmental and social context on the evolution of traits and behaviors, but ignores genetics and neglects evolutionary trajectories, which excludes the opportunity to observe cyclic evolutionary dynamics [Iwasa & Pomiankowski 1999]. Simple genetic approaches assume an unrealistic genetic determinism, but they allow easy modelling of the evolution of genetic variance and covariance, contrary to most quantitative genetic approaches [Charmantier & Sheldon 2006]. Eco-evolutionary approach combine phenotypic and genetic approach, but can seldom be analyzed mathematically, except in very simple cases.

2.1 Phenotypic models

The researchers that study behavior typically adopt phenotypic approaches. In such approaches, the individual behavior is seen as the agent of selection acting on traits in populations by considering fitness effects.

2.1.1 Fisherian optimality analysis

Simple optimality analyses find the life-history trait values that maximize the fitness function at the evolutionary equilibrium (e.g.[Hirshfield & Tinkle 1975, Pianka 1976, Michiels *et al.* 2009]). In such models, fitness is mathematically described with respect to life-history traits (by the Malthusian parameter, r , or by the lifetime reproductive success R_0). This approach assumes that the trait under study is not affected by density-dependence and frequency-dependent interactions.

Box 1: Example of Fisherian optimality analysis: Natural selection of reproductive effort (Pianka 1976)

Question: What factors determine optimal allocation of the available energy into reproductive versus non-reproductive (somatic) tissues, organs, and/or activities?

Optimization criteria: Maximization of the lifetime reproductive success, R_0 , calculated as the cumulative product of survival and fecundity over organism's lifespan, assuming a population with constant size and stable age distribution.

Mechanism: Trade-off between present reproduction (present offspring) versus expectation of future reproduction (future progeny). At each age, an organism is expected to allocate resources to present reproduction in a manner that will maximize reproductive value at that age (Fig.2.1.1).

Result: The form of the trade-off, which is itself sensitive to a multitude of environmental influences including resource availability and the immediate environmental conditions for reproduction and survival, determines the optimal strategy of reproduction at any given age.

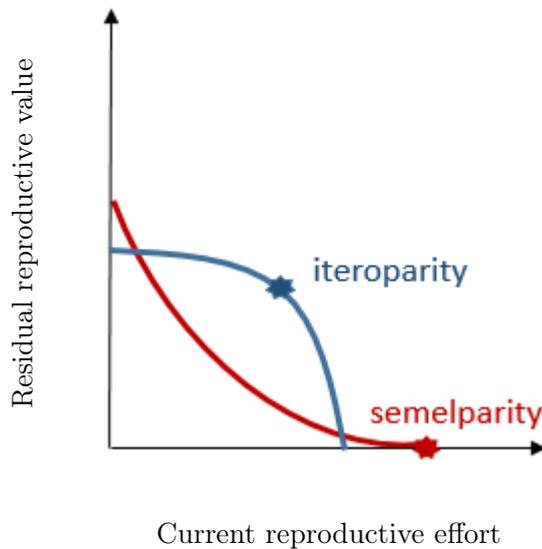


Figure 2.1: Example of hypothetical curves representing the trade-offs between current reproductive effort and expectation of future offspring at any particular instant (or age). Concave curve (in red) leads to only one reproductive event (semelparity), convex one (blue) result in repeated reproduction (iteroparity). Dots mark the reproductive tactics that maximize the lifetime reproductive success. Modified from [Pianka 1976].

In particular, optimality analysis was used to make predictions on the evolution of life histories traits in response to “r-“ and “K-“ environments [Pianka 1976]. Environmental condition would affect the shape of the trade-off between present reproduction and expectation of future reproduction promoting either iteroparity or semelparity (Box.2.1.1). For example, a resource-rich, non-competitive, r-

environment would select for traits that enhance population growth rate, including early maturity, high reproductive effort, high fecundity, and semelparity. Conversely, resource-limited, competitive, K-environments select for traits that enhance persistence of individuals, including delayed maturity, high investment in individual maintenance at the cost of low reproductive effort, low fecundity with a large investment in each offspring, and longer life span. Environments might fluctuate in the extent to which they exert r- or K-selection; and depending on the extent to which organisms experienced either of these environments they fall on a continuum from pure r- to pure K-selection.

2.1.2 Game theory

By contrast, evolutionary game theory allows to consider social environment and interactions between individuals as drivers of selection. Game theory was first developed by mathematicians and economists to solve problems with complex interdependent decision-making [Von Neumann 1992, Nash *et al.* 1950], then used by behavioral ecology with an evolutionary narrative [Smith 1982, Dechaume-moncharmont 2018]. Game theory allows to calculate the best strategy for a player depending on the strategies of others, and therefore accounts for frequency-dependence. In traditional game theory, an equilibrium point is reached when the strategy of the players cannot be strictly outperformed by another strategy (e.g. Nash equilibrium, [Nash *et al.* 1950]). It is assumed that players are aware of the structure of the game and consciously try to predict the moves of their opponents and to maximize their own payoffs.

By contrast, evolutionary game theory propose a different narrative: it presumes that the players' strategies are biologically encoded and heritable. Individual's behavior directly affects its Darwinian fitness, namely, its probability to survive, re-

28 Chapter 2. Several modelling approaches to study sexual selection

produce, and contribute to future generations. The best strategies lead to higher reproductive success, and thus, their frequency increases in the population across generations. On the contrary, the least efficient strategies are progressively washed away by the natural selection. Alternative strategies of the game occasionally occur, via a process like mutation. To be an Evolutionary Stable Strategy (ESS), a strategy must be resistant to these alternatives, that is, no rare mutant strategy can outcompete the resident strategy ¹ [Smith 1982].

In sexual selection, evolutionary game theory has been widely used to predict the level of selectivity (or choosiness) during mate choice. Many models considered that only females choose their mates [Collins & McNamara 1993, Johnstone *et al.* 1996, Kokko *et al.* 2003, Kokko *et al.* 2007, Barry & Kokko 2010, Bleu *et al.* 2012, Etienne *et al.* 2014, Dechaume-Moncharmont *et al.* 2016]. Being choosy would theoretically allow a female to find a high-quality male if she was the only chooser in the population. But if there are other females competing for the same pool of males, the female would be better accepting even a mediocre partner. A greedy female trying to outsmart her non-selective competitors by increasing her choosiness exposes herself to the risk of unsuccessfully searching for her ideal mate, possibly no longer available if less selective females have progressively monopolized all the males [Dechaume-Moncharmont *et al.* 2016]. The average partner quality that a female can expect when following a given decision rule cannot be calculated as an absolute value. It is strongly dependent upon the frequency of the other strategies in the population through the background distribution of the available male's quality. Searching for the optimal choosiness strategy of a given individual thus requires to explicitly consider the behaviors of its competitors.

Considering the possibility that choosiness evolves in both sexes, thereby

¹Every ESS corresponds to a Nash equilibrium, but some Nash equilibria are not ESSes.

giving rise to mutual mate choice, adds a new dimension in the game, wherein individuals have to adjust not only to the choosiness of their same-sex competitors but also to the change in choosiness of the opposite sex [McNamara & Collins 1990, Collins & McNamara 1993, Johnstone 1997, Kokko & Monaghan 2001, Kokko & Johnstone 2002, Alpern & Reyniers 2005, Ramsey 2011, Courtiol *et al.* 2016]. For example, increasing the choosiness of one sex may reduce the mating rate of the opposite sex, forcing it to become less choosy and more competitive [Kokko & Johnstone 2002]. Mate choice is thus the result of a complex dynamic game where each individual tries at any time to maximize its reproductive output by optimizing its choosiness with regard to mate availability (which fluctuates as a function of mating strategies). I am going to develop this example in the following section as I devoted a significant amount of time in my thesis to work on a dynamic game model to understand how much the fluctuations of mate availability - i.e., context-dependence - in different mating systems affect the evolution of choosiness ([Chevalier *et al.* 2020]), Appendix A). My approach allows to represent the feedback between individual's choosiness and the fluctuations of mate availability over the breeding season. This feedback implies that choosiness can potentially be flexible over time. In a nutshell, I show that different mating systems, characterized for instance by contrasted adult sex ratio or differences in latency periods between sexes, can influence the dynamics with regard to the availability of mating partners (both in quantity and quality), making optimal choosiness between sexes, between quality and over time a non-trivial pattern - or at least, extremely context-dependent. An associated yet central benefit of considering quality- and time-dependent choosiness is to bolster interactions between theoretical and empirical research: experiments and most observations in natural environments involve complex situations where individuals interact in a fluctuating

30 Chapter 2. Several modelling approaches to study sexual selection

context, producing choosiness that changes over time, or with the quality or sex of the individual [Byrne & Rice 2006, Labonne *et al.* 2009, Judge *et al.* 2014]. This potentially generates variation in choosiness that is not necessarily captured by the current theoretical models. The strong context-dependency on behaviors appears as an important factor to understand evolution, and should, therefore, be accounted for in predictive models. Part of the developments presented in Chapter 3 were influenced by this observation.

2.1.3 A case study: fluctuations of the mating market affect the evolution of flexible choosiness

The game model

The model is formulated as a dynamic game that describes mating in a population of females and males of variable quality. Both males and females are potentially choosy (i.e. we assume that mutual mate choice is possible), thereby assuming no *a priori* sex-role. All individuals have the possibility to compete for partners of variable quality. As a consequence, their fitness payoffs also depend on the choices made by the other individuals from both sexes in the population. We build a discrete time model with a finite time horizon (i.e., the breeding season) of length T time steps. The choosiness strategy for an individual of quality q is a $Q \times Q \times T$ matrix of acceptance probabilities for every possible partner's quality q' and at each time step t . Mated individuals become unavailable for further mating during their latency period, noted τ_{σ} for males, and τ_{φ} for females. At the end of their latency, they return to the pool of available partners. When τ_{σ} and τ_{φ} are greater than the length of the breeding season, each individual can only expect to mate once during the breeding season, which corresponds to a case of monogamy. Other mating

systems are modeled as follows: polygyny ($\tau_{\sigma} \ll T$, and $\tau_{\varphi} \gg T$), polyandry ($\tau_{\varphi} \ll T$, and $\tau_{\sigma} \gg T$), or polygynandry ($\tau_{\sigma} \ll T$, and $\tau_{\varphi} \ll T$). Contrary to the *ASR*, which is the initial sex-ratio of mature individuals in the population and is defined only at the beginning of the breeding season, the *OSR*(t) is the ratio of available males to available females and changes dynamically during the breeding season. The *OSR*(t) can become increasingly skewed, for instance when the duration of the latency period is shorter in males than in females (available males become more numerous than available females), or when initially the *ASR* is skewed, and individuals from the least common sex mate and are thus removed from the pool of available partners. The *OSR* and the distribution of unpaired individual qualities are therefore emergent properties of the pair formation process, which results from choosiness strategies in the population.

To calculate the Evolutionary Stable Strategy (*ESS*), we used a method of *best response iterations* [Houston & McNamara 1999, Roff 2010]. The core of the optimization algorithm is a two-step process (as illustrated in Fig.2.2). We begin with an arbitrary set of choice strategies $p_{resident\sigma}(q, q', t)$ and $p_{resident\varphi}(q, q', t)$ and, for each time step t during the breeding season, we calculate the distributions of qualities for males ($f_{\sigma}(q, t)$) and females ($f_{\varphi}(q, t)$) which result from this set of strategies (the detailed method is in Appendix A). Assuming these distributions of quality, we then calculate the best response strategies for males and females ($p_{best\sigma}(q, q', t)$ and $p_{best\varphi}(q, q', t)$) as the strategies which maximize the individual's expected payoffs (the detailed method is in Appendix A). The new resident set of strategies to be used in the next iteration is derived from the previous one.

And this new set of strategies is used to calculate the new distributions of partner qualities across the season that, in turn, would lead to new adjustments of the choosiness strategies. We iterate this process until convergence of the sequences of

32 Chapter 2. Several modelling approaches to study sexual selection

choosiness strategies for males and females, such that a male or a female of any quality cannot increase their payoff by changing their choosiness strategy under the current distribution of qualities across the breeding season (*i.e.* ESS).

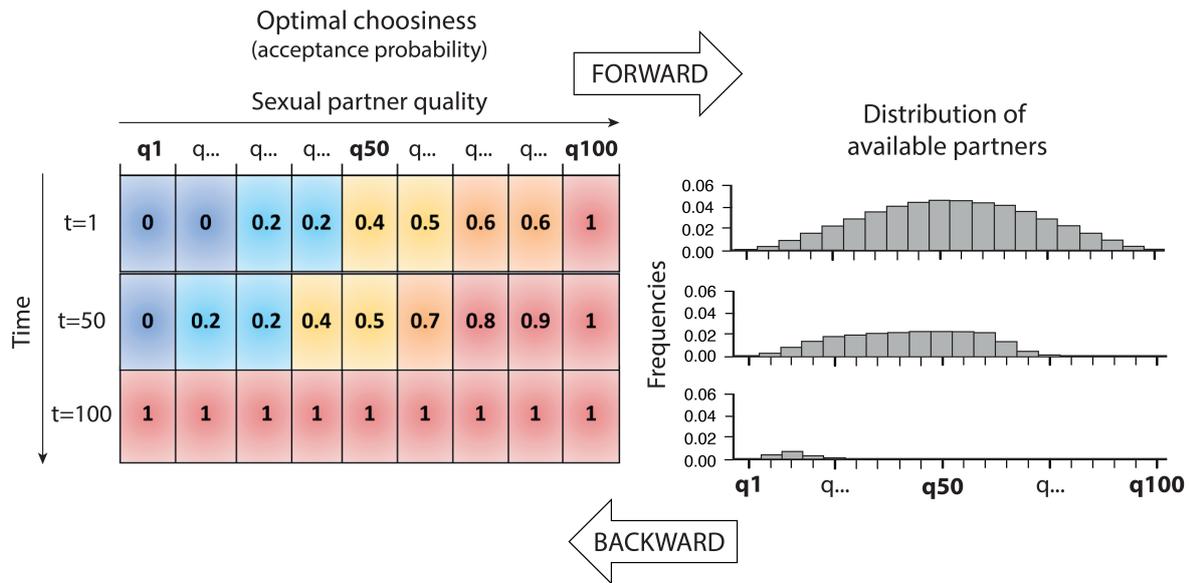


Figure 2.2: An illustration of the optimization approach used to calculate the optimal choosiness. The newly calculated choosiness strategies are used to calculate the new distributions of partner qualities across the season that, in turn, would lead to new adjustments of the choosiness strategies.

Some mains results

Our model reveals some common characteristics between mating systems as we globally observe quality dependence and flexibility in choosiness, controlling for the variance of choosiness. Still, our results are well contrasted between the monogamous system and the polygynandrous system (Fig.2.3). In the former, remaining individuals of higher quality (HQ) constantly adjust their choosiness downward to the dwindling opportunities, while low quality (LQ) individuals are never choosy.

The situation is far more complex in the polygynandrous system, where flexibility is observed for individuals of all qualities, adjusting their choosiness both downward and upward, thereby sustaining variance in choosiness later in the breeding season (Fig.2.3). A polygynous system presents intermediate patterns in terms of flexibility and variance (Fig.2.3). It is also remarkable that in the different mating systems envisioned here, positive assortative mating always occurs (Fig.2.4). Its intensity decreases with the level of unbalance in ASR and in latency periods. In unbalanced mating systems (such as polygynandry), LQ males have almost no mating success, and thus sexual selection against them is strong; but in contrast, sexual selection is relaxed among HQ individuals. Indeed, we observe a particular pattern of apparent assortment wherein the population is divided into random-mating subsets (which result from the evolution of “stepped” choosiness). Interestingly, this pattern emerges through the use of a fine-scale for quality variation. This result is reminiscent of the results obtained by previous analytical models, which found that couples were formed between males and females belonging to the same band (interval) of quality levels in the population [McNamara & Collins 1990, Alpern & Reyniers 2005] under monogamy. [Johnstone *et al.* 1996] also predicted similar apparent assortative mating from a non flexible choosiness strategy throughout the breeding season. On the contrary, our predictions indicate that such clear patterns can be related to quality-dependent flexible choosiness, wherein the population is self-organized into subsets of competitors, especially when mating systems are unbalanced. Note that the number of randomly mating subsets into which the population is divided, and hence the degree of precision of the assortative mating, is higher in monogamous and balanced mating systems (Fig.2.4). In the present approach, where we focus on the intragenerational time scale, the dynamics of the mating market is highly influenced by the type of mating systems, which we here manipulated via ASR and

latency period.

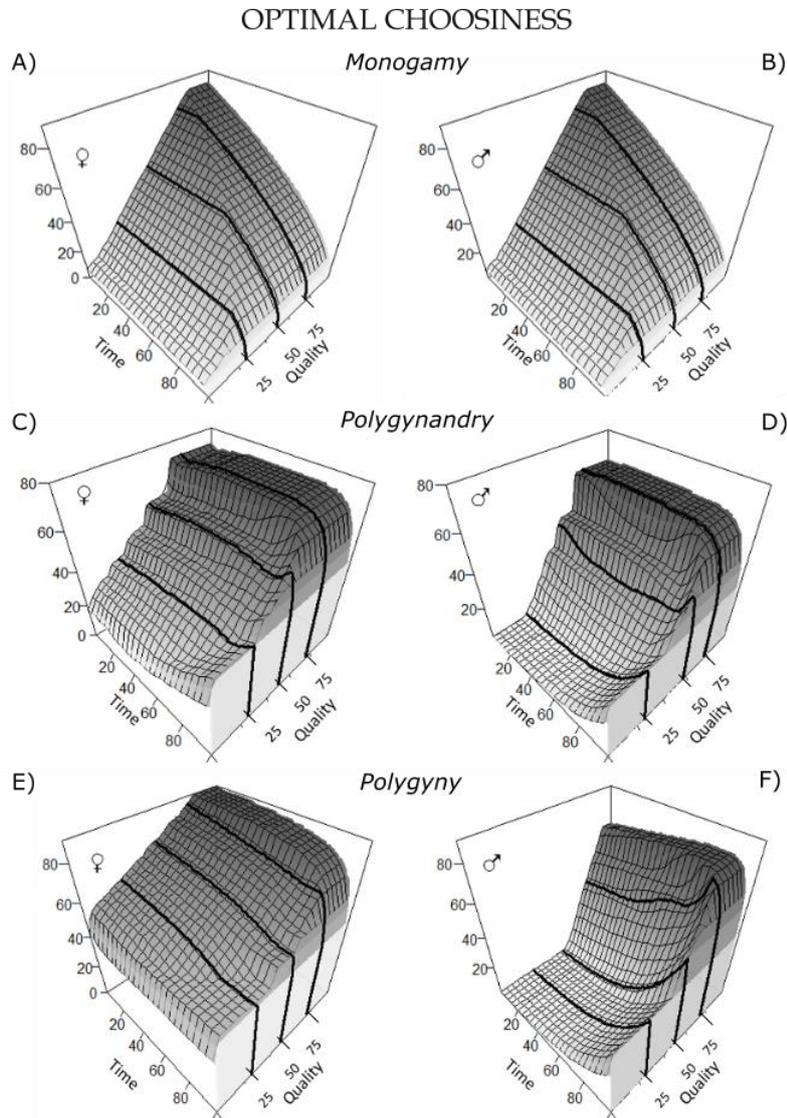


Figure 2.3: Optimal choosiness for females and males as functions of time and individual quality, in three mating systems: monogamy ($\tau_{\sigma} \gg T$, and $\tau_{\varphi} \gg T$), polygynandry ($\tau_{\varphi} \ll T$, and $\tau_{\sigma} \ll T$, polygyny ($\tau_{\sigma} \ll T$, and $\tau_{\varphi} \gg T$)). Optimal choosiness is measured as the partner quality that an individual is ready to accept with at least a probability of 0.5. Thick black lines indicates High Quality (HQ, $q = 75$), Intermediate Quality (IQ, $q = 50$) and Low Quality (LQ, $q = 25$) individuals.

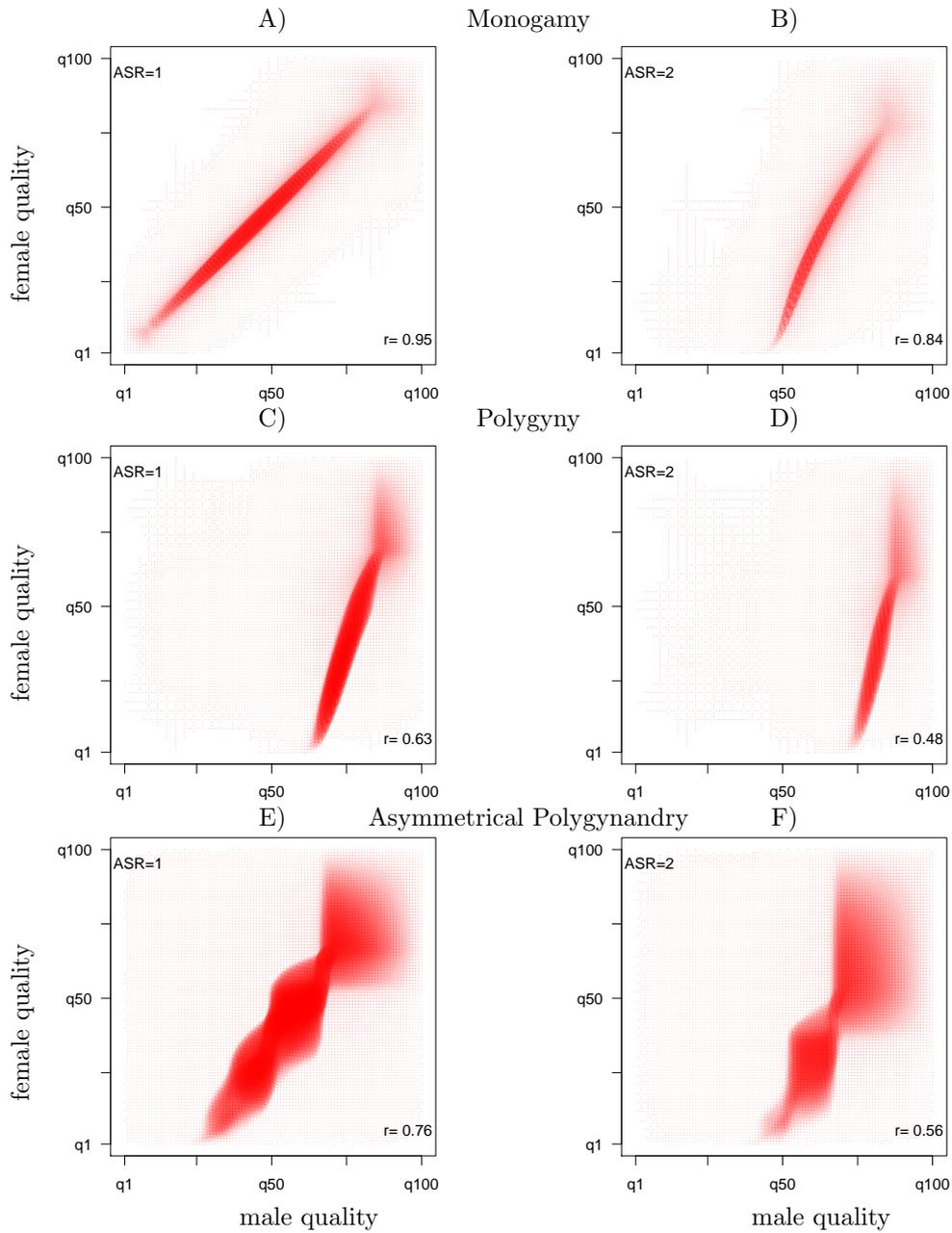


Figure 2.4: Final mating pattern. For a given quality of male and female, the size of the points is proportional to the frequency of pairs formed during the breeding season. r indicates the degree of assortative mating measured as the Pearson coefficient correlation.

Above described optimal methods assume that genetic basis of traits does not affect their evolution (an assumption acknowledge as the "*Phenotypic gambit*" by [Grafen 1984]), and do not question the evolutionary trajectory nor the genetic mechanisms of evolution. Yet, as shown below, the genetic basis of traits are likely to affect their evolution.

2.2 Genetic models

2.2.1 Invasibility analysis

Invasibility analysis is often regarded as a phenotypic approach because it ignores the importance of genes and sex. However, it offers a narrative and hypotheses about the genetic mechanisms of evolution. In particular, mutations are assumed to occur on a time scale which is long relative to the time scale of convergence to an ecological attractor, and reproduction is clonal.

The goal of this approach is to assess whether a clone displaying an alternate life history (the mutant) can invade a resident population [Dieckmann & Ferriere 2004, Roff 2010]. This is estimated by calculating the growth rate of the invader in the population. The rationale is that the fittest phenotype is one that cannot be invaded. This criterion, although more complicated to use, has the advantage that it is robust in scenarios where density dependence, frequency dependence, or environmental stochasticity are important. For instance, [Benton & Grant 1999] simulated the effects of stochasticity and density dependence of fecundity and survival on the evolution of reproductive effort. In their model, fitness is density dependent and population fluctuates (in size and distribution in age classes). Their results indicate that iteroparity is almost always the ESA strategy under stochasticity. When density acts on fecundities, the net result is that the fecundities are more variable than

survivorships. Likewise, when density acts on survivorships, the result is that survivorships become more variable than fecundities. Simple application of bet-hedging models would suggest that in the former case iteroparity should be selected for and in the latter case semelparity. But according to the result iteroparity would evolve in both cases.

Optimal reproductive effort in stochastic, density-dependent environments. Benton and Grant (1999)

Question: What is the effect of both stochastic and density dependent environments on the evolution of optimal reproductive effort?

Optimization criteria: the invasion exponent which estimates the growth rate of the invader population. It allows to find the Evolutionary Stable Attractor (ESA) of the system.

Mechanism: (1) Trade-off between current fecundity and survival (different shapes of trade-off are investigated). (2) Stochastic and density dependent variation in the fecundity and/or survival.

Procedure: A resident population is simulated using a matrix population model until the population dynamics have stabilized on the attractor. An invader with the same life history (i.e. shape of trade-off between reproduction and survival) as the resident, but with higher reproductive effort and so lower survival is then introduced. Invaders does not contribute to the density dependence. The sign of the invasion exponent then measures whether the invader

manages to invade, and the magnitude of the partial differential between the invasion exponent and reproductive effort is a measure of the sensitivity of the invasion exponent to change in reproductive effort (i.e elasticity), and thus a measure of the selection pressure on reproductive effort. The ESA of the system for reproductive effort is reached when the elasticity equal 0.

Results: Iteroparity is almost always the ESA strategy under stochasticity: "extreme" life histories (those with either very low or very high reproductive efforts) tend to become less extreme under selection in a variable environment.

2.2.2 Quantitative genetics

Quantitative genetics adopt a statistical view of the genome to summarize its complexity. Fisher proposed that the complex genetic basis of quantitative traits can be simply represented by the sum of small allelic effects (assuming that epistatic effects on the genome compensate each other). The classic quantitative genetics approach (i.e. populational variance components approach, [?]) focuses at the mean, the variance and covariance of traits in the population. The mean phenotypic trait value in the population and the variance are decomposed into the sum of genetic and environmental components. Both components are assumed normally distributed, the former with a mean varying as a result of selection and drift and the latter with a zero mean. This approach can be extended to multiple traits, in that case, variance and covariance between traits are components of the "G-matrix" which describes the statistical dispersion of genetic values. The change in the mean phenotypic values due to selection across generations are calculated based on the "multivariate breeders' equation" [Lande & Arnold 1983] (Fig.2.5).

$$\Delta\bar{\mathbf{Z}} = \mathbf{G}\boldsymbol{\beta}$$

$$\begin{bmatrix} \Delta\bar{Z}_1 \\ \Delta\bar{Z}_2 \\ \Delta\bar{Z}_3 \end{bmatrix} = \begin{bmatrix} G_{11} & G_{12} & G_{13} \\ G_{21} & G_{22} & G_{23} \\ G_{31} & G_{32} & G_{33} \end{bmatrix} \begin{bmatrix} \beta_1 \\ \beta_2 \\ \beta_3 \end{bmatrix}$$

Vector of evolutionary change in individuals traits
Genetic variance-covariance matrix (G-matrix)
Vector of directional selection gradients

Figure 2.5: The multivariate breeder's equation.

In models interested in sexual selection, selection differential can be calculated as the sum of natural and sexual selection. Natural selection corresponds to the viability of phenotypic values (the adaptive optimum is thus a priori defined), and sexual selection corresponds to the mating success of phenotypic values.

The Lande model (1981) shows, for example, that the variance-covariance matrix between ornament and preference influences the stability of the system. Depending on the ratio between the genetic variance of the ornament and its genetic covariance with the preference, the equilibrium line can be stable or unstable (Fig.2.6); this can strongly influence the dynamics of the system since in the case of an unstable equilibrium the ornament and the preference will evolve together very quickly away from the equilibrium point (as predicted by [Fisher 1915]). Interestingly, adding some realistic features of behaviour and pairing processes can also change the equilibrium, for instance preference-dependent risk that females remain unmated would prevent instable equilibrium to occurs [De Jong & Sabelis 1991].

Darwin-Fisher-Lande model of sexual selection by female choice.
Lande 1981.

Question: What are the combinations of male phenotype and female preference such as the intensity of female sexual preference cancels the force of natural selection on male phenotype?

Mechanism: The maximization criteria is the mean fitness of males in the population. Fitness of males with a phenotype z is the product of their viability (stabilizing natural selection on z) and mating success averaged over the entire female population. The female preferences are not directly selected; they evolve indirectly through the selection pressures on the male trait to which they are genetically correlated.

Main assumptions: All females fully express their preference. Male trait and female preference are sex limited. The genetic variance and covariance remain constant : a loss of variance due to selection is supposed balanced by the input of polygenic mutation and recombination.

Result: For any mean value of the male character there is an average intensity of female sexual preference that will cancel the force of natural selection tending to restore the mean male phenotype to its optimum. The population will evolve along trajectories specified by the genetic covariance between male character and female preference. Genetic covariance arises between male and female characters as a consequence of assortative mating and heritable variation in the two characters. Interestingly, this work demonstrates that the structure

of the G-matrix can profoundly affect the evolutionary outcome as it can trigger stable or unstable equilibrium.

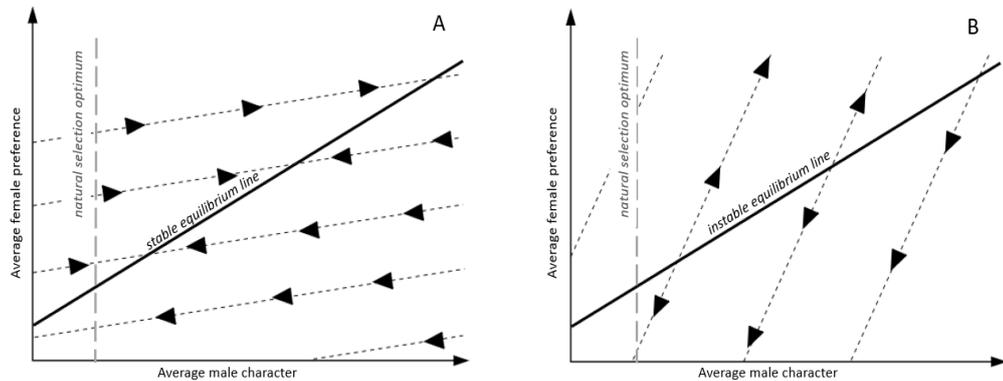


Figure 2.6: The joint evolution of female preferences and male trait. Male trait is subject to an ordinary stabilizing natural selection and to sexual selection. The population will evolve along the dotted lines (following the direction of the arrows). Depending on the genetic variance parameters, the equilibrium line (solid line) can be stable (A) or unstable (B). Unstable equilibrium is triggered when the genetic covariance exceeds a critical value determined by the intensity of female preference and the strength of stabilizing natural selection on male phenotype [Lande 1981, Arnold 1983].

The basic quantitative genetic model captures the influence of genetics through G-matrix which is supposed to be stable (i.e. no change in genetic variance and covariance). The stability of the G-matrix is justified in the case of mutation-selection balance and multivariate Gaussian distribution of allelic effects (i.e. mutational effects are much larger than the standing genetic variation). This allows to show that the genetic variance and covariance of traits affect their evolution [Lande 1981, Iwasa & Pomiankowski 1995].

42 Chapter 2. Several modelling approaches to study sexual selection

However, the genetic variance and covariance of traits could evolve in response to selection because of changes in genetic architecture (e.g. allele frequencies and/or the build up of linkage disequilibrium among important loci [Steppan *et al.* 2002, Careau *et al.* 2015]); and this may either facilitate or constrain future evolutionary changes [Steppan *et al.* 2002, Hansen 2003, Hansen 2006, Walsh & Blows 2009]. Additionally, physical structure of the genome is likely to affect the evolution of such genetic characteristics (number of loci involved, allelic pleiotropy, linkage disequilibrium; [Hansen 2006], e.g. [Servedio & Lande 2006, Gavrillets *et al.* 2007, Labonne & Hendry 2010]).

More explicit genetic models are therefore necessary to account for the physical structure of the genome and for possible changes in the genetic architecture of the population [Arnold *et al.* 2008, Nelson *et al.* 2013].

2.2.3 Population genetic models

Population genetics models simulate the evolution of gene frequencies at the expense of ecological detail. An explicit Mendelian model is used. In the simplest case this would be a single locus with two alleles. Complexity can be introduced first by the addition of more alleles and second by the addition of more loci. Often these models are used to make predictions about the change in frequency of certain alleles that code for a trait. Some models also use the population genetics approach to represent certain allele properties (e.g. pleiotropy, epistasis) and understand how they may have evolved. For instance, [Guillaume & Otto 2012] follow the evolution of a modifier loci, carrying alleles that change the allocation of one or two genes activity for two functions. They predict that pleiotropy is favored in situations where gene product can perform well at the two functions.

2.2.4 Demogenetic individual based models

Previous modelling approaches are either interested in how the phenotype is shaped by the interactions between individuals and their environment (phenotypic approaches) or consider the effect of genetic constraints on evolution (genetic approaches). But feedbacks between phenotypic and genetic processes are less addressed [Govaert *et al.* 2019, Svensson 2019].

The first interest of demogenetic (also termed ecogenetic) Agent Based Models (ABM) is to explicitly represent the relationship between life-history traits (or behaviour) and fitness to understand the building up of fitness in natural populations, rather than assuming a fixed relation between life-history traits and fitness. Indeed, the evolution of life-history traits is tightly linked to population dynamics and to social environment, as the adaptive advantage of those traits depends on social environment and on demography and as the evolution of those traits in turn impact the social environment and the demography.

The second interest of demogenetic ABM is to explicitly represent change in genetic architecture and its effect on trait evolution. Indeed, the significance of feedback between genetic changes and ecological changes (behaviours and demography) for sexual selection has recently been highlighted [Govaert *et al.* 2019, Svensson 2019]. To state an example, in guppies, sexual selection tends to limit local adaptation by maintaining unadapted gene flow, since females do not necessarily prefer adaptive colors in males (i.e., they can choose conspicuous males even when predators are present) [Labonne & Hendry 2010]. This preference, although not adaptive, could be maintained by i/ indirect selection over preference going against natural selection (females make sexier sons by mating with conspicuous males), ii/ the genetic architecture of the color trait which is encoded by genes on the Y chro-

44 Chapter 2. Several modelling approaches to study sexual selection

mosomes and on the autosomes, so that females carry genes for preference and for color (but without expressing them and therefore without predation cost).

Two types of approaches are commonly used to represent the genetic basis of traits in demogenetic ABMs: the quantitative approach and the allelic approach.

Quantitative genetics approach: Most of models adapt the quantitative genetic framework to an individual based approach, notably to realistically represent the genetic and environmental components of the phenotype [Hendry 2020, Oddou-Muratorio *et al.* 2020]. The genetic and environmental variance–covariance matrices are defined. Then, individuals that satisfy the basic quantitative genetic assumptions are generated by randomly picking the phenotypic values from a normal distribution with a given mean and variance. The fitness of individuals is tracked - often under the assumption of random association of gametes and no sex-difference- and changes in both means and the phenotypic distributions can be assessed.

Quantitative genetics approaches are not interested by how the allelic effect are distributed over the genome. Yet, the arrangement of genetic values in the genome can impact the evolution beyond what can be predicted by the G-matrix. Indeed, the association of allelic values for the different traits potentially influences the number of combinations to produce given values of traits, and thus influences the conjoint transmission of traits and the variance of genotypes in the population. Additionally, the observed variability in the pattern of genetic architecture (pleiotropy, polygeny) suggest that genetic architecture can be shaped by selection pressures [Hansen 2003, Guillaume & Otto 2012].

Allelic approach: Recently, few ABMs representing genetic architecture have been developed: Easypop [Balloux 2001], SimuPop [Peng & Kimmel 2005], Nemo

[Guillaume & Rougemont 2006], QuantiNemo [Neuenschwander *et al.* 2008]. Only the last three can handle dynamical demographics and traits under selection while the others are designed as programs to generate genetic datasets in populations of constant size. In all these models, the selective pressure on traits is *a priori* defined and therefore does not emerge from inter-individual interactions, and from trade-off between traits. For the simplest case of a strictly additive model, individuals are explicitly modeled with their trait values determined from the sum of the allelic values plus a normally distributed environmental value with a mean of zero and a variance necessary to generate the required heritability. This approach can also allow to consider the possibility of linkage and epistasis. Epistasis can be incorporated in simple models (e.g., two locus models) but can become difficult to incorporate in models with many loci and alleles in absence of known statistical distribution or physical prediction.

2.3 Discussion

As stated before, each model carries its sets of assumptions. This delineates its potential for application to empirical research as well as to theoretical investigations. I synthesized various characteristics of the different modelling approaches in Tables 2.1 and 2.2. What is striking is that generally the different approaches do not allow to look both at the evolution of traits in a dynamic context (where the advantage of a trait depends on the environment and on the other individuals' traits) and at the evolution of the genetic architecture with a functional vision of it (with the idea that some genetic architectures are advantageous and may be selected).

After having reviewed the different existing models in sexual selection, it seems that (1) to understand the evolution of behaviors it is necessary to represent the

46 Chapter 2. Several modelling approaches to study sexual selection

interactions between individuals and context dependence (as inspired by game theory); (2) to understand the transmission of these behaviors it is also important to take into account the demography of the population and genetic constraints (e.g. covariance between traits, variance); (3) and that genetic constraints are influenced by the genetic architecture which may itself evolve and which could be represented more explicitly. A demogenetic agent-based model is a suitable approach to tackle this challenge, as it allows to explicitly represent behavioral interactions between individuals during reproduction, wherein behavioural decisions can be at least partly determined by individual genome, making this genome effectively under selection, and thus likely to evolve, too. Figure 2.7 summarizes the integrative approach I want to develop, with the different components (demography, traits, genome) and their interactions.

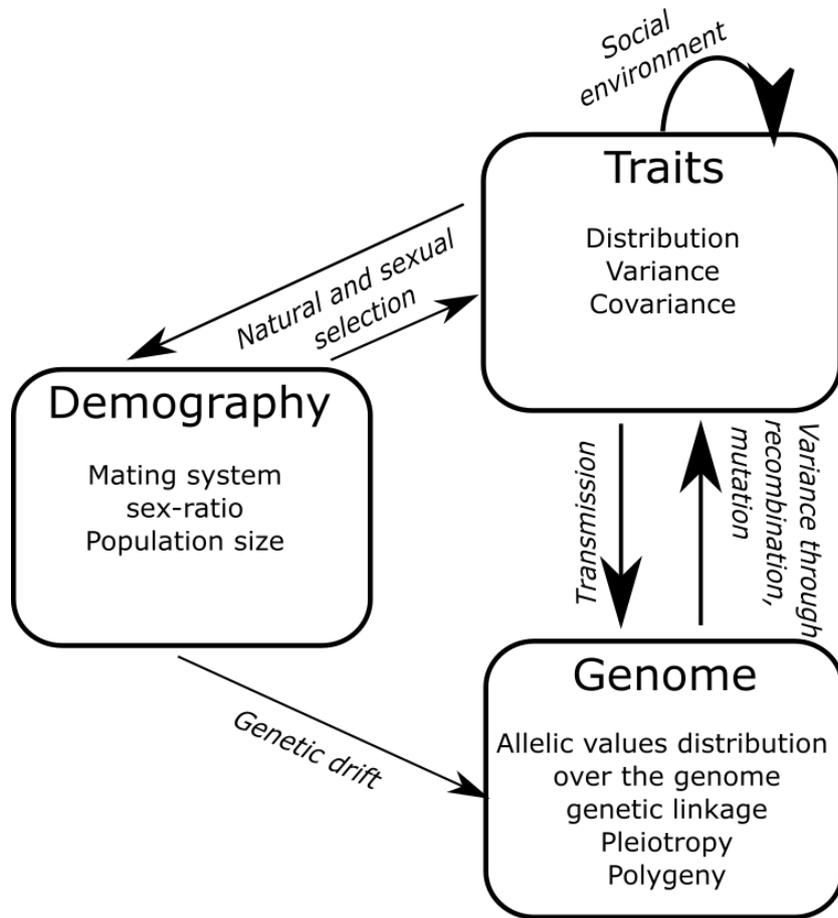


Figure 2.7: The demogenetic conceptual model, integrating population dynamic, behavioral interactions and the evolution of genetic architecture.

	OPTIMAL ANALYSIS	GAME THEORY	ADAPTIVE DYNAMIC	CLASSIC QUANTITATIVE GENE MODELS	POPULATION GENETIC	DEMO GENETIC	
PHENOTYPIC ASSUMPTIONS	Optimum	Fixed Optimum with fixed relation between life history trait and fitness	Optimum with frequency-dependent selection, calculate the ESS	Invasion fitness frequency of a mutant in a population of monomorphic residents	Fixed Optimum with fixed relation between life history trait and fitness	Change in allelic frequency	Emerging optimum: fitness emerge as a consequence of the underlying population dynamics, include frequency-dependent selection
	Outcome	Focus on equilibrium conditions	Focus on equilibrium conditions	Evolutionary trajectory	Focus on equilibrium conditions	Evolutionary trajectory	Evolutionary trajectory
	Demography	Demographic equilibrium (in term of population size and age structure)	Demographic equilibrium (in term of population size and age structure)	Demographic equilibrium (in term of population size and age structure)	Demographic equilibrium (in term of population size and age structure)	Demographic equilibrium (infinite population size)	Demographic changes (e.g. population size, age distribution, migration)
	Transmission	No representation of genetic transmission		Clonal reproduction	Implicit representation of mating process (e.g. mating preferences affect the relation between fitness and phenotype)	Implicit representation of mating process (e.g. mating preferences affect the relation between fitness and phenotype)	Explicit representation of mating processes (rather than approximated by a fitness formula)

Table 2.1: Summary of the main phenotypic assumptions of various evolutionary modelling approaches.

	OPTIMAL ANALYSIS	GAME THEORY	ADAPTIVE DYNAMIC	CLASSIC QUANTITATIVE GENE MODELS	POPULATION GENETIC	DEMO GENETIC
GENETIC ASSUMPTIONS	No genetic assumptions		No genetic representation	Statistical representation of the genome (G-matrix). Infinitesimal number of alleles with small additive effect	Simple explicit representation of the genome (e.g. one bi-allelic locus)	explicit genetic map
				No change in the distribution of allelic effects (Gaussian distribution)	Change in allelic frequencies	Change in allelic frequencies
			rare mutations (a rare mutant invade the population, no impact on resident fitness and demography)	Equilibrium between genetic drift, mutations and selection (i.e. weak selection).	no mutation or genetic drift (no allele loss/gain).	No equilibrium assumption: specified mutation rate, variable genetic drift (depending on effective population size), possible variable selection

Table 2.2: Summary of the main genetic assumptions of various evolutionary modelling approaches.

Modelling mating behaviours and genetic architecture

Contents

3.1	Conception of a demogenetic simulation model	53
3.1.1	An overview of the demogenetic agent based model	53
3.1.2	Detailed model description	55
3.1.3	General concepts underlying the design of the model	65
3.2	Exploring the evolutionary dynamic of a particular simu- lation: coevolution between Gametic investment and Pref- erence	67
3.2.1	Evolution of reproductive traits:	68
3.2.2	Demographic consequences	71
3.2.3	Evolution of genetic architecture	73
3.3	Effect of mating systems on traits and genetic architecture evolution	77
3.3.1	Evolution of the reproductive traits:	77
3.3.2	Genetic architecture evolution:	82
3.4	The sensibility of parameters change	85
3.4.1	Sensibility to reproductive trade-off:	85

3.4.2 Genetic parameters:	86
3.4.3 Shape of the preference function:	87
3.5 Discussion	92

In the light of Chapter 2 conclusions, we here propose a DG-ABM (DemoGenetic Agent Based Model) that includes most of the ingredients required to study the evolution of mating behaviors and genetic architecture under sexual selection. The present description of my modelling approach covers the principles, the details, the assumptions and their justifications. I will also produce some simple simulation results in order to illustrate the potential of the model and its mechanisms. It is noteworthy that this modelling approach is coded as a software, which is available at <https://doi.org/10.15454/6NFGZ9>, and will continue to be developed further on by my lab. While the model itself could be transferred on any software solution, the current software allows an easy manipulation of the model in a rather user friendly context, with user defined simulation choices, and a more austere script mode for more intensive calculation if needed. During my PhD, my contribution to improve the model, which already existed in a less developed state, took place on two levels: Part of my work focused on the theoretical formulation of the model and the choice of the hypotheses formulated, notably to represent the survival-reproduction trade-off, mating behaviors, and genetic architecture. Another part of my work was to improve the model code, in particular, to code mating routines, to add sexual chromosomes in the genome and to calculate and record index to follow the evolution of traits and genetic architecture (evolution rate in haldanes, lifetime reproductive success, allelic correlations etc..).

3.1 Conception of a demogenetic simulation model

3.1.1 An overview of the demogenetic agent based model

We here give an extensive description of the model in the spirit of the ODD protocol [Grimm *et al.* 2006]. The model can be downloaded at <https://forgemia.inra.fr/louise.chevalier/runaway>

Purpose: The purpose of the model is to investigate the co-evolution between reproductive traits under sexual selection and their genetic architecture, taking into account mating behaviour, genetic and demographic characteristics of the population.

State variables and scales: This individual-based model uses a discrete temporal scale and is not spatially explicit. The time horizon of the model is in the order of a thousand generations. Three levels are considered: the gene level, the individual level, and the population level. At the gene level, genes are characterized by their alleles, which code for various genetic values depending on the number of traits simulated. They are also characterized by their position in the genome and their recombination probabilities with other genes. Such a position is invariant during the simulation because we do not wish to simulate the physical evolution of the genome. Individuals are characterized by the following state variables: identity number, sex, age, lifetime reproductive success and the genetic values of three traits: the gametic investment G , the preference P and the competitiveness C . The population level is characterized by the following state variables: population size, allelic diversity, mean allelic values and standard deviations for the two traits on each gene, mean and standard deviation of traits values and mean lifetime reproductive success. Note that the population level can be subdivided during reproduction, to create smaller

mating groups and mimic spatial or temporal isolation during the breeding season.

Process overview and scheduling: The process for simulation is described below and can be seen in Appendix C1. The model proceeds in generational time steps. Within each generation (or time step), four procedures are processed in the following order: survival, mate choice, reproduction, genetic mutations. Within the survival procedure, the default probability of surviving for an individual depends on population size (density dependence) and on its reproductive effort (which is the sum of the genetic values for costly traits). Within the mate choice procedure, mating groups of a user-specified size are formed by drawing lots of individuals from the population, then individuals from the same mating group encounter each other (randomly or according to their values of competitiveness, sequential encounter, [Gimelfarb 1988]), and they choose to mate or not with the encountered partner. If individuals make up a couple, they will become unavailable to mate again for the present time step. Within the reproduction procedure, each parent produces gametes and the offspring are created by the random fusion of the parent gametes, their sex is randomly assigned. Within the genetic mutation procedure, each allele might be substituted by a new one. New alleles are assumed to be created by mutation, and their genetic values for each trait are drawn in independent Beta distributions by default, whose shape parameters are defined at the initialization stage (paragraph *Initialization*). The user-defined mutation rate is assumed to be constant throughout the genome. Because survival (S) partly depends on reproductive effort, it can therefore also evolve, allowing individuals to potentially participate in more than one reproduction (i.e., iteroparity evolution).

3.1.2 Detailed model description

Survival: The probability of surviving to the next reproduction event is determined as follows:

$$S = \frac{1}{(1 + R)(1 + \frac{N}{K})} \quad (3.1)$$

where N is the population size, K an indicator of resource limitation in the environment (akin, but not equal, to carrying capacity), R the sum of the genetic values of costly traits of the individual. Eq. 3.1 states that individual survival results from the interaction between two components. The first component is demographic ($\frac{1}{1+N/K}$), this formula corresponds to the survival rate of the population in considering that survival is solely density-dependent. The second component is individual ($\frac{1}{1+R}$), this formula states that individual survival is maximum when energy invested into reproduction is null, and that S decreases when R increases. Intuitively, if $N/K = 1$ (close to a demographic equilibrium) and if $R = 1$, then both components affect the survival equally. If the population size drops below K but $R = 1$, then survival rate will increase, making R a relatively greater contributor to survival (see Fig. 3.1). As a consequence, the model can evolve towards semelparity (low survival, single reproduction) or iteroparity (higher survival leading to potential multiple reproductions).

Mating behaviour: During reproduction, the population is first divided into mating groups of user-specified size MG . Population sex ratio is 1 : 1 but the sex ratio within each group can vary, due to random sampling. Then, within each mating group, mating processes occur, individuals mating only once per time step. The model can represent different mating systems:

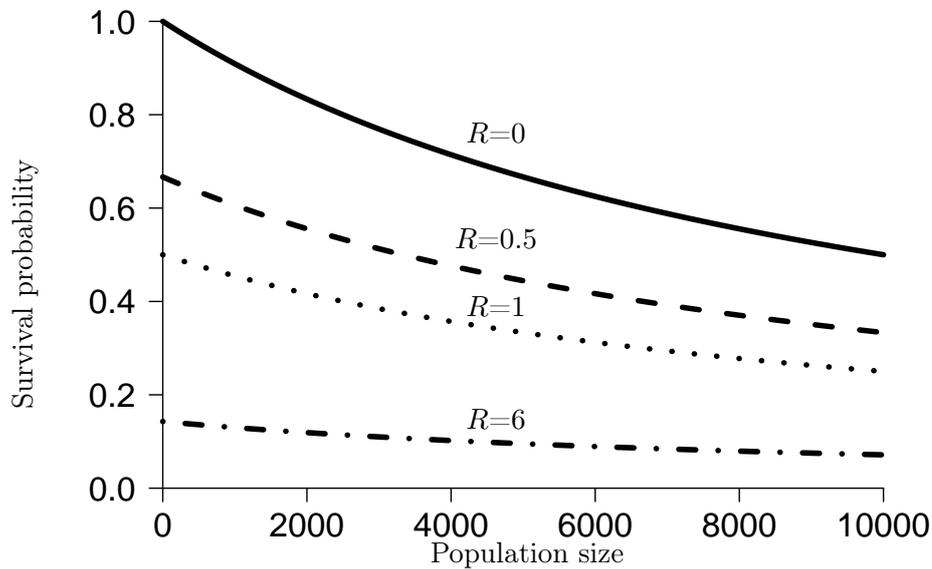


Figure 3.1: survival probability as a function of population size for different values of R , with resource limitation parameter $K=10000$.

(1) **random mating:** pairs of individuals from the same mating group but with different sex are formed randomly. Once a pair is formed, mating will occur. Mated individuals are not available for further mating for the current time step. Note that if MG is not a pair number, or if all partners of one sex have mated, some individuals will remain unmated for the current time step.

(2) **random encounter with preference:** pairs of individuals from the same mating group but of the opposite sex are randomly drawn, but each individual chooses to accept the potential partner based on its preference trait. If both partners mutually accept each other, they become unavailable for further mating for the current time step. If at least one individual rejects the mating, both of them return in the mating group and yet again two individuals are randomly drawn from the

mating group.

(3) competitive encounter without preference: Individuals from the same mating group but with opposite sex encounter each other based on their value of competitiveness C . For each pair of individuals thus formed, mating will occur. Mated individuals are not available for further mating for the current time step. When M is not a pair number, or if one sex is no more available, the less competitive individuals of the mating group will remain unmated.

(4) competitive encounter with preference: Here again, individuals from the same mating group but with opposite sex encounter each other based on their value of competitiveness C . Within each pair thus formed, each individual chooses to accept the potential partner based on its own preference trait. If both partners mutually accept each other, they become unavailable for further mating for the current time step. If at least one individual rejects the mating, both of them return in the mating group and yet again two individuals are randomly drawn from the mating group.

Note that for each of these scenarios, the whole process is sequential: if a first pair is formed and both individuals mate, then the routine proceeds to the next pair. However, if the first pair is formed but mating does not occur, then it will affect the subsequent pair formation and potential related matings.

Preference model: The probability that an individual will accept the mating (Pm) is a function of his genetic value of Preference and his partner's Gametic investment (Eq.3.2).

$$Pm = \exp^{-\frac{(G_{pref}-G)^2}{2\nu^2}} \quad (3.2)$$

This equation indicates that individuals have a unimodal preference, i.e. they

prefer a particular value of the gametic investment (G_{pref}) with a tolerance around this value (ν). The closer the gametic investment of the partner met is to the individual's preferred value, the higher the probability that the individual will accept the mating (Fig. 3.2). The parameter ν is by default set to 0.2 (so that the probability to accept an encountered partner for an individual with a preference peak of 0.5 is equal to 0.5). The shape of the preference function can be changed in the model, and its effect on the evolution is investigated further on (section 3.4.3).

Another possible shape of preference supposes that individual reaction is always stronger for higher values of G (directional preference, Lande 1981). In that case the genetically determined value of preference corresponds to a threshold of G above which partners are accepted with a probability higher than 0.5 and the probability Pm that an individual with a given preference G_{pref} will accept the mating is a sigmoid function of the encountered partner's Gametic investment G (Eq.3.3). α and β are parameters to adjust the slope of the sigmoid function and their default values are 10 and 5 respectively.

$$Pm = \frac{\exp[G_{pref} * G * \alpha - G_{pref} * \beta]}{1 + (\exp[G_{pref} * G * \alpha - G_{pref} * \beta])} \quad (3.3)$$

Additionally, unimodals and directional preferences could also be either an absolute intrinsic property of individuals or could be relative and scaled to the distribution of phenotypes in a population.

Competition model: Within a mating group of size M , the competitiveness C will be used to assess a non-random order of meeting between pairs of individuals. We assume that the probability of meeting between two individuals is dependent on their respective competitiveness trait values. For instance, two individuals with high competitiveness values will probably meet first within the mating group.

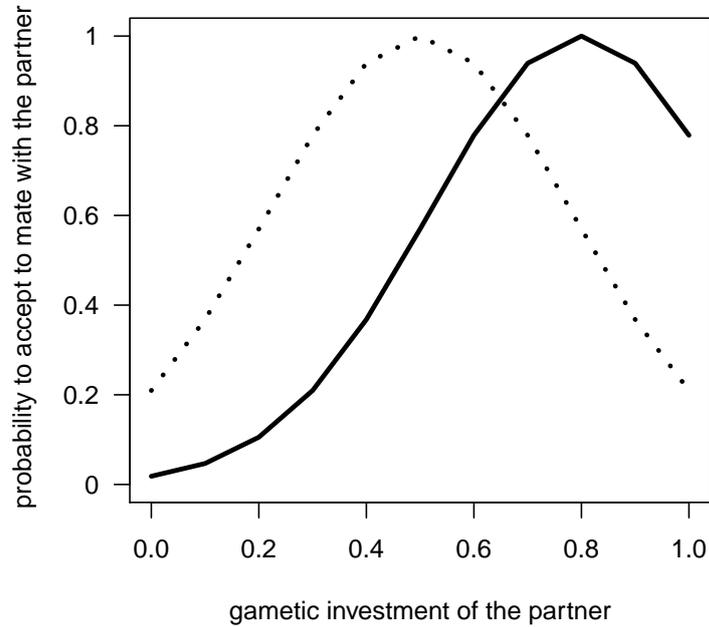


Figure 3.2: Probability that a potential partner will be accepted, as a function of the values of gametic investment of this partner for two different values of preference ($G_{pref} = 0.5$ dotted line, and $G_{pref} = 0.8$ plain line). Individuals differ in the value of G of their most preferred mate, but all individuals share the same tolerance ($\nu = 0.2$).

Then come pairs of individuals with contrasted competitiveness (one high, one low). Finally, two individuals with low competitiveness will meet at the end of the process, mostly. Pairs of individuals are thus ordered according to the product of their competitiveness. Note that if the meeting is not followed by a mating, each individual will be still available for mating. When individuals have mated once however, they are not available for further mating for the current time step and are removed from the list. Consequently, the less competitive individuals may miss reproduction if all opposite-sex partners in the mating group have already mated.

Reproduction: The gametes are composed of randomly chosen strands from each pair of homologous chromosomes. Recombination between successive genes may occur during the meiosis (depending on the values of recombination probabilities between genes). These probabilities are gathered in a table consisting of $n - 1$ lines, n being the number of genes studied. Each line i contains a number between 0 and 0.5 which represents the probability of recombination between the i^{th} gene and the $(i + 1)^{th}$ gene. The number of offspring per couple is set to the average value of the parents' gametic investments multiplied by a demographic constant. The sex of the offspring is randomly attributed.

Genetic mutation: Right after fecundation, mutations might occur on the offspring genome. The rate of mutation is the probability each allele has to be substituted by a new one. Alleles mutation probability is user-defined, but the default value is set to 10^{-4} mutations per loci, which is within the range of spontaneous mutation rates estimated at particular genes in different organisms [Haldane 1935, Muller 1928, Eyre-Walker & Keightley 1999].

Physical structure of the genome: All individuals share the same physical architecture of the genome, which is defined by the number of autosomes, the presence or absence of a sex chromosome, the number of genes, their location on the chromosomes and the probabilities of recombination between these genes. Each individual inherits two alleles for each gene (i.e. diploid). Alleles effects are described by continuous values and are additive within and between loci, i.e. dominance effects are not considered, such as the value of a trait corresponds to the sum of allelic values at every locus:

$$T_g = \sum_{l=1}^{l=n} (l_T^{a1} + l_T^{a2}) \quad (3.4)$$

with n the number of loci ($n = 100$), l_T^{a1} and l_T^{a2} values of the first and the second allele for the trait at the loci l . Individual phenotype is fully determined by the genotype, that is, environmental effects on phenotype are neglected, and heritability is thus assumed to be unity. We define the landscape of allelic values for preference, gametic investment, and competitiveness by Beta distributions of user-specified shape parameters. Default values are $[0.65, 24.5]$, resulting in a right-skewed distribution such as many alleles will have small values for the traits but still, some alleles will have relatively high values for the traits (Fig. 3.3A). As a consequence, under this set of parameters and using 10 loci, initial trait values for gametic investment followed the distribution showed in Fig. 3.3B with mean 0.5 and variance 0.02.

Genetic architecture: The genetic architecture is here defined as the distribution of allelic effects along the genome, and can, therefore, vary between individuals. According to universal pleiotropy assumption [Fisher 1930, Hill & Zhang 2012, Paaby & Rockman 2013], each allele at each locus is specified by a vector of contribution to every trait. For instance in our case, in a two traits model with gametic investment and preference, each allele has two values, one for each trait. Alleles can, therefore, have a pleiotropic effect (if they have high values for both of the traits) or a modular effect (if the allelic value for one of the two traits is close to zero). Thus, an individual's genetic architecture can be in average pleiotropic if he carries alleles that have more similar effects for the different traits than they would have by chance, and conversely, an individual's genetic architecture can be in average modular if he carries alleles that have more different effects for the different traits than randomly expected.

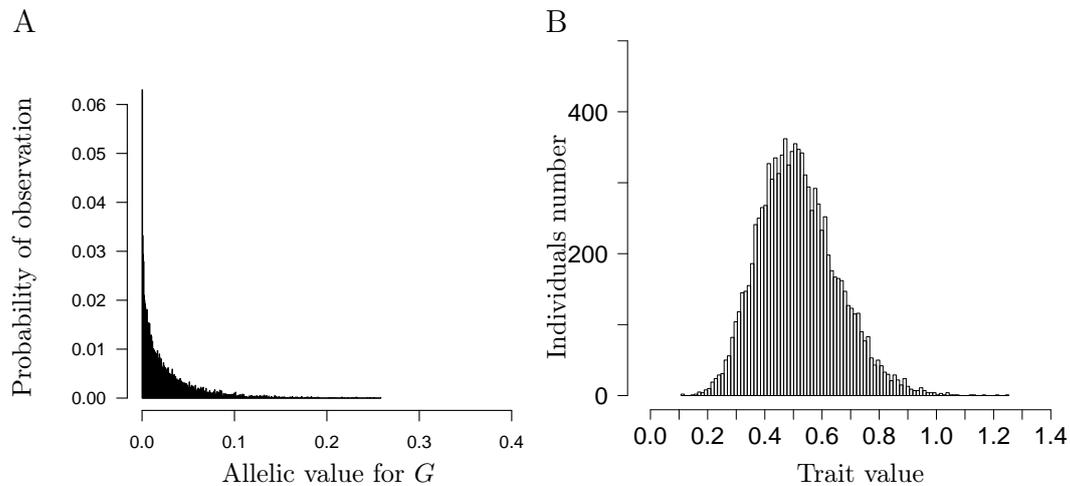


Figure 3.3: Example of allelic values distribution for the gametic investment (A) and consequent distribution of gametic investment trait values in the population (B). This distribution is obtained by drawing allelic values on the Beta distribution of shape parameters [0.65, 24.5].

Genetic architecture evolution is assessed by recording the distribution of allelic values at each locus on average in the population, which gives a statistical view of the genome at the population level. From this data we calculate two indicators to characterize genetic architecture:

(i) The inter-loci relative standard deviation (RSD) in allelic values indicates if all genes contribute equally or very differently to the total genetic value of traits (Fig.3.4 A). The inter-loci RSD is calculated as the standard deviation of allelic values between loci pondered by their mean value over the genome to look at the relative effects of mutations present in the population.

(ii) The Index of allelic correlation for two traits in the population indicates if genes have, in average, similar effects for both traits or if, on the contrary, genes have in average different effects for each of the traits (Fig.3.4 B). It is calculated from the sum of the squared difference between the mean allelic values for the two traits

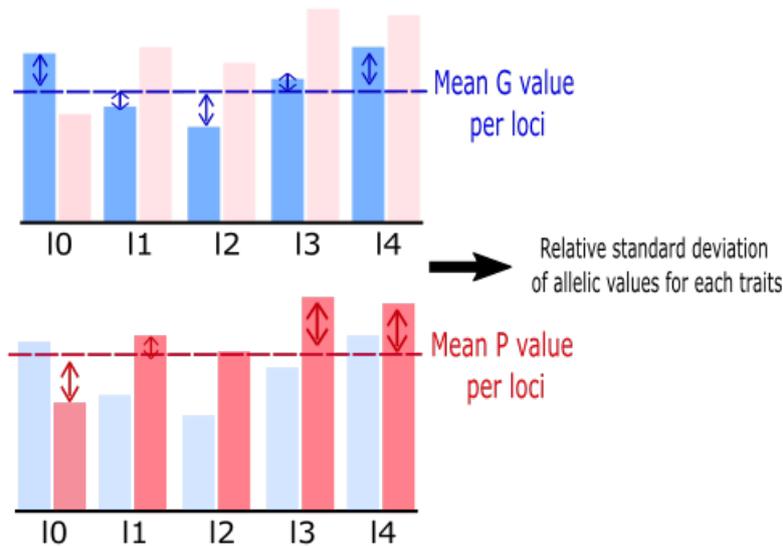
at every locus (Appendix C2). To assess whether this allelic correlation deviates from random expectation, we used a bootstrap approach. We calculated an index corresponding to the rank of the sum of the squared difference in the distribution of the sum of the squared differences calculated by bootstrap. The bootstrap is performed $n * (n - 1)$ times, with n the loci number. A ranking close to 0 means that the sum of differences in the average genetic values at each locus for the two traits is lower than expected by chance. A ranking close to the maximum index value means that the sum of differences in the average genetic values at each locus for the two traits is higher than expected by chance.

Initialization: To initialize a simulation, three types of parameters are specified:

- Demographic parameters: the resource limitation in the environment K and the initial population size.
- Mating parameters: the mating system (e.g. random mating, random encounter with preference, competitive encounter without preference or competitive encounter with preference) and the size of the mating group MG are also required. The minimal model is run using a single trait (G), more complex models can be run adding mating preference (P) and/or competitiveness (C).
- Physical genome structure parameters: the number of autosomes and sex-chromosomes, the number of loci, the probability of recombination between each contiguous pairs of loci, the maximum number of alleles per locus in the population, the distribution of allelic values, and the mutation rate.

Because initialization includes stochastic processes such as sampling in allelic effects distributions, initial states using the same parameters sets may vary. The model, however, allows starting several simulations out of a single initial state. The

A) Inter-loci RSD



B) Index of allelic correlation

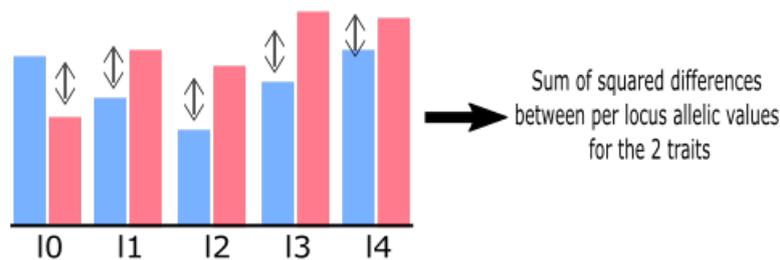


Figure 3.4: Schematic representation of the indexes measured to describe the genetic architecture, A) inter-loci RSD, B) index of allelic correlation. The blue and the red bars represent the mean allelic value of each locus for G and for P respectively in the population.

user thus can either start several simulations with different initial states produced by a same set of parameters, or start several simulations with the exact same initial state (obviously produced by a single set of parameters).

Observation : The model can either be run in graphical user interface mode or script mode. In the former, the user can select if all time steps or only a subset

should be memorized. In the memorized time steps, all objects (and therefore all individuals and their genomes) are observable. A wide panel of data extractors and visualizers is then available to analyze and illustrate the simulations. In script mode, only population-level variables are recorded over time. For each simulation, we record different type of variables to characterize mechanisms of evolution: At the population level, trait evolution is monitored by measuring their average values and standard deviations in the population, their evolution rate in Haldanes ([Hendry & Kinnison 2001], Appendix C3), and the genetic correlations between traits. Genetic architecture evolution is monitored by calculating inter-loci RSD, and the index of allelic correlation. Demography is monitored by measuring population size, the number of breeders at each breeding season, the sex-ratio and the mean survival rate. At the individual level: we record individuals lifetime reproductive success (fitness), individuals values of traits and the covariance between the allelic effects for the pairs of traits.

Installation and execution procedures : The previous description of the model assumptions and mechanisms only covers a part of the settings and tools available to users in the model. A software package is available (package RUNAWAY, running under CAPSIS-4 simulation platform), allowing users to install and interact with the model, at the following address: <https://doi.org/10.15454/6NFGZ9>. A brief documentation, downloading and installation procedures, as well as a quick start guide can be accessed at the following address: http://capsis.cirad.fr/capsis/help_en/runaway

3.1.3 General concepts underlying the design of the model

We here describe the general concepts underlying the design of the model.

Fitness : Individuals do not seek to improve their fitness explicitly. Fitness improvement is obtained through genetic selection. Individual reproductive traits can indirectly improve individual fitness: high values of G enhance individuals reproductive success but have a survival cost, high values of P allow individuals to get a mate with high fecundity but have an *opportunity cost* (i.e. missing mating opportunities, [De Jong & Sabelis 1991, Fawcett *et al.* 2012, Dechaume-Moncharmont *et al.* 2016]), high values of C improve the mating success of individuals through priority access to mating partners, yet they do have a survival cost too.

Sensing, interactions, collectives : As previously described, during reproduction, the population is partitioned in several mating groups. Individuals sense and interact with potential sexual partners and competitors within the scale of the mating group. However, such information will not affect their decision (*i.e.* no adaptive behaviour ABM wise), only the outcome of interactions.

Adaptation : Individual fitness is mainly determined by its reproductive behaviour relative to other strategies. Indeed individual success during reproduction depends on opposite-sex preference and availability and same-sex competitiveness and preference. Individuals are therefore more or less adapted to their social environment. Individuals presenting preference for a very rare trait value in the population may not be socially adapted and entail the risk of not finding a mate, thereby compromising their fitness. Individuals also need to adapt to an extrinsic part of environment which is represented by habitat resources limitation. A typically non-adapted individual would present traits values that do not allow him to survive until reproduction in this environment.

Stochasticity : Because we use an agent-based model, most processes are inherently stochastic. Survival is, for instance, the realization of a Bernoulli random draw. Mating systems are also an important source of stochasticity. First, individuals are sampled randomly to constitute mating groups. Then, when they do not express competitiveness, individuals randomly encounter each other within each mating group. Mate choice itself is a highly stochastic process: it results from mutual acceptance of both partners, through their respective mating preference which is a probabilistic function, so to include an error of assessment of the mating partner quality. Lastly, the transmission of genetic information is also subject to stochasticity because we represent chromosome segregation and recombination during the meiosis and we also account for mutation risk.

3.2 Exploring the evolutionary dynamic of a particular simulation: coevolution between Gametic investment and Preference

This first example introduces the evolutionary mechanics in the model, such as the relationships between demography, individual variation, and genetics, as well as the emergent patterns in the model. We here look at the evolution of gametic investment (G), under two different mating systems. First, with random mating, second, with preference (P) driven mating, wherein individuals select partners on their gametic investment. We track how the inclusion of a non-random mating system influences the evolution of G , and whether P itself co-evolves with the former trait. We examine both the dynamic phase of the evolution during the first generations and the later convergence phase around a pseudo-equilibrium after 5000 generations. For simplicity, the following figures compare the evolutionary dynamics of two single

population (one with preference the other with random mating) but, as shown latter on, independent replication of the simulations follow the same dynamics (section 3.3.1).

Default parameters values used for all the simulations are the following: initial population size equal 10000, resource limitation is $K = 10000$, the size of the mating group M is set to the size of the population. The genetic map is, by default, made of 10 unlinked loci distributed over 10 chromosomes (i.e. recombination probability between adjacent locus is 0.5). The mutation rate is set to 10^{-4} mutations per loci.

3.2.1 Evolution of reproductive traits:

Under random mating, G shows a substantial evolution from its initial value (of 0.5 average in the population) to reach a pseudo equilibrium around 0.7 after 200 generations (Fig.5.5 A). The standard deviation around this value remains stable throughout the simulation (around 0.1). The evolution rate of G calculated in Haldanes is accordingly moderately high during the first 200 generations and then stabilizes to low values. During the early phase of the simulation, we observe a clear positive correlation between individual fitness and G , indicating that values above the mean for this trait are beneficial (i.e. directional selection). Once at the pseudo-equilibrium, this relationship turns to a bell-shaped distribution, indicating stabilizing selection. Note that in this scenario, we also looked at the evolution of P as a non functional trait: this trait shows no evolution whatsoever, its average and standard deviation remaining stable throughout the simulation (Fig.5.5 A).

When mating is driven by P , the two traits increase quickly from their initial values (of 0.5 in average in the population) to reach maximum average values of 1.95 for G and 1.99 for P after about 200 generations (Fig.5.5 B). At this time, the standard deviation of the traits is maximal, around 0.2. Then the mean val-

ues of traits and their standard deviation decrease slowly overtime to reach mean values of 1.61 for G and 1.63 for P and standard deviations of 0.035 and 0.038 respectively. During the whole simulation, P is always greater than G , indicating that on average, the higher values of G are preferred in the population. The evolution rates of traits indicate a rapid evolution during the first 200 generations (with evolution rates that are in the upper range of values reported in the literature, [Hendry & Kinnison 2001]), followed by a much slower evolution of traits. At the 100th generation, a snapshot of the fitness landscape shows that high values of G (of about 2) are advantageous (Fig.5.5 B). Later snapshots of the fitness landscapes confirm stabilizing selection, as well as an erosion of variance of G in the population with time (Fig.5.5 B). The emergence of a genetic correlation between G and P sheds some light on the rapid joint evolution of traits (Fig. 3.6). This correlation arises because individuals with high P choose individuals with high G , and thus, statistically, their offspring will inherit similar P and G values. It accentuates the joint evolution of the traits toward extreme values because P will then evolve under indirect selection (only because of the genetic correlation with G), while G is under increasing sexual selection (as P increases in the population) (e.g. Fisherian mechanism, [Fisher 1915, Lande 1981, Hall *et al.* 2000]). Interestingly, the correlation between traits oscillates during the dynamic phase of evolution (contrary to the random mating scenario where the correlation progressively decreases). The correlation first quickly increases to reach a maximum of 0.7 at time step 100, but then decreases to become negative, before increasing again (Fig. 3.6). The strong directional selection that occurs in the preference driven mating scenario is also apparent when observing the variance of the traits in the population, which is very low in the phase of rapid evolution of the traits (in comparison with the random mating scenario)(Fig.5.5).

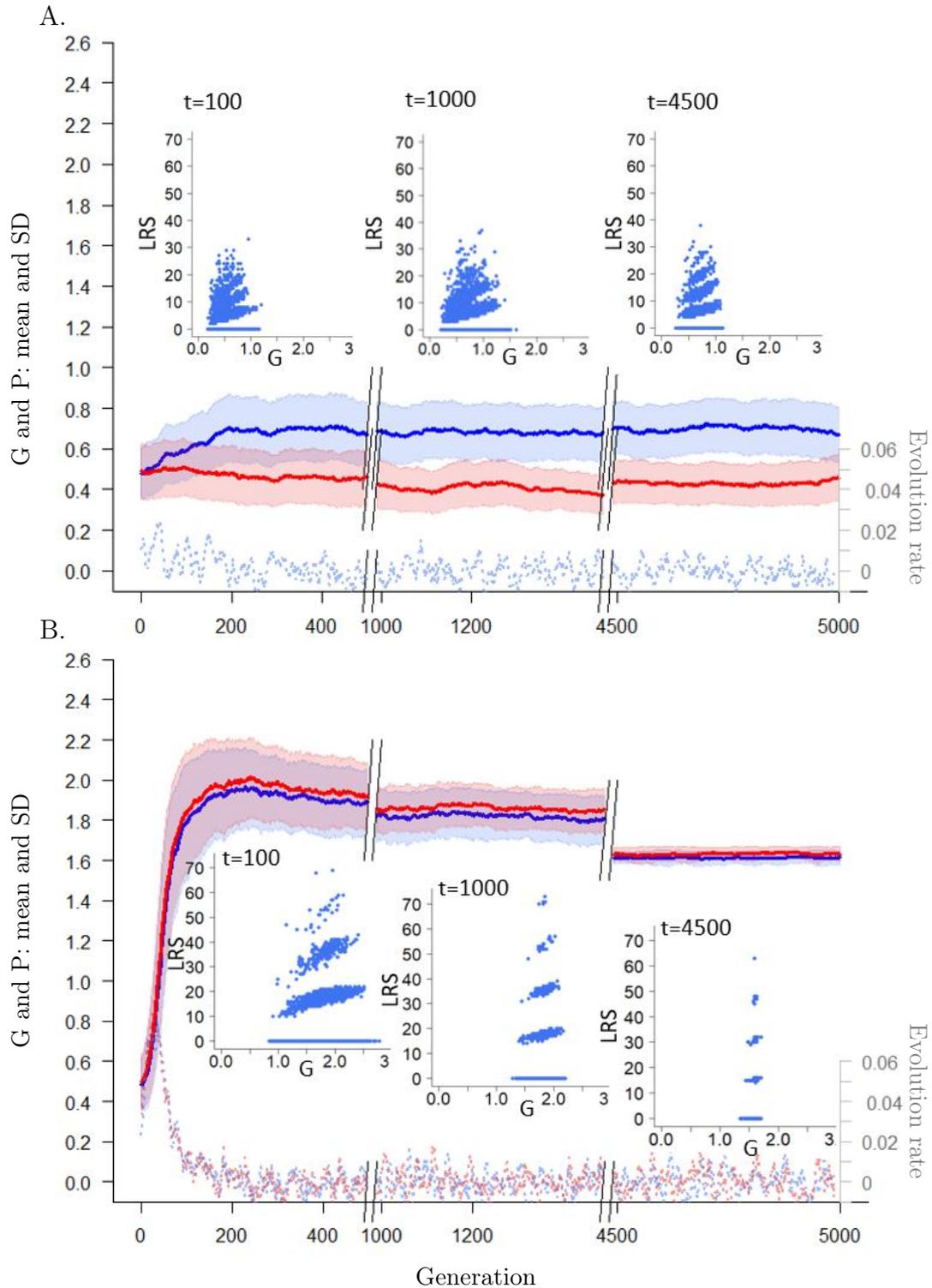


Figure 3.5: Evolution of G (in blue) and P (in red) in the population over 5000 time step, for one simulation of either (A) random mating, (B) preference driven mating. Mean values of traits are showed in thick lines and standard deviation values are represented in transparency. Also appear in the figure the evolution rates in Haldanes for the two traits (dashed lines) and the relationship between lifetime reproductive success (LRS) and G values at different time step during the simulation.

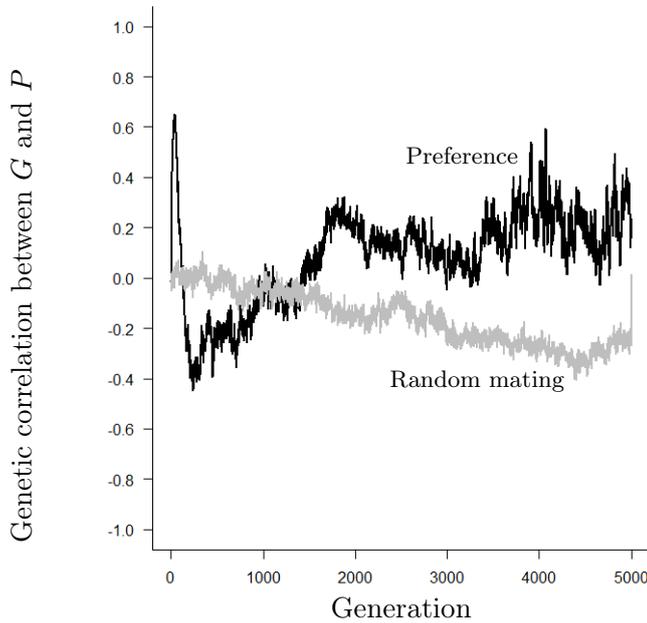


Figure 3.6: Genetic correlation between G and P over generations, for two mating scenarios: random mating in grey, mating preference in black.

3.2.2 Demographic consequences

The evolution of reproductive traits impacts the population size via the trade-off between survival and reproduction. When the evolution of G is driven by the preference, the mean probability of surviving from one reproduction to another is of 0.1 at the pseudo equilibrium, and population size is about 2800 (Fig.3.7). Whereas, when mating is random, the mean survival probability is a bit greater (about 0.25 at the pseudo-equilibrium) because G stabilize at lower values resulting in a higher mean survival, and consequently the population reached bigger size at the pseudo equilibrium (about 3600, Fig.3.7). When mating is random almost all individuals in the population reproduce at each breeding event (with small fluctuations due to fluctuations of the sex ratio). When there is mate choice and the mating group equals the size of the population (individuals can potentially encounter all opposite-sex mem-

bers in the populations), still almost all individuals in the population reproduce at each breeding event, as indicated by the low number of unmated individuals after each breeding event (Fig.3.7).

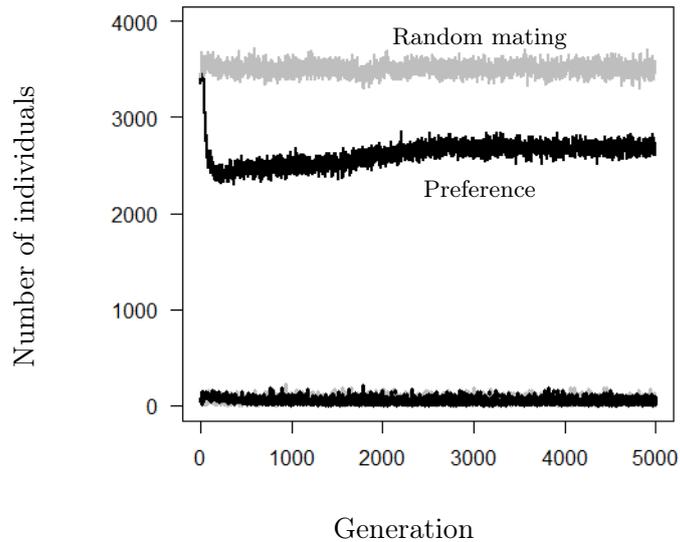


Figure 3.7: Number of mated (top grey and black lines) and unmated individuals (bottom lines) after each breeding event for two mating scenarios: random mating in grey, mating preference in black

3.2.3 Evolution of genetic architecture

Allelic diversity: The allelic diversity measured as the proportion of unique alleles per locus, initially very high as a result of our initialisation parameters (500 different alleles per locus in average) decreases significantly in the two mating scenario (Fig.3.8). In the scenario with mate choice, the allelic diversity decreases more significantly than in the presence of random mating (to reach an average of 10 alleles per locus versus 18 alleles per locus). This difference can be due to stronger sexual selection in the case of preference driven mating but can also be caused by a smaller population size at the pseudo equilibrium. It is noteworthy that the most active phase of evolution happens when allelic diversity has not stabilized and is plentiful. Yet, further evolution also happens when allelic diversity has stabilized. This reinforces the idea that genetic variation is actively controlling the speed of evolution in the model.

Genome spatialization: We look at a statistical view of the mean genome in the population by computing the mean allelic effects per trait at each locus (Fig. 3.9). Initially, due to the random sampling of allelic values in beta distributions, all loci have similar mean genetic values and do not deviate from the mean. After 5000 generations, the allelic values are not evenly distributed over the genome anymore, some locus having strong effect - either in a positive way: locus coding for much higher values than the average for a trait (e.g. *l1*, *l7*, *l9*, *l10* for *G*) or a negative way: locus coding for much lower values than the average (e.g. *l4*, *l5*, *l6*, *l8* for *G*) (Fig. 3.9). Remarkably, loci having a very high effect on one of the traits also have a very low effect on the other trait (e.g. *l4*, *l5*, *l7*, *l10*). Therefore it seems that a negative correlation among allelic values for the two trait is established within loci, with a differentiation of the genes coding for the trait *G* from the gene coding for

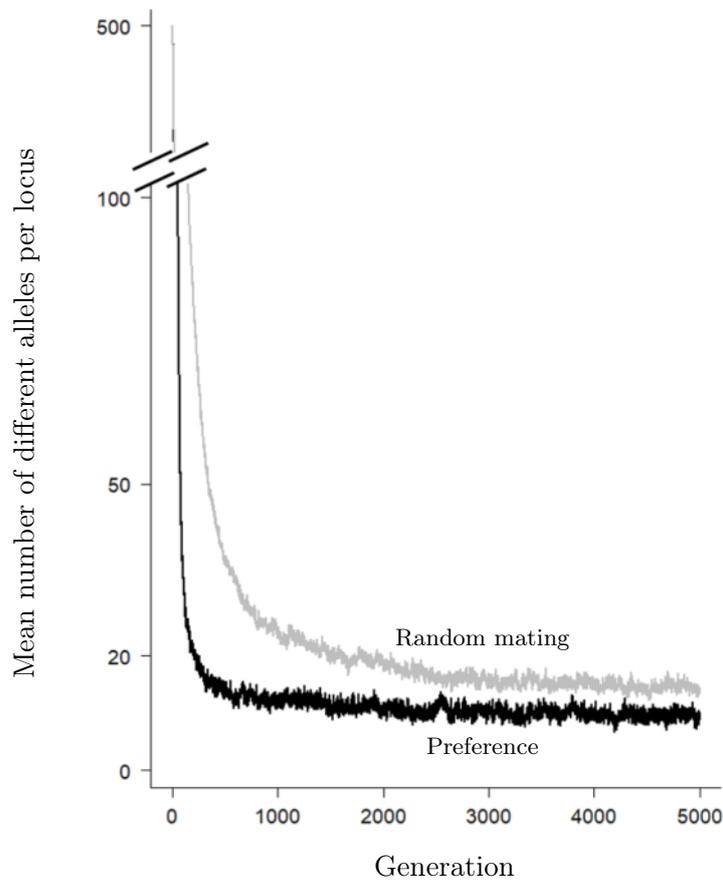


Figure 3.8: Mean number of different alleles per locus in the population for two mating scenarios: random mating in grey, mating preference in black

the trait P . This observation is supported by the fact that the mean correlation among allelic values individuals' genome become negatively correlated with their lifetime reproductive success when mating is driven by preference (Fig. 3.10), which suggests that the negative correlation between the allelic values is selected when the two traits are under selection. Furthermore, this relationship is not observed in the scenario of random mating were preference is not subject to selection, so the emergence of negative allelic correlation is certainly not obtained by chance or caused by an artefact in the model. The fact that allelic values are selected to vary in opposed directions (e.g. negative correlation) while traits are selected towards

3.2. Exploring the evolutionary dynamic of a particular simulation: coevolution between Gametic investment and Preference 75

similar values could be explained by a potential advantage in decoupling the selection that weighs on the genes. This is coherent with the idea that modular genetic architecture can promote adaptive evolution by allowing traits to vary independently and thus be optimized separately without interference [Pepper 2000, Hansen 2003, Jones *et al.* 2003, Otto 2004, Griswold 2006].

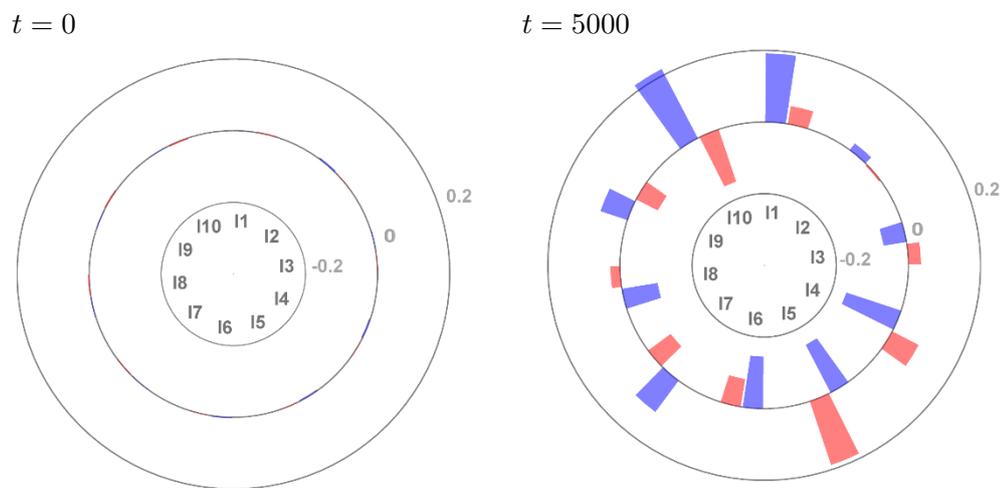


Figure 3.9: The mean genome in the population, at initialization ($t=0$) and after evolution over 5000 generation. The height of bars at each locus represents the mean effect of alleles at each locus for the gametic investment (bar in blue) and preference (bar in red). The mean effect of alleles at a locus being calculated by subtracting the mean allelic value of the loci for the trait in the genome from the mean allelic value at the given locus. Grey circles give the graduations for allelic effects. At the initialization, all loci have average allelic values that do not deviate from the mean. After evolution some locus have strong (positives or negatives) effect for one or both of the traits

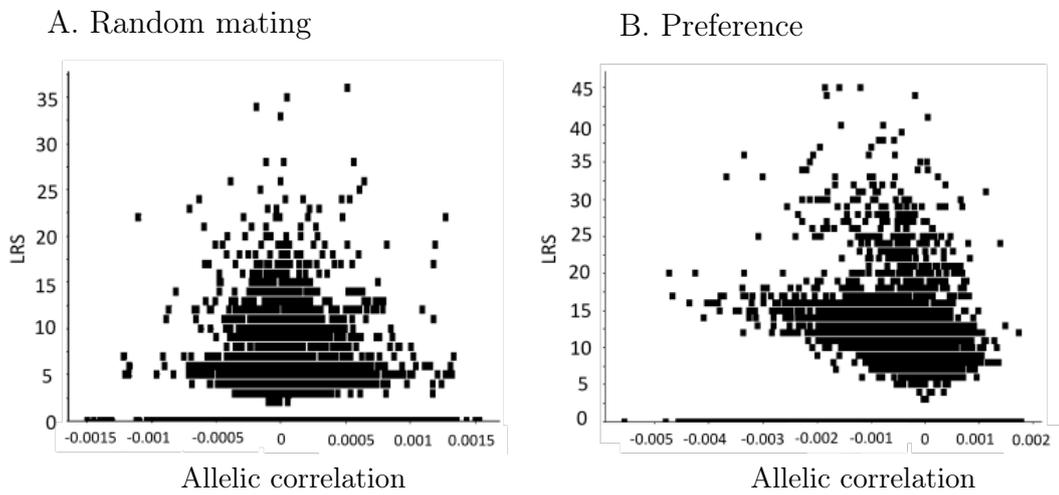


Figure 3.10: The Lifetime reproductive success of individuals as a function of their allelic correlation for G and P at time step $t=50$, for two mating scenarios: A) Random mating, B) Preference driven mating.

3.3 Effect of mating systems on traits and genetic architecture evolution

I generalize the above approach to 4 different mating systems named according to the number of traits expressed in each one (random mating G , random encounter and preference $G + P$, competitive encounter without preference $G + C$, competitive encounter with preference $G + C + P$). The others parameters remain to the same values than in the previous experiment (section 3.2). Our objective is to look at the effects of mating systems on traits evolution, but also at their rippling consequences on alleles values distributions and their correlation in the genome and on population demography. Here however, we perform 30 replications of simulation for each mating system, each over 5000 generations. The simulations are conducted with the same set of parameters but with different initial states. By replicating the simulations, we investigate whether the factor we manipulate (here mating systems) leads to very homogeneous evolutionary outcomes, or whether they generate a diversified set of equilibrium. Stochastic variations in the evolutionary outcome between simulations can be due to random difference at the initial state of the model (in the drawing of genetic values for example) and to stochasticity in the demographic, genetic and behavioral processes.

We here only focus on the final picture of the evolutionary process, without detailing the dynamics leading to it.

3.3.1 Evolution of the reproductive traits:

The traits evolves differently depending on the mating scenario, which is accompanied by different population sizes and survival at the pseudo-equilibrium (Fig. 3.13).

The effect of mating preference: Globally, replications of the simulations confirm the above observations on G and P coevolution. When individuals express their preference (scenario G and $G + P$), P is favored by sexual selection, which promotes the evolution of G toward much higher values than in the random mating system, in which P evolves under genetic drift. As G is costly in terms of survival, its increase in the population leads to lower average survival, which translates into a lower pseudo-equilibrium population size (Fig. 3.13).

As previously explained, the joint evolution of P and G toward extreme values is caused by the build-up of correlation between the traits (Fig. 3.6), which is at the core of the mechanism of Runaway selection proposed by [Fisher 1915].

Beside, the amount of variations between simulations varies depending on the mating scenario: Under random mating, all simulations converge toward the same value of G , the trait P which is not expressed in this scenario present inter-simulation variance due to genetic drift (Fig.3.11).

Under preference driven mating, inter-simulation variance is significant, G and P co-evolving to reach values ranging from 1.4 to 2.6 (Fig.3.11). All the simulations reach a pseudo equilibrium. Inter-simulations differences in the conjoint trait values are certainly caused by stochasticity in demographic, genetic and behavioral processes, and by small random variations in the initial conditions. Interestingly, as theoretically demonstrated by [Lande 1981], the joint evolution of the preference and the trait under preference can reach different combinations of traits values at the equilibrium.

The effect of competitiveness: Competitiveness (C) evolution is also conditioned by the simulation scenario (Fig.3.12). C is selected for (despite the associated survival cost) only when individuals also express their preference P on gametic

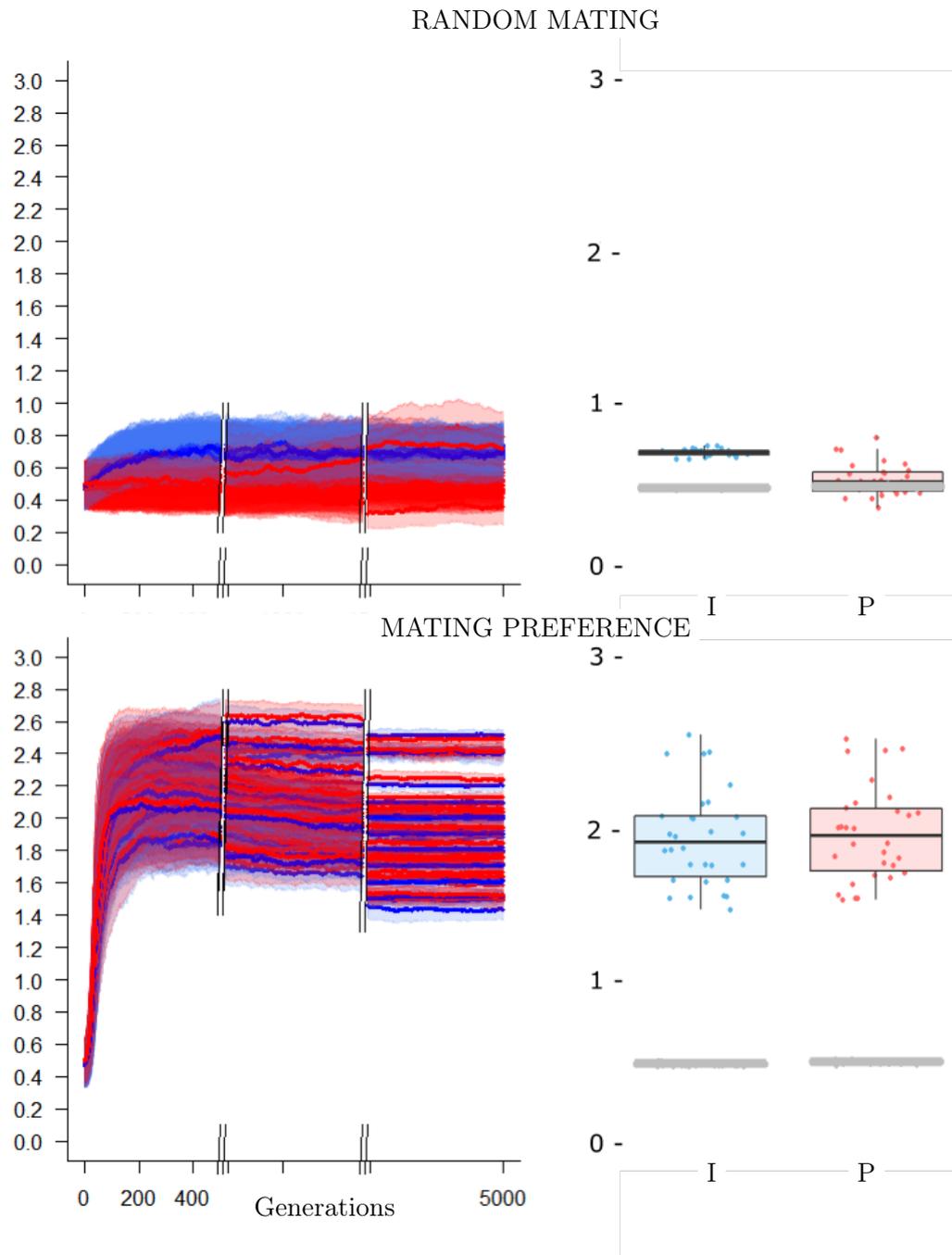


Figure 3.11: Mean traits values in the population for G and P for 30 simulations with two mating scenarios, random mating (P is not expressed) and mating preference. Left panels show the evolution through time, right panel show boxplots of the mean traits values in the simulations initially (in grey) and at the final states (in colors).

investment G (scenario $G + P + C$), because in that case, the most competitive individuals are more likely to find a suitable mate. In the scenario without preference (scenario $G + C$), C is counter selected, its cost is too great when traded off against the possible benefit of reducing opportunity costs (e.g. the risk of not finding a mate). Indeed, in the present simulations, mating group size is equal to the population size, and opportunity cost under random mating is therefore negligible.

Whereas all simulations converge to the same value of G at the pseudo equilibrium under random mating, as soon as there is some preference or some competitive encounter, inter-simulations variance increases. It therefore underlines the importance of the complexity of behavioural interactions on the outcome of evolution.

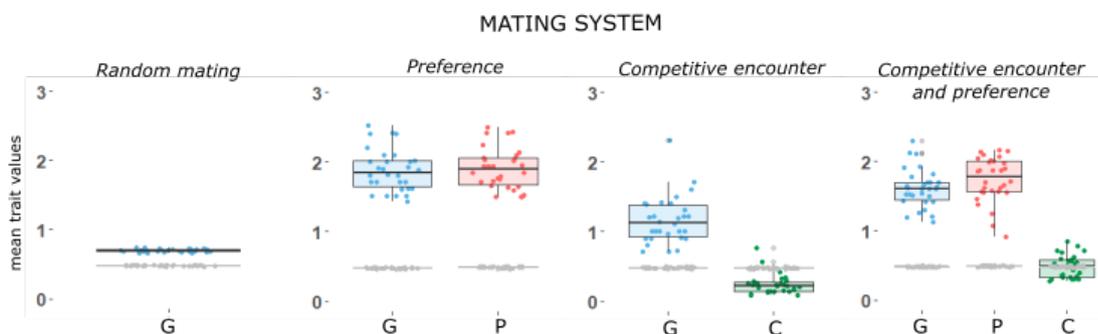


Figure 3.12: Mean values of gametic investment, preference and competitiveness, initially (in grey) after evolution over 5000 time step (in color). Four mating systems simulated : random mating (G), random encounter and preference ($G + P$), competitive encounter without preference ($G + C$), competitive encounter and preference ($G + C + P$). Each dot represents the main trait value in a simulation. A boxplot indicates the 2.5, 25, 50, 75 and 97.5 quantiles of the distribution among simulations.

As previously stated I did not change the mutation rate μ , the carrying capacity K and the mating group size MG , for now. Within these settings, preference reached high values whereas competitiveness barely evolved. I suspect that it would be a whole different matter if we had modified MG . Indeed, in situations where

individuals only meet a subset of the population to find their mate ($MG < \text{population size}$), the risk of not reproducing can be high, and consequently competition is expected to be stronger. In that case, traits may evolve differently; for instance, iteroparity (i.e low G , low P) can be favored as a form of "Bet Hedging" in time [Wilbur & Rudolf 2006] and high competitiveness could be an advantage. This may, in turn, affect the evolution of genetic architecture.

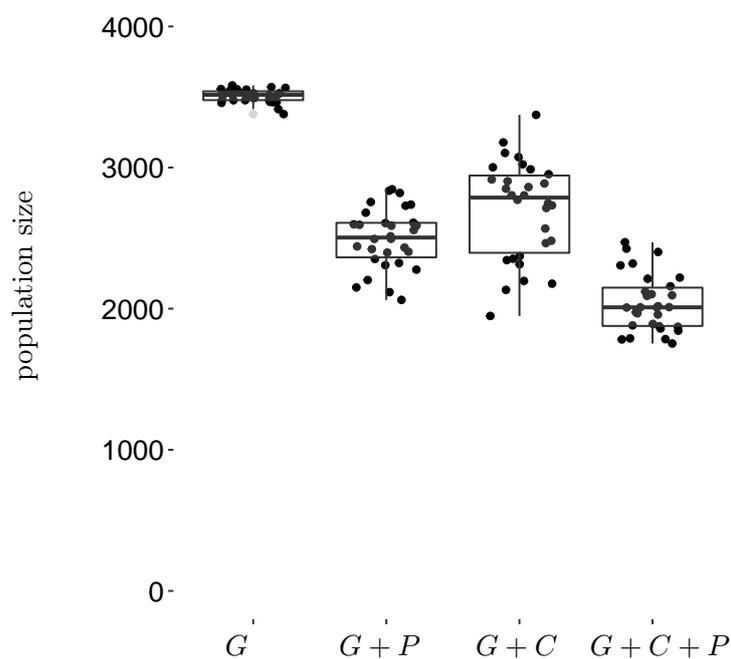


Figure 3.13: Mean population size in the different simulations after evolution over 5000 time step. Boxplots indicate the 2.5, 25, 50, 75 and 97.5 quantiles of the distribution among replications. Four mating systems are simulated : random mating (G), random encounter and preference ($G + P$), competitive encounter without preference ($G + C$), competitive encounter and preference ($G + C + P$).

3.3.2 Genetic architecture evolution:

We also observe different characteristics of the genetic architectures among the different mating scenarios, indicating that mating behaviors and consequent sexual selection shapes the genetic architecture.

The effect of mating preference: A difference of allelic values distribution between mating systems is visible when looking at inter-loci relative standard deviation (RSD) of the mean allelic values (Fig.3.14), the measure allows to show the difference in magnitude of genetic values between loci, independently of the differences in the values of the traits. When mating is random, inter-loci RSD for G and for P is initially low - as after the initialisation all loci have similar allelic values in average - and then inter-loci RSD for G and P increases. Inter-loci RSD becomes slightly higher for G , compared to the trait P that is only subject to genetic drift and mutation in the random mating system (i.e., neutral trait). Under the preference driven mating system, P is expressed, and the inter-loci RSD of the two traits is a bit higher compare to the random mating scenario. The pattern of correlation among allelic values within loci also evolves differently depending on the mating system (Fig. 3.15). When individuals mate randomly, within loci correlation evolves randomly and there is no effect of initial allelic distribution: in simulations where allelic values are initially correlated by chance, the genome can evolve toward negative correlation and *vice versa*, not illustrated here. However, when preference matters, the genome evolves towards negative correlation among allelic values for G and P . Remarkably, such negative correlation occurs in scenarios where there is co-evolution between the G and P and so positive global genetic correlation between the traits (scenarios $G+P$ and $G+P+C$, Fig.3.15). I here predict selection of alleles with opposite effects for the two traits in a situation where I may have intuitively

expected positive correlation to evolve. Indeed, I initially supposed that as the traits evolves conjointly, if a mutation that positively affects G also affects P in a similar direction, then these new mutations will be transmitted and become fixed in the population due to the increased fitness of their bearers. But my results showed that alleles with opposite's effects are fixed during directional selection. This result is robust to other distribution of allelic values (uniform law, Appendix C4) but could be challenged if environmental parameters were changed (K , MG). Within loci negative correlation may confer an adaptive advantage by allowing more combinations of traits values to be transmitted in offspring. Such variability could allow to better respond to the dynamic changes in demography and social environment.

Importantly, this result is robust to other mutation landscapes (Appendix C4), confirming that the emergence of negative correlation between allelic values is not a computational artefact due to the draws in the distribution of allelic values.

The effect of competitiveness: When we turn to a mating system with competitive encounter without preference, inter-loci RSD further increases for both G and C traits. In contrast, when the mating system is driven by both competition and preference, inter-loci RSD for C is much lower, whereas the inter-loci RSD for G and P remain high (Fig.3.14). These results indicate that the different mating systems, mostly due to different ways for individuals to interact for reproduction, will select for non random distribution of genetic values along the genome. This could be of course further investigated by considering different physical structure of the genome (number of chromosomes and location of genes) .

Looking at the pattern of correlation among allelic values within loci we found no clear organisation in the allelic effects for G and for C , when encounter is competitive but in the absence of preference. But here again the addition of preference tend to

select for negative correlation between allelic values and thus for a modular genetic architecture. We, therefore, observe that the mating systems mediates to some extent the evolution of the genome.

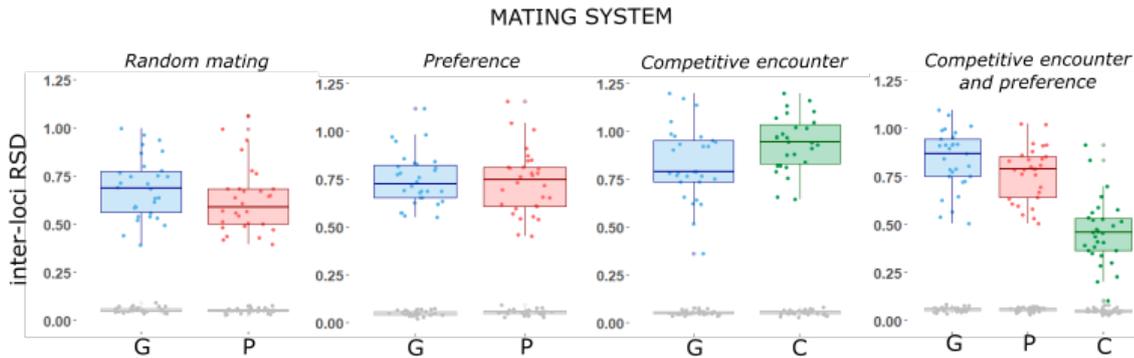


Figure 3.14: The relative standard deviation of the mean allelic values between loci for the different traits present in the scenarios (G, P, C, in the first scenario the preference is not expressed), initially (in grey) and after evolution over 5000 generations (in color). The boxplots are drawn with 30 simulation of the four mating routines (random encounter, preference driven mating, competitive encounter, competitive encounter and preference driven mating) and report the mean and 95% interquartile range interval.

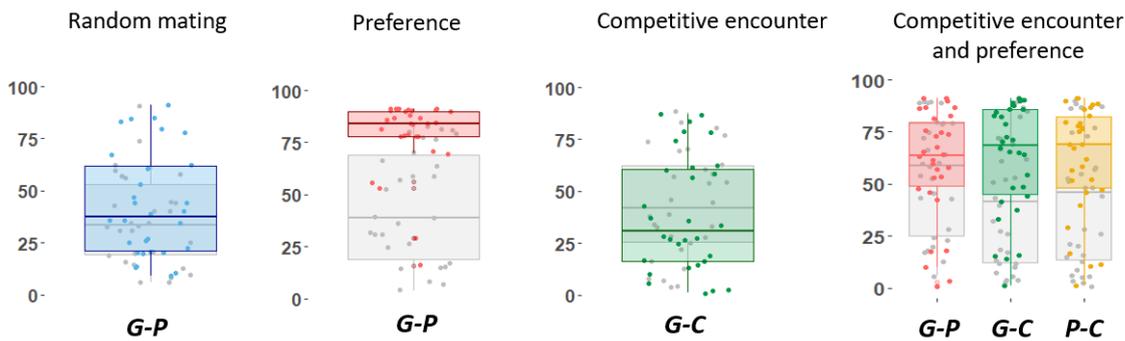


Figure 3.15: Index correlation of allelic values for the pairs of traits. The measures are showed initially (in grey) and after evolution over 5000 time step (in color) for 30 replications for each mating system (random mating, random encounter and preference, competitive encounter without preference, competitive encounter and preference). The boxplots report the mean and 95% interquartile range interval.

3.4 The sensibility of parameters change

To get an idea of the effect of the initial parameters on the evolution, I ran some additional simulations and recorded some global features of the population (mean traits values and SD, genetic correlation between traits, population size, and mean survival rate). Using the mating system of preference driven mating, I first analyze summarily the sensitivity of the results to resource limitation K , demographic constant controlling for fecundity d , and the inclusion of a cost of preference (i.e., influencing directly the reproduction/survival trade-off). Then, I examine the effect of some genetic parameters (mutation rate and mutation distribution). And lastly, I browse the various possible effects of the different shape and assumptions of sexual preference.

3.4.1 Sensibility to reproductive trade-off:

Preference cost: When preference has a direct cost on survival ($R = G + P$), we still observe a rapid conjoint evolution of traits at first (data not shown), but the traits then decrease substantially to stabilize at lower values (Fig.3.16) and genetic correlations between G and P are also a bit lower. This finally, leads to a higher population size and higher survival at the pseudo equilibrium (Fig.3.16). So costly preference changes the reproductive strategy in population, promoting more semelparous strategies. Globally, inter-simulation variance in the mean trait values is lower than in the scenario with cost-free preference (Fig.3.16).

Resource limitation (K) effect: In contrast, K has not a strong effect on the evolution of traits, the mean trait values are somewhat smaller when $K=5000$ compared to when $K=10000$ (Fig.3.16). Yet, population size at the pseudo-equilibrium is reduced to adapt to lower resources, causing the intra-population standard de-

viation in traits to be slightly reduced as well (Fig.3.16).

Demographic parameter d (i.e. mean number of offspring per clutch):

When the relation between reproductive investment values (which directly affect survival) and number of offspring produced per reproduction changes in the direction of a reduce number of offspring per reproduction, the runaway mechanism is not initiated and P and G remain to rather low values (Fig.3.16). This is likely due to a numeric effect, as the number of offspring is equal to the rounding of the product $G*d$, if d is smaller the number of offspring that varies less continuously with G and therefore individuals with slightly different values of G will have the same number of offspring, resulting in a selection on G less efficient. Such situation would require more mutational input or genetic variation to allow the real number of offspring to spring out of the "evolutionary cul de sac".

3.4.2 Genetic parameters:

Mutation rate: Lowering the mutation rate (from 10^{-4} to 10^{-8}) has relatively little effect on evolution. Traits evolve toward slightly higher values, intra-population SD values are lower (close to zero). Genetic correlation between traits however now becomes negative. Population demography (population size and survival) is nearly identical (Fig.3.17).

Mutation distribution: Changing the distribution of allelic values among which mutations are drawn however has important consequences on traits evolution (as it changes the initial value of traits, and the distribution of mutational values) and thus on demography (Fig.3.17). Uniform distribution of allelic values leads to very high value of traits initially; the trait values will then decrease, but the population size is reduced, and the life history strategy becomes extremely semelparous. Genetic

correlation between traits is mainly negative. The $beta(2, 2)$ distribution of the allelic values (bell-shaped around 0.5), leads to initial trait values similar to our reference scenario, but then favours the co-evolution between G and P towards more extreme values. The life history strategy again becomes semelparous as a consequence. Clearly, the shape of the mutation landscape has pervasive effects on evolution in the presented simulations, affecting the runaway process, and the life history strategy.

3.4.3 Shape of the preference function:

Changing the shape of the preference function have strong effect on the evolution of traits and consequently affect population demography.

Tolerance: Individuals with unimodal preference can have different tolerance around the preferred value. In the model of [Lande 1981] the tolerance value is involved in the calculation of the equilibrium line, and may affect the outcome of evolution. In our model the tolerance ν is fixed to 0.2 by default (and does not evolve in the current version of the model, for the sake of simplicity). Allowing a higher tolerance ($\nu = 0.5$, that would mean a lower choosiness) indeed affect the conjoint evolution of traits. The runaway process happens during the dynamic phase (which is not shown here), but the correlation between G and P remains rather low, such as the two traits reach lower values (Fig.3.18). Then the correlation between G and P becomes null and the evolution of the traits is decoupled, leading to low preference in the population (Fig.3.18). When $\nu = 0.5$ tolerance is high and difference in mating success is too small to trigger runaway. This reduced evolution implies a larger population size, with increased survival at equilibrium.

Directional preference: When preference is directional, in the case of the scenario we envision, there is no runaway selection, P and G evolves independently and the correlation between traits becomes slightly negative then remains around zero (Fig.3.18). Indeed, when preference is directional (probabilistic threshold function), globally, individuals with G values below the preference value are rejected. A higher opportunity cost is therefore associated with a high preference value, compared to a unimodal preference which is more closely matched with the distribution of G in the population. Preference evolves toward low values such that individuals are poorly discriminating their mates (in average they accept almost all partners with $G > 0.1$). So globally, preference does not drive the evolution of G which evolves toward the same mean value and variance than under random mating. Consequently, population size is higher, and the population adopt a more iteroparous strategy.

Relative preference: In the scenario tested with our set of parameters, unimodal relative preference are expressed relatively to the distribution of G value in the population, for instance when the preference is 0.5, it means that the median value of G is preferred. Such preference surprisingly prevents the conjoint increase of G as initially $P = 0.5$ so median G value in the population is preferred and so sexual selection tends to work against natural selection which would select for slightly higher value of G . Directional relative preference however leads to extreme evolution of G as individuals with the higher values of G in the population are preferred (data not shown).

The above quick sensitivity analysis shows that, as expected, the different parameters can influence the outcomes of the evolution. The choice of these parameters values will depend on the questions to be addressed.

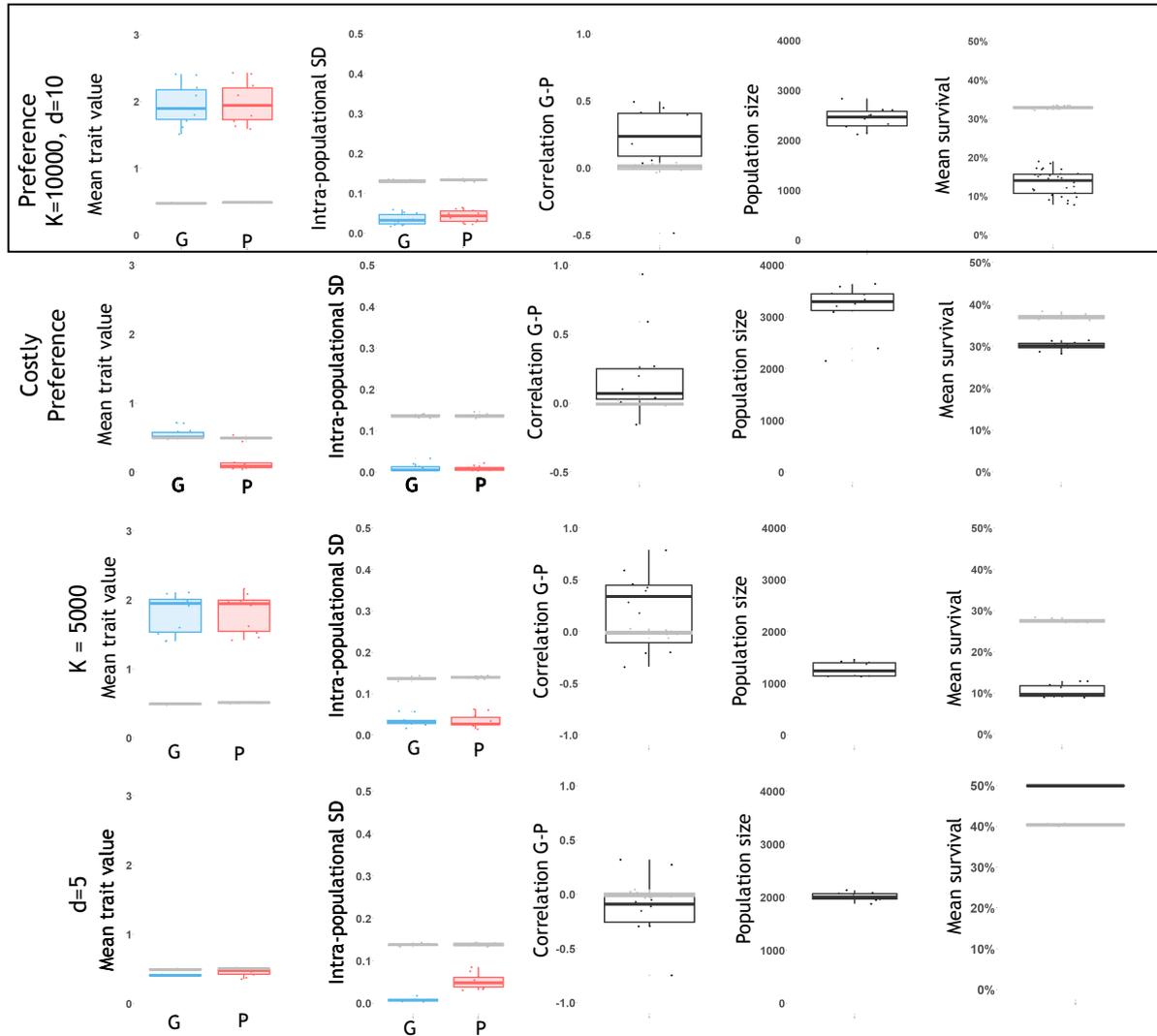


Figure 3.16: The effect of the parameters influencing the trade-off reproduction-survival on evolution. The boxplots show values of representative variable at the pseudo equilibrium for 30 simulations. the variables shown are the mean trait values in the population, their standard deviation, the correlation between G and P , the populations size and the mean probability of survival in the population.

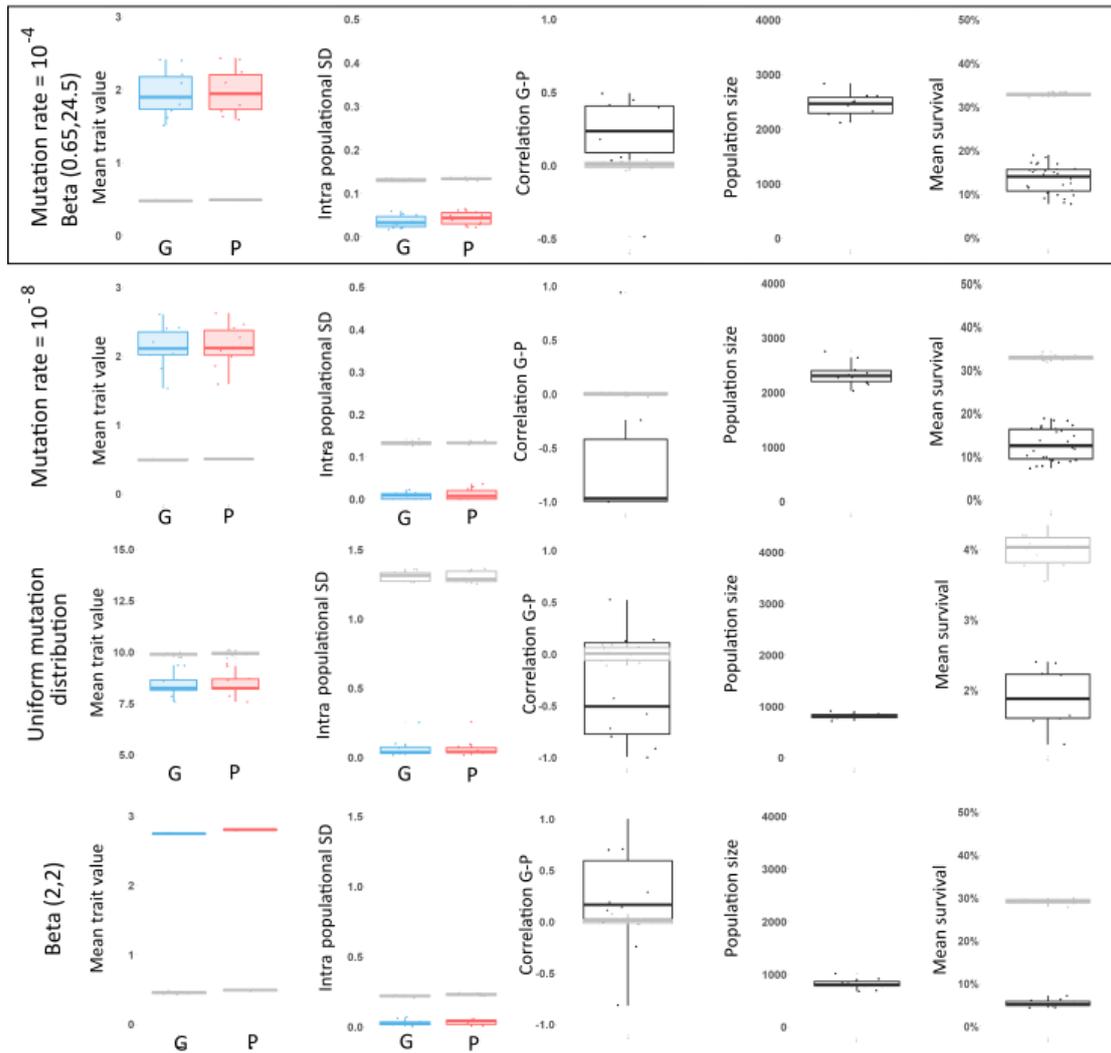


Figure 3.17: The effect of genetic parameters on evolution. The boxplots show values of representative variable at the pseudo equilibrium for 30 simulations. the variables shown are the mean trait values in the population, their standard deviation, the correlation between *G* and *P*, the populations size and the mean probability of survival in the population.

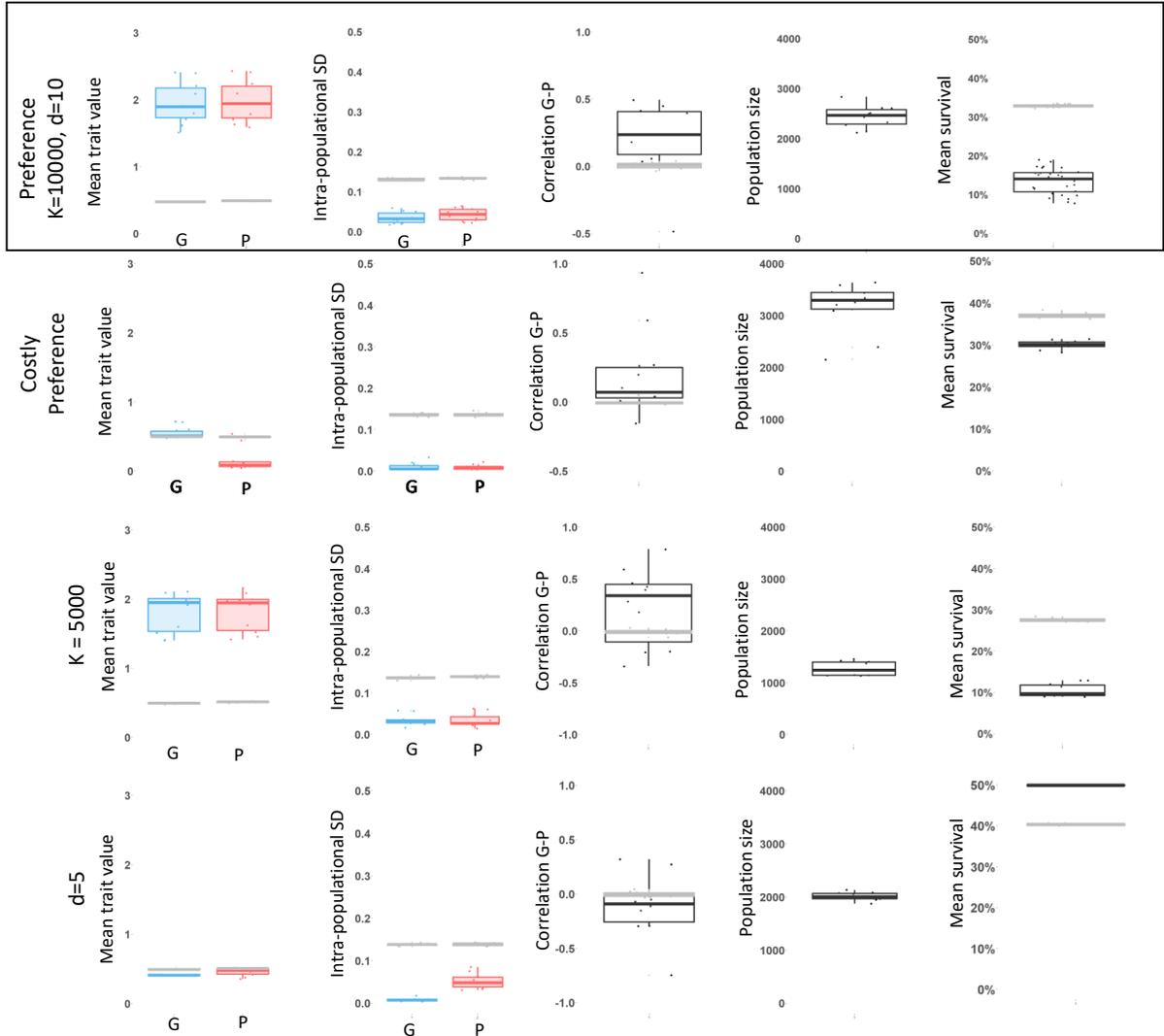


Figure 3.18: The effect of preference shape parameters on evolution. The boxplots show values of representative variable at the pseudo equilibrium for 30 simulations. the variables shown are the mean trait values in the population, their standard deviation, the correlation between G and P , the populations size and the mean probability of survival in the population.

3.5 Discussion

By capturing the processes linking genes, genome, individuals, groups, population, and environment levels, DG-ABMs offer new opportunities to study evolutionary dynamics in an integrative approach. The present model is an endeavour to develop such approach in a domain where it should be especially relevant: sexual selection, an eminently context-dependent process emerging from inter-individual interactions [Otto *et al.* 2008, Kokko *et al.* 2015, Muniz & Machado 2018].

The DG-ABM approach is somehow a middle ground between analytical methods and empirical approaches. On the one hand, analytical methods usually only focus on a limited sets of variables, generally requiring strong assumptions. DG-ABMs can relax some of these assumptions: for instance, they do not need to assume an equilibrium of genetic variance or stable age distribution within populations; they are thus well suited to focus on unstable demographic or genetic situations, that are often of primary importance in evolution [Barton & Charlesworth 1984, Kirkpatrick & Barton 1997, Hendry & Kinnison 2001]. On the other hand, empirical approaches can produce a wealth of patterns, on traits, on behaviour, their genetic basis, on life history trade-offs, or on population demographic and genetic structures. Only a fragment of these data are usually picked up, so to fit theoretical predictions stemming from reductive analytical models, whereas many of these patterns could help testing theoretical expectations more confidently. By allowing to generate patterns on several levels, I hope that DG-ABM way can help strengthen the links between theory and observations.

I sought, by means of examples, to illustrate here how the interplay between genetics, demography, and behaviors is pivotal in predicting the evolution of traits and genetic architecture under sexual selection, whereas too often these dis-

ciplines are treated separately [Rittschof & Robinson 2014, Wilkinson *et al.* 2015, Bengston *et al.* 2018]. And I believe that this approach could allow to make much more comprehensive predictions on biological systems: for instance, instead of solely focusing on the evolutionary equilibria of trait values, we here provide a complete picture of what such equilibria entail also in term of genetic architecture and demography. Namely, for such dynamics to occur, we demonstrate that demographic characteristics will also reach non-random values (level of iteroparity and survival, opportunity cost, population size) and that this may come with the building of non-random allelic structure within the genome.

Our simple simulation experiments revolved around comparing various mating systems. These examples illustrate the potential of the approach to predict micro-evolutionary processes and to thereby better understand the routes towards patterns. The model allows to analyze in detail micro-evolutionary processes at the gene, individual or population level. We measure the variance of fitness throughout evolution, and we can, therefore, reveal the mechanisms that lead to the selection of some trait combinations and some particular genetic architectures. For instance, we can measure the correlation between traits, relate it to fitness at each generation, and simultaneously observe whether these correlations between traits foster – or not - the building of actual non-random allelic structure within the genome. In this way, the model can also be used to assess the robustness of some previous and more simple theoretical models, in a more realistic framework. It allows us to show, among other things, that the building of genetic correlation between mating preferences and a trait - assumed by quantitative genetics approaches and at the core of Runaway selection [Fisher 1915, Lande 1981] - also occurs in our more complex model and explicitly arises from assortative mating process (which itself emerges from the evolution of preference and competitiveness, and interac-

tions between individuals during the breeding season). Our results also support the idea that selection pressures may substantially shape the distribution of pleiotropic effects among genes [Cheverud 1996, Hansen 2003, Pigliucci 2008]. Additionally, predictions at equilibrium are of particular interest when we consider to what extent different replications for the same scenario may or may not converge. The difference between replications can be seen as a proxy of variance between populations, indicating that despite having the same initial conditions, environment, and constraints, two populations may diverge to some extent (when not related by dispersal, in the current model). This is of major interest for empiricists trying to investigate parallel evolution of populations experimenting similar environments [Schwartz & Hendry 2007, Oke *et al.* 2017, Bolnick *et al.* 2018]; in some cases, one should not be surprised to find substantially variable evolutionary outputs. In the core of our model, it is noteworthy to underline that such divergences mainly occur due to interactions between individuals (such as mate choice), and are therefore highly sensitive to stochastic processes. We suggest that such a mechanism could also play a major role in natural populations, stressing the importance to study and understand behavioural interactions in relation to the environment.

Obviously, a central concern in this matter lies in the validity of the mechanisms and assumptions we made, and how well they allow to recreate either theoretical facts or natural patterns. Such question cannot be addressed independently from the simulations scenarios built by the user, since the sets of parameters (physical structure of the genome, ecological parameters, type of mating system, etc.) will directly impact the results. We can however give examples on how to connect some outcomes of the model to either theoretical or empirical expectations. For instance, when we investigated the coevolution between sexual preference and gametic investment, both traits evolved rapidly at the beginning - since we were unlikely to start

from an equilibrium. But during this phase of evolution, genetic variance of the traits increased. It later decreased when the population reached a pseudo-equilibrium. We thus reproduced a known result, which is the transient runaway as trait and preference eventually goes to a new steady state [De Jong & Sabelis 1991]. Additionally, the pseudo-equilibrium reached was admittedly stable. [Lande 1981] predicted in his model that stability in this case was only ensured when the ratio of trait-preference covariance over trait variance in the population was equal or below 1 (in case of equal mutation variance input for both traits). Above that value, the equilibrium becomes unstable. The said ratio was observed to reach values up to 0.8 over our simulations, but it never exceeded 1. The non-occurrence of unstable equilibrium could be due to finite population size and the resulting genetic drift, potentially erasing the genetic correlation [Nichols & Butlin 1989]; or it could be caused by the opportunity cost that emerges from the individual interactions in the model and slows down the evolution of preference [De Jong & Sabelis 1991, Kokko *et al.* 2015]. On a more empirical perspective, on the same example, we were able to measure evolutionary rates of about 0.06 Haldanes over 10 generations during the phase of rapid evolution with preference, and then of 0.005 (over 10 generations) at the pseudo equilibrium. These values are in agreement with empirical studies: for instance, Karim *et al.* (2007) reported rates of male color change following an experimental introduction that ranged from 0.01 to 0.031 Haldanes over 13–26 generations. These values are very close to ours taking into account that we use shorter time frame (10 generations), given that evolutionary rates are higher over short time intervals [Hendry & Kinnison 2001]. Overall, the speed of evolution in the model matches general expectations on rates of evolution [Gingerich 2009].

Lastly, concerning the genetic architecture, we predict variance in the allelic values between loci in all the mating systems, some genes having therefore strong

effects and others small effects. Additionally, some mating systems seems to promote the establishment of modular genetic architecture. Such prediction are difficult to validate at the moment since I could not find previous work relating some general characteristics of mating systems (e.g. the absence or presence of preference or competitiveness during mating) to global characteristics of the genetic architecture (e.g. oligo-genicity, modularity) of sexual traits and behaviours. Fitzpatrick (2004) started to address this question looking at the genomic location and pleiotropic effects of sexually selected genes of *Drosophila melanogaster*. However, to be potentially linked to my predictions a measure of the direction of variation of pleiotropic effects at the gene level would be needed (i.e. when a gene have opposite effects for two traits we can speak about modularity); and/or an estimate of the level of variance of allelic values in the genome. The difficulty I have in relating my predictions to empirical findings also lies in the particular vision of genetic architecture that I adopted. Indeed, I assumed that each allele has values for several traits (i.e. universal pleiotropy) and I look at the emergence of a particular organisation of allelic values. This vision depart from some other representations of genetic architecture. For instance, [Pepper 2000] explicitly represented genetic architecture in an individual based model, assuming that each gene codes for only one trait, and look at the change in the localisation of the genes over the genome. With this approach genetic architecture is modular if sets of genes with epistatic effects on fitness are physically clustered on the chromosomes. [Guillaume & Otto 2012] adopt an other model of genetic architecture allowing pleiotropy being governed by a modifier loci. An other difficulty to compare my results, lies in the absence of descriptors of genetic architecture organization. The measures proposed in genomics (measuring the pleiotropy of certain genes and the polygeny of certain traits) do not really give information on the overall organization of the genome (Phillips 2005) and are difficult to compare

with theoretical predictions (where all the genetic architecture underlying traits is known).

Beside, environmental and genetic parameters significantly affect the evolution, and the model can be used to simultaneously test for the effect of habitat resource (K), social parameter (MG), and genetic constraints (mutation rate μ as well as the physical structure of the genome). All of these parameters will likely affect the genetic variance, which will in turn condition the speed of evolution. The study of these effects is central to the understanding of biodiversity dynamics and resilience: for instance the model could be used to investigate variable mutation rates in time or along the genome, or to explore the effect of fluctuating resource in the environment or factors affecting the distribution of potential mating partners.

To begin these investigations, in the limited time I had, I focused on two particular case studies, which I think should benefit from being studied in a demogenetic framework: First, I investigated the effect of a component of the social environment: the mating group size (which constrains the number of potential partners) on the evolution of traits and on genome architecture. The effect of social environment (in particular the quantity and phenotypic distribution of available mate) on mating behavior has been demonstrated by other approaches notably game theory models. But its effect on genetic architecture evolution has not been extensively investigated yet. Second, I investigate how physical constraints on genome can affect the evolution of traits and of genome architecture. Because physical structure of the genome does not change in the present model, I thus assume that such structure is stable on hundred to thousands of generations, and can be seen as a potential obstacle to reaching evolutionary optima, as opposed to the phenotypic gambit usually invoked in Behavioural Ecology.

The effect of social environment on the evolution of mating behaviours and genetic architecture

Contents

4.1	Introduction	100
4.2	Increasing the fluctuations in mate availability	102
4.3	The effect of mating group size on the mating dynamics . .	103
4.4	Effect on trait evolution	106
4.5	Effect on genetic architecture	109
4.6	Discussion	116

4.1 Introduction

Social environment is a central matter in the evolution of traits, especially traits under sexual selection. It influences both the competition to access sexual partners, and the dynamics of phenotype availability. In essence, the social environment is always in motion, and its dynamics may constantly affect the strength and direction of selection. It thus plays a central role in the evolution of mating behaviours, as demonstrated by game-theory approaches [Fawcett & Johnstone 2003, Barry & Kokko 2010, Etienne *et al.* 2014, Courtiol *et al.* 2016, Dechaume-Moncharmont *et al.* 2016, Chevalier *et al.* 2020] and pointed out by empirical studies [Candolin & Salesto 2009, Wacker & Amundsen 2014, Amundsen 2018]. For instance, in guppies, males appear to decrease their choosiness when competitors attractiveness is high [Gasparini *et al.* 2013]. An important aspect of the social environment is the OSR : its effect on mating behaviour and interactions of individuals is reported in several species [Emlen & Oring 1977, Kokko & Rankin 2006, Weir *et al.* 2011, Arnocky *et al.* 2014, Noë 2017]. The OSR reflects a difference in terms of the number of available partners for each sex influences the intensity of intra-sexual competition to mate. But beyond the unbalance between the sexes reflected by the *OSR*, the number of potentials partners and their phenotypic distribution is an important component of the social environment for both sexes as it directly affects the number of alternatives from which individuals can choose. So the risk of not finding the desirable or desired mate depends on the frequencies of available partners and on their phenotype.

Such context dependent selection is likely to have a strong effect on the evolutionary trajectory, as demonstrated in [De Jong & Sabelis 1991] and [Kokko *et al.* 2015],

where considering a preference-dependent risk that females remain unmated, or a sampling cost of preference (which depends on the number of sampled male rather than being fixed) allows female preference and the male trait to coevolve towards a stable equilibrium in situations where an unstable equilibrium was predicted by other models.

And because mating behaviours are pivotal in determining the way genes are transmitted to the next generation, the social environment is likely to have rippling effects on the evolution of genetic architecture. Indeed, when selection varies over time and space, sex-ratio and population size vary, genetic architecture built up by past selection can become detrimental. For instance, pleiotropy and genetic linkage resulting from previous correlations selection can maintain genetic correlation between traits when it is no longer advantageous (i.e., evolutionary load, [Hendry & Gonzalez 2008]). Genetic correlations between life history traits across environment can be highly variable, possibly underpinning different past and present regimes of selection to uncover [Stearns *et al.* 1991, Sgrò & Hoffmann 2004, Carlson & Seamons 2008], although the part of social environment in this is seldom investigated.[Kopp & Hermisson 2006] however indicated that under frequency-dependent disruptive selection, evolution favors a highly asymmetric genetic architecture, where most of the genetic variation is concentrated on a small number of loci. Some genetic architecture may thus better adapt to dynamic and changing conditions, such as the social environment. A leading hypothesis is that modularity of systems mainly emerges because of rapidly changing environments as higher modularity have higher adaptability and therefore higher survival rates in changing environments [Lipson *et al.* 2002, Kashtan & Alon 2005, Parter *et al.* 2007, Clune *et al.* 2013]. We here investigate if this pattern is found at the scale of the genome.

4.2 Increasing the fluctuations in mate availability

In natura, constraints on mate availability can be due to the characteristics of the mating systems, the density of the population or the spatial distribution of individuals, in relationship with landscape characteristics. Partner availability is therefore partly constrained at the beginning of the breeding season, but it is also dynamic within a mating group as a result of the pairing process over the breeding season: as individuals get paired, they become unavailable within the mating group. In the following simulations scenarios, I manipulate indirectly mate and phenotype availability by changing the mating group size. As a reminder, mating group size determines in the model the size of the group within which an individual will interact and attempt to reproduce during the breeding season. New mating groups are drawn at the beginning of each breeding season. Reducing the mating group size thus mechanically increases the odds of having an unbalanced OSR within mating groups, as well as the variance of phenotypes within and between mating groups. This may in turn increase the variability of mating opportunities between mating groups and from one breeding event to another, and so increases mating uncertainty. By reducing the mating group size we expect a stronger sexual selection as mating opportunities are fewer, so we should observe more individuals that do not reproduce. A variable environment is also expected to select for less risky reproductive strategies, which allow to average the risk of reproductive failure over several breeding seasons for instance. We should then observe evolution toward K strategies and iteroparity [Seger 1987, Benton & Grant 1999, Gremer & Venable 2014]. Yet, this prediction can be modulated according to the traits considered, for example certain traits allowing individuals to reduce their social environment stochasticity could be selected in spite of their potential cost on survival; such as competitiveness because

it may help to ensure a mating partner.

In addition, individuals could on average better pass on their genes to future generations by producing offspring with different strategies from each other, which increases the chances that at least some offspring will perform well in this unpredictable environment [Lande & Shannon 1996] and this offspring would inherit of modular genetic architecture, provided our manipulation of mating group size generates enough stochasticity.

Therefore, at the individual level, I would say individual modularity should have a fitness advantage within the population at any moments. We can test this hypothesis by looking at the relationship between the individual level of allelic correlation and the lifetime reproductive success. At the population level, one may wonder if such selection will lead a non random organisation of the average genome in the population, maximizing variance of allelic values within loci.

To explore those questions, I explore four mating systems (random mating, preference driven mating, competitiveness driven mating, and preference and competitiveness driven mating). I simulate 4 different mating group size (MG), with $MG = [N, 300, 100, 30]$. All other parameters are similar to exploratory simulations in §3.2: $K = 10000$, $\nu = 10^{-4}$, the genetic map is composed of 10 unlinked loci distributed over 10 chromosomes. Simulations are run over 5000 generations.

4.3 The effect of mating group size on the mating dynamics

A smaller mating group size increases the number of unmated individuals relative to the population size, in all mating routines (Fig.4.1). When mating is random, a smaller mating group increases the percentage of unmated individuals after a

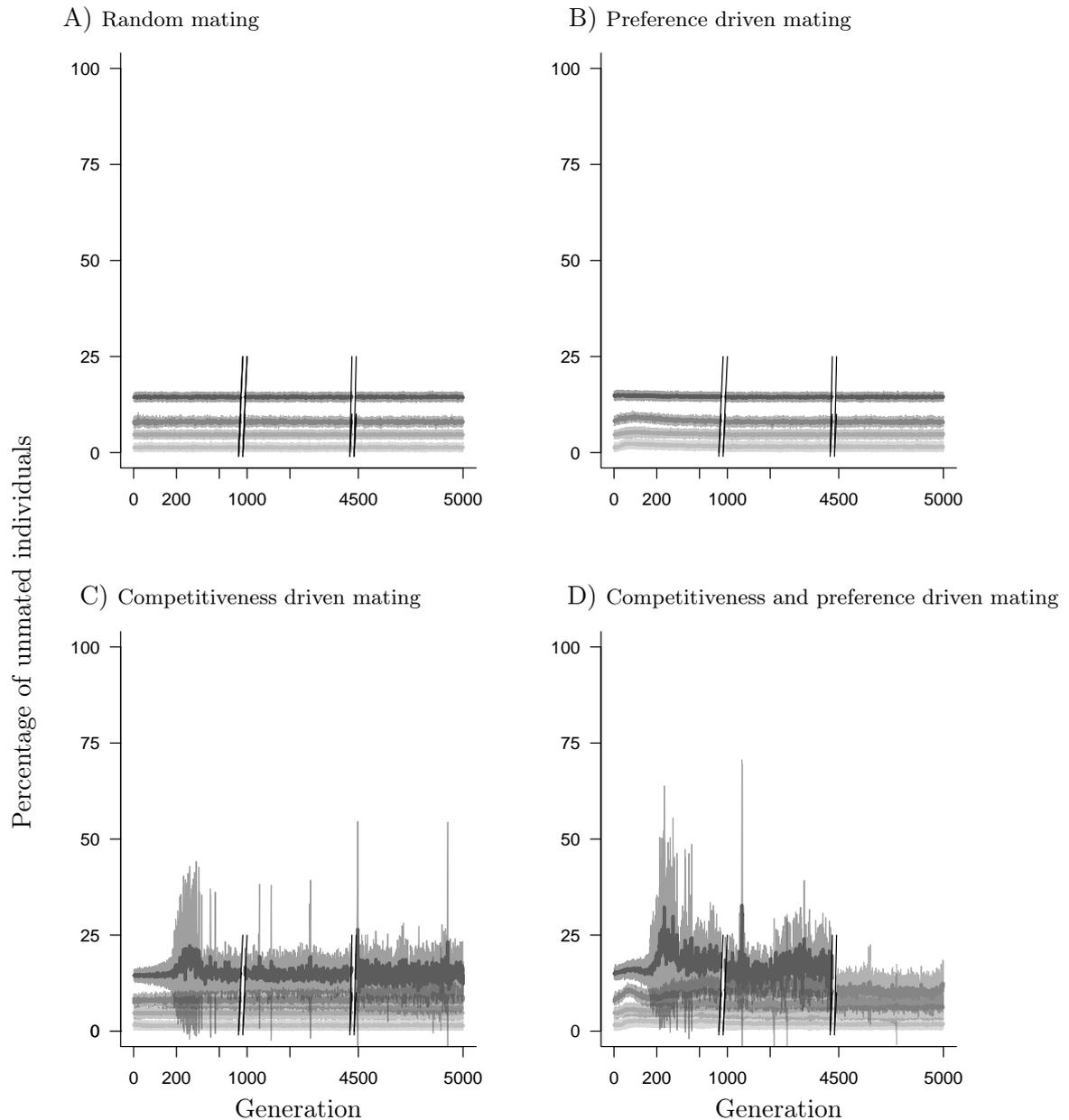


Figure 4.1: Percentage of unmated individuals (i.e., that fail to reproduce during a breeding event) for different mating scenarios and mating group size. The plot show the mean (dark line) and SD values (grey filling around the line) calculated over the 30 replication of simulations conducted for each mating scenario and each mating group size. The different panels correspond to different mating routines: (A) random mating , (B) preference driven mating, (C) competitiveness driven mating, (D) competitiveness and preference driven mating; and the shades of grey correspond to the with mating group sizes (from light gray to dark gray: $MG = N$, $MG = 300$, $MG = 100$, $MG = 30$). In the scenario of competitive encounter with preference and $MG = 30$, in all the simulation the population goes extinct after 4500 generations.

reproduction (up to 15% when $MG = 30$, Fig.4.1, A). Indeed, as the sex ratio can be biased in a mating group, not all individuals can be matched. Almost the same pattern is found when mating involves preferences (Fig.4.1, B). So here the presence of preference does not change the risk of missing a reproduction, as compared to random mating. This can be explained by the unimodal form of preference in our scenario, so that the value of the preference evolves with the distribution of the G trait in the population, allowing the preference to increase without cost. One might expect different results if the preference were a form of threshold function, where only G values above the threshold are accepted, potentially leading to the rejection of more potential partners -if preference were to change under such conditions-.

In these two mating scenarios, a smaller mating group size does not increase the variability of mating success from one breeding event to another as the percentage of unmated individuals remain constant over generations, so a reduction in the mating group size just increases the mean risk of missing a reproduction for individuals. However, because preference targets gametic investment, opportunities for sexual selection may become greater as mating group size is reduced. When mate encounter is based on competitiveness, a small mating group also increases the average risk of missing a reproduction. But, it now also triggers high variability in mating success. It first does so at the inter-simulation level (as indicated by wide SD in the percentage of unmated individuals among simulations especially at some point of the evolution; for instance after 200 time steps), indicating that in these conditions, evolution of competitiveness can be a strong determinant of sexual selection (Fig.4.1, C, D). But it is also the case between the mating groups within a single population (i.e., within a single simulation), or from one breeding event to another. This results hints at the strong context dependence generated by the inclusion of the competitiveness in the model.

4.4 Effect on trait evolution

The size of the mating group affects the evolution of traits as soon as mating is not random. When mating is random G evolve toward the same value at the equilibrium, whatever the mating group size, the population size at the equilibrium slightly decrease with smaller mating groups (being on average equal to 3500 when $MG = N$ and 3250 when $MG = 30$, data not shown), as when the mating group is small some individuals can not mate, therefore reducing the effective population size. When mating is driven by preference, a smaller mating group prevents the conjoint evolution of G and P toward extremes values (Fig.4.2). As the risk of not finding a suitable partner increases, the evolution of the G and P are reduced. Yet G and P still co-evolve as reflected by lower but globally positive genetic correlations between them when MG is reduced (Fig.4.3). It is noteworthy that the reduced values of P and G produce an increased survival, and therefore, an evolution toward more iteroparity, as a response to the increase of stochasticity of the social environment and reduced mating opportunities. And in fine this allows to reach an higher population size at equilibrium (being on average equal to 2900 when $MG = N$ and 3400 when $MG = 30$, data not shown). Inter-simulations (= between populations) variance decreases when mating group size is reduced, as shown by the evolution of P and G toward very similar values in the simulations (Fig.4.3). Yet I had rather thought that more stochasticity in the pairing dynamics with smaller MG would have promoted inter-simulation variability, by amplifying the importance of stochasticity on evolution. Probably, a smaller MG triggers stronger sexual selection (as reproductive opportunities are reduced) leading to a more deterministic outcome of evolution. When mating is driven by competitiveness, a decrease in the mating group size selects clearly for increased competitiveness, as competitiveness provides

an advantage by ensuring reproduction in a context where the number of possible partners is small and fluctuating (Fig. 4.5). Interestingly, the size of the mating group affects the genetic correlation between G and C (data not shown): Relatively big mating group ($MG = N$ and $MG = 300$) favor negative correlation between G and C . This is likely the result of divergent selection for the 2 traits, high values of G and low values of C being favored; and leads to individuals reasonably share their reproductive investment between G and C . Whereas in smaller mating groups ($MG = 100$ and $MG = 30$), a positive genetic correlation between G and C arises during the dynamic phase of evolution, as both high values of G and of C are initially favored. This triggers a co-evolution of G and C toward extremes values - making individuals to invest more and more into reproduction -, and leads to evolutionary suicide in the majority of simulations when $MG=30$ (Fig.4.4) (extinction rate of about 0.84 when $MG = 30$). Such extremely semelparous strategy leads to very low population size in the simulations that do not crash (being on average equal to 2800 when $MG = N$ and 250 when $MG = 30$, data not shown, with an extinction rate of about 0.9 when $MG = 30$). Lastly, when mating is driven by preference and competitiveness, reducing the mating group size still tends to select against preference and to favor high competitiveness. In this scenario the preference increases the occurrence of evolutionary suicide when the mating group size is small (with an extinction rate of about 0.5 when $MG = 100$ and of 1 when $MG = 30$). Preference has not a direct cost of survival here, but reduces the number of mated individuals per generations, reducing the reproductive rate of the population and so increasing the risk of extinction. Even if the role of sexual selection on the extinction risk is rarely considered, it has been acknowledge that sexual selection can drive a a population extinct in the case of variable and unpredictable environment [Kokko & Brooks 2003].

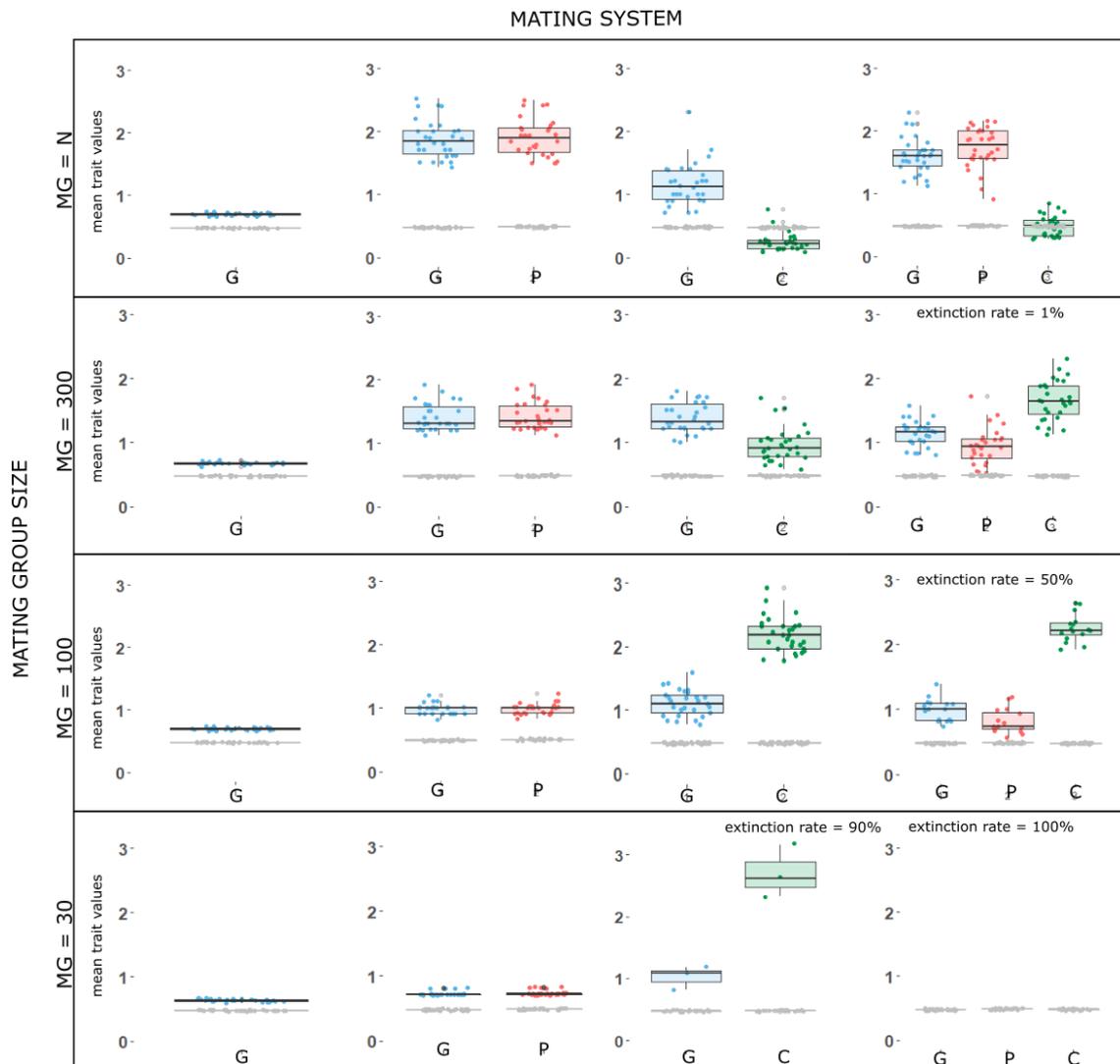


Figure 4.2: Mean values of reproductive traits at the pseudoequilibrium, for the 4 mating routines and for different mating group sizes. 30 replications are conducted for each scenario.

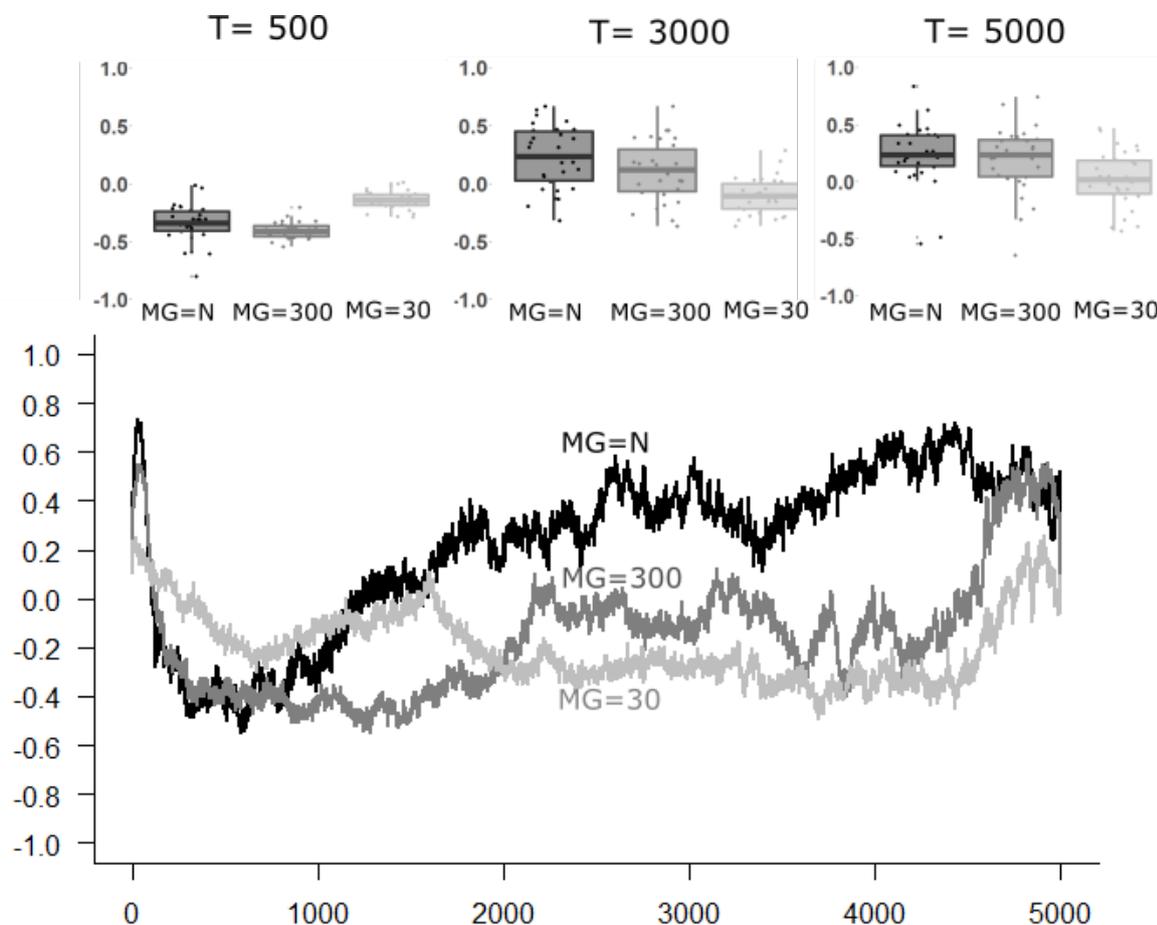


Figure 4.3: Genetic correlation between G and P in simulations where mating is driven by preference with different mating group size. The boxplots show the correlation in the 30 different simulations at different times step, the lower panel show the evolution of the correlation for 3 simulations each with a different MG .

4.5 Effect on genetic architecture

When mating is random there is no special pattern of allelic values correlation for G and any selectively neutral trait (here P which is not expressed), and so whatever the mating group size (Fig.4.8). This scenario serves as a reference as an allelic correlation pattern that is not under selection to which we can compare other scenarios. When mating is driven by preference, a negative correlation between allelic

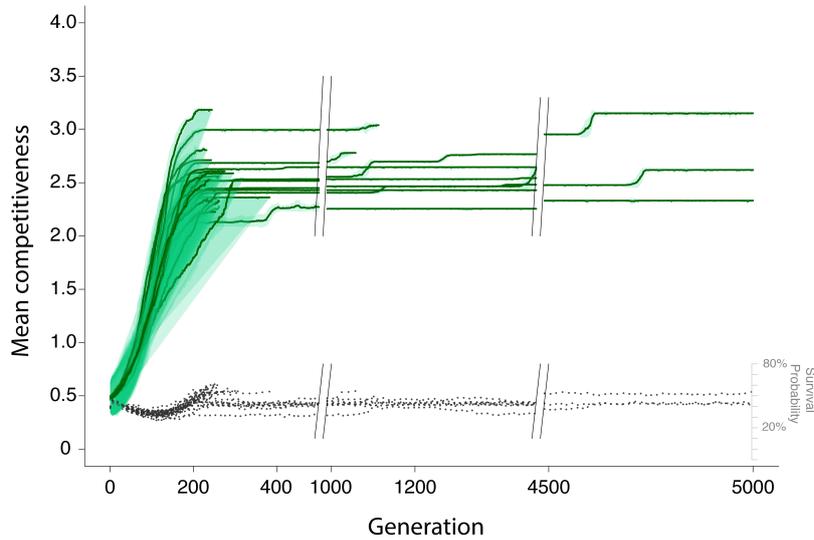


Figure 4.4: The evolution of the mean value of competitiveness (and sd) over generations.

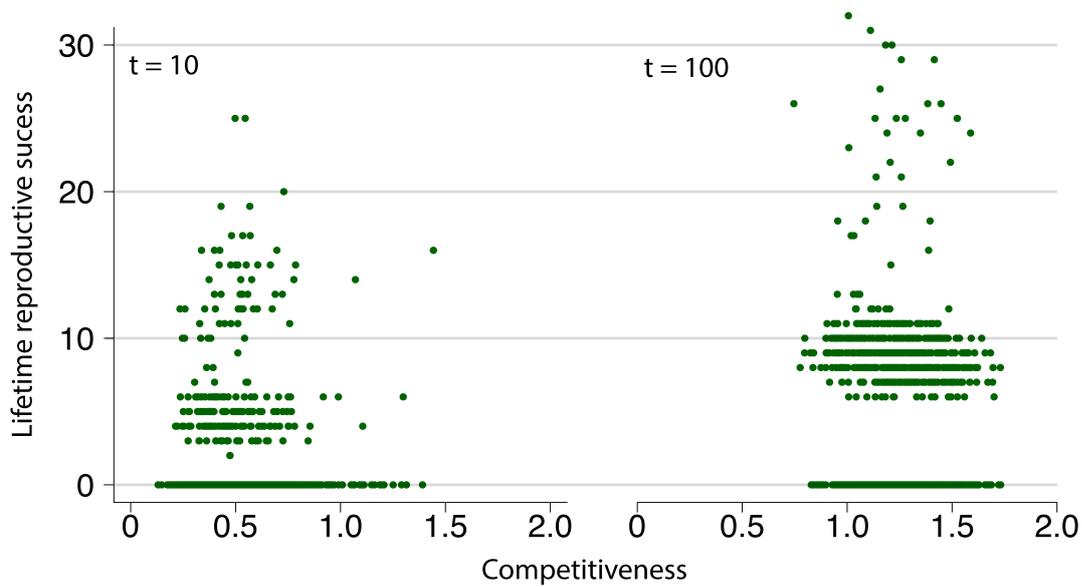


Figure 4.5: Lifetime Reproductive success of individuals as a function of their competitiveness at two different time steps. At $t = 10$, higher values of competitiveness clearly provides higher reproductive success (directional selection), which explains why the distribution of the trait in the population is shifted from the higher values at time $t = 100$. From this time, a stabilizing selection is observed on the Competitiveness.

effects for G and P (i.e. modularity) arises; and is maintained but not amplified with small mating group size, even if, as shown above, the traits themselves evolve towards much less extreme values (Fig.4.8). Additionally, the emergence of modular architecture at the populational scale is also accompanied by a positive relationship between the lifetime reproductive success and the level of negative allelic correlation of individuals (Fig. 4.8). When mating is driven by competitiveness, no strong genetic organization emerges (Fig.4.8) whatever the mating group size. When mating is driven by both competitiveness and preference, a negative correlation between the allelic effects appears for all three pairs of traits, in most of the simulations with $MG = N$. With smaller mating groups ($MG = 300$), a negative correlation between the allelic effects for G and C and for C and P (i.e. modularity) is found in the vast majority of the simulations, whereas more simulations present positive correlation for allelic effects for G and P . An even smaller mating group size ($MG = 100$) will lessen this pattern of modularity in the genetic architecture observed for the $C - P$ and $G - C$ trait pairs. I have verified that the relaxed modularity is not caused by the reduced number of simulation reaching 5000 generations (because of extinction) by launching more simulation to get at least 30 simulations reaching the end. There is also no difference of genetic architecture between extinct and maintained simulations (data not shown). So genetic architecture does not affect the risk of extinction here. When mating group is even smaller, as populations goes rapidly extinct (within the first 1000 generations), we can only look at the genetic architecture before their extinction and there is no clear organization of the genetic architecture that appears (data not shown).

There is globally no strong effect of the mating group size on the inter-loci RSD of allelic effects, except in the mating system with preference and competitiveness driven mating, where reducing the mating group from $MG = N$ to $MG = 300$

increases the inter-loci RSD particularly for traits P and C (Fig.4.7). Remarkably, this corresponds to the traits that are most affected by a reduction in the size of the mating group with respect to allelic correlation (this is where we found the emergence of strong modularity).

Globally, Mating group size does not strongly impact the genetic architecture, yet some mating systems seems to promote modular and oligogenic genetic architecture (when mating is driven by competitiveness and preference and $MG = 300$).

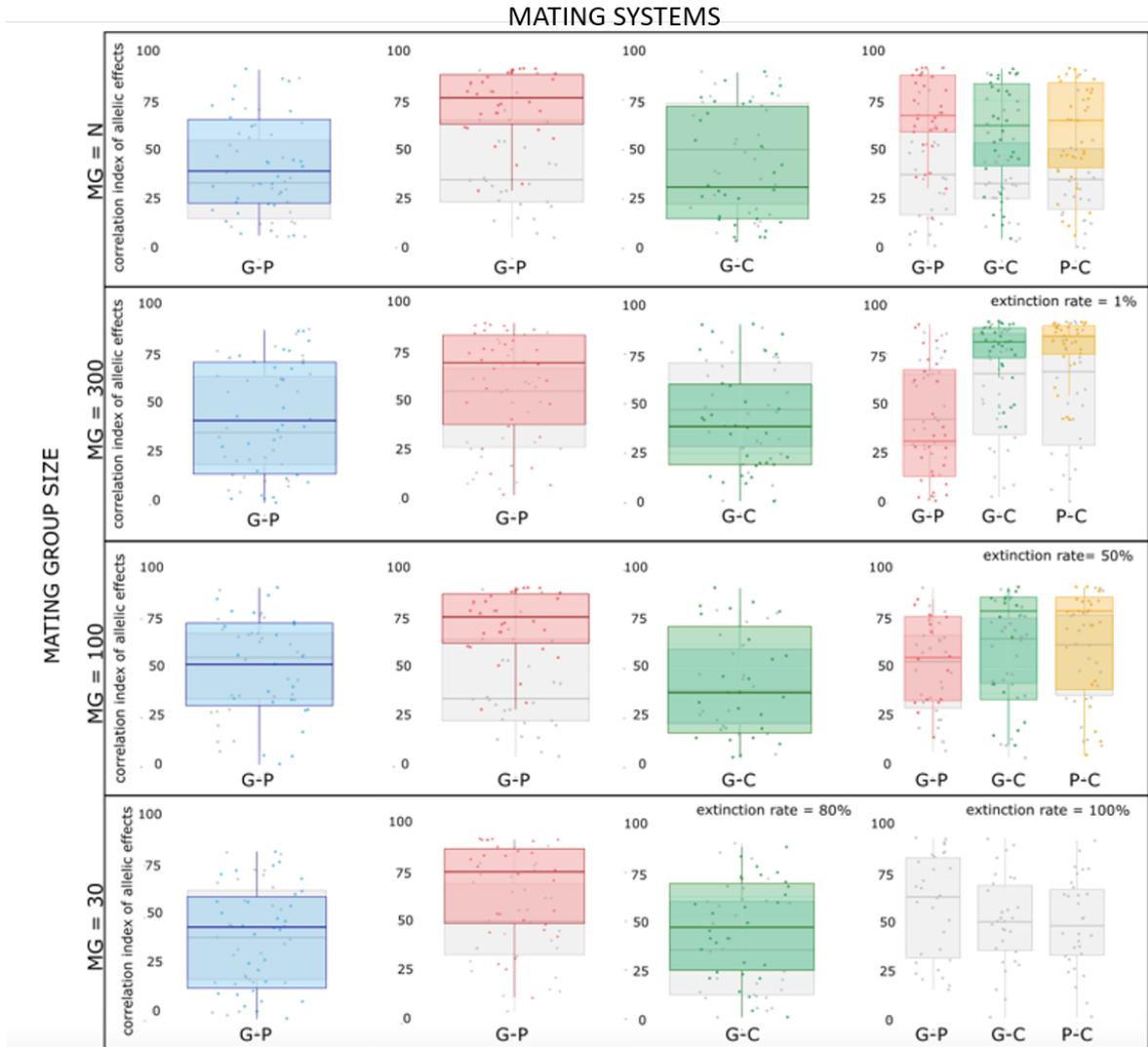


Figure 4.6: Index correlation of allelic values for the traits taken two by two. The measures are showed initially (in grey) and after evolution over 5000 time step (in color). The boxplot are drawn with the indexes calculated for 30 simulations of the four mating routines (random encounter, preference driven mating, competitive encounter, competitive encounter and preference driven mating) and for different mating group sizes ($MG = N$, $MG = 300$, $MG = 100$, $MG = 30$). A high index (100) signals a mean allelic correlation that is more negative than what could be observed by chance in the population (i.e. modularity). Conversely, an index close to 0 signals a mean allelic correlation more positive than what could be observed by chance in the population. 30 replications are conducted for each scenario. In the scenarios with some extinction risk (but which do not crash all the time), I conduct more simulations in order to get 30 simulations that do not crash.

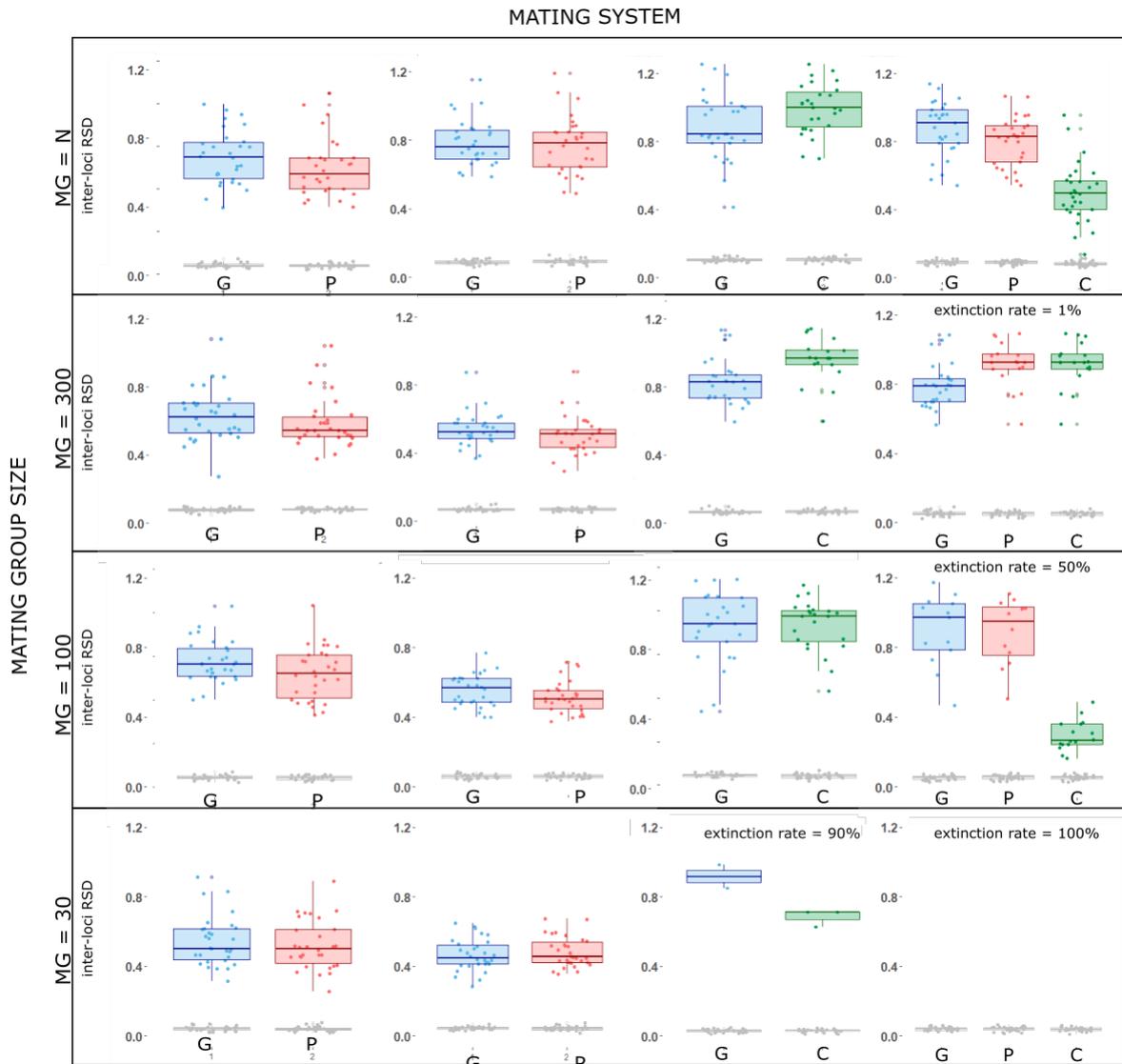


Figure 4.7: The relative standard deviation of the mean allelic values between loci for the different traits present in the scenarios (G, P, C, in the first scenario the preference is not expressed), initially (in grey) and after evolution over 5000 generations (in color). The boxplots are drawn with 30 simulation for the 4 mating systems scenarios (random encounter, preference driven mating, competitive encounter, competitive encounter and preference driven mating) and for different mating group sizes. The boxplots report the mean and 95% interquartile range interval.

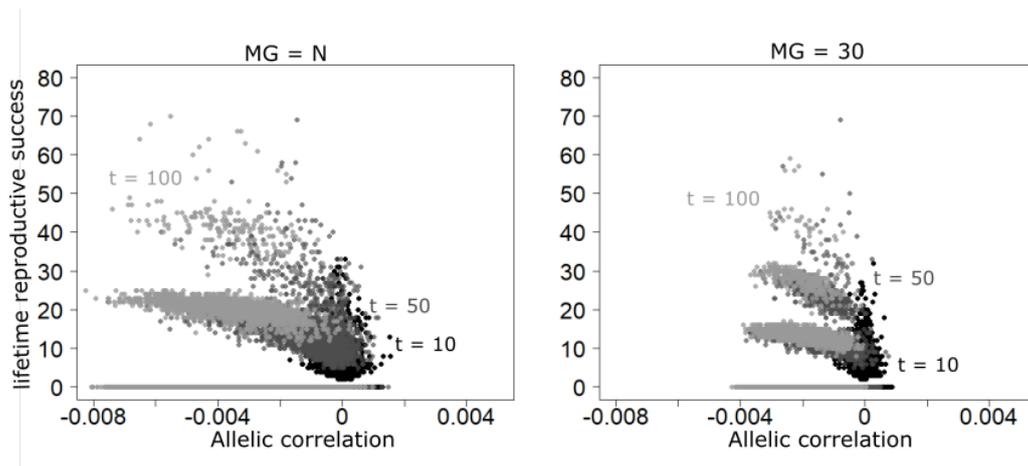


Figure 4.8: Relation between Lifetime reproductive success and individuals' allelic correlation, for the preference driven mating scenario and two mating group size, at two time step of the simulation ($t=10$, $t= 50$, and $t=100$). negative correlation correspond to modular architecture

4.6 Discussion

Changing mating group size has an effect on population dynamics, notably on the percentage of unmated individuals. It is true in a random mating scenario, where a small group generates a low effective population size (N_e in population genetics), and therefore stronger genetic drift. So, social environment affects genetic drift. It is also true for other mating systems, which translates into variance in mating success related to trait values, hence higher sexual selection most of the time.

As hinted above, stronger sexual selection in non random mating systems leads to various outcomes in terms of trait values. The social environment, here mating group size, controls strongly the evolution of both preference and competitiveness, changes the evolution of iteroparity, affect population size, and possibly leads to evolutionary suicide in some cases. Additionally, the genetic correlations between traits are strongly influenced by the social context.

Changing mating group size tends to affect genetic architecture, yet not systematically in all mating scenarios, and sometimes in a non linear way. For instance reducing MG from N to 300 reduces the genetic correlation of allelic values, but decreasing again MG to 100 seems to favour again the emergence of negative correlation. Such non linear effects could be due to different effective population size in the different mating systems according to the size of the mating group. But globally, a decrease in the mating group size - therefore when the social environment is less predictable - leads to a decrease in the modularity of the genome. Or, in other terms, predictable situations tend to select for modularity, whereas less predictable tend to limit such modularity, with correlation between allelic values following a near random pattern. This is in accordance with the idea that maximally variable pleiotropy may optimize evolvability by increasing the potential for genetic variance per trait

[Hansen 2003, Chevin *et al.* 2010]. This prediction on patterns are confirmed by the micro-evolutionary process observed during the dynamic phase of evolution, wherein more (modular) random individuals have a higher fitness during the dynamic phase (4.8). As shown in the previous results, social environment may have consequences on the genetic architecture over a hundred to thousands of generations: absence of reproductive isolation during breeding period appears to build modularity, whereas reducing mating group size tends to randomize the distribution of allelic correlation in the genome. If we broaden the perspective, and consider that for small mating groups, part of the populations can go extinct, it certainly makes evolution less predictable, as we have shown in the present case that the evolutionary suicide did not seem related to the genetic architecture. - Interestingly, high correlation between allelic values, which can be seen as a form of pleiotropy, seems to be never selected for in the present simulations. Browsing the literature, it is in fact difficult to find explicit and supported hypotheses that would conclude to the adaptive evolution of pleiotropy, in particular in relation to sexual selection.

Pleiotropy is partly addressed in literature on sexual selection: It is sometimes considered as a factor of genetic correlation in some quantitative genetic models [Lande 1982, Lande & Arnold 1985]; or as a mechanism linking sexual traits to others fitness related traits. This kind of pleiotropy underlies "magic traits", [Kokko *et al.* 2003, Servedio *et al.* 2011, Shaw *et al.* 2011], could also prevent sexually selected genes from being lost, and could maintain genetic variance in sexually selected traits [Rowe & Houle 1996]. But I did not find papers specifically addressing the evolution of pleiotropy between sexual traits/behaviours evolving as a by product of sexual selection. Generally speaking, it is predicted that pleiotropy evolves when there is a constraint on the genome [Guillaume & Otto 2012], but in our case, the only constraint is the number of loci.

As I had hypothesized, the context-dependence is largely influencing the evolution in the model. Changing variance in social environment has strong effects on population structure and on traits evolution, favoring more iteroparous strategies in the preference driven mating system, but more semelparous strategies when competitiveness is evolved. Some effects on genetic architecture also emerge and more variability in the social environment seems to favor random association of allelic effects, possibly because smaller mating group reduce the effective numbers of breeders N_e , and may limit the target for selection. Yet, more investigations would be necessary in order to fully grasp the reasons that lead to selection of more modular genome when environment is more predictable.

All these findings however could be very influenced by the simulation scenarios envisioned here, and the simulation parameters. It is my opinion that the question of the effects of social environment on evolution of mating behaviours and genetic architecture deserve further investigations, especially in the demogenetic framework.

How physical constraints on the genome architecture affect the evolution of traits and genetic architecture

Contents

5.1	Introduction	120
5.2	The effects of genome size and linkage disequilibrium . . .	123
5.3	The effects of sexual chromosomes:	127
5.4	Discussion	133

5.1 Introduction

As I described in Chapter 2, models in evolutionary ecology envisioned different type of genome physical structure and transmission. Many assume, more or less explicitly, extremely simple models for genetic architectures, up to the phenotypic gambit, where it is acknowledged that genetic architecture, and therefore transmission, is not a constraint on evolution to reach adaptive equilibria. Classical quantitative genetics models make strong assumptions about the underlying physical structure of the genome (infinitesimal number of loci with additive small effects), and make deductions about the net effects of all loci affecting the trait by partitioning the total phenotypic variance into components attributable to additive, dominance and epistatic genetic variance, variance of genotype-environment interactions, and other environmental variances. But the known genetic architectures of traits often differ from this model. For instance, some traits are encoded by a relatively small number of loci. In guppies, a handful of genes control for orange colour in males, a trait under natural and sexual selection [Tripathi *et al.* 2009]. In Atlantic Salmon, a single locus with sex-dependent dominance can control up to 39 percent of variation in age at maturity [Barson *et al.* 2015]. Alternatively, while most of the fully explicit models (representing a finite number of loci and their locations) assume very simplified genetic architecture, the complex physical structure of the genome (loci and chromosome number, mutation rate) can affect the evolution of traits: with several loci, the role of recombination could complicate the pattern of long term equilibria and change predictions of simple optimization model (that often consider one diallelic loci controlling the strategy) (e.g.[Matessi & Eshel 1992]).

Additionally, a high number of loci would allow to maintain a high genetic variance in the face of strong selection that is the ground for traits evolution

[Lande 1975, Gavrilets & Hasting 1995]. Physical linkage among genes experiencing different kinds of selection can facilitate (by favoring co-adapted genes complexes, [Aeschbacher & Bürger 2014]) or hinder adaptation (as recombination could facilitates selection by breaking down the negative associations generated by random drift, [Barton 2010]). Finally, the presence of sexual chromosomes can strongly affect evolutionary dynamics and presents a way to resolve sexual conflict [Mank 2009, Promislow 2006], since some loci are only present and expressed in one sex. It also has been suggested that some genetic systems are especially conducive to sexual selection [Reeve & Pfennig 2003]. It is the case of the "Z" sex chromosome in female heterogametic systems ("ZW" systemes; notably found in birds), because in these systems females transmit their preference alleles to all of their sons, such as half of the sons possess both the "sexy trait" and preference alleles, favoring runaway selection [?]. In other cases, different genes coding for the same trait under sexual selection can be distributed on autosomes and on the male sexual chromosome, as orange color in guppies, which can facilitate evolution of the trait while still retaining some hidden genetic variation, since orange color is not really expressed in females [Hughes *et al.* 2005].

In this chapter, I investigate how some physical characteristics of the genome may impact the evolution of traits and subsequently the distribution of allelic values over the genome.

To that aim I envision several genetic maps, that are characterized by the maximum number of loci that can hold an allele coding for reproductive traits, the probabilities of recombination between loci during meiosis, and the presence or not of sexual chromosomes. Following the above mentioned literature, I hypothesize that the maximum number of loci available will condition genetic variance and therefore the speed of evolution. The linkage disequilibrium may affect the strength of co-

evolution between traits, and the presence of sex chromosomes combined with sex specific selective pressure may allow sex-specific mating strategies to evolve.

To investigate these hypotheses, I built 4 distinct genetic maps:

(1) In the first genetic map : 10 loci are represented and the probabilities of recombination between loci are of 0.5 , meaning that all loci are independently segregated during the meiosis. This scenario corresponds to a case of no linkage and may correspond to a situation where the genome is made of 10 chromosome pairs each carrying a single locus. It is the same genetic map that I already used for chapter 2 and 3.

(2) In the second genetic map: 100 unlinked loci are represented and the probability of recombination between loci are of 0.5. This scenario corresponds to a situation where the genome is made of 100 chromosome pairs each carrying a single locus.

(3) In the third genetic map : 10 loci are represented, distributed on 2 chromosomes (probability of recombination between genes from different chromosomes are 0.5, and recombination probabilities within chromosomes are 0.01. This scenario is therefore a case of strong linkage disequilibrium between some loci.

(4) In the fourth genetic map : Male and female sexual chromosomes are represented in addition to autosomes. Sexual chromosomes are represented as haploid portions of genome that are only expressed in either males or females. 5 loci are located on autosomes (with a recombination probability between each other of 0.5) and 5 loci are located on each of the sexual chromosomes (and are not subject to recombination). The expression of sexual chromosomes are sex-specific, so regardless of gender, individuals inherit both male and female sexual chromosomes, but they express only one of them depending on their sex. The representation of sexual chromosomes here does correspond to some genome structure found in plants

for instance, wherein mitochondrial and chloroplast DNAs are transmitted along nuclear DNA. Other types of chromosomal sex determination are found in nature: male chromosome (XY systems, as in mammals), female chromosome (ZW systems, as in birds), and haploid phase determination (UV systems, as in some algae and bryophytes). They could be investigated as well in the future.

To get the same initial distribution of traits values for all genetic maps, alleles values are drawn in different Beta laws depending on the number of loci in the genome. Alleles values are drawn in a Beta law of shape parameters [0.65, 24.5] when the genome is composed of 10 loci (Fig. 3.3) and of shape parameters [0.06, 25] when the genome is composed of 100 loci (AppendixB4); under this latter distribution, many alleles have very small effect.

With this approach we place ourselves in a temporal framework wherein the physical structure of the genome remains the same: major genomic rearrangements do not occur, and genetic sex determination has already evolved with selection for the suppression of recombinational exchange over sexual chromosomes.

5.2 The effects of genome size and linkage disequilibrium

In this first section, I compare the effects of the first genetic map (10 unlinked loci) to the effects of the second (100 unlinked loci) and of the third (10 linked loci) genetic maps.

Effect on trait evolution:

Under random mating, there is no strong effect of the genetic map on the evolution of G (Fig.5.1). When preference drives mating, the joint evolution between G and P is affected by the different genetic maps: With a genetic map of 100 unlinked loci (versus 10 unlinked loci), the coevolution between traits is boosted towards

much higher values (Fig.5.1). This is so because the genetic correlation between traits reaches higher values and is maintained over a much longer period (Fig.5.2). So individuals invest a lot in reproduction at the expense of survival which is reflected by low population sizes at pseudo equilibrium (around 1000 individuals in the simulations with 100 loci versus 2500 in simulations with 10 loci). Focusing on the dynamic phase of evolution, with a higher number of loci, more time is needed to reach the pseudo equilibrium (about 1000 generations to reach the maximum versus 200) as the evolution of the traits is more extreme. Yet the evolutionary rate in Haldanes is also slightly higher when more genes are involved. For instance, during the most dynamic phases of runaway evolution, a peak rate of 0.08 Haldanes is observed for 100 loci versus 0.06 Haldanes for 10 loci. Likewise, after the runaway phase, during the evolution towards the pseudo equilibrium, the evolutionary rate is higher with 100 loci (0.02 Haldanes) than with 10 loci (0.01 Haldanes, data not shown). More loci also generate increased genetic variance during the dynamic phase of evolution (with SD for G and P situated around 0.9 with a 100 loci genetic map versus 0.4 with a 10 loci genetic map, data not shown). When mating is driven by competitive encounter ($G + C$), the number of genes involved seems to have relatively little influence on the evolution of traits, although a higher number of loci seems to preclude the negative evolution of C observed in the 10 unlinked loci scenario. However, as soon as sexual preference is accounted for ($G + P + C$), a higher number of loci does again promote the evolution of traits toward higher values, even for the competitiveness this time. Additionally, 25 percent of the simulated populations went extinct before 5000 generations in this scenario, pointing out the role of the genetic architecture in the fate of populations.

The inclusion of physical linkage (10 loci, 2 chromosomes) tends to impede the conjoint evolution of the traits in the mating scenario $G + P$ (Fig.5.1) as it limits

the rising of genetic correlation between the traits during the 100 first generations (Fig.5.2). So, in the present context, physical linkage did not favor the build-up of genetic correlation between traits. In the other mating scenarios, physical linkage also seems to limit the evolution of traits (Fig.5.1).

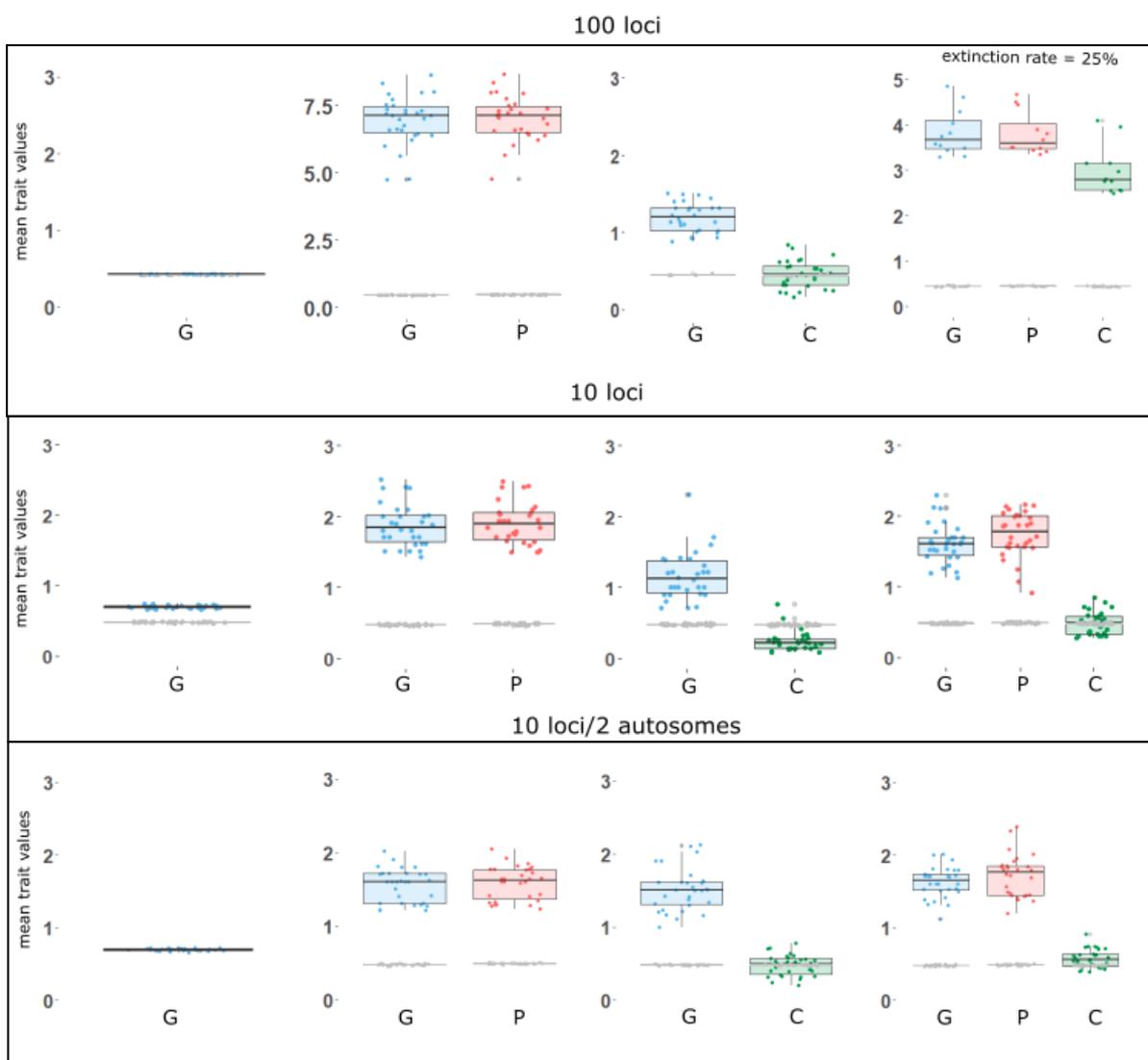


Figure 5.1: Mean values of reproductive traits for the 4 mating routines (random mating G , preference driven mating $G - P$, competitiveness driven mating $G - C$, and competitiveness and preference driven mating $G - P - C$) and for different genetic maps.

The physical structure of the genome affects the evolution of traits in situations where several traits can be co-selected (when preference and/or competitiveness are expressed together with gametic investment) and it has rippling consequences on population demography. For instance, in the case of preference driven mating, the population size at the pseudo-equilibrium goes from about 2900 in simulations with 10 loci to about 1000 individuals in simulations with 100 loci. And in the case of preference and competitive encounter scenario, the population size goes from about 2500 in simulation with 10 loci from 700 in simulations with 100 loci.

Genetic linkage seems to rather limit the evolution, one reason for that could be the "weak selection Hill-Robertson effect" [McVean & Charlesworth 2000]: closely linked selected alleles may interfere with each other, inhibiting the spread of favourable alleles and the elimination of deleterious ones [Fisher 1930, Hill & Robertson 1966, Birky & Walsh 1988]. Whereas a higher number of unlinked loci (via more mutational input) appears to promote the runaway process that leads to smaller population that triggers evolution of C .

Effects on genetic architecture evolution:

The effect of the physical structure of the genome on inter-loci RSD depends on the mating system considered. More loci strongly increase inter-loci RSD in the mating systems where competitiveness is involved (Fig.5.3).

The correlation index of allelic values in the different mating systems is slightly affected by the genetic map. Globally, when the genetic map is composed of 100 unlinked loci, the pattern of negative allelic correlations observed with 10 loci in some mating systems are amplified (in the mating scenario G+P and G+C+P Fig.5.4). More loci allow thus a higher modularity to emerge. This higher modularity may be linked to change in traits values (more extreme evolution) and also to a more

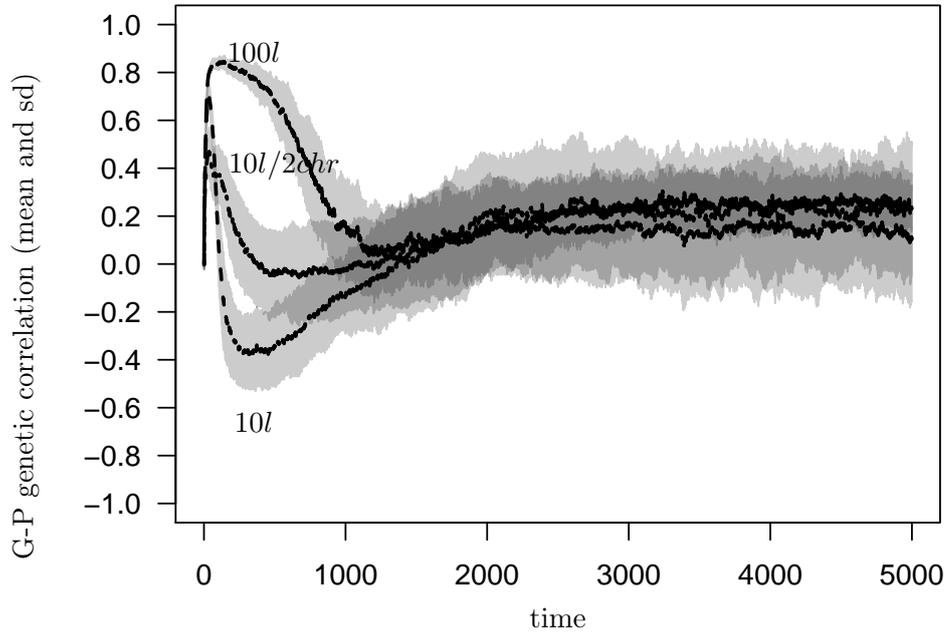


Figure 5.2: Mean genetic correlations between gametic Investment G and preference P over 5000 generations (black line, and standard deviation around the mean, grey area) for 30 replications of the three genetic maps (10 loci on 10 chromosomes, 10 loci on 2 chromosomes and 100 loci on 100 chromosomes).

spacious genome so that the allelic values have more room to be distributed in the genome.

On the contrary when loci are linked, the pattern of genetic correlation observed for the mating systems $G + P$ and $G + C$ are relaxed. The linkage implies that alleles at neighboring loci will be transmitted together with a high probability, thereby offsetting the effect of particular organization of allelic value at the locus level.

5.3 The effects of sexual chromosomes:

Up to now, I admit that the reader might be curious about how much my work revolves around sexual selection without really addressing sex differences in behaviour

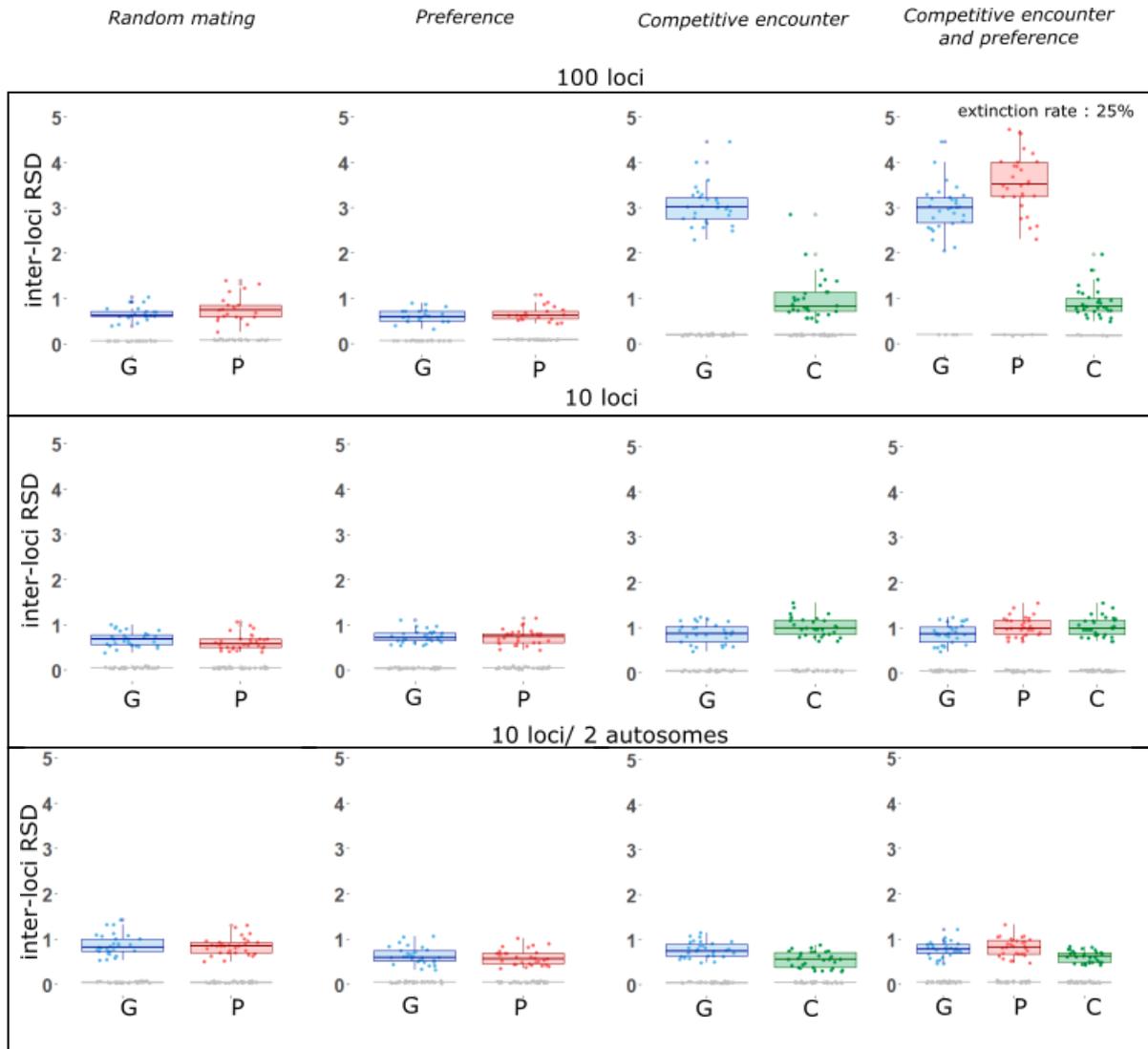


Figure 5.3: The relative standard deviation of the mean allelic values between loci for the different traits present in the different scenarios, initially (in grey) and after evolution over 5000 generations (in color). The boxplots are drawn with 30 simulations of the four mating routines (random encounter, preference driven mating, competitive encounter, competitive encounter and preference driven mating) and for the different genetic maps, and report the mean and 95 % interquartile range interval.

or traits, since the evolution of sexual dimorphism and sex roles is a major topic of interest in sexual selection. For my defence, sexual selection occurs without need for difference between sexes. As mate choice in both male and female can be found in

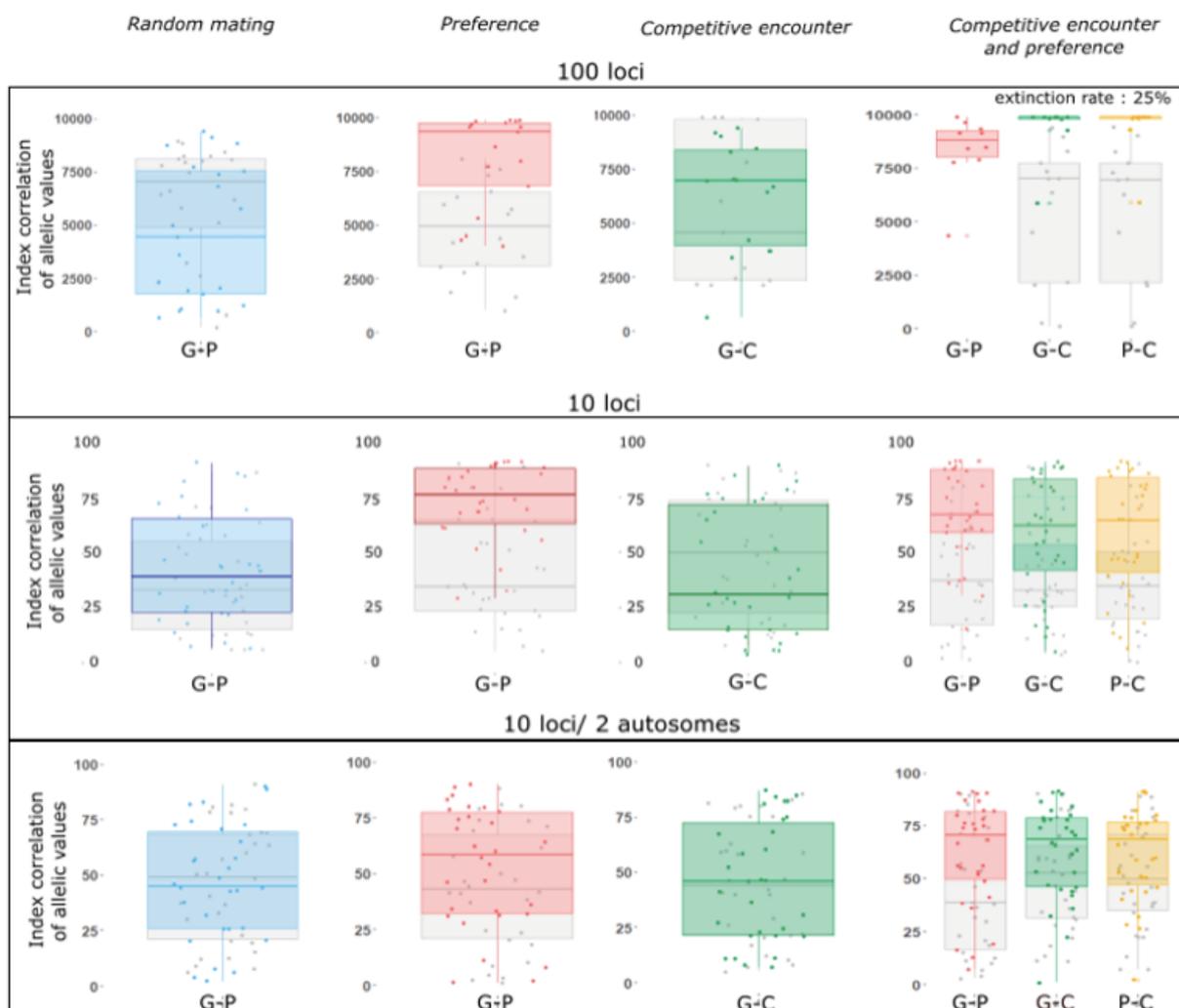


Figure 5.4: Index correlation of allelic values (calculated with a bootstrap approach) for traits for the for different genetic maps. The boxplot are drawn with the indexes calculated for 30 simulations of the four mating routines (random encounter, preference driven mating, competitive encounter, competitive encounter and preference driven mating) and for the differents genetic maps. A high index (10000 or 100) signals that the mean allelic correlation is more negative than what could be observed by chance in the population (i.e. modularity). Conversely, an index close to 0 signals a mean allelic correlation more positive than what could be observed by chance in the population.

many species, as well as competitiveness, I did not want to set in stone traits that are sex-specific (contrary to what is done most often). The sexes are first and foremost

defined by differences in the type of gametes they produce; the female sex produces relatively few, large and usually non-motile gametes (eggs or ovules), whereas the male sex produces many, smaller and often motile gametes (sperm or pollen). The evolution of different behaviours between males and females would then stem from anisogamy and the resulting sex-specific selection [Schärer *et al.* 2012] .

As a preliminary work, I simply suppose an asymmetry in the potential reproductive rate between males and females; females in this scheme incur a latency period after a reproduction making them unavailable for the next breeding event. This difference in the latency period between males and females is justified in many species by a global difference in the time needed to replenish the gametic stock due to anisogamy (which is present present in most taxa). Females thus have a higher breeding cost than males and a lower reproductive rate, being often the rarer sex in the mating pool of available mates. This is usually predicted to favor the evolution of female preference by game-theoretical models [Kokko & Johnstone 2002, Etienne *et al.* 2014, Courtiol *et al.* 2016]. If males and females share the same genetic architecture yet, sex specific selection can not shape the genome of the two sexes independently as the advantageous alleles for females are not the same for males (i.e. Intralocus conflict). A way to solve this conflict would be the evolution of sex linked gene expression (via epistasis and sex chromosome). So I expect that the addition sex chromosome in the genome will allow sex specific strategy to evolve. I also expect to see differences between the distribution of allelic values on autosomes and sex chromosomes. Indeed, genes having differential fitness effects on males and females (sexually antagonistic) are expected to be found on the sex chromosomes [Charlesworth *et al.* 1987, Hurst & Randerson 1999]. Also, sexual chromosomes are subject to a different strength of selection relatively to genetic drift, because each one is expressed in only half of the population (in the

sex or male or female), and this is susceptible to affect their genetic variance.

I thus investigate the effect of sex chromosomes in the genome on the evolution of traits, to understand whether sex-specific selection may shape the distribution of allelic value over autosomes and sexual chromosomes, but also whether a sex-specific portion of genome allows trait optimization for each sex. For this simulation, I choose to focus on the scenario of preference driven mating (with always the same set of default parameters, stated in Chapter 3 section 3.2), and compare the effect of the first genetic map (10 unlinked loci) to the effects of the fourth genetic map (5 nuclear loci, 5 haploid loci on female chromosome, 5 haploid loci on male chromosome).

Effects on traits evolution: The traits G and P conjointly increase in the population but toward lower values (about [1.2-1.4] here) than in the basic scenario without sexual chromosomes (values around 2, Chapter 3). Additionally, the traits values becomes slightly different between males and females. Even if the difference is thin, females globally evolve a higher preference and maintain it. Concerning G , during the dynamic phase higher values are also selected among females, but then the mean trait value of females finally stabilizes slightly below the mean trait value of males.

Effects on genetic architecture evolution: When looking at the spatialization of allelic effects over the genome in one simulation (Fig.5.6), one notices immediately that alleles with the strongest effects are located on autosomes, and that alleles located on maternal chromosome have globally higher positive effects for P than alleles located on male chromosome. This observation is confirmed when we replicate the simulation (data not shown). Additionally, the alleles selected on male chromosome globally have negative effects on G .

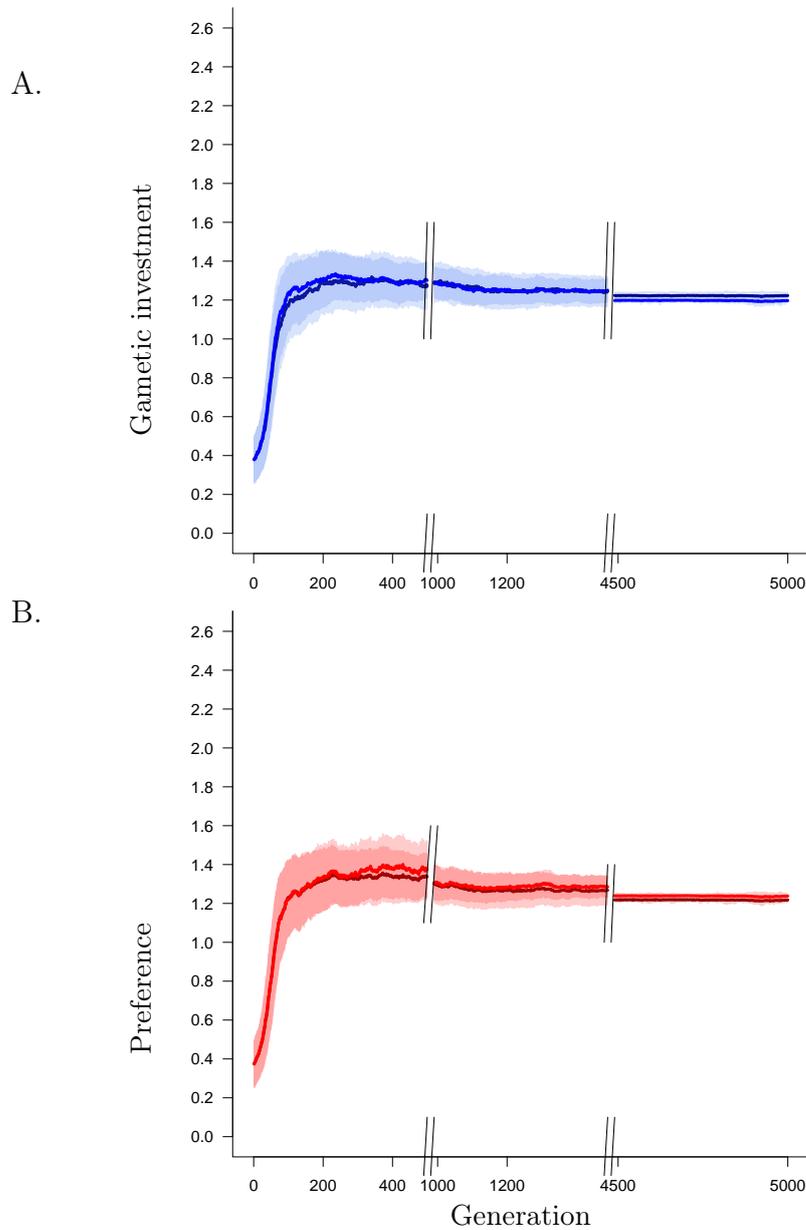


Figure 5.5: Evolution of G for males (in dark blue) and females (in blue) and P for males (in dark red) and females (in red) in the population over 5000 time steps, for one simulation of either (A) random mating, (B) preference driven mating. Mean values of traits are showed in thick lines and standard deviation values are represented in transparency.

The difference in the effect of alleles situated on autosomes and on sexual chromosomes may be due to the difference in the strength of selection and genetic drift.

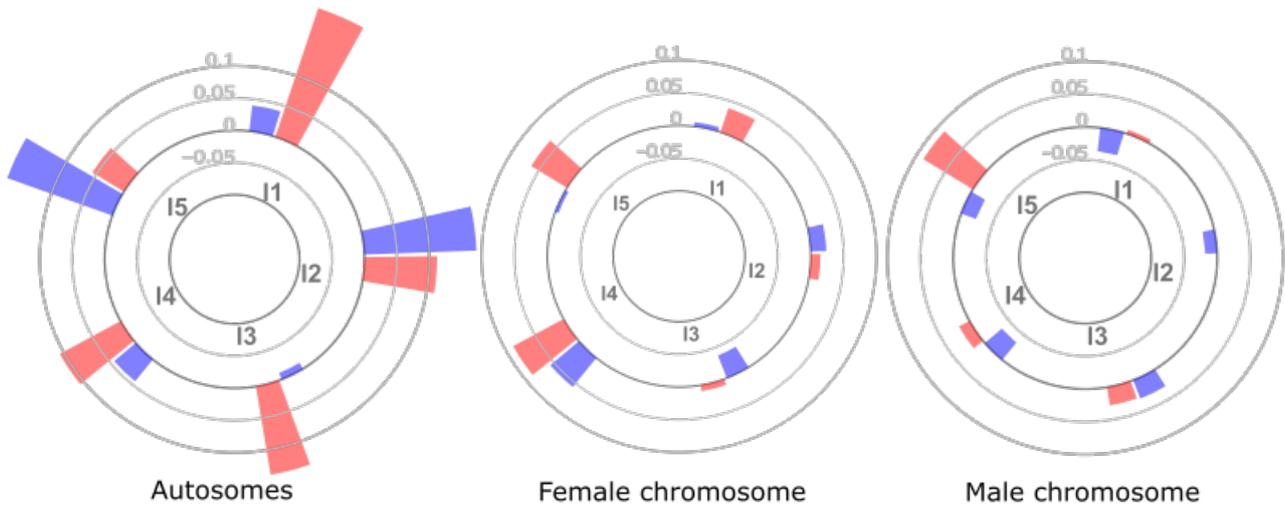


Figure 5.6: The mean allelic effects per loci in the population, on the autosomes and on the sexual chromosomes (wherein loci are linked). The height of bars at each locus represents the mean effect of alleles at each locus for the gametic investment (bar in blue) and preference (bar in red). Grey circles give the graduations for allelic effects. Alleles on sexual chromosomes are only expressed on either males or females.

Indeed, sexual chromosomes are more subject to genetic drift due to their haploid nature, and less subject to selection because they are expressed in only one sex, but physically present in both sexes: part of these genes are thus passed on to the next generations without undergoing selection at all. Nevertheless the allelic effects are indeed selected differently depending on the sex, causing different trait values depending on sex. We still found evolution toward a more modular genetic architecture concerning autosomes.

5.4 Discussion

The number of loci clearly influences evolution, but especially when preference is involved: the coevolution process between G and P is strongly influenced by the physical structure of the genome, and probably by the mutational input (number

of mutations which here depends on loci number, effects of mutations). More loci clearly promote the coevolution of the traits to higher values in the mating scenario of preference driven mating (G+P) and of preference and competitiveness driven mating (G+P+C). It also has an effect on the genetic architecture evolution, by accentuating the establishment of modular architecture in particular (negative correlation between allelic values). This evolution of the genetic architecture is not isolated from the evolution of the traits, but may also be favored by the gain of space in the genome. This potentially allows for a wider distribution of allelic values for traits in the genome.

Physical linkage however, as we simulated it, had limited effect on traits evolution and on genetic architecture.

Including a sex-dependent genetic architecture of the genome, in relationship with lower availability of females due to a longer latency period, affects quantitatively and qualitatively our predictions. Our genetic architecture assumed that all individuals could carry both female and male chromosomes, but with differential expression with respect to sex. This assumption clearly implies that part of the genetic variation is hidden to selection at each generation. Despite this, we found increased preference in females, as we hypothesized, and potential differences in gametic investment between sexes too. Likewise, the distribution of allelic values is contrasted between nuclear DNA and sex-specific DNA: remarkably, the female sexual chromosome harbours more elevated allelic values for the preference trait P than the male's one. In spite of this, autosomes continue to code strongly for G and P values, although it is sometimes predicted that sexually selected genes are preferentially sex linked and that sex chromosomes could be a hot spot with high effects on sexual antagonistic trait, ([Rice 1984, Reeve & Pfennig 2003] but see [Fitzpatrick 2004, Mank 2009]).

As shown in previous literature [Bulmer 1971, Gavrilets & Hasting 1995], the physical structure of the genome partly controls evolution of traits, notably through its effect on genetic variance. According to our result, the physical structure of the genome can strongly impact traits involved in reproduction, which has also consequences on population demography. It is especially obvious as soon as at least two traits are involved in the mating system, it determines the size of traits, and can therefore also strongly control iteroparity. Such prediction could be compared with empirical data linking genome size to life-history traits, for instance, [Rees *et al.* 2008] noted that a simple life cycle is associated with small genomes in species of shrimps. Where fast-growing species with a more primitive life cycle (e.g. eggs shed directly in water) generally had smaller genomes compared with more K-selected species having a partial embryonic development in eggs attached to the pleopods. Yet, some other studies on insects suggest that, advanced species displayed the smallest genomes [Gregory 2002, Gregory 2011].

Additionally, the physical structure of the genome affects the evolution of genetic architecture, more space in the genome favoring more modular genetic architecture, in the sense that it allows more specialization of the loci, each one coding more strongly for a single trait. These results must be however tempered by some initial choices that deserve more investigation: the mutation landscape is modified with the number of loci so to obtain similar trait values at the beginning of the simulation, but this implies that the distribution of allelic values is then different between the 10 and 100 loci scenarios for instance. And because we kept the mutation rate per locus equal between these two scenarios, the total number of mutations increases when the genome is bigger: this can be viewed as a supplementary fuel for evolution to proceed, as well as a destabilizing force from adaptive optimums. In the present case, the runaway process was actually boosted in the 100 loci scenario, this may

indicate that the 10 loci scenario was somehow constrained in its evolution by the mutational input. The 100 loci scenario proposes probably more combinations to increase relative fitness, and it sometimes can even lead to evolutionary suicide when competitiveness is present. Additionally, we did not yet investigate the interaction with different average mutation rates values, or with variable mutation rates along the genome.

It is clear that these first investigations produce interesting results, either because they actually confirm previous expectations in a more integrative context (like the relationship between polygeny, genetic variance and speed of evolution, or the evolution of sex-specific strategies with the presence sexual chromosomes), or because they shed some light on complex retroactions or loops between behaviour, fitness, genetic architecture and demography (like the fact that the increased polygeny can lead to evolutionary suicide by means of fitness improvements when competitiveness is present - or in other terms, arms race). One can however only notice that once again, some modelling choices, as well as the initial conditions used, may have tremendous effects on my conclusions. But at the very least, it strongly suggests that the role of the physical structure of the genome deserve way more investigations.

Discussion

Contents

6.1	Synthesis of the main results	138
6.2	Some remarks concerning our choices of modeling and future development	142
6.3	A short reflection on my work	147

Reproductive traits and behaviours have major impacts on dynamics of biodiversity, and yet much remains to be done to predict the direction of their evolution. My approach is, in short, an attempt to bring together different ideas treated separately by previous theoretical approaches, based on the following observations: First, that sexual selection is strongly context dependent and results from complex processes involving behavioural interactions between individuals. This social part of the environment being one of the most dynamic sources of variation an organism might experience during its lifetime [West-Eberhard 1983, Kent *et al.* 2008, Krupp *et al.* 2008] and having tremendous impacts on evolution [Wolf *et al.* 1999, Moore *et al.* 1997, Bailey & Moore 2012, Kokko *et al.* 2015, Muniz & Machado 2018]. Second, that sexual selection strength and direction should be partly controlled by the physical structure of the genome [Matessi & Di Pasquale 1996, Kirkpatrick & Hall 2004, Servedio & Lande 2006, Hansen 2006]. And lastly, that genome may also be shaped

by sexual selection ([Hammer *et al.* 2008, Hansen 2006, Reeve & Pfennig 2003, Mank *et al.* 2008, Corl & Ellegren 2012, Wright & Mank 2013]), making mating systems and the social environment an important element in the understanding of genetic architecture evolution.

So I endeavoured during my PhD to build an ad-hoc approach, based on demo-genetic Agent Based Modelling, to further our understanding of reproductive traits evolution and how it affects the genome. I hope that the present contribution can motivate further work on the link between the physical structure of the genome and variations in mating systems or life histories (e.g [Charlesworth & Wright 2001, Sinervo & Svensson 2002, Misevic *et al.* 2006, Lamichhane *et al.* 2016, Plomion *et al.* 2018]).

6.1 Synthesis of the main results

Based on the comparison of simulation scenarios, with different reproductive routines, and different social environments and genetic maps, I investigated the dynamic feedback between social environment, population demography, mating behaviors and genetic architecture. I here summarize some findings which best illustrate the interest and complexity of demogenetics, focusing on two mating systems.

Preference driven mating: When the evolution of preference is allowed, sexual selection takes place (i.e. higher fitness for individuals with a preference for relatively high values of G) accompanied by the establishment of a genetic correlation between G and P , which drives the evolution of behavior, and will also change the size of the population. The population follows a more semelparous strategy than a random mating population. This will also have repercussions on genetic architecture: it slightly reduces allelic diversity but slightly increases the inter-loci variance of allelic

values and leads to a more modular and oligogenic organization of the genome. The social environment, which corresponds here to the size of the mating group (and conditions the quantity and phenotypic distribution of potential partners), affects this pattern. A reduction in the mating group size decreases the advantage conferred by high preference (by increasing the opportunity cost), and thus decreases the genetic correlation between G and P. By preventing the evolution of G and P towards high values, a smaller mating group size allows larger population sizes, as the population adopts a more iteroparous strategy. The social environment will finally have effects on the evolution of the genetic architecture by decreasing the emergence of modularity (possibly through a reduction of the effective number of breeders, Ne). The physical structure of the genome also has an important effect on the coevolution of G and P. More loci allows for more allelic variance and favor the rising of genetic correlation between G and P leading to the evolution of extreme trait values and low population size (semelparous strategies). On the contrary, genetic linkage, which causes a constraint during meiotic recombination, tends to prevent the rising of genetic correlation and of G and P.

Preference driven mating with competitiveness: When the evolution of both preference and competitiveness is allowed, sexual selection is strong. Individuals with greater competitiveness have a reproductive advantage, but the cost of competitiveness in terms of survival prevents this trait from increasing too much in the population. Adding an additional trait changes the trade-off between survival and reproduction and consequently changes the population size. The presence of competitiveness also changes the genetic architecture by reducing the modularity. Certainly because the best combination to maximize the modularity between all the traits implies a slight decrease in modularity between pairs of traits. Small mating

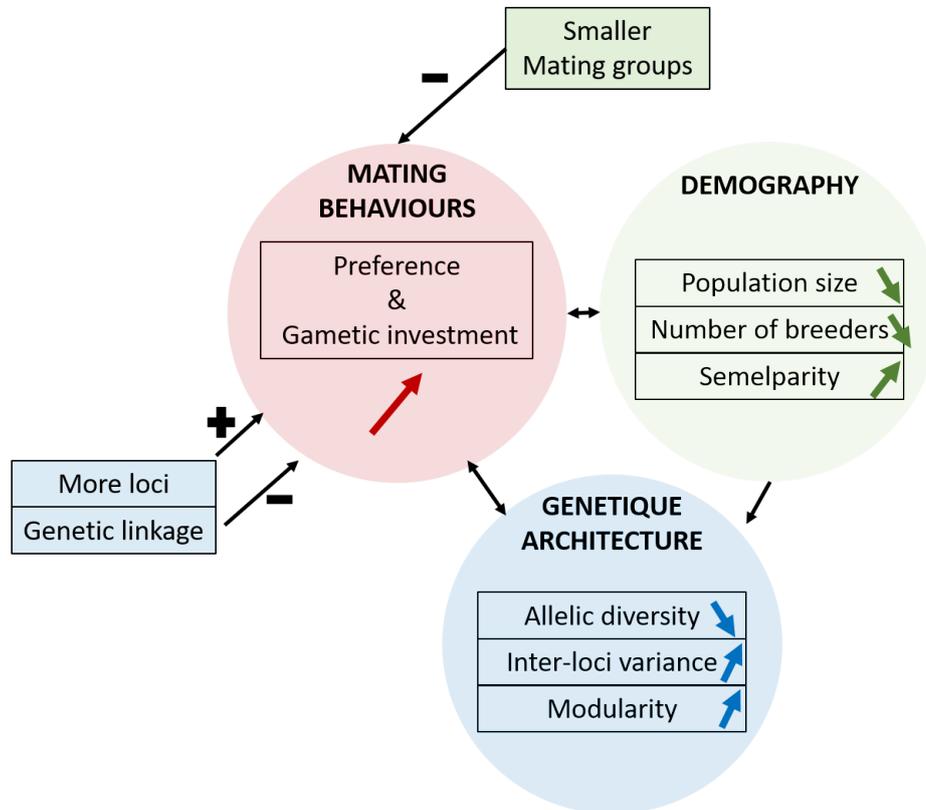


Figure 6.1: A schematic illustration of feedbacks between mating behaviours, demography and genetic architecture with the mating scenario of preference driven mating

groups, and consequent mating uncertainty, increase the advantage of expressing a high competitiveness, leading to an increase of competitiveness in the population to the detriment of the investment in G . More loci also promote the evolution of C toward higher values through its coevolution with G and P .

Variance in mating behaviours: Studies of conditional strategies and inter-population variations in behaviour often explain these variations by differences in ecological factors and social environment (e.g.;[Jackson 1992]).

Different behaviours appear to be adaptively fine tuned to the specific conditions. Accordingly, I demonstrated that choosiness could vary according to the

characteristics of the mating system which influences the dynamics of availability of the partners (Chapter 2 section 2.1.3). However, and contrarily to the model presented in Chapter 2, if these behavioural differences are based on genetic differences, then they are not simply the result of plastic response by individuals to different circumstances and, instead, can result from divergent local adaptation between the populations. Of paramount importance is usually to determine what are the factors driving such divergence. The inter-simulation variance I observed using the DG-ABM approach, as soon as mating routines are driven by preference or/and competitiveness, shows that interpopulation variation in behaviours can arise even if the populations experimented similar environmental condition. Interestingly such interpopulation variance is not predicted when mating is random. Meaning that sexual selection arising from non random mating (due to preference and competitiveness) and consequent co-evolution between traits can lead populations to various and contrasted equilibriums. [Lande 1981] found that the values reached by female preference and males traits at equilibrium depended on the initial condition of trait and preference values in the population. My approach however differs: even with similar initial conditions (drawn with the same parameters), because stochastic processes occur explicitly in the model, the conditions are permanently shuffled to some extent, and this becomes a driving force in evolution. And this force is clearly magnified when mating behaviours are allowed to evolve, producing a diversity of outcomes. This is all the more true since replicating a simulation starting from the same initial state (and not from 2 initial states drawn randomly with identical parameters) also evolves toward two different final states (data not shown). This is, in my opinion, a striking demonstration of the importance of context-dependence, wherein the interaction of stochasticity and reproductive behaviour plays a wondrous part.

Interestingly, when populations are structured into small mating group (which increase inter-individual variance of mating success), inter-simulation variance is reduced, such as all simulations converge toward the same pseudo equilibrium.

6.2 Some remarks concerning our choices of modeling and future development

During my PhD, I have been thinking a lot about how to represent mating behaviours and genetic architecture, I had to make some modelling choices (that have often raised a lot of doubts for me), in particular to define the trade-off between reproduction and survival, to determine the reproductive traits considered and their genetic basis and to represent the social environment. These choices are not without consequences, and I would like to discuss them, and the possible alternatives for further work.

About the representation of social environment: With a game theory approach (Chapter 2), I first highlighted that pairing dynamics is an important process to account for when studying the evolution of choosiness (a component of the preference function). Based on this reflection, I coded different algorithms in the DG-ABM to represent the pairing dynamics for different behavioral scenario (random mating, preference and or competitiveness driven mating, Chapter 3). The social part of environment is still envisioned in a relatively simplistic way, as it is summarized by the different mating routines and by the mating group size. Mating group size allows to represent the fact that these individual interactions are influenced by external factors that may reduce the number of accessible partners at the onset of each breeding season. A more realistic approach would be to make mate

accessibility an individual variable or trait, individuals varying in their capacity to sample mate. This trait would thus be part of the reproductive strategy and could potentially evolve. I also elected not to represent any flexibility of behavior during the breeding season. Here again, with a more complex approach, the shape of the individual's preference could be adjusted during the pairing process depending on the availability of partners. But the genetic basis of this trait would also need to be considered. This is still an avenue to be investigated, especially if one wishes to study the evolution of choosiness.

About the trade-off between reproduction and survival: The equation linking survival to reproduction is inspired by both energy allocation theory and demographic models where population growth depends on population size and habitat capacity. The equation combines a population component (logistic growth equation) and an individual component (trade-off: the more energy invested in reproduction, the lower the survival rate). The balance between the effect of a given value of gametic investment on survival and on reproduction can be tuned by a demographic constant, and exploratory simulations allowed us to fix a default value allowing population persistence and trait evolution. I think that this trade-off is an interesting approach, since it was key in linking iteroparity, population size, allocation among various traits, and genome energetics. But we assumed that traits can evolve freely within the compromise between survival and reproduction, which means that they can potentially have a very important cost on survival in comparison to demographic cost. This potential unbalance may have favored the frequent emergence of semelparous strategies in my results.

About the genetic basis of the traits: I adopted a fully explicit, allelic model that can handle both the simplest systems, and can mimic quantitative genetics too. To be able to study the evolution of the genetic architecture, there must be a direct link between genome and fitness. Like in Fisher's model, any simulated locus has an effect since it contributes to the value of the trait. Additionally, I adopted the hypothesis of universal pleiotropy by giving alleles a value for each trait. I was interested by the organisation allelic values in the genome, and therefore investigated pattern of correlation at the gene level. My idea is that it can impact the evolution beyond what can be predicted by the more traditional G-matrix. The G-matrix sums up all necessary information to describe correlation between traits at condition that the distribution of allele effects is normally spread around mean values, and that traits are coded by an infinitesimal number of loci with small additive effects. But the association of allelic values at a potentially limited number of loci for the different traits influences the number of combinations to produce given values of traits, and thus influences the conjoint transmission of traits and the variance of genotypes in the population. Beside, correlation between allelic values can not be shuffled by recombination. By looking at allelic correlation, we see if there is particular associations in allelic values due to selection. Interestingly, in some mating systems, we found a relation between correlation of allelic values in individuals genome and their lifetime reproductive success. In particular, the coevolution between the gametic investment and the preference seems to favor the emergence of alleles with negative correlation in their genetic value. I suggest that individuals with negative allelic correlation in their genome achieved a greater lifetime reproductive success, as they might also inherit of an advantageous combination of traits values. The evolution of such association is lessened when reducing the mating group size, possibly as a result of demogenetic retroactions (reduction of the effective population size

and consequent reduction of genetic variation). This microevolutionary mechanism generates a particular pattern of genetic architecture at the population level. My bootstrap approach accounts for the mutations retained in the population versus what would be obtained by randomly sampling into the mutations distributions. It therefore describes how the evolution of the population selects for some particular arrangement of correlated allelic values for the various traits. I found that some situations favor the emergence of particular arrangement wherein allelic values for 2 traits in the population are globally negatively correlated, more than it would be by chance. I directly relate this pattern to the concept of modularity: the general definition of modularity is the division of a product into independent components, that allow to easily create product variety [Hansen 2003, Kashtan & Alon 2005]. It would be interesting to investigate further the evolution of genome spatialization, to see if particular allelic values becomes segregated on some chromosomes for instance. I started to address this question by envisioning more complex genetic map (with physical linkage) and by adding some sex chromosomes. What would be particularly interesting would be to characterize certain situations / physical structures of the genome that lead to the evolution of functional ilots (e.g. group of genes that have similar effect for a trait).

Admittedly, the simple additive allelic model adopted in our approach does not allow to represent some mechanisms of dominant and epistatic gene actions. Such phenomenon is likely the result of intralocus and interlocus interactions. I actually wondered about including such interactions. But the establishment of mathematical rules to represent the phenomena of genetic regulation opens the door to even more complexity, and it remains difficult to find general physical or statistical principles in the literature to represent these interactions in a generic way. Finally, I also had to define a framework concerning the evolution of the genome, the physical

structure of the genome being here a constraint that cannot evolve. I elected that this framework fits correctly empirical studies on trait evolution that take place within species or populations (i.e. microevolution [Hendry & Kinnison 2001]). Yet, chromosomal rearrangements may happen, and can be a potential driving forces of genetic architecture evolution. Modeling the microevolutionary dynamics of such rearrangements, fission or fusion of chromosomes for instance, would also require general knowledge and principles about the odds of occurrence, genomes compatibility and fitness of offspring. Again, it is difficult to find some general rules and models applicable for this matter in the literature at the moment.

About the representation the sexes: The evolution of sexual dimorphism and sex roles being a major topic of interest in sexual selection, I guess that the reader might be curious about how much my work revolves around sexual selection without really addressing sex differences in behaviour or traits. For my defence, sexual selection occurs without need for difference between sexes. As mate choice in both male and female can be found in many species, as well as competitiveness, I did not want to set in stone traits that are sex-specific (contrary to what is done most often). Sexes are primarily defined by the two types of gametes they produce; the female sex produces relatively few, large and usually non-motile gametes (eggs or ovules), whereas the male sex produces many, smaller and often motile gametes (sperm or pollen).

I first thought to make anisogamy evolve in the model. Anisogamy is predicted to evolve as a result of task division between the male and female gametes to maximize both the number of gametes finding each other and the reserves contained by the gametes to allow the growth of the zygote [Lehtonen & Kokko 2011]. So I tried to define a trade-off between mobility and number of gametes (which would influence

the probability of fertilization) and the amount of reserve in the egg (which would influence the survival of the eggs) but this required to add two traits as well as to define relationships between the number and size of gametes, the amount of gamete and the fertilization probability, the amount of reserve and the survival of the eggs; I preferred not to include such a complexity in the first place.

However a parsimonious way to let sex differences emerge in the model is to introduce an asymmetry between the sexes accompanied by a genetic map that allows a different expression of traits according to sex (sex chromosomes). In the Chapter 5, I thus proposed a first road to create sex differences in traits and behaviour by introducing a difference in latency period between sexes and by adding males and females chromosomes with sex biased expression.

Lastly, the evolution of parental care has raised much interest in the evolution of mating systems and sex roles [Parker *et al.* 1972, Kokko & Jennions 2008, Fromhage & Jennions 2016, Lehtonen *et al.* 2016]. For now, parental care is implicitly assumed in the model, it is somehow factored in the current fitness (number of offspring), because the calculation of fecundity (as the mean of both partners gametic investments) implies that both parents contribute to reproductive success. The model also offers the option to only select one gametic investment, but I did not explore that option yet.

6.3 A short reflection on my work

One of the potential benefit of my modelling approach was that as much as possible would "emerge from the model"; such as pairing processes, the cost and the benefits to express a certain trait value, and the distribution of genetic effect over the genome. I now consider that it was rather successful, and it allowed to relax

strong assumptions that are usually made in other studies (see Chapter 2). Yet, to reach that objective, I still had to make choices that bear consequences. I here, with the example of pairing formation process, illustrate how wanting to explicitly formulate certain mechanisms in turns triggers many new questions, opening the way to even more complexity: The process of pair formation, which is implicitly assumed in genetics models may involve complex sampling routines. And this is not trivial, as different sampling routines can result in different mating pattern even if individuals have similar preferences [Gimelfarb 1988]. In the model, I integrated the dynamics aspect of the process, individuals pairing sequentially during breeding seasons, thus leading to a progressive decrease in mate availability. But when coding the pairing algorithm, I had to define how to stop the algorithm in the case where all the individuals do not match up. This can happen if phenotypes do not match with preferences. Because preference is a probabilistic function, one can always hope that all individuals will pair after a large number of iterations, but such large number is biologically unrealistic, and can also increase computation time. I thus defined a maximum number of sampled partners for males and females, which correspond to the number of individuals of the opposite sex present in the mating group before individuals get paired (in Chapter 3). This number could of course have an influence on my results. Moreover, such a number could also be seen as a trait that evolve, and it opens the way for more complexity.

As my approach is original, it is sometimes difficult to relate it to classical formulations that can be found in many other models (which define for example a trait-fitness relationship). Our results are therefore difficult to compare with analytical predictions that apply in a very particular framework where several variables are controlled (such as the strength and direction of selection, the distribution of allelic effect etc..). I also came across some unaddressed issues in the literature. In

particular, I was unable to find previous work relating some general characteristics of mating systems (e.g. the absence or presence of preference or competitiveness during mating) or social environment to global characteristics of the genetic architecture (e.g. oligogenecity, modularity) of sexual traits and behaviours. [Fitzpatrick 2004] started to address this question looking at the genomic location and pleiotropic effects of sexually selected genes of *Drosophila melanogaster*; and some databases could now possibly make it possible to address this question. However, I tried to measure a global organization of the genome that goes beyond the description of the pleiotropic effects of certain genes. Inspired by the notion of modularity of a system, I tried to quantify the direction of variation in allelic effects within the genome. This notion is a little different from that of pleiotropy, because it aims to see if the values of the alleles for the different traits vary in the same directions or not. I also tried to measure the polygeny, which is classically defined as the number of genes coding for a trait. But I came up against the difficulty of how to measure it: since everything is explicit in the model and all the alleles have a genetic value. I would therefore have had to decide on a threshold for the detection of allelic effects (e.g. an allele whose value deviates from the mean value of the trait). But in any case the polygeny would have given little information on the distribution of allelic effects among loci. I thus proposed to measure inter-loci variance of allelic values, to obtain an indication of the oligogenicity of the genome. I think it would be interesting to further develop this reflection on measures of the functional organization of the genetic architecture, which could facilitate the formulation and testability of some theoretical predictions on the evolution of the genetic architecture.

The DG-ABM approach to sexual selection I built during my Philosophical Dissertation has obviously potential for improvement, and also can be now used in many ways, developed, adapted. I believe that this kind of approach can help at understanding complex situations, and complex outputs, something that is definitely observed in natural populations too. Indeed, empirical approaches can produce a wealth of patterns, on traits, on behaviour, their genetic basis, on life history trade-offs, or on population demographic and genetic structures. Only a fragment of these data are usually picked up, so to fit theoretical predictions stemming from reductive analytical models, whereas many of these patterns could help testing theoretical expectations more confidently.

Bibliography

- [Aeschbacher & Bürger 2014] Simon Aeschbacher and Reinhard Bürger. *The effect of linkage on establishment and survival of locally beneficial mutations*. *Genetics*, vol. 197, no. 1, pages 317–336, 2014. (Cited on page [121](#).)
- [Albert *et al.* 2008] Arianne YK Albert, Sterling Sawaya, Timothy H Vines, Anne K Knecht, Craig T Miller, Brian R Summers, Sarita Balabhadra, David M Kingsley and Dolph Schluter. *The genetics of adaptive shape shift in stickleback: pleiotropy and effect size*. *Evolution: International Journal of Organic Evolution*, vol. 62, no. 1, pages 76–85, 2008. (Cited on page [18](#).)
- [Alpern & Reyniers 2005] Steve Alpern and Diane Reyniers. *Strategic mating with common preferences*. *Journal of Theoretical Biology*, vol. 237, no. 4, pages 337–354, 2005. (Cited on pages [29](#) and [33](#).)
- [Amundsen *et al.* 1997] T. Amundsen, E. Forsgren and L. T.T. Hansen. *On the function of female ornaments: Male bluethroats prefer colourful females*. *Proceedings of the Royal Society B: Biological Sciences*, vol. 264, no. 1388, pages 1579–1586, 1997. (Cited on page [13](#).)
- [Amundsen 2018] Trond Amundsen. *Sex roles and sexual selection: lessons from a dynamic model system*. *Current zoology*, vol. 64, no. 3, pages 363–392, 2018. (Cited on page [100](#).)
- [Andersson 1994] Malte B Andersson. *Sexual selection*. *Monographs in Behavior and Ecology*. Princeton Univ. Press, Princeton, New Jersey, 1994. (Cited on pages [3](#), [5](#), [7](#), [10](#) and [18](#).)

- [Arnocky *et al.* 2014] Steven Arnocky, Angela Ribout, Reehan S Mirza and Jennifer M Knack. *Perceived mate availability influences intrasexual competition, jealousy and mate-guarding behavior*. *Journal of Evolutionary Psychology*, vol. 12, no. 1, pages 45–64, 2014. (Cited on page 100.)
- [Arnold *et al.* 2008] Stevan J Arnold, Reinhard Bürger, Paul A Hohenlohe, Beverley C Ajie and Adam G Jones. *Understanding the evolution and stability of the G-matrix*. *Evolution: International Journal of Organic Evolution*, vol. 62, no. 10, pages 2451–2461, 2008. (Cited on page 42.)
- [Arnold 1983] Stevan J. Arnold. *Sexual selection: the interface between theory and empiricism*. Bateson PP, ed. *Mate choice*. Cambridge: Cambridge University Press, pages 67–107, 1983. (Cited on pages 7, 41 and 250.)
- [Arnqvist & Rowe 1995] Göran Arnqvist and Locke Rowe. *Sexual conflict and arms races between the sexes: a morphological adaptation for control of mating in a female insect*. *Proceedings of the Royal Society of London. Series B: Biological Sciences*, vol. 261, no. 1360, pages 123–127, 1995. (Cited on page 9.)
- [Ashman *et al.* 2014] Tia-lynn Ashman, Doris Bachtrog and Heath Blackmon. *Tree of Sex: a database of sexual systems*. *Scientific data*, vol. 1, 2014. (Cited on page 14.)
- [Bailey & Moore 2012] Nathan W Bailey and Allen J Moore. *Runaway sexual selection without genetic correlations: social environments and flexible mate choice initiate and enhance the Fisher process*. *Evolution: International Journal of Organic Evolution*, vol. 66, no. 9, pages 2674–2684, 2012. (Cited on page 137.)

- [Balloux 2001] Francois Balloux. *EASYPPOP (version 1.7): a computer program for population genetics simulations*. *Journal of heredity*, vol. 92, no. 3, pages 301–302, 2001. (Cited on page 44.)
- [Barry & Kokko 2010] Katherine L Barry and Hanna Kokko. *Male mate choice: why sequential choice can make its evolution difficult*. *Animal Behaviour*, vol. 80, no. 1, pages 163–169, 2010. (Cited on pages 28 and 100.)
- [Barson *et al.* 2015] Nicola J Barson, Tutku Aykanat, Kjetil Hindar, Matthew Baranski, Geir H Bolstad, Peder Fiske, Céleste Jacq, Arne J Jensen, Susan E Johnston, Sten Karlsson *et al.* *Sex-dependent dominance at a single locus maintains variation in age at maturity in salmon*. *Nature*, vol. 528, no. 7582, pages 405–408, 2015. (Cited on page 120.)
- [Barton & Charlesworth 1984] Nicholas H Barton and Brian Charlesworth. *Genetic revolutions, founder effects, and speciation*. *Annual Review of Ecology and Systematics*, vol. 15, no. 1, pages 133–164, 1984. (Cited on page 92.)
- [Barton 2010] Nicholas H Barton. *Genetic linkage and natural selection*. *Philosophical Transactions of the Royal Society B: Biological Sciences*, vol. 365, no. 1552, pages 2559–2569, 2010. (Cited on page 121.)
- [Bateman 1948] Angus J Bateman. *Intra-sexual selection in *Drosophila**. *Heredity*, vol. 2, no. 3, pages 349–368, 1948. (Cited on page 12.)
- [Benenson 2013] Joyce F Benenson. *The development of human female competition: allies and adversaries*. *Philosophical Transactions of the Royal Society B: Biological Sciences*, vol. 368, no. 1631, page 20130079, 2013. (Cited on page 13.)

- [Bengston *et al.* 2018] Sarah E Bengston, Romain A Dahan, Zoe Donaldson, Steven M Phelps, Kees Van Oers, Andrew Sih and Alison M Bell. *Genomic tools for behavioural ecologists to understand repeatable individual differences in behaviour*. *Nature ecology & evolution*, vol. 2, no. 6, pages 944–955, 2018. (Cited on page 93.)
- [Benton & Grant 1999] TG Benton and Alastair Grant. *Optimal reproductive effort in stochastic, density-dependent environments*. *Evolution*, vol. 53, no. 3, pages 677–688, 1999. (Cited on pages 36 and 102.)
- [Berglund *et al.* 1989] Anders Berglund, Gunilla Rosenqvist and Ingrid Svensson. *Reproductive Success of Females Limited by Males in Two Pipefish Species*. *The American Naturalist*, vol. 133, no. 4, page 506, 1989. (Cited on page 12.)
- [Birky & Walsh 1988] C William Birky and J Bruce Walsh. *Effects of linkage on rates of molecular evolution*. *Proceedings of the National Academy of Sciences*, vol. 85, no. 17, pages 6414–6418, 1988. (Cited on page 126.)
- [Bleu *et al.* 2012] Josefa Bleu, Carmen Bessa-Gomes and David Laloi. *Evolution of female choosiness and mating frequency: effects of mating cost, density and sex ratio*. *Animal Behaviour*, vol. 83, no. 1, pages 131–136, 2012. (Cited on page 28.)
- [Bolnick *et al.* 2018] Daniel I Bolnick, Rowan DH Barrett, Krista B Oke, Diana J Rennison and Yoel E Stuart. *(Non) parallel evolution*. *Annual Review of Ecology, Evolution, and Systematics*, vol. 49, pages 303–330, 2018. (Cited on page 94.)

- [Bonduriansky 2001] Russell Bonduriansky. *The evolution of male mate choice in insects: a synthesis of ideas and evidence*. *Biological Reviews*, vol. 76, no. 03, pages 305–339, 2001. (Cited on page 13.)
- [Booksmythe *et al.* 2008] Isobel Booksmythe, Tanya Detto and Patricia RY Backwell. *Female fiddler crabs settle for less: the travel costs of mate choice*. *Animal Behaviour*, vol. 76, no. 6, pages 1775–1781, 2008. (Cited on pages 7 and 8.)
- [Brooks 2002] Robert Brooks. *Variation in female mate choice within guppy populations: Population divergence, multiple ornaments and the maintenance of polymorphism*. *Genetica*, vol. 116, no. 2-3, pages 343–358, 2002. (Cited on page 9.)
- [Bulmer 1971] MG1 Bulmer. *The effect of selection on genetic variability*. *The American Naturalist*, vol. 105, no. 943, pages 201–211, 1971. (Cited on page 135.)
- [Byrne & Rice 2006] Phillip G Byrne and William R Rice. *Evidence for adaptive male mate choice in the fruit fly *Drosophila melanogaster**. *Proceedings of the Royal Society B: Biological Sciences*, vol. 273, no. 1589, pages 917–922, 2006. (Cited on page 30.)
- [Campbell 2004] Anne Campbell. *Female competition: Causes, constraints, content, and contexts*. *Journal of sex research*, vol. 41, no. 1, pages 16–26, 2004. (Cited on page 13.)
- [Candolin & Salesto 2009] Ulrika Candolin and Tiina Salesto. *Does Competition Allow Male Mate Choosiness in Threespine Sticklebacks?* *The American Naturalist*, vol. 173, no. 2, pages 273–277, 2009. (Cited on page 100.)

- [Careau *et al.* 2015] Vincent Careau, Matthew E Wolak, Patrick A Carter and Theodore Garland Jr. *Evolution of the additive genetic variance–covariance matrix under continuous directional selection on a complex behavioural phenotype*. *Proceedings of the Royal Society B: Biological Sciences*, vol. 282, no. 1819, page 20151119, 2015. (Cited on page 42.)
- [Carlson & Seamons 2008] Stephanie M Carlson and Todd R Seamons. *A review of quantitative genetic components of fitness in salmonids: implications for adaptation to future change*. *Evolutionary Applications*, vol. 1, no. 2, pages 222–238, 2008. (Cited on page 101.)
- [Chapman *et al.* 2003] Tracey Chapman, Göran Arnqvist, Jenny Bangham and Locke Rowe. *Sexual conflict*. *Trends in Ecology & Evolution*, vol. 18, no. 1, pages 41–47, 2003. (Cited on page 9.)
- [Charlesworth & Wright 2001] Deborah Charlesworth and Stephen I Wright. *Breeding systems and genome evolution*. *Current opinion in genetics & development*, vol. 11, no. 6, pages 685–690, 2001. (Cited on page 138.)
- [Charlesworth *et al.* 1987] Brian Charlesworth, Jerry A Coyne and Nicholas H Barton. *The relative rates of evolution of sex chromosomes and autosomes*. *The American Naturalist*, vol. 130, no. 1, pages 113–146, 1987. (Cited on page 130.)
- [Charmantier & Sheldon 2006] Anne Charmantier and Ben C Sheldon. *Testing genetic models of mate choice evolution in the wild*. *Trends in Ecology & Evolution*, vol. 21, no. 8, pages 417–419, 2006. (Cited on page 24.)
- [Chevalier *et al.* 2020] Louise Chevalier, Jacques Labonne, Matthias Galipaud and Dechaume-Moncharmont. *Fluctuating dynamics of mate availability promote*

the evolution of flexible choosiness in both sexes. preprint in *The American Naturalist*, vol. 196, 2020. (Cited on pages 4, 8, 19, 29 and 100.)

[Cheverud 1996] James M. Cheverud. *Developmental Integration and the Evolution of Pleiotropy.* *Integrative and Comparative Biology*, vol. 36, no. 1, pages 44–50, 1996. (Cited on page 94.)

[Chevin *et al.* 2010] Luis-Miguel Chevin, Guillaume Martin and Thomas Lenormand. *Fisher's model and the genomics of adaptation: restricted pleiotropy, heterogenous mutation, and parallel evolution.* *Evolution: International Journal of Organic Evolution*, vol. 64, no. 11, pages 3213–3231, 2010. (Cited on page 117.)

[Clune *et al.* 2013] Jeff Clune, Jean-Baptiste Mouret and Hod Lipson. *The evolutionary origins of modularity.* *Proceedings of the Royal Society b: Biological sciences*, vol. 280, no. 1755, page 20122863, 2013. (Cited on page 101.)

[Clutton-Brock & Parker 1992] Tim H Clutton-Brock and Geoffrey A Parker. *Potential reproductive rates and the operation of sexual selection.* *The Quarterly Review of Biology*, vol. 67, no. 4, pages 437–456, 1992. (Cited on page 12.)

[Collins & McNamara 1993] Edward J Collins and John M McNamara. *The job-search problem with competition: an evolutionarily stable dynamic strategy.* *Advances in Applied Probability*, vol. 25, no. 2, pages 314–333, 1993. (Cited on pages 28 and 29.)

[Corl & Ellegren 2012] Ammon Corl and Hans Ellegren. *The genomic signature of sexual selection in the genetic diversity of the sex chromosomes and autosomes.* *Evolution: International Journal of Organic Evolution*, vol. 66, no. 7, pages 2138–2149, 2012. (Cited on pages 15 and 138.)

- [Cotton *et al.* 2006] Samuel Cotton, Jennifer Small and Andrew Pomiankowski. *Sexual selection and condition-dependent mate preferences*. *Current Biology*, vol. 16, no. 17, pages R755–R765, 2006. (Cited on page 19.)
- [Courtiol *et al.* 2016] Alexandre Courtiol, Loïc Etienne, Romain Feron, Bernard Godelle and François Rousset. *The Evolution of Mutual Mate Choice under Direct Benefits*. *The American Naturalist*, vol. 188, no. 5, pages 521–538, 2016. (Cited on pages 7, 19, 29, 100 and 130.)
- [Darwin 1871] Charles Darwin. *The descent of man and selection in relation to sex*. Murray, London, 1871. (Cited on pages 3, 11 and 12.)
- [Davidian *et al.* 2016] Eve Davidian, Alexandre Courtiol, Bettina Wachter, Heribert Hofer and Oliver P Höner. *Why do some males choose to breed at home when most other males disperse?* *Science Advances*, vol. 2, no. 3, page e1501236, 2016. (Cited on page 19.)
- [De Jong & Sabelis 1991] Mart CM De Jong and Maurice W Sabelis. *Limits to runaway sexual selection: the wallflower paradox*. *Journal of Evolutionary Biology*, vol. 4, no. 4, pages 637–655, 1991. (Cited on pages 20, 39, 66, 95 and 100.)
- [Dechaume-Moncharmont *et al.* 2016] François-Xavier Dechaume-Moncharmont, Thomas Brom and Frank Cézilly. *Opportunity costs resulting from scramble competition within the choosy sex severely impair mate choosiness*. *Animal Behaviour*, vol. 114, no. April, pages 249–260, 2016. (Cited on pages 4, 8, 19, 28, 66 and 100.)

- [Dechaume-moncharmont 2018] François-Xavier Dechaume-moncharmont. *Evolutionarily Stable Strategies*. Encyclopedia of Animal Cognition and Behavior, no. January, 2018. (Cited on page 27.)
- [Dieckmann & Ferriere 2004] U Dieckmann and R Ferriere. *Adaptive dynamics and evolving biodiversity*. In Régis Ferrière, Ulf Dieckmann and Denis Couvet, editors, Evolutionary conservation biology, page 188–224. Cambridge University Press, Cambridge, UK, 2004. (Cited on page 36.)
- [Dougherty & Shuker 2015] Liam R Dougherty and David M Shuker. *The effect of experimental design on the measurement of mate choice: a meta-analysis*. Behavioral Ecology, vol. 26, no. 2, pages 311–319, 2015. (Cited on page 5.)
- [Edward & Chapman 2011] Dominic A. Edward and Tracey Chapman. *The evolution and significance of male mate choice*. Trends in Ecology and Evolution, vol. 26, no. 12, pages 647–654, 2011. (Cited on page 13.)
- [Ellegren 2009] Hans Ellegren. *The different levels of genetic diversity in sex chromosomes and autosomes*. Trends in Genetics, vol. 25, no. 6, pages 278–284, 2009. (Cited on page 15.)
- [Emlen & Oring 1977] Stephen T Emlen and Lewis W Oring. *Ecology, sexual selection, and the evolution of mating systems*. Science, vol. 197, no. 4300, pages 215–223, 1977. (Cited on pages 4, 13 and 100.)
- [Endler & Basolo 1998] John A Endler and Alexandra L Basolo. *Sensory ecology, receiver biases and sexual selection*. Trends in ecology & evolution, vol. 13, no. 10, pages 415–420, 1998. (Cited on page 11.)
- [Etienne *et al.* 2014] L. Etienne, F. Rousset, B. Godelle and A. Courtiol. *How choosy should I be? The relative searching time predicts evolution of choosi-*

- ness under direct sexual selection*. Proceedings of the Royal Society B: Biological Sciences, vol. 281, no. 1785, pages 20140190–20140190, 2014. (Cited on pages 7, 28, 100 and 130.)
- [Eyre-Walker & Keightley 1999] Adam Eyre-Walker and Peter D Keightley. *High genomic deleterious mutation rates in hominids*. Nature, vol. 397, no. 6717, pages 344–347, 1999. (Cited on page 60.)
- [Fawcett & Johnstone 2003] Tim W. Fawcett and Rufus A. Johnstone. *Mate choice in the face of costly competition*. Behavioral Ecology, vol. 14, no. 6, pages 771–779, 2003. (Cited on page 100.)
- [Fawcett *et al.* 2012] Tim W Fawcett, John M McNamara and Alasdair I Houston. *When is it adaptive to be patient? A general framework for evaluating delayed rewards*. Behavioural processes, vol. 89, no. 2, pages 128–136, 2012. (Cited on pages 4, 7, 8 and 66.)
- [Fisher 1915] Ronald A Fisher. *The evolution of sexual preference*. The Eugenics Review, vol. 7, no. 3, page 184, 1915. (Cited on pages 10, 11, 14, 39, 69, 78 and 93.)
- [Fisher 1930] RA Fisher. *The Genetical Theory of Natural Selection Oxford Univ. Press* (Clarendon), London (Reprinted and revised, 1958), 1930. (Cited on pages 5, 15, 18, 61 and 126.)
- [Fitzpatrick 2004] MarkÂJ. Fitzpatrick. *Pleiotropy and the Genomic Location of Sexually Selected Genes*. The American Naturalist, vol. 163, no. 6, pages 800–808, 2004. (Cited on pages 18, 134 and 149.)
- [Fontaneto *et al.* 2012] Diego Fontaneto, Cuong Q Tang, Ulrike Obertegger, Francesca Leasi and Timothy G Barraclough. *Different diversification rates*

between sexual and asexual organisms. *Evolutionary Biology*, vol. 39, no. 2, pages 262–270, 2012. (Cited on page 2.)

[Fromhage & Jennions 2016] Lutz Fromhage and Michael D Jennions. *Coevolution of parental investment and sexually selected traits drives sex-role divergence*. *Nature communications*, vol. 7, no. 1, pages 1–11, 2016. (Cited on page 147.)

[Gadgil & Bossert 1970] Madhav Gadgil and William H Bossert. *Life historical consequences of natural selection*. *The American Naturalist*, vol. 104, no. 935, pages 1–24, 1970. (Cited on page 8.)

[Gascoigne *et al.* 2009] Joanna Gascoigne, Ludek Berec, Stephen Gregory and Franck Courchamp. *Dangerously few liaisons: a review of mate-finding Allee effects*. *Population Ecology*, vol. 51, no. 3, pages 355–372, 2009. (Cited on page 19.)

[Gasparini *et al.* 2013] Clelia Gasparini, Giovanna Serena and Andrea Pilastro. *Do unattractive friends make you look better? Context-dependent male mating preferences in the guppy*. *Proceedings of the Royal Society B: Biological Sciences*, vol. 280, no. 1756, page 20123072, 2013. (Cited on page 100.)

[Gauthey *et al.* 2015] Zoé Gauthey, Marine Freychet, Aurélie Manicki, Alexandre Herman, Olivier Lepais, Stéphane Panserat, Arturo Elosegi, Cédric Tente-lier and Jacques Labonne. *The concentration of plasma metabolites varies throughout reproduction and affects offspring number in wild brown trout (*Salmo trutta*)*. *Comparative Biochemistry and Physiology Part A: Molecular & Integrative Physiology*, vol. 184, pages 90–96, 2015. (Cited on page 19.)

- [Gauthey 2014] Zoé Gauthey. *Effect of environment on sexual selection in brown trout (*Salmo trutta*)*. PhD thesis, Université de Pau et des Pays de l'Adour; Universidad del País Vasco-Euskal, 2014. (Cited on page 19.)
- [Gavrilets & Hasting 1995] Sergey Gavrilets and Alan Hasting. *Dynamics of polygenic variability under stabilizing selection, recombination, and drift*. *Genetics Research*, vol. 65, no. 1, pages 63–74, 1995. (Cited on pages 121 and 135.)
- [Gavrilets *et al.* 2007] Sergey Gavrilets, Aaron Vose, Marta Barluenga, Walter Salzburger and Axel Meyer. *Case studies and mathematical models of ecological speciation. 1. Cichlids in a crater lake*. *Molecular Ecology*, vol. 16, no. 14, pages 2893–2909, 2007. (Cited on page 42.)
- [Gibson *et al.* 2017] Amanda K Gibson, Lynda F Delph and Curtis M Lively. *The two-fold cost of sex: experimental evidence from a natural system*. *Evolution letters*, vol. 1, no. 1, pages 6–15, 2017. (Cited on page 2.)
- [Gil *et al.* 2016] Jordi Gil, Arnaud Caudron and Jacques Labonne. *Can female preference drive intraspecific diversity dynamics in brown trout (*Salmo trutta*, *L.*)?* *Ecology of Freshwater Fish*, vol. 25, no. 3, pages 352–359, 2016. (Cited on page 19.)
- [Gimelfarb 1988] A. Gimelfarb. *Processes of pair formation leading to assortative mating in biological populations: encounter-mating model*. *The American naturalist*, vol. 131, no. 6, pages 865–884, 1988. (Cited on pages 54 and 148.)
- [Gingerich 2009] Philip D Gingerich. *Rates of evolution*. *Annual Review of Ecology, Evolution, and Systematics*, vol. 40, pages 657–675, 2009. (Cited on page 95.)

- [Glutton-Brock & Vincent 1991] T. H. Glutton-Brock and A. C. J. Vincent. *Sexual selection and the potential reproductive rates of males and females*. *Nature*, vol. 351, no. 6321, pages 58–60, 1991. (Cited on page 12.)
- [Govaert *et al.* 2019] Lynn Govaert, Emanuel A Fronhofer, Sébastien Lion, Christophe Eizaguirre, Dries Bonte, Martijn Egas, Andrew P Hendry, Ayana De Brito Martins, Carlos J Melián, Joost AM Raeymaekers *et al.* *Eco-evolutionary feedbacks—Theoretical models and perspectives*. *Functional Ecology*, vol. 33, no. 1, pages 13–30, 2019. (Cited on pages 20 and 43.)
- [Gowaty 2003] P A Gowaty. *Sexual natures: how feminism changed evolutionary biology*. *Signs*, vol. 28, no. 3, pages 901–921, 2003. (Cited on pages 10 and 13.)
- [Grafen 1984] Alan Grafen. *Natural selection, kin selection and group selection*. *Behavioural ecology: An evolutionary approach*, vol. 2, pages 62–84, 1984. (Cited on page 36.)
- [Gregory 2002] T Ryan Gregory. *Genome size and developmental complexity*. *Genetica*, vol. 115, no. 1, pages 131–146, 2002. (Cited on page 135.)
- [Gregory 2011] T Ryan Gregory. *The evolution of the genome*. Elsevier, 2011. (Cited on page 135.)
- [Gremer & Venable 2014] Jennifer R Gremer and D Lawrence Venable. *Bet hedging in desert winter annual plants: optimal germination strategies in a variable environment*. *Ecology Letters*, vol. 17, no. 3, pages 380–387, 2014. (Cited on page 102.)
- [Grimm *et al.* 2006] Volker Grimm, Uta Berger, Finn Bastiansen, Sigrunn Eliassen, Vincent Ginot, Jarl Giske, John Goss-Custard, Tamara Grand, Simone K

- Heinz, Geir Huse *et al.* *A standard protocol for describing individual-based and agent-based models.* *Ecological modelling*, vol. 198, no. 1-2, pages 115–126, 2006. (Cited on page 53.)
- [Griswold 2006] Cortland K Griswold. *Pleiotropic mutation, modularity and evolvability.* *Evolution & development*, vol. 8, no. 1, pages 81–93, 2006. (Cited on page 75.)
- [Guillaume & Otto 2012] Frédéric Guillaume and Sarah P Otto. *Gene functional trade-offs and the evolution of pleiotropy.* *Genetics*, vol. 192, no. 4, pages 1389–1409, 2012. (Cited on pages 15, 42, 44, 96 and 117.)
- [Guillaume & Rougemont 2006] Frédéric Guillaume and Jacques Rougemont. *Nemo: an evolutionary and population genetics programming framework.* *Bioinformatics*, vol. 22, no. 20, pages 2556–2557, 2006. (Cited on pages 15 and 45.)
- [Haldane 1935] Joseph BS Haldane. *The rate of spontaneous mutation of a human gene.* *Journal of genetics*, vol. 31, no. 3, page 317, 1935. (Cited on page 60.)
- [Hall *et al.* 2000] David W Hall, Mark Kirkpatrick and Brian West. *Runaway sexual selection when female preferences are directly selected.* *Evolution*, vol. 54, no. 6, pages 1862–1869, 2000. (Cited on page 69.)
- [Hamilton 1980] William D Hamilton. *Sex versus non-sex versus parasite.* *Oikos*, pages 282–290, 1980. (Cited on page 1.)
- [Hammer *et al.* 2008] Michael F Hammer, Fernando L Mendez, Murray P Cox, August E Woerner and Jeffrey D Wall. *Sex-biased evolutionary forces shape genomic patterns of human diversity.* *PLoS genetics*, vol. 4, no. 9, 2008. (Cited on page 138.)

- [Hansen 2003] Thomas F. Hansen. *Is modularity necessary for evolvability? Remarks on the relationship between pleiotropy and evolvability*. *BioSystems*, vol. 69, no. 2-3, pages 83–94, 2003. (Cited on pages 14, 16, 42, 44, 75, 94, 117 and 145.)
- [Hansen 2006] Thomas F Hansen. *The evolution of genetic architecture*. *Annu. Rev. Ecol. Evol. Syst.*, vol. 37, pages 123–157, 2006. (Cited on pages 14, 42, 137 and 138.)
- [Hendry & Gonzalez 2008] Andrew P Hendry and Andrew Gonzalez. *Whither adaptation?* *Biology & Philosophy*, vol. 23, no. 5, page 673, 2008. (Cited on page 101.)
- [Hendry & Kinnison 2001] Andrew P Hendry and Michael T Kinnison. *Perspective: the pace of modern life: measuring rates of contemporary microevolution*. *Evolution*, vol. 53, no. 6, pages 1637–1653, 2001. (Cited on pages 65, 69, 92, 95 and 146.)
- [Hendry 2020] Andrew P Hendry. *Eco-evolutionary dynamics*. Princeton university press, 2020. (Cited on page 44.)
- [Hill & Robertson 1966] William G Hill and Alan Robertson. *The effect of linkage on limits to artificial selection*. *Genetics Research*, vol. 8, no. 3, pages 269–294, 1966. (Cited on page 126.)
- [Hill & Zhang 2012] William G Hill and Xu-Sheng Zhang. *On the pleiotropic structure of the genotype–phenotype map and the evolvability of complex organisms*. *Genetics*, vol. 190, no. 3, pages 1131–1137, 2012. (Cited on page 61.)
- [Hirshfield & Tinkle 1975] Michael F Hirshfield and Donald W Tinkle. *Natural selection and the evolution of reproductive effort*. *Proceedings of the National*

- Academy of Sciences of the United States of America, vol. 72, no. 6, pages 2227–2231, 1975. (Cited on page 25.)
- [Houle 1998] David Houle. *How should we explain variation in the genetic variance of traits?* *Genetica*, vol. 102, page 241, 1998. (Cited on page 16.)
- [Houston & McNamara 1982] Alasdair I Houston and John McNamara. *A sequential approach to risk-taking*. *Animal Behaviour*, 1982. (Cited on page 8.)
- [Houston & McNamara 1999] AI Houston and JM McNamara. *Models of adaptive behaviour: an approach based on state*. Cambridge University Press, United Kingdom, 1999. (Cited on page 31.)
- [Hughes *et al.* 2005] KA Hughes, FH Rodd and DN Reznick. *Genetic and environmental effects on secondary sex traits in guppies (*Poecilia reticulata*)*. *Journal of evolutionary biology*, vol. 18, no. 1, pages 35–45, 2005. (Cited on page 121.)
- [Hurst & Randerson 1999] Laurence D Hurst and James P Randerson. *An exceptional chromosome*. *Trends in Genetics*, vol. 15, no. 10, pages 383–385, 1999. (Cited on page 130.)
- [Iwasa & Pomiankowski 1995] Yoh Iwasa and Andrew Pomiankowski. *Continual change in mate preferences*. *Nature*, vol. 377, no. 6548, page 420, 1995. (Cited on page 41.)
- [Iwasa & Pomiankowski 1999] Yoh Iwasa and Andrew Pomiankowski. *Good parent and good genes models of handicap evolution*. *Journal of Theoretical Biology*, vol. 200, no. 1, pages 97–109, 1999. (Cited on pages 10 and 24.)

- [Jackson 1992] RR Jackson. *Conditional strategies and interpopulation variation in the behaviour of jumping spiders*. New Zealand Journal of Zoology, vol. 19, no. 3-4, pages 99–111, 1992. (Cited on page 140.)
- [Janetos 1980] A.C. Janetos. *Strategies of female mate choice: a theoretical analysis*. Behavioral Ecology and Sociobiology 7, vol. 12, pages 107–112, 1980. (Cited on page 19.)
- [Jennions & Petrie 1997] M D Jennions and M Petrie. *Variation in mate choice and mating preferences: A review of causes and consequences*. Biological reviews of the Cambridge Philosophical Society, vol. 72, no. 2, pages 283–327, 1997. (Cited on pages 5, 6 and 7.)
- [Johnstone *et al.* 1996] Rufus A Johnstone, John D Reynolds and James C Deutsch. *Mutual mate choice and sex differences in choosiness*. Evolution, vol. 50, no. 4, pages 1382–1391, 1996. (Cited on pages 7, 28 and 33.)
- [Johnstone 1997] Rufus A. Johnstone. *The tactics of mutual mate choice and competitive search*. Behavioral Ecology and Sociobiology, vol. 40, no. 1, pages 51–59, 1997. (Cited on pages 7, 8 and 29.)
- [Jones *et al.* 2003] Adam G Jones, Stevan J Arnold and Reinhard Bürger. *Stability of the G-matrix in a population experiencing pleiotropic mutation, stabilizing selection, and genetic drift*. Evolution, vol. 57, no. 8, pages 1747–1760, 2003. (Cited on page 75.)
- [Judge *et al.* 2014] KA Judge, JJ Ting and DT Gwynne. *Condition dependence of female choosiness in a field cricket*. Journal of evolutionary biology, vol. 27, no. 11, pages 2529–2540, 2014. (Cited on pages 7 and 30.)

- [Kashtan & Alon 2005] Nadav Kashtan and Uri Alon. *Spontaneous evolution of modularity and network motifs*. Proceedings of the National Academy of Sciences, vol. 102, no. 39, pages 13773–13778, 2005. (Cited on pages 101 and 145.)
- [Kent *et al.* 2008] Clement Kent, Reza Azanchi, Ben Smith, Amanda Formosa and Joel D Levine. *Social context influences chemical communication in *D. melanogaster* males*. Current Biology, vol. 18, no. 18, pages 1384–1389, 2008. (Cited on page 137.)
- [Kilmer *et al.* 2017] JT Kilmer, KD Fowler-Finn, DA Gray, G Höbel, D Rebar, MS Reichert and RL Rodríguez. *Describing mate preference functions and other function-valued traits*. Journal of evolutionary biology, vol. 30, no. 9, pages 1658–1673, 2017. (Cited on pages 5, 6 and 249.)
- [Kirkpatrick & Barton 1997] Mark Kirkpatrick and Nicholas H Barton. *Evolution of a species' range*. The American Naturalist, vol. 150, no. 1, pages 1–23, 1997. (Cited on page 92.)
- [Kirkpatrick & Hall 2004] Mark Kirkpatrick and David W. Hall. *Sexual selection and sex linkage*. Evolution, vol. 58, no. 4, pages 683–691, 2004. (Cited on page 137.)
- [Kirkpatrick 1996] Mark Kirkpatrick. *Good genes and direct selection in the evolution of mating preferences*. Evolution, vol. 50, no. 6, pages 2125–2140, 1996. (Cited on page 10.)
- [Kokko & Brooks 2003] Hanna Kokko and Robert Brooks. *Sexy to die for? Sexual selection and the risk of extinction*. Annales Zoologici Fennici, vol. 40, no. April, pages 207–219, 2003. (Cited on pages 18, 19 and 107.)

- [Kokko & Jennions 2008] Hanna Kokko and Michael D. Jennions. *Parental investment, sexual selection and sex ratios*. *Journal of Evolutionary Biology*, vol. 21, no. 4, pages 919–948, 2008. (Cited on pages 12 and 147.)
- [Kokko & Johnstone 2002] Hanna Kokko and Rufus A Johnstone. *Why is mutual mate choice not the norm? Operational sex ratios, sex roles and the evolution of sexually dimorphic and monomorphic signalling*. *Philosophical Transactions of the Royal Society of London. Series B: Biological Sciences*, vol. 357, no. 1419, pages 319–330, 2002. (Cited on pages 7, 19, 29 and 130.)
- [Kokko & Monaghan 2001] H. Kokko and P. Monaghan. *Predicting the direction of sexual selection*. *Ecology Letters*, vol. 4, no. 2, pages 159–165, 2001. (Cited on pages 7 and 29.)
- [Kokko & Rankin 2006] Hanna Kokko and Daniel J Rankin. *Lonely hearts or sex in the city? Density-dependent effects in mating systems*. *Philosophical Transactions of the Royal Society B: Biological Sciences*, vol. 361, no. 1466, pages 319–334, 2006. (Cited on pages 19 and 100.)
- [Kokko *et al.* 2002] Hanna Kokko, Robert Brooks, John M McNamara and Alasdair I Houston. *The sexual selection continuum*. *Proceedings of the Royal Society of London. Series B: Biological Sciences*, vol. 269, no. 1498, pages 1331–1340, 2002. (Cited on pages 5 and 10.)
- [Kokko *et al.* 2003] Hanna Kokko, Robert Brooks, Michael D Jennions and Josephine Morley. *The evolution of mate choice and mating biases*. *Proceedings of the Royal Society of London. Series B: Biological Sciences*, vol. 270, no. 1515, pages 653–664, 2003. (Cited on pages 28 and 117.)

- [Kokko *et al.* 2007] Hanna Kokko, Michael D Jennions and Anne Houde. *Evolution of frequency-dependent mate choice: keeping up with fashion trends*. Proceedings of the Royal Society B: Biological Sciences, vol. 274, no. 1615, pages 1317–1324, 2007. (Cited on pages 20 and 28.)
- [Kokko *et al.* 2015] Hanna Kokko, Isobel Booksmythe and Michael D Jennions. *Mate-sampling costs and sexy sons*. Journal of evolutionary biology, vol. 28, no. 1, pages 259–266, 2015. (Cited on pages 20, 92, 95, 100 and 137.)
- [Kopp & Hermisson 2006] Michael Kopp and Joachim Hermisson. *The evolution of genetic architecture under frequency-dependent disruptive selection*. Evolution, vol. 60, no. 8, pages 1537–1550, 2006. (Cited on page 101.)
- [Krupp *et al.* 2008] Joshua J Krupp, Clement Kent, Jean-Christophe Billeter, Reza Azanchi, Anthony K-C So, Julia A Schonfeld, Benjamin P Smith, Christophe Lucas and Joel D Levine. *Social experience modifies pheromone expression and mating behavior in male Drosophila melanogaster*. Current Biology, vol. 18, no. 18, pages 1373–1383, 2008. (Cited on page 137.)
- [Kvarnemo & Ahnesjo 1996] Charlotta Kvarnemo and Ingrid Ahnesjo. *The dynamics of operational sex ratios and competition for mates*. Trends in Ecology & Evolution, vol. 11, no. 10, pages 404–408, 1996. (Cited on page 13.)
- [Labonne & Hendry 2010] Jacques Labonne and Andrew P Hendry. *Natural and sexual selection giveth and taketh away reproductive barriers: models of population divergence in guppies*. The American Naturalist, vol. 176, no. 1, pages 26–39, 2010. (Cited on pages 42 and 43.)
- [Labonne *et al.* 2009] Jacques Labonne, Matthieu Augery, Michel Parade, Stéphane Brinkert, Etienne Prevost, Michel Héland and Edward Beall. *Female prefer-*

- ence for male body size in brown trout, Salmo trutta: is big still fashionable?* Animal Behaviour, vol. 77, no. 1, pages 129–137, 2009. (Cited on page 30.)
- [Lamichhaney *et al.* 2016] Sangeet Lamichhaney, Guangyi Fan, Fredrik Widemo, Ulrika Gunnarsson, Doreen Schwochow Thalmann, Marc P Hoepfner, Susanne Kerje, Ulla Gustafson, Chengcheng Shi, He Zhanget *al.* *Structural genomic changes underlie alternative reproductive strategies in the ruff (Philo-machus pugnax)*. Nature Genetics, vol. 48, no. 1, pages 84–88, 2016. (Cited on page 138.)
- [Lande & Arnold 1983] Russell Lande and Stevan J Arnold. *The measurement of selection on correlated characters*. Evolution, pages 1210–1226, 1983. (Cited on page 38.)
- [Lande & Arnold 1985] Russell Lande and Stevan J. Arnold. *Evolution of mating preferences and sexual dimorphism*. Journal of Theoretical Biology, vol. 117, no. 4, pages 651–664, 1985. (Cited on page 117.)
- [Lande & Shannon 1996] Russell Lande and Susan Shannon. *The role of genetic variation in adaptation and population persistence in a changing environment*. Evolution, vol. 50, no. 1, pages 434–437, 1996. (Cited on page 103.)
- [Lande 1975] Russell Lande. *The maintenance of genetic variability by mutation in a polygenic character with linked loci*. Genetics Research, vol. 26, no. 3, pages 221–235, 1975. (Cited on page 121.)
- [Lande 1981] Russell Lande. *Models of speciation by sexual selection on polygenic traits*. Proceedings of the National Academy of Sciences, vol. 78, no. 6, pages 3721–3725, 1981. (Cited on pages 14, 15, 41, 69, 78, 87, 93, 95, 141 and 250.)

- [Lande 1982] Russell Lande. *A quantitative genetic theory of life history evolution*. Ecology, vol. 63, no. 3, pages 607–615, 1982. (Cited on pages 15 and 117.)
- [Lehtonen & Kokko 2011] Jussi Lehtonen and Hanna Kokko. *Two roads to two sexes: Unifying gamete competition and gamete limitation in a single model of anisogamy evolution*. Behavioral Ecology and Sociobiology, vol. 65, no. 3, pages 445–459, 2011. (Cited on pages 12 and 146.)
- [Lehtonen *et al.* 2016] Jussi Lehtonen, Geoff A Parker and Lukas Schärer. *Why anisogamy drives ancestral sex roles*. Evolution, vol. 70, no. 5, pages 1129–1135, 2016. (Cited on page 147.)
- [Lindström & Lehtonen 2013] Kai Lindström and Topi K Lehtonen. *Mate sampling and choosiness in the sand goby*. Proceedings of the Royal Society B: Biological Sciences, vol. 280, no. 1765, page 20130983, 2013. (Cited on page 7.)
- [Liou & Price 1994] Lily W Liou and Trevor D Price. *Speciation by reinforcement of premating isolation*. Evolution, vol. 48, no. 5, pages 1451–1459, 1994. (Cited on page 19.)
- [Lipson *et al.* 2002] Hod Lipson, Jordan B Pollack and Nam P Suh. *On the origin of modular variation*. Evolution, vol. 56, no. 8, pages 1549–1556, 2002. (Cited on page 101.)
- [Lively & Lloyd 1990] Curtis M Lively and David G Lloyd. *The cost of biparental sex under individual selection*. The American Naturalist, vol. 135, no. 4, pages 489–500, 1990. (Cited on page 2.)
- [Mank *et al.* 2007] Judith E Mank, Erik Axelsson and Hans Ellegren. *Fast-X on the Z: rapid evolution of sex-linked genes in birds*. Genome Research, vol. 17, no. 5, pages 618–624, 2007. (Cited on page 15.)

- [Mank *et al.* 2008] Judith ÅE. Mank, Lina Hultin Å€Rosenberg, Martin Zwahlen and Hans Ellegren. *Pleiotropic Constraint Hampers the Resolution of Sexual Antagonism in Vertebrate Gene Expression*. *The American Naturalist*, vol. 171, no. 1, pages 35–43, 2008. (Cited on page 138.)
- [Mank 2009] Judith E Mank. *Sex chromosomes and the evolution of sexual dimorphism: lessons from the genome*. *The American Naturalist*, vol. 173, no. 2, pages 141–150, 2009. (Cited on pages 15, 121 and 134.)
- [Matessi & Di Pasquale 1996] Carlo Matessi and Cristina Di Pasquale. *Long-term evolution of multilocus traits*. *Journal of Mathematical Biology*, vol. 34, no. 5-6, pages 613–653, 1996. (Cited on page 137.)
- [Matessi & Eshel 1992] Carlo Matessi and Ilan Eshel. *Sex ratio in the social Hymenoptera: a population-genetics study of long-term evolution*. *The American Naturalist*, vol. 139, no. 2, pages 276–312, 1992. (Cited on page 120.)
- [McNamara & Collins 1990] J M McNamara and E J Collins. *the Job Search Problem As an Employer Candidate Game*. *Journal of Applied Probability*, vol. 27, no. 4, pages 815–827, 1990. (Cited on pages 29 and 33.)
- [McVean & Charlesworth 2000] Gilean AT McVean and Brian Charlesworth. *The effects of Hill-Robertson interference between weakly selected mutations on patterns of molecular evolution and variation*. *Genetics*, vol. 155, no. 2, pages 929–944, 2000. (Cited on page 126.)
- [Mead & Arnold 2004] Louise S Mead and Stevan J Arnold. *Quantitative genetic models of sexual selection*. *Trends in ecology & evolution*, vol. 19, no. 5, pages 264–271, 2004. (Cited on page 15.)

- [Michiels *et al.* 2009] Nico K Michiels, Philip H Crowley and Nils Anthes. *Accessory male investment can undermine the evolutionary stability of simultaneous hermaphroditism*. *Biology Letters*, vol. 5, no. 5, pages 709–712, 2009. (Cited on page 25.)
- [Milinski & Bakker 1992] Manfred Milinski and Theo CM Bakker. *Costs influences sequential mate choice in sticklebacks, *Gasterosteus aculeatus**. *Proceedings of the Royal Society of London. Series B: Biological Sciences*, vol. 250, no. 1329, pages 229–233, 1992. (Cited on page 7.)
- [Misevic *et al.* 2006] Dusan Misevic, Charles Ofria and Richard E Lenski. *Sexual reproduction reshapes the genetic architecture of digital organisms*. *Proceedings of the Royal Society B: Biological Sciences*, vol. 273, no. 1585, pages 457–464, 2006. (Cited on page 138.)
- [Møller & Legendre 2001] Anders Pape Møller and Stéphane Legendre. *Allee effect, sexual selection and demographic stochasticity*. *Oikos*, vol. 92, no. 1, pages 27–34, 2001. (Cited on page 19.)
- [Moore *et al.* 1997] Allen J Moore, Edmund D Brodie III and Jason B Wolf. *Interacting phenotypes and the evolutionary process: I. Direct and indirect genetic effects of social interactions*. *Evolution*, vol. 51, no. 5, pages 1352–1362, 1997. (Cited on page 137.)
- [Muller 1928] Hermann J Muller. *The measurement of gene mutation rate in *Drosophila*, its high variability, and its dependence upon temperature*. *Genetics*, vol. 13, no. 4, page 279, 1928. (Cited on page 60.)
- [Muniz & Machado 2018] Danilo G Muniz and Glauco Machado. *Mate sampling influences the intensity of sexual selection and the evolution of costly sexual*

- ornaments*. Journal of theoretical biology, vol. 447, pages 74–83, 2018. (Cited on pages 92 and 137.)
- [Nash *et al.* 1950] John F Nash *et al.* *Equilibrium points in n-person games*. Proceedings of the national academy of sciences, vol. 36, no. 1, pages 48–49, 1950. (Cited on page 27.)
- [Neelon *et al.* 2019] Daniel P Neelon, Rafael L Rodríguez and Gerlinde Höbel. *On the architecture of mate choice decisions: preference functions and choosiness are distinct traits*. Proceedings of the Royal Society B, vol. 286, no. 1897, page 20182830, 2019. (Cited on page 5.)
- [Nelson *et al.* 2013] Ronald M Nelson, Mats E Pettersson and Örjan Carlborg. *A century after Fisher: time for a new paradigm in quantitative genetics*. Trends in Genetics, vol. 29, no. 12, pages 669–676, 2013. (Cited on page 42.)
- [Neuenschwander *et al.* 2008] Samuel Neuenschwander, Frédéric Hospital, Frédéric Guillaume and Jérôme Goudet. *quantiNemo: an individual-based program to simulate quantitative traits with explicit genetic architecture in a dynamic metapopulation*. Bioinformatics, vol. 24, no. 13, pages 1552–1553, 2008. (Cited on page 45.)
- [Nichols & Butlin 1989] Richard A Nichols and Roger K Butlin. *Does runaway sexual selection work in finite populations?* Journal of Evolutionary Biology, vol. 2, no. 4, pages 299–313, 1989. (Cited on page 95.)
- [Noë 2017] Ronald Noë. *Local mating markets in humans and non-human animals*. Behavioral Ecology and Sociobiology, vol. 71, no. 10, page 148, 2017. (Cited on page 100.)

- [Nordling *et al.* 1998] Dag Nordling, Måns Andersson, Siamak Zohari and Gustafsson Lars. *Reproductive effort reduces specific immune response and parasite resistance*. Proceedings of the Royal Society of London. Series B: Biological Sciences, vol. 265, no. 1403, pages 1291–1298, 1998. (Cited on page 8.)
- [Oddou-Muratorio *et al.* 2020] Sylvie Oddou-Muratorio, Hendrik Davi and François Lefèvre. *Integrating evolutionary, demographic and ecophysiological processes to predict the adaptive dynamics of forest tree populations under global change*. Tree Genetics & Genomes, vol. 16, no. 5, pages 1–22, 2020. (Cited on page 44.)
- [Oke *et al.* 2017] Krista B Oke, Gregor Rolshausen, Caroline LeBlond and Andrew P Hendry. *How parallel is parallel evolution? A comparative analysis in fishes*. The American Naturalist, vol. 190, no. 1, pages 1–16, 2017. (Cited on page 94.)
- [Orr 2000] H. Allen Orr. *Adaptation and the Cost of Complexity*. Evolution, vol. 54, no. 1, pages 13–20, 2000. (Cited on page 18.)
- [Otto *et al.* 2008] Sarah P Otto, Maria R Servedio and Scott L Nuismer. *Frequency-dependent selection and the evolution of assortative mating*. Genetics, vol. 179, no. 4, pages 2091–2112, 2008. (Cited on page 92.)
- [Otto 2004] Sarah P Otto. *Two steps forward, one step back: the pleiotropic effects of favoured alleles*. Proceedings of the Royal Society of London. Series B: Biological Sciences, vol. 271, no. 1540, pages 705–714, 2004. (Cited on page 75.)
- [Otto 2009] Sarah P Otto. *The evolutionary enigma of sex*. the american naturalist, vol. 174, no. S1, pages S1–S14, 2009. (Cited on page 2.)

- [Paaby & Rockman 2013] Annalise B Paaby and Matthew V Rockman. *The many faces of pleiotropy*. Trends in Genetics, vol. 29, no. 2, pages 66–73, 2013. (Cited on pages 17 and 61.)
- [Parker *et al.* 1972] G. A. Parker, R. R. Baker and V. G F Smith. *The origin and evolution of gamete dimorphism and the male-female phenomenon*. Journal of Theoretical Biology, vol. 36, no. 3, pages 529–553, 1972. (Cited on page 147.)
- [Parker 1983] GA Parker. *Mate quality and mating decisions*. Mate choice, vol. 141, page 166, 1983. (Cited on page 19.)
- [Parry 1981] Gregory D Parry. *The meanings of r-and K-selection*. Oecologia, vol. 48, no. 2, pages 260–264, 1981. (Cited on page 8.)
- [Parter *et al.* 2007] Merav Parter, Nadav Kashtan and Uri Alon. *Environmental variability and modularity of bacterial metabolic networks*. BMC evolutionary biology, vol. 7, no. 1, page 169, 2007. (Cited on page 101.)
- [Partridge & Hurst 1998] Linda Partridge and Laurence D Hurst. *Sex and conflict*. Science, vol. 281, no. 5385, pages 2003–2008, 1998. (Cited on page 2.)
- [Pavlicev *et al.* 2011] Mihaela Pavlicev, Elizabeth A Norgard, Gloria L Fawcett and James M Cheverud. *Evolution of pleiotropy: epistatic interaction pattern supports a mechanistic model underlying variation in genotype–phenotype map*. Journal of Experimental Zoology Part B: Molecular and Developmental Evolution, vol. 316, no. 5, pages 371–385, 2011. (Cited on pages 15 and 16.)
- [Peng & Kimmel 2005] Bo Peng and Marek Kimmel. *simuPOP: a forward-time population genetics simulation environment*. Bioinformatics, vol. 21, no. 18, pages 3686–3687, 2005. (Cited on page 44.)

- [Pepper 2000] John W Pepper. *The evolution of modularity in genome architecture*. Artificial Life, vol. 7, pages 9–12, 2000. (Cited on pages 75 and 96.)
- [Phillips 2005] Patrick C Phillips. *Testing hypotheses regarding the genetics of adaptation*. Genetica, vol. 123, no. 1-2, pages 15–24, 2005. (Cited on page 18.)
- [Pianka 1976] Eric R Pianka. *Natural selection of optimal reproductive tactics*. American Zoologist, vol. 16, no. 4, pages 775–784, 1976. (Cited on pages 25, 26 and 249.)
- [Pigliucci 2008] Massimo Pigliucci. *Is evolvability evolvable?* Nature Reviews Genetics, vol. 9, no. 1, pages 75–82, 2008. (Cited on page 94.)
- [Plomion *et al.* 2018] Christophe Plomion, Jean-Marc Aury, Joelle Amsellem, Thibault Leroy, Florent Murat, Sébastien Duplessis, Sébastien Faye, Nicolas Francillonne, Karine Labadie, Grégoire Le Provost *et al.* *Oak genome reveals facets of long lifespan*. Nature Plants, vol. 4, no. 7, pages 440–452, 2018. (Cited on page 138.)
- [Pomiankowski 1987] Andrew Pomiankowski. *The costs of choice in sexual selection*. Journal of Theoretical Biology, vol. 128, no. 2, pages 195–218, 1987. (Cited on page 7.)
- [Promislow 1992] Daniel EL Promislow. *Costs of sexual selection in natural populations of mammals*. Proceedings of the Royal Society of London. Series B: Biological Sciences, vol. 247, no. 1320, pages 203–210, 1992. (Cited on page 18.)
- [Promislow 2006] Daniel EL Promislow. *Sex chromosomes and male ornaments: a comparative evaluation in ray-finned fishes*. Proceedings of the Royal Society

B: Biological Sciences 273.1583, vol. 273, no. 1583, pages 233–236, 2006.

(Cited on page 121.)

[Rajon & Plotkin 2013] Etienne Rajon and Joshua B Plotkin. *The evolution of genetic architectures underlying quantitative traits*. Proceedings of the Royal Society B: Biological Sciences, vol. 280, no. 1769, page 20131552, 2013. (Cited on pages 14 and 15.)

[Ramsey 2011] David M Ramsey. *Mutual mate choice with multiple criteria*. In Advances in dynamic games, pages 337–355. Springer, 2011. (Cited on page 29.)

[Real 1990] Leslie Real. *Search Theory and Mate Choice. I. Models of Single-Sex Discrimination*. The American Naturalist, vol. 136, no. 3, pages 376–405, 1990. (Cited on page 19.)

[Rees *et al.* 2008] David J Rees, Claude Belzile, Helene Glemet and France Dufresne. *Large genomes among caridean shrimp*. Genome, vol. 51, no. 2, pages 159–163, 2008. (Cited on page 135.)

[Reeve & Pfennig 2003] Hudson Kern Reeve and David W Pfennig. *Genetic biases for showy males: Are some genetic systems especially conducive to sexual selection?* Proceedings of the National Academy of Sciences, vol. 100, no. 3, pages 1089–1094, 2003. (Cited on pages 121, 134 and 138.)

[Reinhold & Schielzeth 2015] Klaus Reinhold and Holger Schielzeth. *Choosiness, a neglected aspect of preference functions: a review of methods, challenges and statistical approaches*. Journal of Comparative Physiology A, vol. 201, no. 1, pages 171–182, 2015. (Cited on page 7.)

- [Rice 1984] William Rice. *Sex Chromosomes and the Evolution of Sexual Dimorphism*. Society for the Study of Evolution, vol. 38, no. 4, pages 735–742, 1984. (Cited on page 134.)
- [Rittschof & Robinson 2014] Clare C. Rittschof and Gene E. Robinson. *Genomics: Moving behavioural ecology beyond the phenotypic gambit*. Animal Behaviour, vol. 92, pages 263–270, 2014. (Cited on pages 15 and 93.)
- [Rodd *et al.* 2002] F Helen Rodd, Kimberly A Hughes, Gregory F Grether and Collette T Baril. *A possible non-sexual origin of mate preference: are male guppies mimicking fruit?* Proceedings of the Royal Society of London. Series B: Biological Sciences, vol. 269, no. 1490, pages 475–481, 2002. (Cited on page 11.)
- [Roff & Fairbairn 2012] Derek A Roff and Daphne J Fairbairn. *A test of the hypothesis that correlational selection generates genetic correlations*. Evolution: International Journal of Organic Evolution, vol. 66, no. 9, pages 2953–2960, 2012. (Cited on page 20.)
- [Roff 2010] Derek A Roff. *Modeling evolution: an introduction to numerical methods*. Oxford University Press, 2010. (Cited on pages 31 and 36.)
- [Rosvall 2011] Kimberly A Rosvall. *Intrasexual competition in females: evidence for sexual selection?* Behavioral Ecology, vol. 22, no. 6, pages 1131–1140, 2011. (Cited on page 13.)
- [Rowe & Houle 1996] Locke Rowe and David Houle. *The lek paradox and the capture of genetic variance by condition dependent traits*. Proceedings of the Royal Society of London. Series B: Biological Sciences, vol. 263, no. 1375, pages 1415–1421, 1996. (Cited on page 117.)

- [Schärer *et al.* 2012] Lukas Schärer, Locke Rowe and Göran Arnqvist. *Anisogamy, chance and the evolution of sex roles*. Trends in Ecology and Evolution, vol. 27, no. 5, pages 260–264, 2012. (Cited on page 130.)
- [Schwartz & Hendry 2007] Amy K Schwartz and Andrew P Hendry. *A test for the parallel co-evolution of male colour and female preference in Trinidadian guppies (*Poecilia reticulata*)*. Evolutionary Ecology Research, vol. 9, no. 1, pages 71–90, 2007. (Cited on page 94.)
- [Seger 1987] HJ Seger J & Brockmann. *What is bet-hedging*. Oxford surveys in evolutionary biology, vol. 4, 1987. (Cited on page 102.)
- [Servedio & Lande 2006] Maria R. Servedio and Russell Lande. *Population genetic models of male and mutual mate choice*. Evolution; international journal of organic evolution, vol. 60, no. 4, pages 674–685, April 2006. (Cited on pages 42 and 137.)
- [Servedio *et al.* 2011] Maria R Servedio, G Sander Van Doorn, Michael Kopp, Alicia M Frame and Patrik Nosil. *Magic traits in speciation: ‘magic’ but not rare?* Trends in ecology & evolution, vol. 26, no. 8, pages 389–397, 2011. (Cited on page 117.)
- [Servedio 2001] Maria R Servedio. *Beyond reinforcement: the evolution of pre-mating isolation by direct selection on preferences and postmating, prezygotic incompatibilities*. Evolution, vol. 55, no. 10, pages 1909–1920, 2001. (Cited on page 19.)
- [Sgrò & Hoffmann 2004] Carla Maria Sgrò and Ary A Hoffmann. *Genetic correlations, tradeoffs and environmental variation*. Heredity, vol. 93, no. 3, pages 241–248, 2004. (Cited on page 101.)

- [Shaw *et al.* 2011] Kerry L Shaw, Christopher K Ellison, Kevin P Oh and Chris Wiley. *Pleiotropy, “sexy” traits, and speciation*. Behavioral Ecology, vol. 22, no. 6, pages 1154–1155, 2011. (Cited on page 117.)
- [Sih 1994] Andrew Sih. *Predation risk and the evolutionary ecology of reproductive behaviour*. Journal of fish biology, vol. 45, pages 111–130, 1994. (Cited on page 8.)
- [Sinervo & Svensson 2002] B Sinervo and Erik Svensson. *Correlational selection and the evolution of genomic architecture*. Heredity, vol. 89, no. 5, pages 329–338, 2002. (Cited on pages 20 and 138.)
- [Smith & Maynard-Smith 1978] John Maynard Smith and John Maynard-Smith. *The evolution of sex*, volume 4. Cambridge University Press Cambridge, 1978. (Cited on page 1.)
- [Smith 1978] John Maynard Smith. *Optimization Theory in Evolution*. Annual Review of Ecology and Systematics, vol. 9, no. 1, pages 31–56, 1978. (Cited on page 2.)
- [Smith 1982] John Maynard Smith. *Evolution and the theory of games*. Cambridge university press, 1982. (Cited on pages 27 and 28.)
- [Smith 1991] John Maynard Smith. *Theories of sexual selection*. Trends in Ecology & Evolution, vol. 6, no. 5, pages 146–151, 1991. (Cited on page 10.)
- [Souroukis & Murray 1994] Konstantine Souroukis and Anne-Marie Murray. *Female mating behavior in the field cricket, *Gryllus pennsylvanicus* (Orthoptera: Gryllidae) at different operational sex ratios*. Journal of Insect Behavior, vol. 8, no. 2, pages 269–279, 1994. (Cited on page 12.)

- [Springer *et al.* 2011] Stevan A Springer, Bernard J Crespi and Willie J Swanson. *Beyond the phenotypic gambit: molecular behavioural ecology and the evolution of genetic architecture*. *Molecular Ecology*, vol. 20, no. 11, pages 2240–2257, 2011. (Cited on page 14.)
- [Stearns *et al.* 1991] Steve Stearns, Gerdien de Jong and Bob Newman. *The effects of phenotypic plasticity on genetic correlations*. *Trends in ecology & evolution*, vol. 6, no. 4, pages 122–126, 1991. (Cited on page 101.)
- [Stearns 2000] Stephen C. Stearns. *Life history evolution: Successes, limitations, and prospects*. *Naturwissenschaften*, vol. 87, no. 11, pages 476–486, 2000. (Cited on page 8.)
- [Steppan *et al.* 2002] Scott J Steppan, Patrick C Phillips and David Houle. *Comparative quantitative genetics: evolution of the G matrix*. *Trends in Ecology & Evolution*, vol. 17, no. 7, pages 320–327, 2002. (Cited on page 42.)
- [Stockley & Bro-Jørgensen 2011] Paula Stockley and Jakob Bro-Jørgensen. *Female competition and its evolutionary consequences in mammals*. *Biological Reviews*, vol. 86, no. 2, pages 341–366, 2011. (Cited on page 13.)
- [Svensson 2019] Erik I Svensson. *Eco-evolutionary dynamics of sexual selection and sexual conflict*. *Functional Ecology*, vol. 33, no. 1, pages 60–72, 2019. (Cited on pages 20 and 43.)
- [Tang-Martinez & Ryder 2005] Zuleyma Tang-Martinez and T Brandt Ryder. *The problem with paradigms: Bateman’s worldview as a case study*. *Integrative and Comparative Biology*, vol. 45, no. 5, pages 821–830, 2005. (Cited on page 10.)

- [Tazzyman & Iwasa 2010] Samuel J Tazzyman and Yoh Iwasa. *Sexual selection can increase the effect of random genetic drift—A quantitative genetic model of polymorphism in *Oophaga pumilio*, the strawberry poison-dart frog*. *Evolution: International Journal of Organic Evolution*, vol. 64, no. 6, pages 1719–1728, 2010. (Cited on page 20.)
- [Tripathi *et al.* 2009] Namita Tripathi, Margarete Hoffmann, Eva-Maria Willing, Christa Lanz, Detlef Weigel and Christine Dreyer. *Genetic linkage map of the guppy, *Poecilia reticulata*, and quantitative trait loci analysis of male size and colour variation*. *Proceedings of the Royal Society B: Biological Sciences*, vol. 276, no. 1665, pages 2195–2208, 2009. (Cited on page 120.)
- [Trivers 1972] Robert Trivers. *Parental investment and sexual selection*, volume 136. *Biological Laboratories, Harvard University Cambridge*, 1972. (Cited on pages 12 and 13.)
- [Valette *et al.* 2020] Theo Valette, Maeva LEITWEIN, Jean-Marc LASCAUX, Erick DESMARAIS, Patrick BERREBI and Bruno GUINAND. *Spotting genome-wide pigmentation variation in a brown trout admixture context*. *bioRxiv*, 2020. (Cited on page 15.)
- [Von Neumann 1992] John Von Neumann. *"Sur la théorie des jeux."* *Comptes rendus de l'académie des sciences*, pages 1689–91, 1992. (Cited on page 27.)
- [Wacker & Amundsen 2014] Sebastian Wacker and Trond Amundsen. *Mate competition and resource competition are inter-related in sexual selection*. *Journal of evolutionary biology*, vol. 27, no. 3, pages 466–477, 2014. (Cited on page 100.)

- [Wagner & Altenberg 1996] Günter P Wagner and Lee Altenberg. *Perspective: complex adaptations and the evolution of evolvability*. *Evolution*, vol. 50, no. 3, pages 967–976, 1996. (Cited on page 16.)
- [Wagner *et al.* 2007] Günter P Wagner, Mihaela Pavlicev and James M Cheverud. *The road to modularity*. *Nature Reviews Genetics*, vol. 8, no. 12, pages 921–931, 2007. (Cited on page 18.)
- [Wagner 1998] We E J Wagner. *Measuring female mating preferences*. *Animal Behaviour*, vol. 55, no. February 1997, pages 1029–1042, 1998. (Cited on page 5.)
- [Walsh & Blows 2009] Bruce Walsh and Mark W Blows. *Abundant genetic variation+ strong selection= multivariate genetic constraints: a geometric view of adaptation*. *Annual Review of Ecology, Evolution, and Systematics*, vol. 40, pages 41–59, 2009. (Cited on pages 20 and 42.)
- [Wang *et al.* 2010] Zhi Wang, Ben-Yang Liao and Jianzhi Zhang. *Genomic patterns of pleiotropy and the evolution of complexity*. *Proceedings of the National Academy of Sciences of the United States of America*, vol. 107, no. 42, pages 18034–18039, 2010. (Cited on page 18.)
- [Wedekind & Penn 2000] Claus Wedekind and Dustin Penn. *MHC genes, body odours, and odour preferences*. *Nephrology Dialysis Transplantation*, vol. 15, no. 9, pages 1269–1271, 2000. (Cited on page 14.)
- [Weir *et al.* 2011] Laura K. Weir, James W. Grant and Jeffrey A. Hutchings. *The Influence of Operational Sex Ratio on the Intensity of Competition for Mates*. *The American Naturalist*, vol. 177, no. 2, pages 167–176, 2011. (Cited on page 100.)

- [Weismann 1889] A Weismann. *The significance of sexual reproduction in the theory of natural selection* In: Poulton EB, Schönland S, Shipley AE., editors. *Essays upon heredity and kindred biological problems*, 1889. (Cited on page 2.)
- [Welch & Waxman 2003] John J Welch and David Waxman. *Modularity and the cost of complexity*. *Evolution*, vol. 57, no. 8, pages 1723–1734, 2003. (Cited on page 16.)
- [Wellenreuther *et al.* 2014] Maren Wellenreuther, Erik I Svensson and Bengt Hansson. *Sexual selection and genetic colour polymorphisms in animals*. *Molecular ecology*, vol. 23, no. 22, pages 5398–5414, 2014. (Cited on page 15.)
- [West-Eberhard 1983] Mary Jane West-Eberhard. *Sexual selection, social competition, and speciation*. *The Quarterly Review of Biology*, vol. 58, no. 2, pages 155–183, 1983. (Cited on page 137.)
- [Whitlock & Agrawal 2009] Michael C Whitlock and Aneil F Agrawal. *Purging the genome with sexual selection: reducing mutation load through selection on males*. *Evolution: International Journal of Organic Evolution*, vol. 63, no. 3, pages 569–582, 2009. (Cited on page 15.)
- [Wilbur & Rudolf 2006] Henry M Wilbur and Volker HW Rudolf. *Life-history evolution in uncertain environments: bet hedging in time*. *The American Naturalist*, vol. 168, no. 3, pages 398–411, 2006. (Cited on page 81.)
- [Wilkinson *et al.* 2015] Gerald S Wilkinson, Felix Breden, Judith E Mank, Michael G Ritchie, Andrew D Higginson, Jacek Radwan, Julie Jaquiere, Walter Salzburger, Elena Arriero, SM Barribeau *et al.* *The locus of sexual selection: moving sexual selection studies into the post-genomics era*. *Jour-*

- nal of evolutionary biology, vol. 28, no. 4, pages 739–755, 2015. (Cited on page 93.)
- [Williams 1966] G.C Williams. *Natural Selection , the Costs of Reproduction , and a Refinement of Lack ' s Principle*. American Society of Naturalists, vol. 100, no. 916, pages 687–690, 1966. (Cited on pages 8 and 12.)
- [Williams 1975] GC Williams. *Sex and Evolution Princeton University Press Princeton*. New Jersey, vol. 200, 1975. (Cited on pages 2 and 12.)
- [Wilson & MacArthur 1967] Edward O Wilson and Robert H MacArthur. The theory of island biogeography. Princeton University Press, 1967. (Cited on page 8.)
- [Wolf *et al.* 1999] Jason B Wolf, Edmund D Brodie III and Allen J Moore. *Interacting phenotypes and the evolutionary process. II. Selection resulting from social interactions*. The American Naturalist, vol. 153, no. 3, pages 254–266, 1999. (Cited on page 137.)
- [Wright & Mank 2013] AE Wright and JE Mank. *The scope and strength of sex-specific selection in genome evolution*. Journal of Evolutionary Biology, vol. 26, no. 9, pages 1841–1853, 2013. (Cited on pages 15 and 138.)
- [Wright 1984] Sewall Wright. Evolution and the genetics of populations, volume 1: genetic and biometric foundations, volume 1. University of Chicago press, 1984. (Cited on page 17.)
- [Zahavi 1975] Amotz Zahavi. *Mate selection—a selection for a handicap*. Journal of theoretical Biology, vol. 53, no. 1, pages 205–214, 1975. (Cited on pages 9 and 11.)

Appendices

**Article 1: Fluctuating Dynamics
of Mate Availability Promote the
Evolution of Flexible Choosiness
in Both Sexes**

Fluctuating Dynamics of Mate Availability Promote the Evolution of Flexible Choosiness in Both Sexes

Louise Chevalier,^{1,*} Jacques Labonne,¹ Matthias Galipaud,²
and François-Xavier Dechaume-Moncharmont³

1. Université de Pau et des Pays de l'Adour, UMR INRAE-UPPA, Ecobiop, FR-64310 Saint-Pée sur Nivelle, France; 2. University of Zurich, Department of Evolutionary Biology and Environmental Studies, Winterthurerstrasse 190, CH-8057 Zurich, Switzerland; 3. University of Lyon, Université Claude Bernard Lyon 1, CNRS, ENTPE, UMR 5023 LEHNA, F-69622 Villeurbanne, France

Submitted September 26, 2019; Accepted July 17, 2020; Electronically published October 23, 2020

Online enhancements: appendix, supplemental PDF, R code.

ABSTRACT: The evolution of choosiness has a strong effect on sexual selection, as it promotes variance in mating success among individuals. The context in which choosiness is expressed, and therefore the associated gain and cost, is highly variable. An overlooked mechanism by current models is the rapid fluctuations in the availability and quality of partners, which generates a dynamic mating market to which each individual must optimally respond. We argue that the rapid fluctuations of the mating market are central to the evolution of optimal choosiness. Using a dynamic game approach, we investigate this hypothesis for various mating systems (characterized by different adult sex ratio and latency period combinations), allowing feedback between the choosiness and partner availability throughout a breeding season while taking into account the fine variation in individual quality. Our results indicate that quality-dependent and flexible choosiness usually evolve in both sexes for various mating systems and that a significant amount of variance in choosiness is observed, especially in males, even when courtship is costly. Accounting for the fluctuating dynamics of the mating market therefore allows envisioning a much wider range of choosiness variation in natural populations and may explain a number of recent empirical results regarding choosiness in the less common sex or its variance within sexes.

Keywords: choosiness, mutual mate choice, flexibility, context dependency, game theory, scramble competition.

Introduction

In most animal species, individuals mate preferentially with some individuals over others, thereby displaying a choosy behavior. Choosiness can be defined as the probability that an encountered individual of a given quality

is considered an appropriate mate and is therefore accepted rather than rejected. Understanding the evolution of choosiness and underlying rules is paramount to understanding sexual selection, because mate choice leads to interindividual variability in reproductive success in the chosen sex (Darwin 1871; Andersson 1994; Jennions and Petrie 1997). The evolution of mate choice has been shown to depend on different factors. Optimal choosiness is predicted to increase: (i) when the survival cost of mate searching (or attraction) decreases (Real 1990; Crowley et al. 1991; Kokko and Johnstone 2002; Kopp and Hermisson 2008), (ii) when the costs of mating associated with lower survival when breeding becomes greater (Kokko and Monaghan 2001; Kokko and Johnstone 2002; Kopp and Hermisson 2008; Bleu et al. 2011), (iii) with increasing variance in mate quality in the population (Real 1990; Owens and Thompson 1994; Johnstone et al. 1996; Kokko and Johnstone 2002), or (iv) when mate availability increases as the risk of not finding an alternative mate decreases (Bleu et al. 2011; Fawcett et al. 2011; Etienne et al. 2014; Courtiol et al. 2016; Dechaume-Moncharmont et al. 2016).

Most of these factors are characteristics of the population and can therefore be considered constant on a relatively short timescale, such that their effect on choosiness should be significant across rather than within generations. For instance, the mating rate of individuals will be restricted by a period of unavailability after mating, dedicated to the replenishment of the gametic stock or to parental care, hereafter called the latency period, which will depend mainly on the physiology of the species. When factors affecting mate choice are constant, models predict that the evolution of mutual mate choice should only be observed under a restrictive set of conditions, specifically when latency periods are long and equal between sexes (thus the breeding cost is high) and when adult sex ratio (ASR) at maturation is balanced (Kokko and Johnstone

* Corresponding author; email: louise.chevalier@inra.fr.

ORCID: Chevalier, <https://orcid.org/0000-0002-5157-0228>; Labonne, <https://orcid.org/0000-0001-5953-3029>; Dechaume-Moncharmont, <https://orcid.org/0000-0001-7607-8224>.

Am. Nat. 2020. Vol. 196, pp. 000–000. © 2020 by The University of Chicago. 0003-0147/2020/19606-5951\$15.00. All rights reserved.

DOI: 10.1086/711417

2002; Kokko and Jennions 2008; Fromhage and Jennions 2016). When population characteristics depart from such specific conditions (e.g., owing to different latency periods between sexes, unbalanced ASR, or unbalanced maturation sex ratio because of differences in mortality between sexes), choosiness is usually favored in the less common sex. The expected differences in choosiness between the sexes can be amplified by a mechanism at the center of some previous models: the increase in choosiness in one sex is expected to cause a decrease in its mating rate, which also mechanically reduces the mating rate in the other sex, in which the choice therefore becomes even less favored (Kokko and Johnstone 2002; Courtiol et al. 2016). Importantly, the reduction in the mating rate associated with choosiness (i.e., the opportunity cost; Fawcett et al. 2011; Etienne et al. 2014; Dechaume-Moncharmont et al. 2016) can vary within generations and among individuals too. In particular, the number and quality distribution of mates available to a focal individual may change drastically among individuals and over time. Indeed, individuals who differ in their attractiveness may also vary in the number and average quality of mating partners they get access to (Johnstone 1997; McNamara et al. 1997). Rather than depending on fixed and homogeneous strategies, mating can be context dependent (Fricke et al. 2009; Rowe and Arnqvist 2015; Gauthey et al. 2016) and is therefore likely the result of a complex dynamic game where each individual tries at any time to maximize its reproductive output by optimizing its choosiness with regard to the choosiness of its competitors (Johnstone 1997; Fawcett and Johnstone 2003; Alpern and Reyniers 2005; Gowaty and Hubbell 2009; Bleu et al. 2011; Ramsey 2011). Of great interest is the possibility that choosiness evolves in both sexes, thereby giving rise to mutual mate choice, adding a new dimension in the game wherein individuals have to adjust not only to the choosiness of their same-sex competitors but also to the change in choosiness of the opposite sex (McNamara and Collins 1990; Collins and McNamara 1993; Johnstone 1997; Kokko and Monaghan 2001; Kokko and Johnstone 2002; Alpern and Reyniers 2005; Ramsey 2011; Courtiol et al. 2016). There are now several experimental publications suggesting that individuals can respond to such fluctuations by flexibly modifying their selection criteria (Chaine and Lyon 2008; Bailey and Zuk 2009; Tinghitella et al. 2013).

Finally, different mating systems, characterized for instance by contrasted ASR or differences in latency periods between sexes, can influence the dynamics with regard to the availability of mating partners (both in quantity and quality). This will in turn strongly affect the mating market, making optimal choosiness between sexes, between quality, and over time a nontrivial pattern. An associated yet central benefit of considering quality- and time-dependent choosiness is to bolster interactions be-

tween theoretical and empirical research: experiments and most observations in natural environments involve complex situations where individuals interact in a fluctuating context, producing choosiness that changes over time, or with the quality or sex of the individual (Byrne and Rice 2006; Labonne et al. 2009; Judge et al. 2014). This potentially generates variation in choosiness that is not necessarily captured by the current theoretical models. We argue that the existence of such variation is a fertile ground to improve our understanding of choosiness evolution and should therefore be accounted for in predictive models. To that end, we here present a dynamic game-theoretical model explicitly accounting for fluctuations in mate availability over a finite breeding season, where individuals of both sexes and of different quality can flexibly change their choosiness as a response to those fluctuations. Although we apply our model to a large range of mating systems and competitive conditions, we focus specifically on a polygynandrous mating system to demonstrate the complex relationship between the mating market dynamics, the evolution of quality-dependent and flexible choosiness, and the resulting patterns of mate choice.

Methods

The Game Model

The model is formulated as a dynamic game that describes mating in a population of females and males of different qualities. Both sexes are potentially choosy. All individuals have the possibility to compete for partners of variable quality. As a consequence, their fitness payoffs also depend on the choices made by the other individuals from both sexes in the population. We build a discrete time model with a finite time horizon (i.e., the breeding season) of length T time steps. Each individual is characterized by its quality (denoted q), which is a discrete value among $Q = 100$ possible classes of qualities regularly spaced between 0 and 1. The lowest class, noted $q = 0$, corresponds to the interval of quality $[0, 0.01]$ and the highest class, noted $q = 100$, corresponds to the interval of quality $[0.99, 1]$. Individual quality remains constant over the breeding season. We here assume that the fitness payoff from mating for an individual depends only on the quality of its mate or mates (but we also explored a fitness payoff equal to the product of qualities between partners; supplement 5; supplements 1–5 are available online). Initially, $f(q)$ specifies the probability distribution of individuals of quality for both males and females. The notations $f(q, t)_{\text{♂}}$ and $f(q, t)_{\text{♀}}$, respectively, are the relative frequency of males of quality q among available males and the relative frequency of females of quality q among available females, at time t . The model describes a mate encounter process

wherein, at each time step, individuals have a probability of encountering an available partner of a given quality (i.e., mass encounter; Gimelfarb 1988). When they meet, individuals independently and immediately (within the time step of the encounter) decide whether they accept mating or not. At a given time step t , when courtship is costless, their choice depends on their own quality and the quality of the encountered partner, as well as on the quality of prospective partners they could expect to meet before the end of the breeding season. Therefore, the model implicitly assumes that individuals know the opposite-sex distribution of quality throughout the breeding season. The choosiness strategy for an individual of quality q , denoted $p_{\sigma}(q, q', t)$ for males and $p_{\varphi}(q, q', t)$ for females, is therefore a $Q \times Q \times T$ matrix of acceptance probabilities for every possible partner's quality q' and at each time step t . Mated individuals become unavailable for further mating during their latency period, denoted τ_{σ} for males and τ_{φ} for females. At the end of their latency, they return to the pool of available partners. When τ_{σ} and τ_{φ} are greater than the length of the breeding season, each individual can only expect to mate once during the breeding season, which corresponds to a case of monogamy. Other mating systems are modeled as follows: polygyny ($\tau_{\sigma} \ll T$ and $\tau_{\varphi} \gg T$), polyandry ($\tau_{\varphi} \ll T$ and $\tau_{\sigma} \gg T$), or polygynandry ($\tau_{\sigma} \ll T$ and $\tau_{\varphi} \ll T$). Contrary to the ASR, which is the initial sex ratio of mature individuals in the population and is defined only at the beginning of the breeding season, the operational sex ratio(t), or OSR(t), is the ratio of available males to available females and changes dynamically during the breeding season, depending on latency periods, ASR, and the dynamics of the market. The OSR and the distribution of unpaired individual qualities are therefore emergent properties of the pair formation process, which results from choosiness strategies in the population.

Evolutionarily Stable Strategy of Choosiness

The reproductive success of an individual depends on the strategies of other players through direct interactions between encountered partners (because the choice is mutual) or indirect interactions between competitors via the removal of available partners in the population. To calculate the evolutionarily stable strategy, we used a method of best response iterations (Houston and McNamara 1999; Roff 2010). The core of the optimization algorithm is a two-step process. We begin with an arbitrary set of choice strategies, $p_{\text{resident}\sigma}(q, q', t)$ and $p_{\text{resident}\varphi}(q, q', t)$ and, for each time step t , we calculate the distributions of qualities for males ($f_{\sigma}(q, t)$) and females ($f_{\varphi}(q, t)$) that result from this set of strategies (app. A, available online). Assuming these distributions of quality, we then calculate the best response strategies for

both sexes, $p_{\text{best}\sigma}(q, q', t)$ and $p_{\text{best}\varphi}(q, q', t)$, as the strategies that maximize the individual's expected payoffs (see details below). The new resident set of strategies to be used in the next iteration is derived from the previous one as follows:

$$p_{\text{new}\sigma}(q, q', t) = (1 - \lambda)p_{\text{resident}\sigma}(q, q', t) + \lambda p_{\text{best}\sigma}(q, q', t), \quad (1)$$

where λ is a damping term that prevents oscillations in the iterative calculation of the optimal strategy (McNamara et al. 1997). This new set of strategies is used to calculate the new distributions of partner qualities across the season that in turn would lead to new adjustments of the choosiness strategies. We iterate this process until convergence of the sequences of choosiness strategies for males and females, such that a male or a female of any quality cannot increase their payoff by changing their choosiness strategy under the current distribution of qualities across the breeding season (i.e., when the difference in the set of strategies calculated in two successive iterations is < 0.001). Fewer than 2,000 iterations are necessary to reach convergence.

Backward Iterations: Computing the Best Response Strategy of Choosiness

Because the best choice at a given time depends on future gains, which in turn depend on future choices, the best choices at each time step are calculated backward from the end of the breeding season, using dynamic programming (Houston and McNamara 1999; Roff 2010). For example, we describe briefly the backward iteration procedure for males. The final time step T is the last opportunity for reproduction, so individuals should mate with whomever they encounter and gain a payoff q' . Individuals are assumed to choose the option leading to the highest expected payoff at each time step of the season. But errors in partners' quality assessment are possible. We therefore consider a stochastic outcome of the mating decision process instead of a deterministic step function defining a clear-cut discrimination between accepted and rejected partners' qualities (McNamara et al. 1997; Dechaume-Moncharmont et al. 2016). The probability that a potential partner will be accepted is thus expressed as a sigmoid function of the difference in payoffs between accepting and rejecting the mate:

$$p_{\text{best}\sigma}(q, q', t) = \frac{1 + \tanh\left\{\frac{1}{k}\left[w_{\text{accept}\sigma}(q, q', t) - w_{\text{reject}\sigma}(q, q', t)\right]\right\}}{2}, \quad (2)$$

where k corresponds to the slope of the sigmoid function at its inflection point (supplement 1) and specifies the

degree of error in assessing $w_{\text{accept}_\delta}(q, q', t) - w_{\text{reject}_\delta}(q, q', t)$; (Johnstone 1997; McNamara et al. 1997). The default value of k is 0.01. Sensitivity analyses (not shown herein) revealed that considering different values for k did not change model predictions qualitatively. The expected payoff at each time step of the breeding season is defined by backward recursion from the final time step T . The expected payoff at t corresponds to the sum of the payoff acquired at t and the prospective payoff acquired from $t + 1$ onward until the end of the season. At time t , individuals encounter a partner with a probability $P_E(t)$, which is calculated as follows for males and females:

$$P_{E_\delta}(t) = \min\left(1, \frac{1}{\text{OSR}(t)}\right), \quad (3)$$

$$P_{E_\varnothing}(t) = \min(1, \text{OSR}(t)).$$

The $\text{OSR}(t)$ is the ratio between available males and available females and is calculated by the ratio of the sums of the frequencies of available individuals by quality class in each sex, noted $f_{\text{abs}_\delta}(q, t)$ and $f_{\text{abs}_\varnothing}(q, t)$ (eq. [4]):

$$\text{OSR}(t) = \frac{\sum_q f_{\text{abs}_\delta}(q, t)}{\sum_q f_{\text{abs}_\varnothing}(q, t)}. \quad (4)$$

Upon encountering a potential partner, individuals can then decide to accept or reject this partner depending on its quality, on its own quality, and on the distribution of available qualities. If individuals reject a partner or get rejected at a time step $t < T$, they nevertheless receive expected future payoffs calculated from time step $t + 1$ (eq. [5]):

$$w_{\text{reject}_\delta}(q, q', t) = w_\delta(q, t + 1). \quad (5)$$

If an individual accepts a partner and gets accepted back, both of them gain an immediate payoff corresponding to the quality of their partner q' . They then have to wait until the end of their respective latency period to have another opportunity for reproduction (eq.[6]):

$$w_{\text{accept}_\delta}(q, q', t) = q' + \sum_{i=1}^{T-t} P_{A_\delta}(t + i)w_\delta(q, t + i), \quad (6)$$

where $P_{A_\delta}(t + i) = [1 - (1/\tau_\delta)]^{i-1}(1/\tau_\delta)$ is the probability that a male who entered in latency period at time step t becomes newly available for reproduction at time step $t + i$. To calculate this probability, we assumed that the latency period ends at a constant rate. As the average duration of latency period is τ_δ , at each time step t , the probability that the individual becomes newly available for reproduction during this time step is $1/\tau_\delta$. The fitness for an individual of quality q at time t is therefore calcu-

lated considering all possible situations weighted by their probabilities of occurrence (eq. [7]):

$$w_\delta(q, t) = P_{E_\delta}(t) \sum_q f_\varnothing(q', t) \times [p_{\text{best}_\delta}(q, q', t)p_{\text{resident}_\varnothing}(q', q, t)w_{\text{accept}_\delta}(q, q', t) + (1 - p_{\text{best}_\delta}(q, q', t)p_{\text{resident}_\varnothing}(q', q, t))w_{\text{reject}_\delta}(q, q', t)] + (1 - P_{E_\delta}(t))w_\delta(q, t + 1). \quad (7)$$

The first part of the equation corresponds to the situation where individuals encounter a partner at time t (with a probability $P_E(t)$). In that case, we sum the payoffs in all the possible situations weighted by their probabilities of occurrences: depending on the quality q' of the encountered partner, individuals can either mate or not. The second part of the equation corresponds to the situation where individuals do not encounter a partner at time step $t < T$, (with a probability $1 - P_E(t)$), individuals can still expect a future payoff at the next time step. Notice that if $t + 1 > T$, $w_\delta(q, t + 1) = 0$.

Costly Courtship

The previous calculations assume no direct courtship cost. It is however also possible that costly courtship may have already evolved, wherein the cost of courtship would be related to the difference in quality between potential partners (Fawcett and Johnstone 2003; Hårdling and Kokko 2005), assuming, for instance, that high-quality partners attract more rivals. We here represent such cost as a simple linear cost function. When individuals of quality q choose a mate of quality q' , they now have a direct cost to pay (eq. [8]):

$$c(q, q') = \begin{cases} g \times (q' - q) & \text{if } q' > q, \\ 1 & \text{if } q' \leq q. \end{cases} \quad (8)$$

And their payoff is thus as follows (eq. [9]):

$$w_{\text{accept}_\delta}(q, q', t) = q' - c(q, q') + \sum_{i=1}^{T-t} P_{A_\delta}(t + i)w_\delta(q, t + i). \quad (9)$$

The shape of the choosiness function (i.e., probability of acceptance as a function of partner quality) now depends on the intensity of the cost (i.e., value of g) and goes from threshold (when $g \leq 1$) to unimodal (when $g > 1$), as individuals pay a cost to court a partner of better quality than themselves. The exact shape of the choosiness depends on the value of g relative to $w_{\text{accept}}(q, q', t)$ and $w_{\text{reject}}(q, q', t)$, so it will vary with the chooser's quality and the time in the season (see fig. 1).

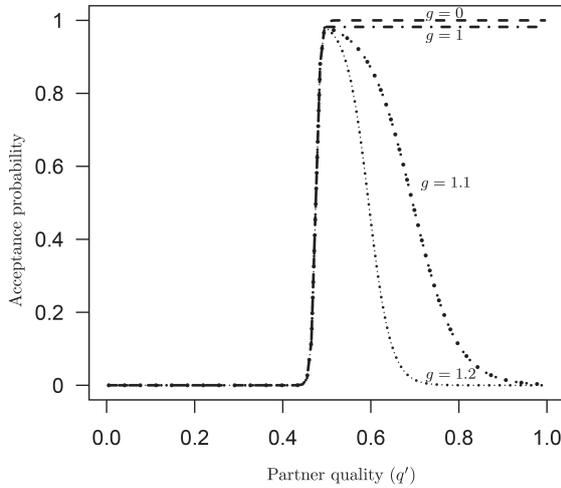


Figure 1: Probability to accept a partner as a function of its quality q' . The present example is calculated on the fiftieth time step of the breeding season, for different cost intensities ($g = 0$, $g = 1$, $g = 1.1$, $g = 1.2$), and the quality of the individual expressing his choosiness is $q50$. $w_{\text{accept}} = q' - c(q, q')$, (i.e., monogamy), and $w_{\text{reject}} = 0.445$.

Modeling Scenarios and Measure of Choosiness

To illustrate the effect of pairing dynamics on the evolution of choosiness, we focus on a polygynandrous mating system, wherein both females and males can mate and potentially return to the mating pool after a latency period that differs between sexes ($[\tau_{\delta} = T/4, \tau_{\varphi} = T/2]$). Such a dynamic mating system is common in nature and is ideal to represent potential asymmetrical fluctuations in pairing dynamics: it is indeed expected that—because they generally have a shorter latency period—males will become newly available for mating more quickly than females and will therefore be in excess in the mating market. The R code and a Shiny application, provided as online enhancements (Chevalier et al. 2020), can be used to explore the predictions for other mating systems, such as monogamy (single mating for both males and females, $\tau_{\delta} = \tau_{\varphi} \gg T$) and polygyny (multiple mating for males only $[\tau_{\delta} = T/4, \tau_{\varphi} \gg T]$), and for different ASR. (R code is also provided in a zip file, available online.)¹ We also compare noncostly ($g = 0$) and costly ($g = 1.1$) courtship. The optimal choosiness strategy for an individual is a set of acceptance probabilities, with one probability for every partner quality q' and time of

1. Code that appears in *The American Naturalist* is provided as a convenience to readers. It has not necessarily been tested as part of peer review.

season t . For illustration purposes, we simplified this multidimensional metric as the inflection point of equation (2), that is, a threshold of quality (varying between $q = 0$ and $q = 100$) at which a partner will be accepted with a probability of 0.5 (fig. 1). We keep this simple metric of choosiness even when the function is unimodal (because of costly courtship). To keep things manageable, we will use discrete, representative categories of quality: high-quality (HQ, $q = 75$), intermediate-quality (IQ, $q = 50$), and low-quality (LQ, $q = 25$) individuals.

Results

Quality-Dependent and Flexible Optimal Choosiness

Here we first examine whether the optimal choosiness depends on the individual quality and whether this choosiness is flexible during the breeding season. In the polygynandrous mating system investigated here, with noncostly courtship, the optimal choosiness strategy is both quality dependent and flexible (fig. 2A, 2B). HQ individuals display high choosiness most of the time, but decrease it at the end of the breeding season. IQ and LQ individuals are more sensitive to the fluctuation of the mating market and decrease their choosiness when partner availability decreases. However, they take advantage of the decline in HQ individuals' choosiness to increase their choosiness near the end of the season (fig. 2A, 2B). In the present mating system, LQ males have very low choosiness, because they struggle to find females that accept them, which is demonstrated by their low probabilities to mate and to be courted and their high probability to be rejected (fig. 3). HQ individuals more easily find mates, as they are more courted and less rejected (fig. 3). It thus appears that the dynamics of mate availability generates quality dependence and flexibility of choosiness. It is consequently also influenced by the ASR and latency periods of males and females, which implies specific patterns depending on the mating system (supplement 2). For instance, in monogamy, the distribution of available partners' quality decreases continuously over time, generating a monotonous decline in choosiness and precise quality dependency (supplement 2). By contrast, in polygyny, individuals of all qualities adjust their choosiness to fluctuations in partner availability, both downward and upward, thereby displaying high flexibility (supplement 2). The Shiny application is available to visualize distribution of available partners (https://louise64.shinyapps.io/frequency_available_individuals/) and optimal choosiness (<https://louise64.shinyapps.io/choosiness/>) in different mating systems (Chevalier et al. 2020).

With costly courtship, optimal choosiness varies even more continuously with the chooser's quality (i.e., increased quality dependence). But the greater the cost of

OPTIMAL CHOOSINESS

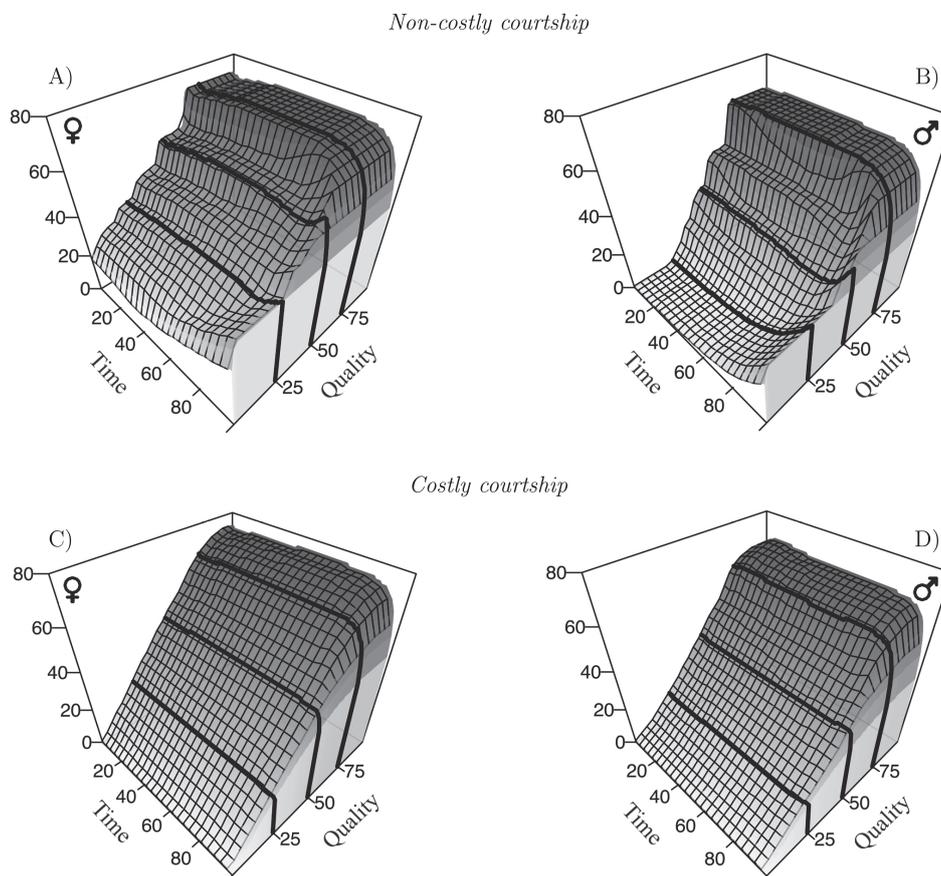


Figure 2: Optimal choosiness for females and males as functions of time and individual quality, with noncostly courtship (A, B) or with costly courtship (C, D). Optimal choosiness is measured as the partner quality that an individual is ready to accept with a probability of at least 0.5. Thick black lines indicate high-quality ($q = 75$), intermediate-quality ($q = 50$), and low-quality ($q = 25$) individuals.

courtship, the lower the flexibility of choosiness (fig. 2C, 2D for $g = 1.1$). The cost of courtship also substantially changes some behavioral aspects in the model: rejection probability even for LQ individuals (males and females) is now reduced, as they tend to select more accessible partners (fig. 2).

Changing initial distribution for individual quality $f(q)$ —either bell shaped ($\beta(2, 2)$), right skewed ($\beta(2, 3)$), or uniform ($\beta(1, 1)$)—does not have a strong qualitative effect on the evolution of choosiness, even though, as expected, optimal choosiness decreases when the distribution of quality is biased toward LQ individuals (fig. S8; figs. S1–S8 are available online). Moreover, changing the fitness gain (initially equal to the quality of the chosen

partner) to a multiplicative gain (equal to the product of both partners' quality) does not qualitatively affect the results (fig. S7). Finally, the length of the breeding season does not qualitatively modify the choosiness either: the temporal pattern in choosiness is simply adjusted to the length of the season (data are not shown).

Emerging Patterns of Interest

As a consequence of the pairing dynamics, several observable patterns arise from the model. As expected, the unequal latency period led to differences in choosiness between sexes. Female choosiness is globally higher than

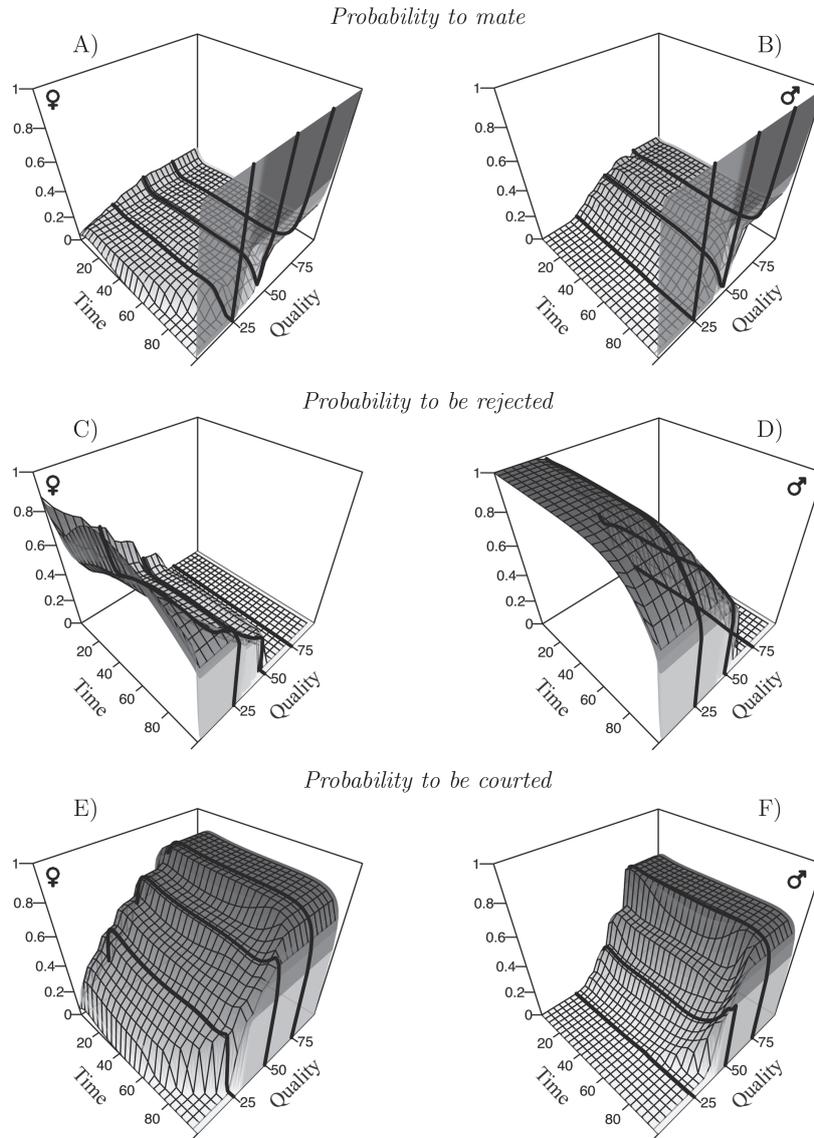


Figure 3: Probability to mate, be rejected, and be courted following an encounter, for females (A, C, E) and males (B, D, F). Courtship is cost free. The probability to mate is the chance an individual of quality q has to mate following an encounter (i.e., mutual choice). The probability to be rejected is the chance of being rejected by a partner that the individual would have accepted. The probability to be courted is the chance that an individual refuses a partner who would have agreed to mate. Thick black lines indicate high-quality ($q = 75$), intermediate-quality ($q = 50$), and low-quality ($q = 25$) individuals.

male choosiness (fig. 2A, 2B). Despite this imbalance, HQ males maintain a high level of choosiness during most of the breeding season (fig. 2B). That is because the high choosiness displayed in general by females reduces the ability of less attractive males to mate with preferred females, which partially relaxes scramble competition among HQ males. It is noteworthy that the OSR, which

is becoming increasingly biased toward an excess of males (supplement 3), poorly describes how the competition is distributed among quality classes. Maintenance of choosiness in the HQ individuals of the common sex is also observed for other mating systems (supplement 2). Even in heavily unbalanced scenarios (extreme ASR values and very short latency periods for males), HQ males remain choosy (except

when the latency period in males is equal to 1; data not shown; R code available to explore all combinations of parameters). This makes the choosiness difference between same-sex individuals higher in males than in females (as reflected by higher variance in choosiness; supplement 3). Another significant result is that—when courtship is cost free—for both males and females, optimal choosiness is not a monotonic function of quality but instead presents some regular thresholds. For instance, females belonging to some adjacent quality classes have the same optimal choosiness (e.g., between females of quality 60 and higher; fig. 2A), meaning that these individuals compete for the same quality of mates. On some other part of the quality range, however (e.g., the quality between 50 and 60; fig. 2A), the optimal choosiness declines steeply, indicating that these females do not really compete over males of the same quality. The population (males and females) can thus be divided into subgroups of individuals competing for the same mates, such that lower-quality individuals from each competing subgroup would have a harder time finding a mate. It operates as soon as both sexes have the opportunity to have more than one mating during the breeding season (<https://louise64.shinyapps.io/choosiness/>; Chevalier et al. 2020). Interestingly, this pattern of stepped choosiness occurs despite using a fine scale for quality variation (with 100 discrete classes of quality) and is less detectable if the population is divided into fewer discrete classes of quality (such

as 10 classes; data not shown). However, as shown previously, quality dependence of choosiness increases with costly courtship by segmenting competition between individuals of close quality and thus erases this stepped choosiness pattern.

Assortative Mating

From the pairing dynamics of the mating market, we can also predict the total frequency of mated couples between each pair of qualities during the breeding season (fig. 4). First, as expected in a polygynandrous mating system, sexual selection appears stronger in males (LQ males have negligible mating success). A general assortative mating pattern is observed in the present situation ($r = 0.76$). However, its distribution with regard to female and to male quality is not linear, and we observe quality-related subgroups within which assortative mating is weaker (fig. 4A). This pattern echoes the abovementioned effect of the pairing dynamics, which splits the population into subgroups of individuals that reproduce among themselves. Interestingly, this particular motif of a quality-related subgroup of individuals mating among themselves is characteristic of the polygynandrous mating system and is relaxed when the latency period increases (supplement 4). With costly courtship, because quality dependence of choosiness is increased, these subgroups disappear and assortative mating increases substantially ($r = 0.92$, fig. 4B). This

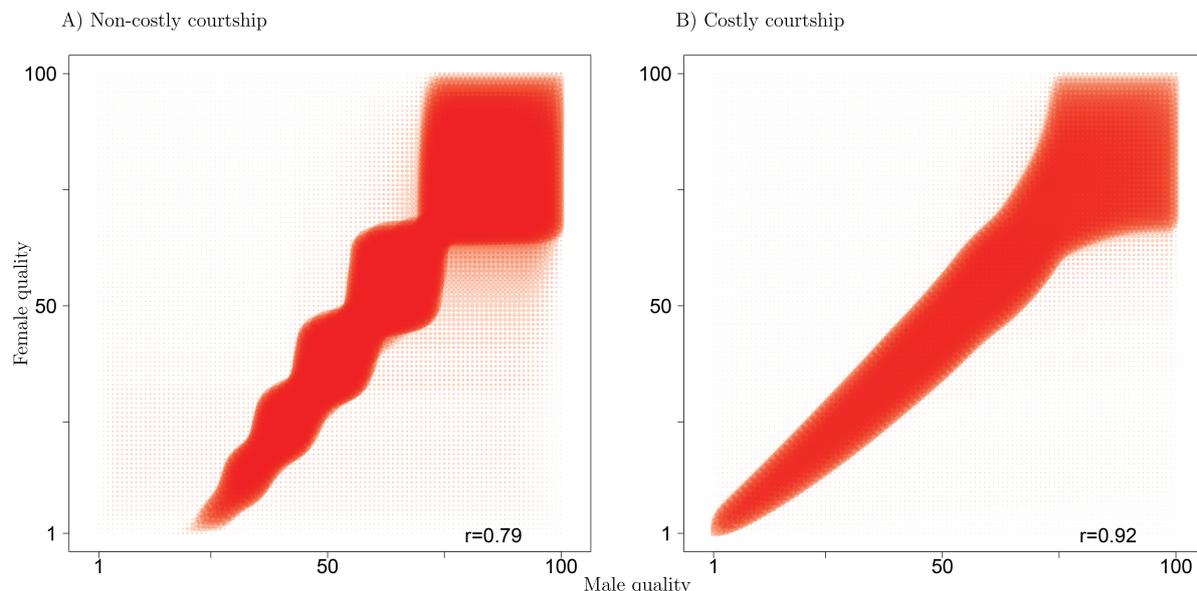


Figure 4: Final mating pattern with noncostly courtship (A) and costly courtship (B; $g = 1.1$). For a given quality of male and female, the size of the points is proportional to the frequency of pairs formed during the breeding season.

also favors the mating success of LQ males, which are now chosen by LQ females.

Discussion

To correctly represent the pairing dynamics within the mating market, and therefore the availability of potential partners of a given sex and quality at a given time, it is essential to allow (1) choosiness to evolve in both sexes (Parker 1983; McNamara and Collins 1990; Johnstone et al. 1996; Johnstone 1997; Kokko and Monaghan 2001; Lande et al. 2001; Kokko and Johnstone 2002; Alpern and Reyniers 2005; Servedio and Lande 2006; Fawcett and Bleay 2009; Ramsey 2011; Courtiol et al. 2016), (2) realistic variation in quality within each sex (McNamara and Collins 1990; Johnstone et al. 1996; Alpern and Reyniers 2005; Ramsey 2011), and (3) potential flexibility in choosiness (Johnstone 1997; Fawcett and Johnstone 2003; Alpern and Reyniers 2005; Fawcett and Bleay 2009; Bleu et al. 2011; Ramsey 2011). Previous studies that have encompassed the three points only addressed very particular cases (i.e., monogamous balanced mating systems; Johnstone 1997; Alpern and Reyniers 2005; Ramsey 2008). By extending the integration of these three mechanisms to a wide range of mating systems, spanning a range of ASR and latency periods, we demonstrated that choosiness is likely to evolve in both sexes in most mating systems, but also to be flexible, depending on the cost of courtship. In a nutshell, our approach aims to capture the fine dynamics of the mating market, whereas previous models confined choosiness to only one sex or limited the quality variation or the extent of plasticity in choosiness.

Quality-Dependent and Flexible Optimal Choosiness

Quality dependence and flexibility in choosiness were predicted for monogamous mating systems (McNamara and Collins 1990; Johnstone 1997; Fawcett and Johnstone 2003; Alpern and Reyniers 2005; Ramsey 2011). By contrast, our model also addresses the role of ecological or physiological constraints (such as mating latency period) on the interplay between mate choice and the dynamics of the mate quality distributions, and investigates sex differences in choosiness. By allowing individuals in each quality class to fine-tune their choosiness to the variation of scramble competition experienced throughout the breeding season, we also reveal some counterintuitive strategies. Specifically, we predict an increase in choosiness for LQ and IQ individuals over the breeding season. To our knowledge, this trend has only been clearly predicted when sampling incurs a direct searching cost (Johnstone 1997)

or when individuals need to defend their mates from rivals (Fawcett and Johnstone 2003). Our model implies that when choosiness is allowed to evolve in both sexes, such an increase could in fact arise from the pairing dynamics alone without these additional costs. However, the addition of a courtship cost, which is constant throughout the breeding season and does not emerge from individual interactions (contrary to Fawcett and Johnstone 2003), will limit this flexibility. Our results also indicate that as soon as both sexes have the opportunity to mate more than once, choosiness varies noncontinuously with quality, as individuals belonging to some adjacent quality classes have the same optimal choosiness. Here, the addition of a courtship cost increases the quality dependence of choosiness.

Perspectives

The fluctuating dynamics of the mating market and the associated opportunity costs are powerful drivers of the choosiness evolution in our model. Future efforts should be dedicated to bridging the gap between these fluctuations that operate on a short timescale, as we envisioned, and longer timescale models that optimize latency periods, for instance, so as to balance the contribution of both short- and long-term approaches in choosiness evolution.

Model Assumptions

As in any modeling exercise, a set of assumptions underpins our analysis and findings. First, individuals are assumed to know the quality distribution of the opposite sex throughout the breeding season (within the margin of the assessment error we simulate). This depends on the presence of accurate cues and honest signals, with any sensory costs sufficiently modest to not alter the decision-making process (Shugan 1980; Wang et al. 2017). In poeciliid fish, for instance, females experiencing more variable male courtship display enhanced brain function (Wang et al. 2014; Cummings 2015), even if they may do so at a significant energetic cost. Likewise, the model calculates fitness horizons on the basis of the quality of the focal individual: this requires that each individual knows its own quality (a rather unlikely scenario; Fawcett and Bleay 2009) or that selection has generated genetic covariation in which HQ individuals are choosy and LQ individuals are less so (Servedio and Lande 2006). For a monogamous and balanced mating system, Fawcett and Bleay (2009) have explored how choosiness could evolve, on the basis of learning one's attractiveness through acceptance/rejection trials. They show that, in such conditions, variance between quality classes is increased during the first few time steps. However, when the mating market is more dynamic and less

predictable, as in most mating systems, a learning process must lag behind the fluctuations of the market, making choosiness always suboptimal (as compared with a system where individuals know their own quality). Under costly courtship, strategies become deliberately assortative, a result consistent with Fawcett and Johnstone (2003) and Härdling and Kokko (2005), where HQ individuals are better able to defend the most attractive partners. Yet costly courtship—as modeled here—does not promote flexibility. Flexibility might be favored if the cost of courtship would emerge from the interaction with rival and vary throughout the breeding season (as in Fawcett and Johnstone 2003). Still, our approach demonstrates that variance in choosiness and consequent assortative mating can be generated by the pairing dynamics alone, without needing to invoke cost constraints and interference effects. Finally, by focusing on the short-term dynamics of mate availability, we assumed constant values for variables characterizing the different mating systems, such as latency period and ASR. Other models, focusing instead on life histories and therefore on longer timescales, assume that latency period can be optimized too by considering the trade-off between mating opportunities and parental care (Kokko and Johnstone 2002; Kokko and Jennions 2003, 2008). This trade-off can affect the mating market dynamics and thus the evolution of choosiness (Kokko and Jennions 2008; Fromhage and Jennions 2016).

Emerging Patterns

Evolving toward quality-dependent and flexible choosiness strategies produces distinct patterns of interest for the study of mating systems. First, choosiness should evolve frequently in both sexes, in a wide span of mating systems. Previously, many models have investigated how differences in choosiness between sex could evolve (Owens and Thompson 1994; Kokko and Monaghan 2001; Kokko and Johnstone 2002; Courtiol et al. 2016; Fromhage and Jennions 2016). In most of these results, the latency period, sex ratio at maturation, and sex-dependent breeding costs explain differences in choosiness between sexes, with the general expectation that the most common sex will be less choosy, if choosy at all. For instance, Dechaume-Moncharmont et al. (2016) showed that scramble competition suppresses choosiness in the most common sex, but they did not include the influence of other-sex choosiness on the evolution of the focal-sex choosiness. Alternatively, Courtiol et al. (2016) and Kokko and Johnstone (2002) predicted that mutual choice would mainly evolve in situations where mating latencies are very long-lasting in both sexes—thus incurring an increasing mortality probability (i.e., monogamy)—but they assumed nonflexible

choosiness. By relaxing the abovementioned assumptions, we showed that the dynamics of the mating market along the breeding season can change these expectations. As a consequence of the interplay between the choosiness of the two sexes, and of quality-dependent flexible strategies, we observed the evolution of choosiness in both sexes for a wide range of parameter spaces. Despite the fact that empirical studies addressing choosiness in both sexes remain uncommon, male choosiness has been reported even when males are the most common sex (i.e., male-biased OSR; WearingWilde 1996; Bel-Venner et al. 2008; Venner et al. 2010). Under the assumptions of the present model, optimal choosiness differs among individuals of different qualities, generating ample variance in choosiness in both sexes.

Second, whereas models investigating quality dependence in choosiness consider either only one sex (Fawcett and Johnstone 2003; Härdling and Kokko 2005; Bleu et al. 2011) or monogamous balanced mating systems (Johnstone 1997; Alpern and Reyniers 2005), our result implies that opportunity costs resulting from pairing dynamics can be a strong driver maintaining interindividual variance in choosiness within populations (which might affect the direction and strength of sexual selection (Jennions and Petrie 1997; Murphy and Gerhardt 2000)). We emphasize that interindividual variance in choosiness is not predicted to be equal between sexes (with the trivial exception of the scenario of monogamy with balanced ASR). Female variance in choosiness is indeed found to be smaller than the male variance in choosiness. Variance in choosiness has been documented in either females or males, with a possible underlying relationship to the individual quality or competitive ability (Widemo and Sæther 1999; Amundsen and Forsgren 2001; Bonduriansky 2001; Brooks and Endler 2001; Brooks 2002; Ritchie et al. 2005; Cotton et al. 2006; Bel-Venner et al. 2008; Labonne et al. 2009; Ratterman et al. 2014).

Third, there are some general relationships between mating systems and the level of quality dependence and flexibility in choosiness. In the present approach, where we focus on the intragenerational timescale, the dynamics of the mating market are highly influenced by the type of mating systems, which we here manipulated via ASR and latency period. The general picture vividly contrasts the monogamous and polygynandrous systems. In the former, remaining individuals of higher quality constantly adjust their choosiness downward to the dwindling opportunities, while LQ individuals are never choosy. The situation is far more complex in the polygynandrous and polygynous system, where flexibility is observed for individuals of all qualities, adjusting their choosiness both downward and upward, thereby sustaining variance in choosiness later in the breeding season.

Fourth, it is remarkable that in the various mating systems envisioned here, positive assortative mating always occurs (supplement 4). Its intensity (measured by the coefficient of correlation, r) decreases with the level of unbalance in ASR and in latency periods. In unbalanced mating systems (such as polygynandry), LQ males have almost no mating success, and thus sexual selection against them is strong; but in contrast, sexual selection is relaxed among HQ individuals. Indeed, we observe a particular pattern of apparent assortment wherein the population is divided into random-mating subsets (which result from the evolution of stepped choosiness). In such case, r is not a precise indicator of assortative mating. This result is reminiscent of the results obtained by previous analytical models, which found that couples were formed between males and females belonging to the same band (interval) of quality levels in the population (McNamara and Collins 1990; Alpern and Reyniers 2005) under monogamy. Johnstone et al. (1996) also predicted similar apparent assortative mating from a nonflexible choosiness strategy throughout the breeding season. On the contrary, our predictions indicate that such clear patterns can be related to quality-dependent flexible choosiness, wherein the population is self-organized into subsets of individuals competing for the same range of partner quality. This population partition is more coarse for unbalanced mating systems, such as polygynandry, where there are a few large groups of competitors, since individuals of relatively distant quality compete (e.g., from q_{60} to q_{100} ; supplement 4). In monogamy, by contrast, the groups are much smaller and more numerous, as only individuals of very close quality are competing together (supplement 4). The fact that costly courtship might counteract this partitioning of the population is of interest for empirical observations: the shape of assortative mating plots might in fact directly hint at the existence and the extent of courtship cost in the studied population.

Acknowledgments

We thank Alexandre Courtiol, Philip H. Crowley, and the reviewers for their valuable comments on the manuscript.

Statement of Authorship

L.C. and F.X. developed the question and research approach. L.C. developed the model structure and wrote the scripts (in R) to run simulations, conduct analyses, and visualize model output. L.C. wrote the manuscript with input from all coauthors. J.L. and M.G. contributed to defining the scope of the study, interpreting the results, and writing the manuscript. All coauthors edited and provided significant comments on the manuscript.

Data and Code Availability

Shiny app: https://louise64.shinyapps.io/frequency_available_individuals/, <https://louise64.shinyapps.io/choosiness/>. GitLab: https://gitlab.com/louiseche/mate_choice.git. These are also backed up at <https://doi.org/10.15454/JHDCQM>. Portail Data INRAE: <https://data.inrae.fr/dataset.xhtml?persistentId=doi:10.15454/JHDCQM>.

Literature Cited

- Alpern, S., and D. Reyniers. 2005. Strategic mating with common preferences. *Journal of Theoretical Biology* 237:337–354.
- Amundsen, T., and E. Forsgren. 2001. Male mate choice selects for female coloration in a fish. *Proceedings of the National Academy of Sciences of the USA* 98:13155–13160.
- Andersson, M. B. 1994. *Sexual selection*. Monographs in Behavior and Ecology. Princeton University Press, Princeton, NJ.
- Bailey, N. W., and M. Zuk. 2009. Field crickets change mating preferences using remembered social information. *Biology Letters* 5:449–451.
- Bel-Venner, M., S. Dray, D. Allaine, F. Menu, and S. Venner. 2008. Unexpected male choosiness for mates in a spider. *Proceedings of the Royal Society B* 275:77–82.
- Bleu, J., C. Bessa-Gomes, and D. Laloï. 2011. Evolution of female choosiness and mating frequency: effects of mating cost, density and sex ratio. *Animal Behaviour* 83:131–136.
- Bonduriansky, R. 2001. The evolution of male mate choice in insects: a synthesis of ideas and evidence. *Biological Reviews* 76:305–339.
- Brooks, R. 2002. Variation in female mate choice within guppy populations: population divergence, multiple ornaments and the maintenance of polymorphism. *Genetica* 116:343–358.
- Brooks, R., and J. A. Endler. 2001. Female guppies agree to differ: phenotypic and genetic variation in mate-choice behavior and the consequences for sexual selection. *Evolution* 55:1644–1655.
- Byrne, P. G., and W. R. Rice. 2006. Evidence for adaptive male mate choice in the fruit fly *Drosophila melanogaster*. *Proceedings of the Royal Society B* 273:917–922.
- Chaine, A. S., and B. E. Lyon. 2008. Adaptive plasticity in female mate choice dampens sexual selection on male ornaments in the lark bunting. *Science* 319:459–462.
- Chevalier, L., F. X. Dechaume-Moncharmont, M. Galipaud, and J. Labonne. 2020. Data from fluctuating dynamics of mate availability promote the evolution of flexible choosiness in both sexes. Portail Data INRAE, vol. 1. <https://doi.org/10.15454/JHDCQM>.
- Collins, E. J., and J. M. McNamara. 1993. The job-search problem with competition: an evolutionarily stable dynamic strategy. *Advances in Applied Probability* 25:314–333.
- Cotton, S., J. Small, and A. Pomiankowski. 2006. Sexual selection and condition-dependent mate preferences. *Current Biology* 16: 755–765.
- Courtio, A., L. Etienne, R. Feron, B. Godelle, and F. Rousset. 2016. The evolution of mutual mate choice under direct benefits. *American Naturalist* 188:521–538.
- Crowley, P. H., S. E. Travers, M. C. Linton, S. L. Cohn, A. Sih, and R. C. Sargent. 1991. Mate density, predation risk, and the seasonal sequence of mate choices: a dynamic game. *American Naturalist* 137:567–596.

- Cummings, M. E. 2015. The mate choice mind: studying mate preference, aversion and social cognition in the female poeciliid brain. *Animal Behaviour* 103:249–258.
- Darwin, C. 1871. *The descent of man and selection in relation to sex*. J. Murray, London.
- Dechaume-Moncharmont, F. X., T. Brom, and F. Cézilly. 2016. Opportunity costs resulting from scramble competition within the choosy sex severely impair mate choosiness. *Animal Behaviour* 114:249–260.
- Etienne, L., F. Rousset, B. Godelle, and A. Courtiol. 2014. How choosy should I be? the relative searching time predicts evolution of choosiness under direct sexual selection. *Proceedings of the Royal Society B* 281:20140190.
- Fawcett, T. W., and C. Bleay. 2009. Previous experiences shape adaptive mate preferences. *Behavioral Ecology* 20:68–78.
- Fawcett, T. W., and R. A. Johnstone. 2003. Mate choice in the face of costly competition. *Behavioral Ecology* 14:771–779.
- Fawcett, T. W., J. M. McNamara, and A. I. Houston. 2011. When is it adaptive to be patient? a general framework for evaluating delayed rewards. *Behavioural Processes* 89:128–136.
- Fricke, C., J. Perry, T. Chapman, and L. Rowe. 2009. The conditional economics of sexual conflict. *Biology Letters* 5:671–674.
- Fromhage, L., and M. D. Jennions. 2016. Coevolution of parental investment and sexually selected traits drives sex-role divergence. *Nature Communications* 7:1–11.
- Gauthey, Z., A. Hendry, A. Eloşegi, C. Tentelier, and J. Labonne. 2016. The context dependence of assortative mating: a demonstration with conspecific salmonid populations. *Journal of Evolutionary Biology* 29:1827–1835.
- Gimelfarb, A. 1988. Processes of pair formation leading to assortative mating in biological populations: encounter-mating model. *American Naturalist* 131:865–884.
- Gowaty, P. A., and S. P. Hubbell. 2009. Reproductive decisions under ecological constraints: it's about time. *Proceedings of the National Academy of Sciences of the USA* 106 (suppl.):10017–10024.
- Hårdling, R., and H. Kokko. 2005. The evolution of prudent choice. *Evolutionary Ecology Research* 7:697–715.
- Houston, A., and J. McNamara. 1999. *Models of adaptive behaviour: an approach based on state*. Cambridge University Press, Cambridge.
- Jennions, M. D., and M. Petrie. 1997. Variation in mate choice and mating preferences: a review of causes and consequences. *Biological reviews of the Cambridge Philosophical Society* 72:283–327.
- Johnstone, R. A. 1997. The tactics of mutual mate choice and competitive search. *Behavioral Ecology and Sociobiology* 40:51–59.
- Johnstone, R. A., J. D. Reynolds, and J. C. Deutsch. 1996. Mutual mate choice and sex differences in choosiness. *Evolution* 50:1382–1391.
- Judge, K., J. Ting, and D. Gwynne. 2014. Condition dependence of female choosiness in a field cricket. *Journal of Evolutionary Biology* 27:2529–2540.
- Kokko, H., and M. D. Jennions. 2008. Parental investment, sexual selection and sex ratios. *Journal of Evolutionary Biology* 21:919–948.
- . 2003. It takes two to tango. *Trends in Ecology and Evolution* 18:103–104.
- Kokko, H., and R. A. Johnstone. 2002. Why is mutual mate choice not the norm? operational sex ratios, sex roles and the evolution of sexually dimorphic and monomorphic signalling. *Philosophical Transactions of the Royal Society B* 357:319–330.
- Kokko, H., and P. Monaghan. 2001. Predicting the direction of sexual selection. *Ecology Letters* 4:159–165.
- Kopp, M., and J. Hermisson. 2008. Competitive speciation and costs of choosiness. *Journal of Evolutionary Biology* 21:1005–1023.
- Labonne, J., M. Augery, M. Parade, S. Brinkert, E. Prevost, M. Héland, and E. Beall. 2009. Female preference for male body size in brown trout, *Salmo trutta*: is big still fashionable? *Animal Behaviour* 77:129–137.
- Lande, R., O. Seehausen, and J. J. Alphen. 2001. Mechanisms of rapid sympatric speciation by sex reversal and sexual selection in cichlid fish. *Genetica* 112–113:435–443.
- McNamara, J. M., and E. J. Collins. 1990. The job search problem as an employer candidate game. *Journal of Applied Probability* 27:815–827.
- McNamara, J. M., J. N. Webb, E. J. Collins, T. Székely, and A. I. Houston. 1997. A general technique for computing evolutionarily stable strategies based on errors in decision-making. *Journal of Theoretical Biology* 189:211–225.
- Murphy, C. G., and H. C. Gerhardt. 2000. Mating preference functions of individual female barking treefrogs, *Hyla gratiosa*, for two properties of male advertisement calls. *Evolution* 54:660–669.
- Owens, I. P. F., and D. B. A. Thompson. 1994. Sex differences, sex ratios and sex roles. *Proceedings of the Royal Society B* 258:93–99.
- Parker, G. 1983. Mate quality and mating decisions. *Mate Choice* 141:166.
- Ramsey, D. M. 2008. A large population job search game with discrete time. *European Journal of Operational Research* 188:586–602.
- . 2011. Mutual mate choice with multiple criteria. Pages 337–355 in *Advances in dynamic games: theory, applications, and numerical methods for differential and stochastic games*. Springer, New York.
- Ratterman, N. L., G. G. Rosenthal, G. E. Carney, and A. G. Jones. 2014. Genetic variation and covariation in male attractiveness and female mating preferences in *Drosophila melanogaster*. *G3: Genes, Genomes, Genetics* 4:79–88.
- Real, L. 1990. Search theory and mate choice. I. models of single-sex discrimination. *American Naturalist* 136:376–405.
- Ritchie, M. G., M. Saarikettu, and A. Hoikkala. 2005. Variation, but no covariance, in female preference functions and male song in a natural population of *Drosophila montana*. *Animal Behaviour* 70:849–854.
- Roff, D. A. 2010. *Modeling evolution: an introduction to numerical methods*. Oxford University Press, Oxford.
- Rowe, L., and G. Arnqvist. 2015. Two faces of environmental effects on mate choice: a comment on Dougherty and Shuker. *Behavioral Ecology* 26:324–324.
- Servedio, M. R., and R. Lande. 2006. Population genetic models of male and mutual mate choice. *Evolution* 60:674–685.
- Shugan, S. M. 1980. The cost of thinking. *Journal of Consumer Research* 7:99–111.
- Tinghitella, R. M., E. G. Weigel, M. Head, and J. W. Boughman. 2013. Flexible mate choice when mates are rare and time is short. *Ecology and Evolution* 3:2820–2831.
- Venner, S., C. Bernstein, S. Dray, and M.-C. Bel-Venner. 2010. Make love not war: when should less competitive males choose low-quality but defensible females? *American Naturalist* 175:650–661.
- Wang, D., W. Forstmeier, and B. Kempenaers. 2017. No mutual mate choice for quality in zebra finches: time to question a widely held assumption. *Evolution* 71:2661–2676.

Appendix from L. Chevalier et al., “Fluctuating Dynamics of Mate Availability Promote the Evolution of Flexible Choosiness in Both Sexes” (Am. Nat., vol. 196, no. 6, p. 000)

Additional Methods

Method to Calculate Quality Distribution Change throughout the Breeding Season

During the iterative calculation of the evolutionarily stable strategy, every time the best strategies are changed, one needs to update the new distributions of qualities across the breeding season $f(q, t)_\delta$, and $f(q, t)_\varphi$. We need to introduce a second notation, $m_\delta(q, t)$, as the proportion of males of quality q available among the males of this quality at time step t , and $m_\varphi(q, t)$ as the proportion of females of quality q available among the females of this quality at time step t . Initially, all individuals are unmated, so that $m_\delta(q, 1) = m_\varphi(q, 1) = 1$ and $f_\delta(q, 1) = f_\varphi(q, 1) = f(q)/\sum_q f(q)$. We express the relative frequency of males and females $f(q, t)_\delta$, and $f(q, t)_\varphi$ as a function of $m(q, t)_\delta$, and $m(q, t)_\varphi$:

$$\begin{aligned} f_\delta(q, t) &= \frac{m_\delta(q, t)f_\delta(q, 1)}{\sum_q m_\delta(q, t)f_\delta(q, 1)}, \\ f_\varphi(q, t) &= \frac{m_\varphi(q, t)f_\varphi(q, 1)}{\sum_q m_\varphi(q, t)f_\varphi(q, 1)}. \end{aligned} \tag{A1}$$

In equation (A1), $m_\delta(q, t)f_\delta(q, 1)$ gives the proportion of males of quality q available at time step t among all the males, and $\sum_q m_\delta(q, t)f_\delta(q, 1)$ gives the proportion of male (all qualities combined) available among all the males. The proportion of individuals available of each quality q is recursively calculated as follows:

$$\begin{aligned} m_\delta(q, t+1) &= m_\delta(q, t) \left[1 - P_{E_\delta}(t) \sum_{q'} f_\varphi(q', 1) p_{\text{new}\delta}(q, q', t) p_{\text{new}\varphi}(q', q, t) \right] \\ &\quad + [1 - m_\delta(q, t)] \times \frac{1}{\tau_\delta}, \\ m_\varphi(q, t+1) &= m_\varphi(q, t) \left[1 - P_{E_\varphi}(t) \sum_{q'} f_\delta(q', 1) p_{\text{new}\varphi}(q, q', t) p_{\text{new}\delta}(q', q, t) \right] \\ &\quad + [1 - m_\varphi(q, t)] \times \frac{1}{\tau_\varphi}. \end{aligned} \tag{A2}$$

In equation (A2), the proportion of individuals of quality q available at $t+1$, $m_\delta(q, t+1)$, is computed from the probability of individuals to encounter a partner ($P_{E_\delta}(t)$ for males and $P_{E_\varphi}(t)$ for females) and to mate with an encountered partner, plus the probability that individuals in refractory period becomes available.

Table A1: Symbol table

Symbol	Definition	Default value
$f_{\varnothing}(q, q', t)$	Frequency of males of quality q among all males available (all quality combined) at time t	Variable
$m_{\varnothing}(q, t)$	Proportion of males of quality q available among all males of quality q (i.e., available males plus mated males)	Variable
$P_{\text{resident}\varnothing}(q, q', t)$	Choosiness strategy for males (respectively females) of quality q , which is a matrix of acceptances probabilities for every possible partner's quality q' and at each time step	Variable
$P_{\text{best}\varnothing}(q, q', t)$	"Best response" choosiness strategy for males that yields the maximum payoff in a population of competitors and mates using "resident" strategy	Variable
$P_{\text{new}\varnothing}(q, q', t)$	New resident strategy calculated with the resident choosiness strategy of the previous iteration and the best response choosiness for this resident strategy	Variable
λ	Damping term in the iterative calculation of the optimal strategy	.1
$P_{E,\varnothing}(t)$	Probability a male encounter a partner at time t	Variable
$w_{\text{accept}\varnothing}(q, q', t)$	Expected payoff for a male of quality q if he mates with a partner of quality q' at time t	Variable
$w_{\text{reject}\varnothing}(q, q', t)$	Expected payoff for a male of quality q if he rejects the mating with a partner of quality q' at time t	Variable
$w_{\text{reject}\varnothing}(q, t)$	Expected fitness for a male of quality q at time t	Variable
$c(q, q')$	Cost of courtship	Variable
g	Parameter tuning the intensity of courtship cost	1.1
OSR	Operational sex ratio (ratio of available males over available females at time step t)	Variable
ASR	Adult sex ratio (ratio of available males over available females at the beginning of the breeding season)	1
T	Length of the breeding season	100
τ_{\varnothing}	Mean time out for males	$\frac{1}{4}T$ or T
τ_{\varnothing}	Mean time out for females	$\frac{1}{2}T$ or T
Q	Number of quality classes	100
k	Degree of error when making choice	.01

**Article 2: A demogenetic agent
based model for the evolution of
traits and genome architecture
under sexual selection**

RESEARCH ARTICLE

 Open Access

 Open Peer-Review

A demogenetic individual based model for the evolution of traits and genome architecture under sexual selection

Cite as: Chevalier L et al. (2020). A demogenetic individual based model for the evolution of traits and genome architecture under sexual selection. *bioRxiv* 2020.04.01.014514, ver. 3 peer-reviewed and recommended by *PCIEvolBiol*. <https://doi.org/10.1101/2020.0401014514>. DOI: 10.24072/pci.evolbiol.100112

Posted: 13th November 2020

Recommender:
Michael Greenfield

Reviewers:
Frédéric Guillaume and Gil Rosenthal

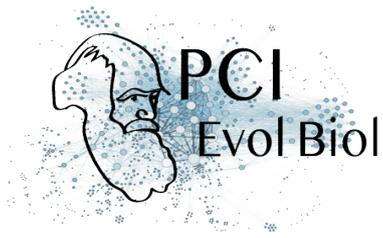
Correspondence:
jacques.labonne@inrae.fr

Louise Chevalier¹, François De Coligny¹, & Jacques Labonne²

¹ Université de Pau et des Pays de l'Adour, E2S UPPA, INRAE, ECOBIOP, Saint-Pée-sur-Nivelle, France

² AMAP, Univ Montpellier, CIRAD, CNRS, INRAE, IRD, 34000 Montpellier, France

This article has been peer-reviewed and recommended by
Peer Community In Evolutionary Biology



Abstract

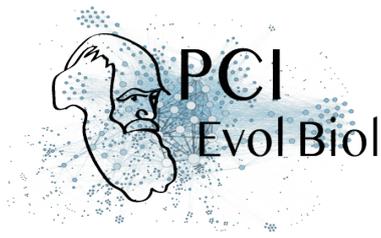
Sexual selection has long been known to favor the evolution of mating behaviors such as mate preference and competitiveness, and to affect their genetic architecture, for instance by favoring genetic correlation between some traits. Reciprocally, genetic architecture can affect the expression and the evolvability of traits and mating behaviors. But sexual selection is highly context-dependent, making interactions between individuals a central process in evolution, governing the transmission of genotypes to the next generation. This loop between the genetic structure conditioning the expression and evolution of traits and behaviour, and the feedback of this phenotypic evolution on the architecture of the genome in the dynamic context of sexual selection, has yet to be thoroughly investigated. We argue that demogenetic agent-based models (DG-ABM) are especially suited to tackle such a challenge because they allow explicit modelling of both the genetic architecture of traits and the behavioural interactions in a dynamic population context. We here present a DG-ABM able to simultaneously track individual variation in traits (such as gametic investment, preference, competitiveness), fitness and genetic architecture throughout evolution. Using two simulation experiments, we compare various mating systems and show that behavioral interactions during mating triggered some complex feedback in our model, between fitness, population demography, and genetic architecture, placing interactions between individuals at the core of evolution through sexual selection. DG-ABMs can, therefore, relate to theoretical patterns expected at the population level from simpler analytical models in evolutionary biology, and at the same time provide a more comprehensive framework regarding individual trait and behaviour variation, that is usually envisioned separately from genome architecture in behavioural ecology.

Keywords: sexual selection, individual-based model, mating preference, genetic architecture, modularity.

Introduction

Sexual selection has long been recognized as an evolutionary force shaping mating behaviours and morphological traits in populations (Darwin, 1872; Fisher, 1915; Jones and Ratterman, 2009), and has been invoked as a driving force behind speciation (Lande, 1981; Ritchie, 2007). Evolutionary biology, therefore, predicts change in the genotypic and phenotypic composition of a population due to sexual selection (Lande, 1981; Lande and Arnold, 1985; Pomiankowski and Iwasa, 1998; Tazzyman and Iwasa, 2010). Such predictions, based on population genetics, quantitative genetics, or adaptive dynamics rely on simplifying assumptions concerning mating processes and do not explicitly represent the pairing dynamics during mating.

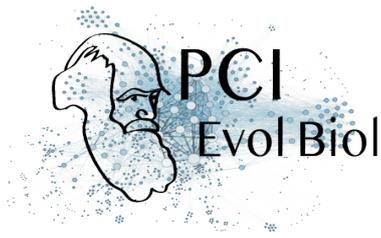
And yet, sexual selection is fundamentally the result of complex processes involving be-



havioural interactions between individuals, with tremendous impacts on evolution (Bailey and Moore, 2012; Kokko, Booksmythe, et al., 2015; Moore et al., 1997; Muniz and Machado, 2018; Wolf et al., 1999). This social part of the environment is indeed one of the most dynamic sources of variation an organism might experience during its lifetime (Kent et al., 2008; Krupp et al., 2008; West-Eberhard, 1983). As a response, behavioral ecology particularly aims at understanding the effect of this variable social environment (e.g., the availability of partners of various qualities) and frequency-dependent strategies (i.e. the outcome of a tactic depends on the tactics of others) on the evolution of mating behaviours, usually through the use of game-theory approach (Ramsey, 2011; Smith, 1976). Alternatively, if models in evolutionary biology pay little attention to the interactions between individuals, they highlight the role of genetic architecture in determining the trajectory of evolutionary change and show that genetic variance and covariance have a potentially strong impact on evolution (Hansen, 2003; Iwasa and Pomiankowski, 1995; Iwasa, Pomiankowski, and Nee, 1991; Lande, 1976, 1981; Matessi and Di Pasquale, 1996; Walsh and Blows, 2009). Yet, mathematical resolution often requires a simplified representation of genetic architecture. For instance, in quantitative genetics, the genetic architecture of traits is described in terms of genetic variance and covariance which are supposed constant (Falconer et al., 1996, e.g. Connallon, 2015; Iwasa and Pomiankowski, 1995; Iwasa, Pomiankowski, and Nee, 1991; Lande, 1976, 1981). A set of simplifying assumptions justify this approximation (additivity, linkage equilibrium, infinite population size, multivariate Gaussian distribution of allelic effects, evolutionary equilibrium). However, in a more realistic view of the world, finite population are subject to dynamic fluctuations in the genetic variance and covariance, as a result of genetic drift and variable selection (Jones, Arnold, et al., 2003, 2007; Roff and Mousseau, 1999; Shaw et al., 1995; Steppan et al., 2002). In particular, frequency-dependent selection can increase genetic variance by favoring rare variants that differ from the most common (Sasaki and Dieckmann, 2011), and correlational selection can increase genetic covariance by favoring genetic correlation between traits (Lande, 1980; Matuszewski et al., 2014). Importantly, such changes in genetic characteristics certainly result in changes in genetic architecture and may in turn impact evolution (Debarre and Otto, 2016; Jones, Bürger, et al., 2014; Wakano and Iwasa, 2013; Wakano and Lehmann, 2014).

To better characterize the role of sexual selection in shaping mating behaviours, genetic architecture, and demography of the population, we, therefore, need to address the following questions: How do social interactions during reproduction affect the architecture of the genome? How does the evolution of genetic architecture, in turn, impact the evolution of traits and consequently affect demographic characteristics of the population? And how does the physical structure of the genome (number of genes involved, mutation rate, linkage disequilibrium) constrain this evolution?

The current challenge is thus to explicitly take into account the context-dependence effect of sexual selection (generated by mating dynamics) on the transmission of genotypes to the next generation and on the evolution of the genetic architecture.



Agent-based models (DeAngelis and Mooij, 2005) are an interesting approach to tackle this challenge, as soon as they integrate genetic transmission (Labonne and Hendry, 2010; Oddou-Muratorio and Davi, 2014; Piou and Prévost, 2012; Romero-Mujalli et al., 2019). Eco-evolutionary models using quantitative genetics showed the importance of inter individual interactions and environment on trait evolution, focusing generally on an applied or isolated question (Aguilée et al., 2013; Holt and Barfield, 2011; Labonne and Hendry, 2010; Labonne, Ravigné, et al., 2008; Oddou-Muratorio and Davi, 2014). Some genetically-explicit (i.e., allelic models) ABMs have been developed to investigate demography and evolutionary change via selection pressures from the ecological settings, but without explicitly modelling interactions between individuals, notably during reproduction (Guillaume and Rougemont, 2006; Neuenchwander et al., 2008; Peng and Kimmel, 2005). So the selective pressure on traits is *a priori* defined and therefore does not emerge from inter-individual interactions, and from trade-off between traits. By contrast, if traits values directly influence mating behaviors and survival of individuals, the selection pressure changes with the distribution of the traits values in the population; organisms thus modify their social environment and are able to respond dynamically to this change. Additionally, to our knowledge, none of these models particularly emphasizes the general role of sexual selection as a driver of traits and genetic architecture, whereas it is known to be central in evolution.

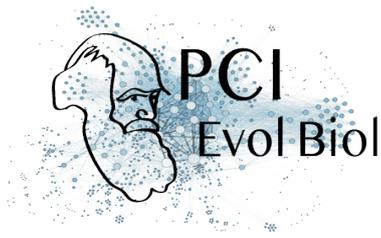
Methods

We here give an extensive description of the model in the spirit of the ODD protocol (Grimm et al., 2006).

An overview of the demogenetic individual based model

Purpose: The purpose of the present model is to investigate the co-evolution between reproductive traits under sexual selection and their genetic architecture, taking into account mating behaviour, genetic and demographic characteristics of the population. We use hereafter the term "demo-genetic agent-based model" (DG-ABM) to indicate that the present model integrates the full retroactive loops between these elements.

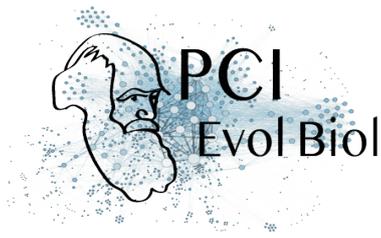
State variables and scales: This agent-based model uses a discrete temporal scale and is not spatially explicit. The time horizon of the model is in the order of tenths to a few thousand time steps; the time step can be interpreted as the minimum generation time. Three levels are considered: the gene level, the individual level, and the population level. At the gene level, genes are characterized by their alleles, which code for various genetic values depending on the number of traits simulated. They are also characterized by their position in the genome and their recombination probabilities with other genes. Such a position is invariant during



the simulation because we do not wish to simulate the physical evolution of the genome. Individuals are characterized by the following state variables: identity number, sex, age, lifetime reproductive success and the genetic values of up to three traits: the gametic investment G , the preference P and the competitiveness C . The population level is characterized by the following state variables: population size, allelic diversity, mean allelic values and standard deviations for the various traits on each gene, mean and standard deviation of traits values and mean lifetime reproductive success. Note that the population level can be subdivided during reproduction, to create smaller mating groups and mimic spatial or temporal isolation during the breeding season.

Process overview and scheduling: The simulation process is described below and can be seen in Appendix A1. The model proceeds in generational time steps. Within each generation or time step, four life cycle events are processed in the following order: survival, mate choice, reproduction, mutations. Within the survival procedure, the default individual survival probability depends on population size (density dependence) and on the individual's reproductive effort (which is the sum of the genetic values for costly traits). Within the mate choice procedure, mating groups of a user-specified size are randomly formed.

Individuals from the same mating group encounter each other (randomly, or according to their values of competitiveness), and they choose to mate or not with the encountered partner. If individuals make up a couple they will become unavailable to mate again for the present time step. Within the reproduction procedure, each parent produces gametes and the offspring are created by the random fusion of the parent gametes. Their sex is randomly assigned. Within the genetic mutation procedure, each allele might be substituted by a new one. The genetic values at each locus for each trait are drawn in independent Beta distributions by default, whose shape parameters are defined at the initialization stage (paragraph *Initialization*). This choice allows an explicit definition of the trait value, without referring to any equilibrium or average in the population. The user-defined mutation rate is assumed to be constant throughout the genome. Because survival (S) partly depends on reproductive effort, it can therefore also evolve, allowing individuals to potentially participate in more than one reproduction (i.e., iteroparity evolution).



Details

Survival: The probability of surviving to the next reproduction event is determined as follows:

$$S = \frac{1}{(1 + R)(1 + \frac{N}{K})} \quad (1)$$

where N is the population size, K an indicator of resource limitation in the environment (akin, but not equal, to carrying capacity), R the sum of the genetic values of costly traits of the individual. Eq. 1 states that individual survival results from the interaction between two components. The first component is demographic ($\frac{1}{1+N/K}$), this formula corresponds to the survival rate of the population when considering that survival is solely density-dependent. The second component is ($\frac{1}{1+R}$), this formula states that individual survival is maximum when energy invested into reproduction is null, and that S decreases when R increases. Intuitively, if $N/K = 1$ (close to a demographic equilibrium) and if $R = 1$, then both components affect the survival equally. If the population size drops below K but $R = 1$, then survival rate will increase, making R a relatively greater contributor to survival (see Fig. 1). As a consequence, the model can evolve towards semelparity (low survival, single reproduction) or iteroparity (higher survival leading to potential multiple reproductions).

Mating behaviour: During reproduction, the population is first divided into mating groups of user-specified size M . Subdividing the population into mating groups at the time of reproduction represents the fact that individuals can potentially sample only a restricted number of mates (due to time, space, or energetic constraints, Alcock, 1991; Byers et al., 2005; Deb and Balakrishnan, 2014; Janetos, 1980; Kokko, Booksmythe, et al., 2015; Kokko and Rankin, 2006; Rintamäki et al., 1995), a central limitation when considering social interactions. Population sex ratio is 1 : 1 but the sex ratio within each group can vary, due to random sampling. Under mating systems involving intra-sexual competition and/or inter-sexual preference (Adler, 2011; Emlen and Oring, 1977; Kokko and Rankin, 2006), the number of potential partners encountered will be conditioned by the outcome of inter-individual interactions within the mating group, and is therefore not easily predictable. Individuals can mate only once per time step. They are thus either strictly monogamous (if they die after their first reproduction) or serially monogamous (i.e., they achieve a sequence of non-overlapping monogamous relationships and mate with a different partner at each time step). The model can represent different mating routines:

(1) random mating: pairs of individuals from the same mating group but with different sex are formed randomly. Once a pair is formed, mating will occur. Mated individuals are not available for further mating for the current time step. Note that if M is not a pair number, or if all partners of one sex have mated, some individuals will remain unmated for the current time step.

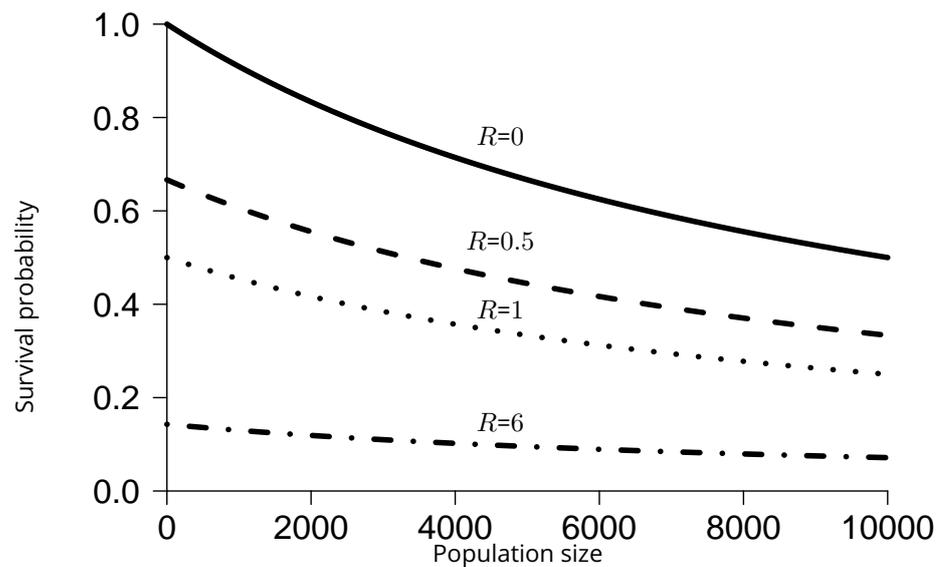
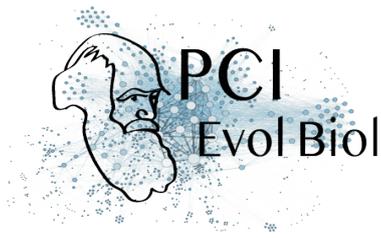


Figure 1. Survival probability as a function of population size for different values of R , with resource limitation parameter $K=10000$.

(2) random encounter with preference: pairs of individuals from the same mating group but of the opposite sex are randomly drawn, but each individual decides to accept or reject the potential partner, conditional on its own preference, and on the gametic investment of the partner. If both partners accept each other, they become unavailable for further mating for the current time step. If at least one individual rejects the mating, both of them return in the mating group and yet again two individuals are randomly drawn from the mating group. The iterative procedure stops when all individuals of one sex are depleted. To prevent the loop from spinning infinitely if individuals remain unpaired, each individual can only encounter x potential partners, x being the initial number of members of the opposite sex in the mating group. The smaller the mating group, the greater the variability of the sex ratio within the mating group, and the more variable the mating opportunities during a season (time step) will be.

(3) competitive encounter without preference: Individuals from the same mating group but with opposite sex encounter each other in an order based on their respective values of competitiveness C . For each pair of individuals thus formed, mating will occur. Mated individuals are not available for further mating for the current time step. When M is not a



pair number, or if one sex is no more available, the less competitive individuals of the mating group will remain unmated.

(4) competitive encounter with preference: Here again, individuals from the same mating group encounter each other based on their value of competitiveness C . Within each pair thus formed, each individual chooses to accept the potential partner based on its preference and the partner's gametic investment. If both partners mutually accept each other, they become unavailable for further mating for the current time step. If at least one individual rejects the mating, individuals remain free to encounter less competitive individuals.

Preference model: The probability that an individual will accept the mating (P_m) is a function of its genetic value of preference and its partner's gametic investment (Eq.2).

$$P_m = \exp^{-\frac{(G_{pref}-G)^2}{2\nu^2}} \quad (2)$$

This equation indicates that individuals have a unimodal preference, i.e. they prefer a particular value of the gametic investment (G_{pref}) with a tolerance around this value (ν). The closer the gametic investment of the partner met is to the individual's preferred value, the higher the probability that the individual will accept the mating (Fig. 2). The parameter ν is set to 0.2. Other forms of preference are also available in the model and can be user specified, such as monotonously increasing preference or threshold preference.

Competition model: Within a mating group of size M , the competitiveness C will be used to assess a non-random order of meeting between pairs of individuals. We assume that the probability of meeting between two individuals is dependent on their respective competitiveness trait values. For instance, two individuals with high competitiveness values will probably meet first within the mating group. Then come pairs of individuals with contrasted competitiveness (one high, one low). Finally, two individuals with low competitiveness will meet at the end of the process, mostly. Pairs of individuals are thus ordered according to the product of their competitiveness. Note that if the meeting is not followed by a mating, each individual will be still available for mating. When individuals have mated once however, they are not available for further mating for the current time step and are removed from the list. Consequently, the less competitive individuals may miss reproduction if all opposite-sex partners in the mating group have already mated.

Reproduction: The gametes are composed of randomly chosen strands from each pair of homologous chromosomes. Recombination between successive genes may occur during the meiosis (depending on the values of recombination probabilities between genes). These probabilities are gathered in a table consisting of $n - 1$ lines, n being the number of genes studied. Each line i contains a number between 0 and 0.5 which represents the probability of recombination between the i^{th} gene and the $(i + 1)^{th}$ gene. The number of offspring per couple

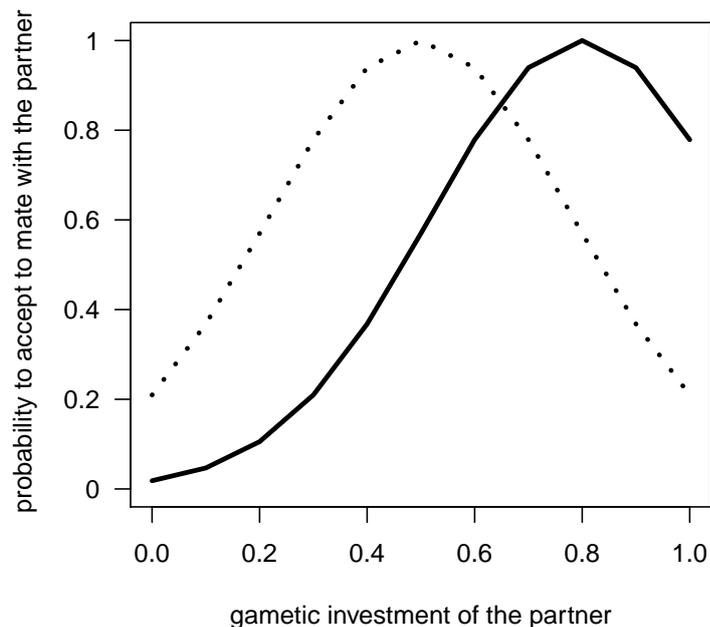
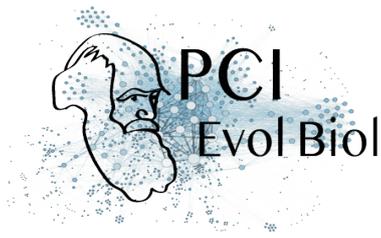


Figure 2. Probability that a potential partner will be accepted, as a function of the values of gametic investment of this partner for two different values of preference ($G_{pref} = 0.5$ dotted line, and $G_{pref} = 0.8$ plain line). Individuals differ in the value of G of their most preferred mate, but all individuals share the same tolerance ($\nu = 0.2$).

is set to the average value of the parents' gametic investments multiplied by a demographic constant. The sex of the offspring is randomly attributed.

Genetic mutation: Right after fecundation, mutations might occur on the offspring genome. The rate of mutation is the probability each allele has to be substituted by a new one. Alleles mutation probability is user-defined, but the default value is set to 10^{-4} mutations per loci, which is within the upper range of spontaneous mutation rates estimated at particular genes in different organisms (Haldane, 1935; Lynch, 2010; Nachman and Crowell, 2000).

Physical structure of the genome: All individuals share the same physical architecture of the genome, which is defined by the number of autosomes, the presence or absence of a sex chromosome, the number of genes, their location on the chromosomes and the probabilities of recombination between these genes. Each individual inherits two alleles for each gene (i.e. diploidy). Allelic effects are described by continuous values and are additive within and be-



tween loci, i.e. dominance effects are not considered, such as the value of a trait corresponds to the sum of allelic values at every locus:

$$T_g = \sum_{l=1}^{l=n} (l_T^{a1} + l_T^{a2}) \quad (3)$$

with n the number of loci ($n = 100$), l_T^{a1} and l_T^{a2} values of the first and the second allele for the trait at the loci l . Individual value of trait is fully determined by the genotype, meanings that environmental effects on phenotype are neglected, and heritability is thus assumed to be unity. Mutations in the model are explicitly defined as a change in allelic value, and we do not draw the effects of mutations in a distribution around a population mean value (as is done in quantitative genetics). In this framework, any mutation (i.e. any allele substitution), has an effect on the trait since the trait is built as the sum of allelic value. This approach contrasts with the other definitions of mutation (for instance, in the quantitative genetics framework), but is required to make explicit the effect of each locus on the trade-off between reproductive investment and survival that is defined at the phenotype level. We define the landscape of allelic values for preference, gametic investment, and competitiveness by Beta distributions of user-specified shape parameters. Default values are [0.65, 24.5], resulting in a right-skewed distribution such as many alleles will have small values for the traits but still, some alleles will have relatively high values for the traits (Fig. 3A). As a consequence, under this set of parameters and using 10 loci, initial trait values for gametic investment followed the distribution showed in Fig. 3B with mean 0.5 and variance 0.02. The skewed Beta distribution is selected to start from relatively low values of traits so to ensure that the initial trade-off between traits values and survival will not crash the population, and to balance the initial conditions between the investment into reproduction and the probability to survive. The user can however specify different values for the Beta distribution, and simulate uniform or bell-shaped distributions.

Genetic architecture: The genetic architecture is here defined as the distribution of allelic effects along the genome, and can, therefore, vary between individuals. According to universal pleiotropy assumption (Fisher, 1930; Hill and XS Zhang, 2012; Paaby and Rockman, 2013), each allele at each locus is specified by a vector of contribution to every trait. For instance in our case, in a two traits model with gametic investment and preference, each allele has two values, one for each trait. All alleles have, therefore, a pleiotropic effect in our model. However, they can code for similar values for the simulated traits, in which case allelic correlation will be high, or they can code for contrasted values among traits, displaying low allelic correlation. This is so, because if selection acts on a trait, an allele with high correlation will not impact the fitness as will a allele with low correlation, depending on the context.

Initialization: To initialize a simulation, three types of parameters are specified:

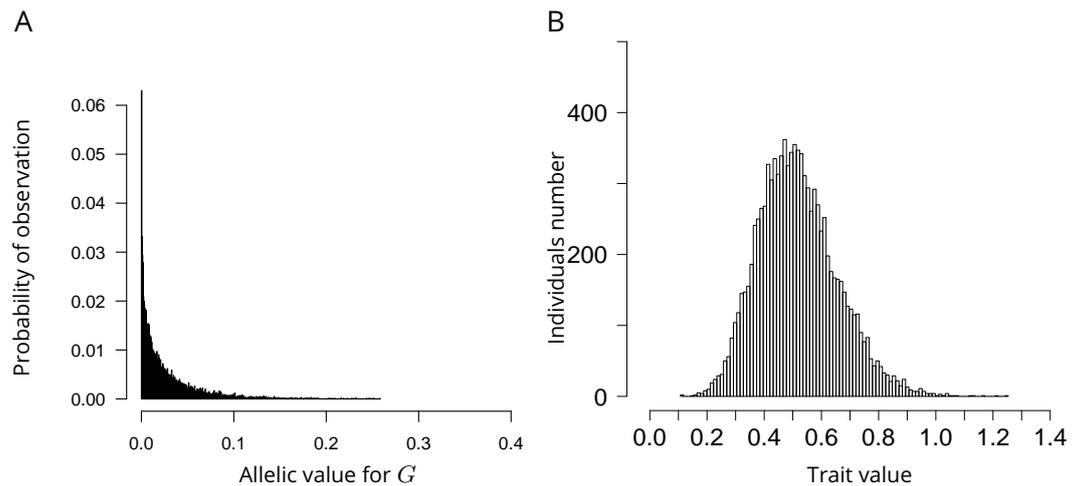
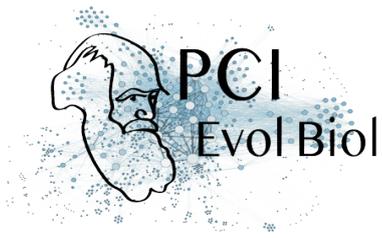


Figure 3. Example of allelic values distribution for the gametic investment (A) and consequent distribution of gametic investment trait values in the population (B). This distribution is obtained by drawing allelic values on the Beta distribution of shape parameters [0.65, 24.5].

- demographic parameters: the resource limitation in the environment K and the initial population size.
- mating parameters: the mating system (e.g. random mating, random encounter with preference, competitive encounter without preference or competitive encounter with preference) and the size of the mating group M are also required. The minimal model is run using a single trait (G), more complex models can be run adding mating preference (P) and/or competitiveness (C).
- parameters for the physical structure of the genome: the number of chromosomes, the number of loci in the genome, the probabilities of recombination between each contiguous pairs of loci, the maximum number of alleles per locus in the population, the distribution of allelic values, and the mutation rate.

Because initialization includes stochastic processes such as sampling in allelic effects distributions, initial states using the same parameters may vary. The model, however, allows starting several simulations out of a single initial state.



Design concept

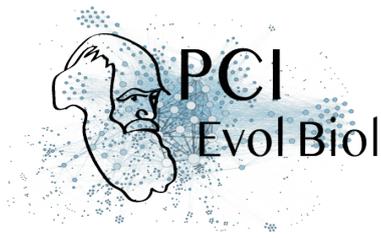
We here describe the general concepts underlying the design of the model.

Adaptation: In our model, fitness is determined by traits, social environment and demography. Individual reproductive traits can indirectly improve individual fitness: high values of G enhance individuals reproductive success but have a survival cost (i.e. higher probability to die per time step). High values of C can improve the mating success of individuals through priority access to mating partners, yet they do have a survival cost too. High values of P allow individuals to get a mate with high fecundity but may have an *opportunity cost* (i.e. loss of mating opportunities, De Jong and Sabelis, 1991; Dechaume-Moncharmont et al., 2016; Fawcett et al., 2012). The survival cost of G and C comes from the function of survival probability (Eq.1). The *opportunity cost* of P emerges from individuals interactions. The user can however also simulates scenarios wherein P has a direct cost too.

Sensing, interactions, collectives: As previously described, during reproduction, the population is partitioned in several mating groups. Individuals sense and interact with potential sexual partners and competitors within the scale of the mating group. However, phenotypic distribution of available partners and of competitors will not affect their decision (i.e. no adaptive behaviour ABM wise), it only affects the outcome of interactions.

Evolutionary dynamics: Fitness variation drives traits evolution which in return changes the social environment (i.e. phenotypic distribution of available partners and competitors) and the demography. This feedback loop prevents 'optimization' in general. Rather, evolutionary dynamics can potentially converge to a pseudo equilibrium. A pseudo-equilibrium is decreed reached when evolutionary rates of traits in Haldane oscillate around zero (Hendry and Kinnison, 1999). It indicates that the product of natural selection, sexual selection, genetic drift and mutation has reached a stable balance.

Stochasticity: Because we use an individual-based model, most processes are inherently stochastic. Survival is, for instance, the realization of a Bernouilli random draw. Mating systems are also an important source of stochasticity. First, individuals are sampled randomly to constitute mating groups. Then, when they do not express competitiveness, individuals randomly encounter each other within each mating group. Mate choice itself is a highly stochastic process: it results from mutual acceptance of both partners, through their respective mating preference which is a probabilistic function (in most cases), so to include an error of assessment of the mating partner quality. Lastly, the transmission of genetic information is also subject to stochasticity because we represent chromosome segregation and recombination during the meiosis and we also account for mutation risk.



Observation: The model can either be run in graphical user interface mode or script mode. In the former, the user can select if all time steps or only a subset should be memorized. In the memorized time steps, all objects (and therefore all individuals and their genomes) are observable. A wide panel of data extractors and visualizers is then available to analyze and illustrate the simulations. In script mode, only population-level variables are recorded over time.

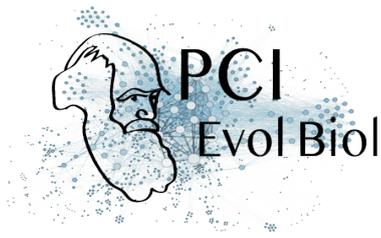
For each simulation, we record different type of variables to characterize mechanisms of evolution: at the population level, trait evolution is monitored by measuring the average trait values in the population and its standard deviations, and the evolutionary rate of traits in Haldanes (Hendry and Kinnison 1999, Appendix 3); genetic architecture evolution is assessed by recording the distribution of allelic values at each locus on average in the population, which gives a statistical view of the genome at the population level. From this data we calculate two indicators to characterize genetic architecture:

(i) The inter-loci relative standard deviation (RSD) in genetic value indicates if all genes contribute equally or very differently to the total genetic value of traits. The inter-loci RSD is calculated as the standard deviation of genetic values between loci pondered by their mean value to look at the relative effects of mutations present in the population.

(ii) The allelic correlation for two traits indicates if genes have, in average, similar effects for both traits or if, on the contrary, genes have in average different effects for each of the traits. It is calculated from the sum of the squared difference between the mean allelic values for the two traits at every locus (Appendix A 2). To assess whether this allelic correlation deviates from random expectation, we used a bootstrap approach. We calculated an index corresponding to the rank of the sum of the squared difference in the distribution of the sum of the squared differences calculated by bootstrap. The bootstrap is performed $n * (n - 1)$ times, with n the loci number. A ranking close to 0 means that the sum of differences in the average genetic values at each locus for the two traits is lower than expected by chance. A ranking close to the maximum index value means that the sum of differences in the average genetic values at each locus for the two traits is higher than expected by chance.

At the individual level: we record individuals lifetime reproductive success (fitness), individuals values of traits and individual level of allelic correlation between each couple of traits. The individual level of allelic correlation is calculated as the covariance between the allelic effects for the two traits.

Installation and execution procedures: The previous description of the model assumptions and mechanisms only covers a part of the settings and tools available to users in the model. A software package is available (package RUNAWAY, running under CAPSIS-4 simulation platform), allowing users to install and interact with the model, at the following address: <https://doi.org/10.15454/6NFGZ9>. A brief documentation, downloading and installation procedures, as well as a quick start guide can be accessed at the following address: http://capsis.cirad.fr/capsis/help_en/runaway



Simulation experiments

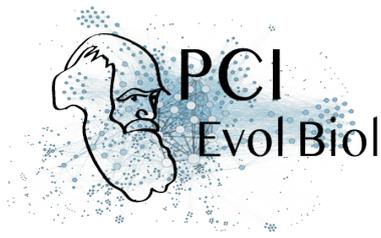
To illustrate the potential of this modelling approach, we here detail two simulation experiments. Default parameters values used for all the simulations are the following: initial population size equal 10000, resource limitation is $K = 10000$, the size of the mating group M is set to the size of the population. The genetic map is, by default, made of 10 unlinked loci distributed over 10 chromosomes (i.e. recombination probability between adjacent locus is 0.5). The mutation rate is set to 10^{-4} mutations per loci.

Experiment 1: coevolution between preference and gametic investment

This first example introduces the evolutionary mechanics in the model, such as the relationships between demography, individual variation, and genetics, as well as the emergent patterns in the model. We here look at the evolution of gametic investment (G), under two different mating systems. First, with random mating, second, with preference (P) driven mating, wherein individuals select partners on their gametic investment. We track how the inclusion of a non-random mating system influences the evolution of G , and whether P itself co-evolves with the former trait. We examine both the dynamic phase of the evolution during the first generations and the later convergence phase around a pseudo-equilibrium after 5000 generations, in a single population.

Evolution of reproductive traits: Under random mating, G shows a substantial evolution from its initial value (of 0.5 average in the population) to reach a pseudo equilibrium around 0.7 after 200 generations (Fig.4 A). The standard deviation around this value remains stable throughout the simulation (around 0.1). The evolutionary rate of G calculated in Haldanes is accordingly moderately high during the first 200 generations and then stabilize to low values. During the early phase of the simulation, we observe a clear positive correlation between individual fitness and G , indicating that values above the mean for this trait are beneficial (i.e. directional selection). Once at the pseudo-equilibrium, this relationship turns to a bell-shaped distribution, indicating stabilizing selection. Note that in this scenario, we also looked at the evolution of P as a non functional trait: this trait shows no evolution whatsoever, its average and standard deviation remaining stable throughout the simulation (Fig.4 A).

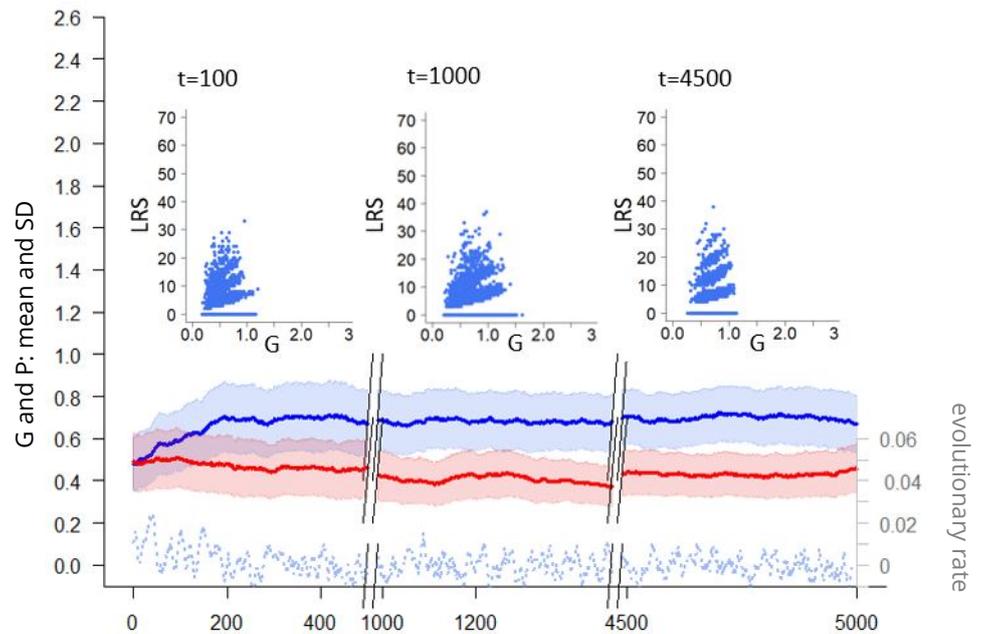
When mating is driven by P , the two traits increase quickly from their initial values (of 0.5 in average in the population) to reach maximum average values of 1.95 for G and 1.99 for P after about 200 generations (Fig.4 B). At this time, the standard deviation of the traits is maximal, around 0.2. Then the mean values of traits and their standard deviation decrease slowly overtime to reach mean values of 1.61 for G and 1.63 for P and standard deviations of 0.035 and 0.038 respectively. During the whole simulation, P is always greater than G , indicating that on average, the higher values of G are preferred in the population. The evolutionary rates of traits indicate a rapid evolution during the first 200 generations (with evolutionary



rates that are in the upper range of values reported in the literature, Hendry and Kinnison, 1999), followed by a much slower evolution of traits. At the 100th generation, a snapshot of the fitness landscape shows that high values of G (of about 2) are advantageous (Fig.4 B). Later snapshots of the fitness landscapes confirm stabilizing selection, as well as an erosion of variance of G in the population with time (Fig.4 B). The emergence of a genetic correlation between G and P sheds some light on the rapid joint evolution of traits (Appendix A4). This correlation arises because individuals with high P choose individuals with high G , and thus, statistically, their offspring will inherit similar P and G values. It accentuates the joint evolution of the traits toward extreme values because P will then evolve under indirect selection (only because of the genetic correlation with G), while G is under increasing sexual selection (as P increases in the population) (e.g. Fisherian mechanism, Fisher, 1915; Hall et al., 2000; Lande, 1981). Interestingly, the positive correlation between the traits is transient as it reaches a maximum of 0.7 at time step 100, and then decreases to become negative (Appendix A4). Then, during the phase of stabilizing selection for the traits, the correlation oscillates around 0.

Genetic architecture evolution: A statistical view of the mean genome of the population shows the mean allelic effect at each locus for the two traits, in regard to the average genetic value per locus (Fig. 5). Initially, due to the sampling of allelic values in random distributions, allelic values are quasi uniformly distributed along the genome and the average allelic values per locus do not deviate from the mean (average allelic effects of loci are thus very close to 0, Fig. 5 at $t = 1$). After 5000 generations, the allelic values are not evenly distributed over the genome, some loci having strong effects; either by coding for much higher value than the average for a trait (e.g. $l1$, $l10$ for G and $l5$ for P) or by coding for much lower value than the average (e.g. $l4$, $l5$, $l6$, for G and $l10$ for P). The increasing variance of genetic value between loci reveals an evolution toward oligogenic architecture (and in this simulation the inter-loci RSD increases from 0.04 for G and 0.05 for P at $t = 1$ to 0.7 and 0.8 respectively). Remarkably, loci having a very high effect on one of the traits also have a very low effect on the other trait (e.g. $l4$, $l5$, $l10$). Therefore it seems that a negative correlation among allelic values for the two trait is established within loci. This observation is supported by the fact that the mean correlation among allelic values individuals' genome is positively correlated with their lifetime reproductive success (Fig. 6). So, in the present context, alleles with opposites effects seems fixed during directional selection. This result is robust to other distribution of allelic values (uniform law, Appendix A5) but could be challenged if environmental parameters were changed (K , M). Within loci negative correlation may confer an adaptive advantage by allowing more combinations of traits values to be transmitted in offspring. Such modular genetic architecture could allow to better respond to the dynamic changes in demography and social environment (Clune et al., 2013; Kashtan and Alon, 2005; Lipson et al., 2002; Parter et al., 2007).

A.



B.

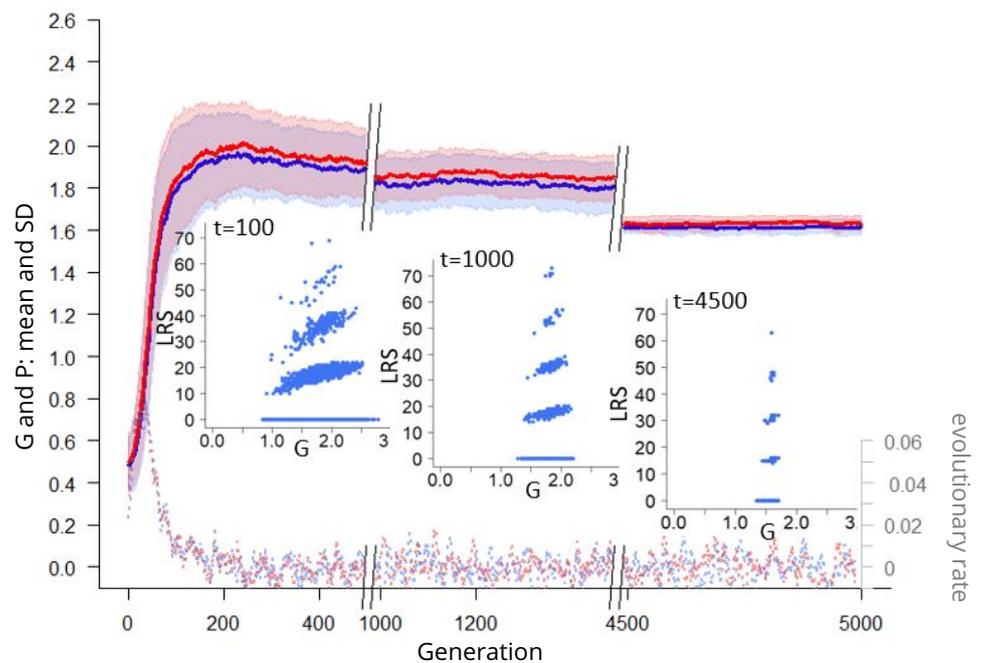


Figure 4. Evolution of G (in blue) and P (in red) in the population over 5000 time step, for one simulation of either (A) random mating, (B) preference driven mating. Mean values of traits are showed in thick lines and standard deviation values are represented in transparency. Also appear in the figure the evolutionary rates in Haldanes for the two traits (dashed lines) and the relationship between lifetime reproductive success (LRS) and G values at different time step during the simulation.

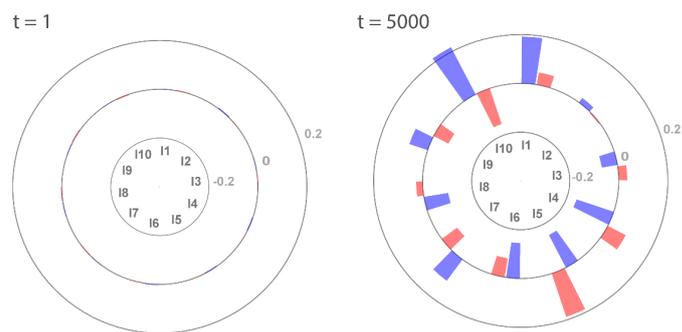


Figure 5. The mean allelic effects per locus in the population, at initialization ($t=0$) and after evolution over 5000 generation. The height of bars at each locus represents the mean value of alleles for this locus centered with respect to the average genetic value per locus for the gametic investment (blue bars) and preference (red bars). The grey lines indicate the scale.

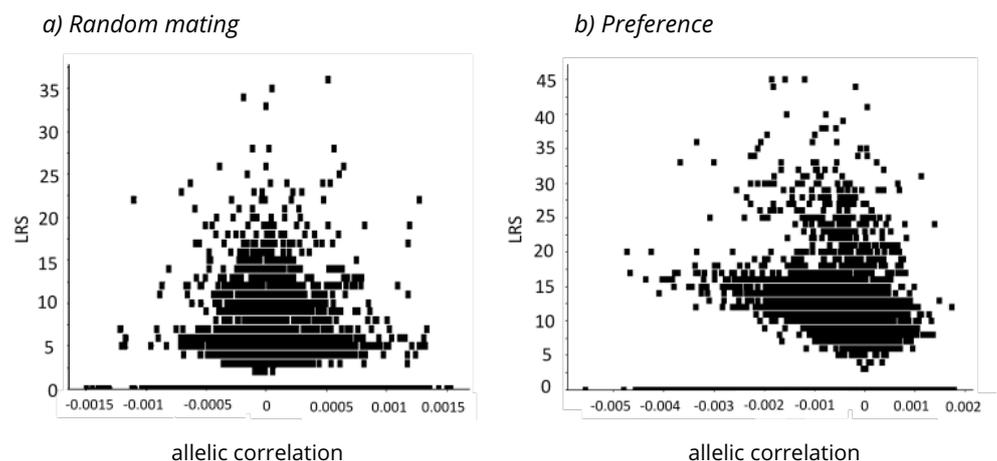
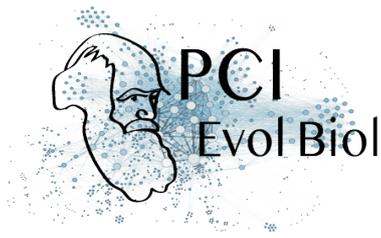


Figure 6. Lifetime reproductive success of individuals at timestep 50 as a function of their G and P allelic values correlation. (a) when mating is random, (b) when mating is driven by preference. In the latter case, we observe a negative correlation between individual lifetime reproductive success allelic correlation.



Experiment 2: Effect of mating systems on traits and genetic architecture evolution

In this experiment, we generalize the above approach to 4 different mating systems, named according to the number of traits expressed in each one (random mating G , random encounter and preference $G + P$, competitive encounter without preference $G + C$, competitive encounter with preference $G + C + P$). Our objective is to look at the effects of mating systems on traits evolution, but also at their rippling consequences on alleles values distributions and their correlation in the genome. Here however, we perform 30 replications of simulation for each mating system, each over 5000 generations. By replicating the simulations, we investigate whether the factor we manipulate (here mating systems) leads to very homogeneous evolutionary outcomes, or whether they generate a diversified set of equilibrium. We here only focus on the final picture of the evolutionary process, without detailing the dynamics leading to it.

Evolution of the reproductive traits: G evolves toward different values depending on the scenario, which is accompanied by different population sizes at the pseudo-equilibrium (Fig. 8). When individuals express their preference (scenario G and $G + P$), P is favored by sexual selection, which promotes the evolution of G toward much higher values than in the random mating system, in which P evolves under genetic drift. As G is costly in terms of survival, its increase in the population leads to lower average survival, which translates into a lower pseudo-equilibrium population size (Fig. 8).

As previously explained, the joint evolution of P and G toward extreme values is caused by the build-up of correlation between the traits (Appendix A4), which is at the core of the mechanism of Runaway selection proposed by Fisher (1915).

Competitiveness (C) evolution is also conditioned by the simulation scenario. C is selected for (despite the associated survival cost) only when individuals also express their preference P on gametic investment G (scenario $G + P + C$), because in that case, the most competitive individuals are more likely to find a suitable mate. In the scenario without preference (scenario $G + C$), C is counter selected, its cost is too great when traded off against the possible benefit of reducing opportunity costs (e.g. the risk of not finding a mate). Indeed, in the present simulations, mating group size is equal to the population size, and opportunity cost under random mating is therefore negligible.

Whereas all simulations converge to the same value of G at the pseudo equilibrium under random mating, as soon as there is some preference or some competitive encounter, inter-simulations variance increases. It therefore underlines the importance of the complexity of behavioural interactions on the outcome of evolution.

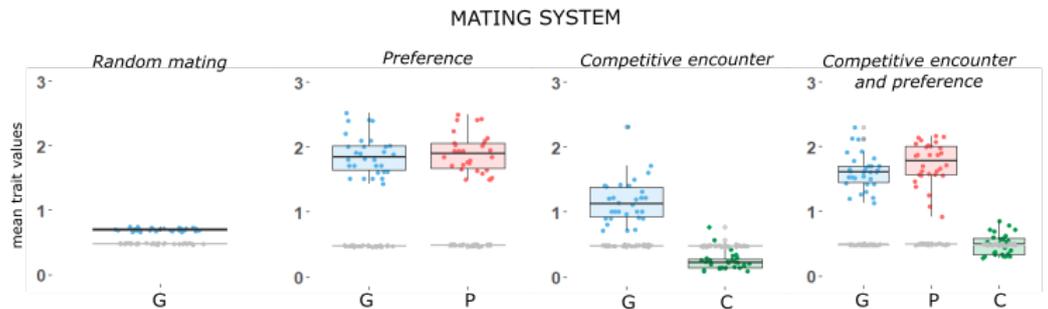


Figure 7. Mean values of gametic investment, preference and competitiveness, initially (in grey) after evolution over 5000 time step (in color). Four mating systems simulated : random mating (G), random encounter and preference ($G + P$), competitive encounter without preference ($G + C$), competitive encounter and preference ($G + C + P$). Each dot represents the main trait value in a simulation. A boxplot indicates the 2.5, 25, 50, 75 and 97.5 quantiles of the distribution among simulations.

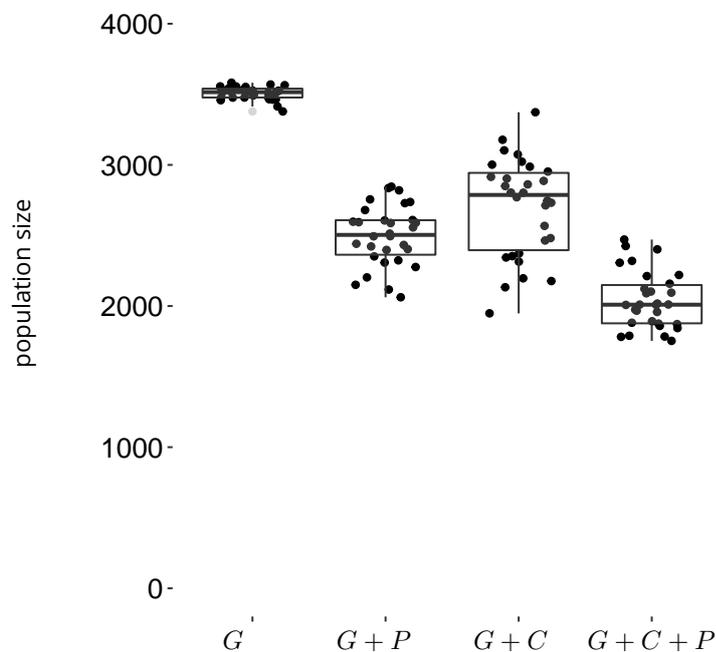
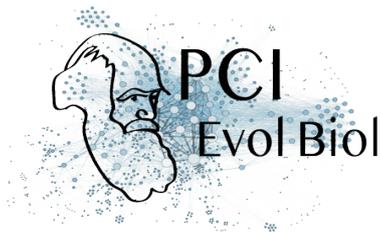


Figure 8. Mean population size in the different simulations after evolution over 5000 time step. Four mating systems are simulated : random mating (G), random encounter and preference ($G + P$), competitive encounter without preference ($G + C$), competitive encounter and preference ($G + C + P$). Boxplots indicate the 2.5, 25, 50, 75 and 97.5 quantiles of the distribution among replications.



Genetic architecture evolution: Whatever the mating system investigated the relative contribution of loci to the total genetic value of each trait has somehow evolved, with some loci presenting a stronger relative contribution than in the initial situation. A difference of allelic effects distribution between mating systems is visible when looking at inter-loci relative standard deviation (RSD) of the mean allelic values (Fig.9), the measure allows to show the difference in magnitude of genetic values between loci, independently of the differences in the values of the traits between scenarios. When mating is random, inter-loci RSD for G and for P increases, but it is slightly higher for G , compared to the trait P that is only subject to genetic drift and mutation in the random mating system (i.e., neutral trait). Under the preference driven mating system, P is expressed, and the inter-loci RSD of the two traits is a bit higher. When we turn to a mating system with competitive encounter without preference, inter-loci RSD further increase for both G and C traits. In contrast, when the mating system is driven by both competition and preference, inter-loci RSD for C is much lower, whereas the inter-loci RSD for G and P remain high (Fig.9). These results indicate that the different mating systems, mostly due to different ways for individuals to interact for reproduction, will select for non random distribution of genetic values along the genome. This could be of course further investigated by considering different physical structure of the genome (number of chromosomes and location of genes) .

The pattern of correlation among allelic values within loci also evolves differently depending on the mating system (Fig. 10). When individuals mate randomly, within loci allelic correlation evolves randomly and there is no effect of initial allelic distribution: in simulations where allelic values are initially correlated by chance, the genome can evolve toward negative correlation and *vice versa*, not illustrated here.

However, when preference matters, negative allelic correlation for G and P evolves. Remarkably, such negative correlation occurs in scenarios where there is co-evolution between the G and P and so positive global genetic correlation between the traits (scenarios $G + P$ and $G + P + C$, Fig.10). In contrast, when encounter is competitive but in the absence of preference, no particular organisation in the allelic effects for G and for C emerge. But here again the addition of preference select for negative correlation between allelic values and thus for a modular genetic architecture. We, therefore, observe that the mating systems mediates to some extent the evolution of the genome. Importantly, this result is robust to other mutation landscape (Appendix A5), confirming that the emergence of negative correlation between allelic values is not a computational artefact due to our draws in the distribution of allelic values.

Within the scope of this paper, we did not change the mutation rate μ , the carrying capacity K and the mating group size M . Within these settings, preference reached high values whereas competitiveness barely evolved. We suspect that it would be a whole different matter if we had modified M . Indeed, in situations where individuals only meet a subset of the population to find their mate ($M < \text{population size}$) the risk of not reproducing can be high, and consequently competition is expected to be stronger. In that case, traits may evolve dif-

ferently; for instance, iteroparity (i.e low G , low P) can be favored as a form of "Bet Hedging" in time (Wilbur and Rudolf, 2006) and high competitiveness could be an advantage. This may, in turn, affect the evolution of genetic architecture.

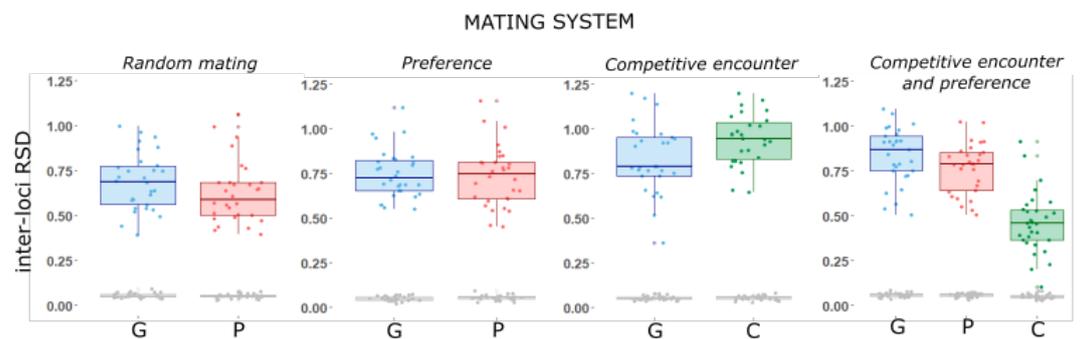


Figure 9. The relative standard deviation of the mean allelic values between loci for the different traits present in the scenarios (G, P, C, in the first scenario the preference is not expressed), initially (in grey) and after evolution over 5000 generations (in color). The boxplots are drawn with 30 simulations of the four mating systems (random encounter, preference driven mating, competitive encounter, competitive encounter and preference driven mating) and report the mean and 95% interquartile range interval.

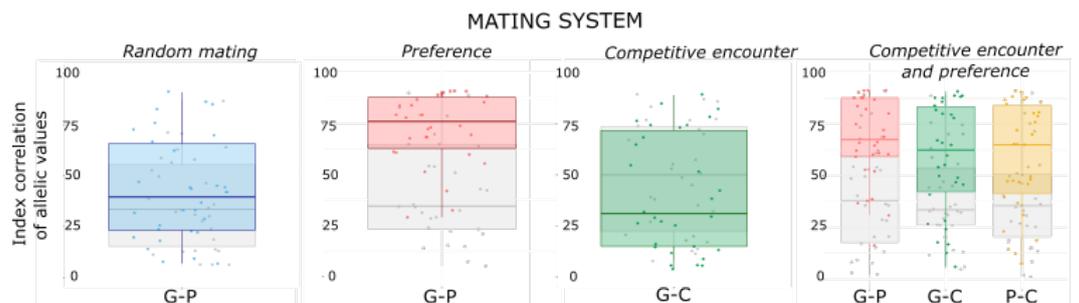
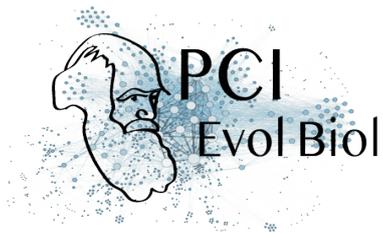


Figure 10. Index correlation of allelic values for the pairs of trait. The measures are shown initially (in grey) and after evolution over 5000 time step (in color). The boxplots are drawn with 30 simulations of the four mating systems (random encounter, preference driven mating, competitive encounter, competitive encounter and preference driven mating) and report the mean and 95% interquartile range interval of index values.

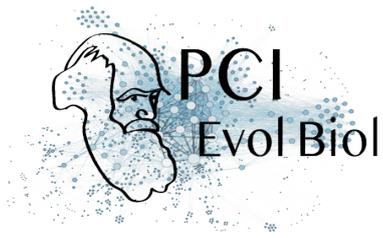


Discussion

By capturing the processes linking genes, genome, individuals, groups, population, and environment levels, DG-ABMs offer new opportunities to study evolutionary dynamics in an integrative approach. The present model is an endeavour to develop such approach in a domain where it should be especially relevant: sexual selection, an eminently context-dependent process emerging from inter-individual interactions (Kokko, Booksmythe, et al., 2015; Muniz and Machado, 2018; Otto et al., 2008). We sought, by means of examples, to illustrate how the interplay between genetics, demography, and behaviors is pivotal in predicting the evolution of traits and genetic architecture under sexual selection, whereas too often these disciplines are treated separately (Bengston et al., 2018; Rittschof and Robinson, 2014; Wilkinson et al., 2015).

Our simple simulation experiments revolved around comparing various mating systems. In these examples, wherein we kept control parameters constant along the simulations (K , μ , M , physical structure of the genome), two distinct evolutionary phases could be distinguished, and they illustrate the potential of the approach to simulate both micro-evolutionary patterns and so called evolutionary equilibrium.

The first phase is often a directional change, wherein micro-evolutionary processes can be analyzed in details, at the gene, individual or population scale. It can be perceived as a possible route towards a pseudo-equilibrium (although such pseudo-equilibrium is not certain). It depends actively on initial conditions (values of traits, based on initial genetic architecture). The direction and speed of this route are of major interest: traits evolve through optimization of fitness through selection, although we do not specify the evolutionary optimum or the adaptive landscape *per se*, contrary to most models which assume a known evolutionary optimum (e.g; Guillaume and Otto 2012; Jones, Arnold, et al. 2003; Jones, Bürger, et al. 2014; Lande and Arnold 1985; Lorch et al. 2003; Matuszewski et al. 2014; Mead and Arnold 2004). We measure the variance of fitness throughout evolution, and we can, therefore, better understand the mechanisms that lead to the selection of some trait combinations and some particular genetic architectures. In this way, the model can also be used to assess the robustness of some previous and more simple theoretical models, in a more realistic framework. For instance, we can measure the correlation between traits, relate it to fitness at each generation, and simultaneously observe whether these correlations between traits foster – or not – the building of actual non-random allelic structure within the genome. It allows us to show, among other things, that the building of genetic correlation between mating preferences and a trait - assumed by quantitative genetics approaches and at the core of Runaway selection (Fisher, 1915; Lande, 1981) - also occurs in our more complex model and explicitly arises from assortative mating process. But in our model, assortative mating is not assumed: it can potentially emerges from the evolution of preference and competitiveness, and interactions between individuals during the breeding season, achieving variable mating and reproductive



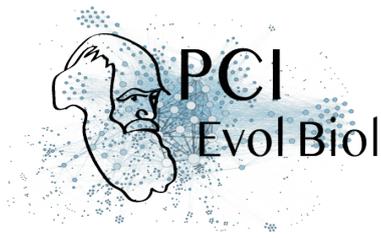
success. Additionally, in the presented simulations, the rising of such genetic correlation is accompanied by the evolution of a modular genetic architecture.

In addition, the role of environmental parameters on this dynamic phase is paramount, and the model can be used to simultaneously test for the effect of habitat resource (K), social parameter (M), and genetic constraints (mutation rate μ as well as the physical structure of the genome). All of these parameters will likely affect the genetic variance, which will in turn condition the speed of evolution. The study of these effects is central to the understanding of biodiversity dynamics and resilience: for instance the model could be used to investigate variable mutation rates in time or along the genome, or to explore the effect of fluctuating resource in the environment or factors affecting the distribution of potential mating partners.

The second phase, in our current examples, is characterized by having reached an evolutionary pseudo equilibrium, the mean and variance of traits being stable within a population. Likewise, K , mutation rate, the physical structure of the genome and mating group size may have a strong impact on the outcome of the simulations. First, it can affect what are the mean values for traits, population size, and genetic architecture, but also their respective variances at equilibrium. Although these equilibria can seldom be observed in nature, where many environmental parameters fluctuate, they can nevertheless be used as a reference point to compare (qualitatively) our predictions with theoretical models. For instance, our results on genetic architecture suggest that allelic correlation can be counter selected in some situations even in the absence of functional trade-off in gene activities (Guillaume and Otto, 2012), and support the idea that selection pressures may substantially shape the distribution of pleiotropic effects among genes (Cheverud, 1996; Hansen, 2003; Pigliucci, 2008).

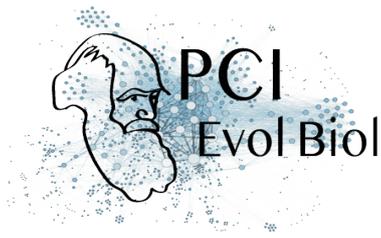
Additionally, predictions at equilibrium are of particular interest when we consider to what extent different replications for the same scenario may or may not converge. The difference between replications can be seen as a proxy of variance between populations, indicating that despite having the same initial conditions, environment, and constraints, two populations may diverge to some extent (when not related by dispersal, in the current model). This is of major interest for empiricists trying to investigate parallel evolution of populations experimenting similar environments (Bolnick et al., 2018; Oke et al., 2017; Schwartz and Hendry, 2007); in some cases, one should not be surprised to find substantially variable evolutionary outputs. In the core of our model, it is noteworthy to underline that such divergences mainly occur due to interactions between individuals (such as mate choice), and are therefore highly sensitive to stochastic processes. We suggest that such a mechanism could also play a major role in natural populations, stressing the importance to study and understand behavioural interactions in relation to the environment.

Arguably, the two simulation experiments only envision a small span of the possible variation in mating behaviours, the physical structure of the genome or environmental control. In its present shape, our model already includes alternative scenarios to explore, of potential



interest to evolutionary and behavioural ecologists. For instance, the shape and modalities of the preference function can be easily modified by users to address fixed or probabilistic threshold responses. By modifying the physical structure of the genome, we can simulate sexual chromosomes, thereby authorizing sexual differentiation in traits genetic values. Such differences may, in turn, affect survival differently between sexes, which would, therefore, affect the operational sex ratio within mating groups, possibly modifying the strength of sexual selection, and retroactively participating in the divergent evolution of the genome between males and females (Bonduriansky, 2009; Chapman et al., 2003; Gibson et al., 2002; Hammer et al., 2008). This example indicates how all levels are naturally entangled in the individual based demogenetic approach. Notably, we have not yet included explicitly the role of parental care as well as the question of gamete size, two questions that can be central in the evolution of mating systems (Fromhage and Jennions, 2016; Kokko and Jennions, 2008; Lehtonen, Jennions, et al., 2012; Lehtonen, Parker, et al., 2016; Parker et al., 1972). For now, parental care is implicitly assumed, since the calculation of fecundity (as the mean of both partners gametic investments) somehow implies that both parents contribute to reproductive success.

The DG-ABM approach is somehow a middle ground between analytical methods and empirical approaches. On the one hand, analytical methods usually only focus on a limited sets of variables, requiring generally strong assumptions. DG-ABMs can relax some of these assumptions: for instance, there is no need to assume an equilibrium of genetic variance or stable age distribution within populations; and they are thus well suited to focus on unstable demographic or genetic situations, that are often of primary importance in evolution (Barton and B Charlesworth, 1984; Hendry and Kinnison, 1999; Kirkpatrick and Barton, 1997). On the other hand, empirical approaches can produce a wealth of patterns, on traits, on behaviour, their genetic basis, on life history trade-offs, or on population demographic and genetic structures. Only a fragment of these data are usually picked up, so to fit theoretical predictions stemming from reductive analytical models, whereas many of these patterns could help testing theoretical expectations more confidently. By allowing to generate patterns on several levels, we hope that DG-ABM way can help strengthen the links between theory and observations. Obviously, a central concern in this matter lies in the validity of the mechanisms and assumptions we made, and how well they allow to recreate either theoretical facts or natural patterns. Such question cannot be addressed independently from the simulations scenarios built by the user, since the sets of parameters (physical structure of the genome, ecological parameters, type of mating system, etc.) will directly impact the results. We can however give examples on how to connect some outcomes of the model to either theoretical or empirical expectations. For instance, when we investigated the coevolution between sexual preference and gametic investment, both traits evolved rapidly at the beginning - since we were unlikely to start from an equilibrium. But during this phase of evolution, genetic variance increased. It later decreased when the population reached a pseudo-equilibrium. We thus reproduced a known result, which is the transient runaway as trait and preference eventually goes to a new

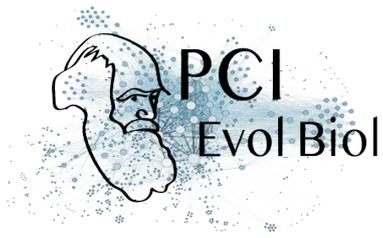


steady state (De Jong and Sabelis, 1991). Additionally, the pseudo-equilibrium reached was admittedly stable. Lande (1981) predicted in his model that stability in this case was only ensured when the ratio of trait-preference covariance over trait variance in the population was equal or below 1 (in case of equal mutation variance input for both traits). Above that value, the equilibrium becomes unstable. The said ratio was observed to reach values up to 0.8 over our simulations, but it never exceeded 1. We also explored different physical structure of the genome (with different loci numbers, linkage disequilibrium, mutation rates; data not shown) but in the range of parameters explored, no simulation produces runaway selection toward unstable equilibrium. The non-occurrence of unstable equilibrium could be due to finite population size and the resulting genetic drift, potentially erasing the genetic correlation (Nichols and Butlin, 1989); or it could be caused by the opportunity cost that emerges from the individual interactions in the model and slows down the evolution of preference (Kokko, Booksmythe, et al., 2015).

On a more empirical perspective, on the same example, we were able to measure evolutionary rates of about 0.06 Haldanes over 10 generations during the phase of rapid evolution with preference, and then of 0.005 (over 10 generations) at the pseudo equilibrium. These values are in agreement with empirical studies: for instance, Karim et al. (2007) reported rates of male color change following an experimental introduction that ranged from 0.01 to 0.031 Haldanes over 13–26 generations. These values are close to those we observe during the dynamic phase of evolution.

Conclusion

Beyond the question of co-evolution between gametic investment and preference, the DG-ABM modelling approach allows to make much more comprehensive predictions on biological systems: for instance, instead of solely focusing on the evolutionary equilibria of trait values, we here provide a complete picture of what such equilibria entail also in term of genetic architecture and demography. Namely, for such dynamics to occur, we demonstrate that demographic characteristics will also reach non-random values (level of iteroparity and survival, effective numbers of breeders, population size) and that this may come with the building of non-random allelic structure within the genome. Such projection also allows capturing more efficiently the evolutionary constraints that will control the outcome of adaptive evolution (genetic architecture, phenotypic correlation, demographic processes). We hope that the present contribution can motivate further work on the link between the physical structure of the genome and variations in mating systems or life histories (e.g. D Charlesworth and Wright 2001; Lamichhane et al. 2016; Misevic et al. 2006; Plomion et al. 2018; Sinervo and Svensson 2002).



Acknowledgements

The present model was coded on the CAPSIS-4 modelling platform (<http://capsis.cirad.fr>, Dufour-Kowalski et al. 2012) maintained by F. de Coligny and N. Beudez, who also helped in developing parts of the code. We also thank A. Courtiol for thoughtful suggestions on an earlier version of the model. Version 3 of this preprint has been peer-reviewed and recommended by Peer Community In Evolutionary Biology (<https://doi.org/10.24072/pci.evolbiol.100112>).

Data accessibility

Simulation results and R scripts are available on demand. A software package is available (package RUNAWAY, running under CAPSIS-4 simulation platform), allowing users to install and interact with the model, at the following address: <https://doi.org/10.15454/6NFGZ9>. A brief documentation, downloading and installation procedures, as well as a quick start guide can be accessed at the following address: http://capsis.cirad.fr/capsis/help_en/runaway

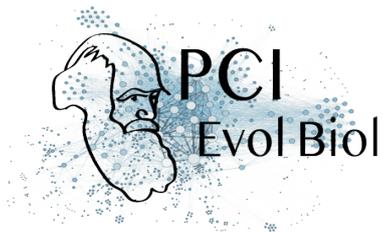
Conflict of interest disclosure

The authors of this preprint declare that they have no financial conflict of interest with the content of this article.

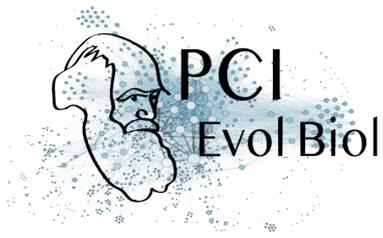
References

- Adler GH (2011). Spacing patterns and social mating systems of echimyid rodents. *Journal of Mammalogy* 92, 31–38.
- Aguilée R, D Claessen, and A Lambert (2013). Adaptive radiation driven by the interplay of eco-evolutionary and landscape dynamics. *Evolution* 67, 1291–1306.
- Alcock J (1991). Mate-locating behavior of *Xylocopa californica arizonensis* Cresson (Hymenoptera: Anthophoridae). *Journal of the Kansas Entomological Society*, 349–356.
- Bailey NW and AJ Moore (2012). Runaway sexual selection without genetic correlations: social environments and flexible mate choice initiate and enhance the Fisher process. *Evolution: International Journal of Organic Evolution* 66, 2674–2684.
- Barton NH and B Charlesworth (1984). Genetic revolutions, founder effects, and speciation. *Annual Review of Ecology and Systematics* 15, 133–164.
- Bengston SE, RA Dahan, Z Donaldson, SM Phelps, K Van Oers, A Sih, and AM Bell (2018). Genomic tools for behavioural ecologists to understand repeatable individual differences in behaviour. *Nature ecology & evolution* 2, 944–955.
- Bolnick DI, RD Barrett, KB Oke, DJ Rennison, and YE Stuart (2018). (Non) parallel evolution. *Annual Review of Ecology, Evolution, and Systematics* 49, 303–330.

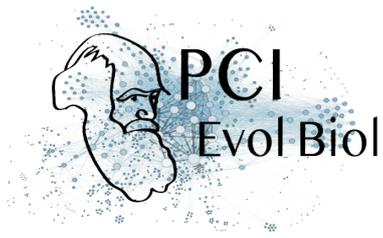
- Bonduriansky R (2009). Reappraising sexual coevolution and the sex roles. *PLoS Biology* 7, 2–4. issn: 15449173. doi: 10.1371/journal.pbio.1000255.
- Byers JA, PA Wiseman, L Jones, and TJ Roffe (2005). A large cost of female mate sampling in pronghorn. *The American Naturalist* 166, 661–668.
- Chapman T, G Arnqvist, J Bangham, and L Rowe (2003). Sexual conflict. *Trends in Ecology & Evolution* 18, 41–47.
- Charlesworth D and SI Wright (2001). Breeding systems and genome evolution. *Current opinion in genetics & development* 11, 685–690.
- Chevalier L, F De Coligny, and J Labonne (2020). A demogenetic individual based model for the evolution of traits and genome architecture under sexual selection. *bioRxiv* 2020.04.01.014514, ver. 3 peer-reviewed and recommended by *PCIEvolBiol*. <https://doi.org/10.1101/2020.0401014514>. doi: 10.24072/pci.evolbiol.100112.
- Cheverud JM (1996). Developmental Integration and the Evolution of Pleiotropy. *Integrative and Comparative Biology* 36, 44–50. issn: 1540-7063. doi: 10.1093/icb/36.1.44.
- Clune J, JB Mouret, and H Lipson (2013). The evolutionary origins of modularity. *Proceedings of the Royal Society b: Biological sciences* 280, 20122863.
- Connallon T (2015). The geography of sex-specific selection, local adaptation, and sexual dimorphism. *Evolution* 69, 2333–2344.
- Darwin C (1872). *The descent of man, and selection in relation to sex*. Vol. 2. D. Appleton.
- De Jong MC and MW Sabelis (1991). Limits to runaway sexual selection: the wallflower paradox. *Journal of Evolutionary Biology* 4, 637–655.
- DeAngelis DL and WM Mooij (2005). Individual-based modeling of ecological and evolutionary processes. *Annu. Rev. Ecol. Evol. Syst.* 36, 147–168.
- Deb R and R Balakrishnan (2014). The opportunity for sampling: the ecological context of female mate choice. *Behavioral Ecology* 25, 967–974.
- Debarre F and SP Otto (2016). Evolutionary dynamics of a quantitative trait in a finite asexual population. *Theoretical population biology* 108, 75–88.
- Dechaume-Moncharmont FX, T Brom, and F Cézilly (2016). Opportunity costs resulting from scramble competition within the choosy sex severely impair mate choosiness. *Animal Behaviour* 114, 249–260. issn: 00033472. doi: 10.1016/j.anbehav.2016.02.019.
- Dufour-Kowalski S, B Courbaud, P Dreyfus, C Meredieu, and F De Coligny (2012). Capsis: an open software framework and community for forest growth modelling. *Annals of forest science* 69, 221–233.
- Emlen ST and LW Oring (1977). Ecology, sexual selection, and the evolution of mating systems. *Science* 197, 215–223.
- Falconer DS, TF Mackay, and R Frankham (1996). Introduction to quantitative genetics (4th edn). *Trends in Genetics* 12, 280.
- Fawcett TW, JM McNamara, and AI Houston (Feb. 2012). When is it adaptive to be patient? A general framework for evaluating delayed rewards. *Behavioural Processes* 89, 128–136. issn: 03766357. doi: 10.1016/j.beproc.2011.08.015.



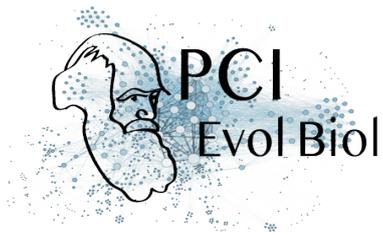
- Fisher RA (1915). The evolution of sexual preference. *The Eugenics Review* 7, 184.
- Fisher RA (1930). *The Genetical Theory of Natural Selection* Oxford Univ. Press (Clarendon), London (Reprinted and revised, 1958).
- Fromhage L and MD Jennions (2016). Coevolution of parental investment and sexually selected traits drives sex-role divergence. *Nature communications* 7, 1–11.
- Gibson JR, AK Chippindale, and WR Rice (2002). The X chromosome is a hot spot for sexually antagonistic fitness variation. *Proceedings of the Royal Society of London. Series B: Biological Sciences* 269, 499–505.
- Grimm V, U Berger, F Bastiansen, S Eliassen, V Ginot, J Giske, J Goss-Custard, T Grand, SK Heinz, G Huse, et al. (2006). A standard protocol for describing individual-based and agent-based models. *Ecological modelling* 198, 115–126.
- Guillaume F and SP Otto (2012). Gene functional trade-offs and the evolution of pleiotropy. *Genetics* 192, 1389–1409.
- Guillaume F and J Rougemont (2006). Nemo: an evolutionary and population genetics programming framework. *Bioinformatics* 22, 2556–2557.
- Haldane JB (1935). The rate of spontaneous mutation of a human gene. *Journal of genetics* 31, 317.
- Hall DW, M Kirkpatrick, and B West (2000). Runaway sexual selection when female preferences are directly selected. *Evolution* 54, 1862–1869.
- Hammer MF, FL Mendez, MP Cox, AE Woerner, and JD Wall (2008). Sex-biased evolutionary forces shape genomic patterns of human diversity. *PLoS genetics* 4.
- Hansen TF (2003). Is modularity necessary for evolvability? Remarks on the relationship between pleiotropy and evolvability. *BioSystems* 69, 83–94. issn: 03032647. doi: 10.1016/S0303-2647(02)00132-6.
- Hendry AP and MT Kinnison (1999). Perspective: the pace of modern life: measuring rates of contemporary microevolution. *Evolution* 53, 1637–1653.
- Hill WG and XS Zhang (2012). On the pleiotropic structure of the genotype–phenotype map and the evolvability of complex organisms. *Genetics* 190, 1131–1137.
- Holt RD and M Barfield (2011). Theoretical perspectives on the statics and dynamics of species' borders in patchy environments. *The American Naturalist* 178, S6–S25.
- Iwasa Y and A Pomiankowski (1995). Continual change in mate preferences. *Nature* 377, 420.
- Iwasa Y, A Pomiankowski, and S Nee (1991). The Evolution of Costly Mate Preferences II. The 'Handicap' Principle. *Evolution* 45, 1431. issn: 00143820. doi: 10.2307/2409890.
- Janetos A (1980). Strategies of female mate choice: a theoretical analysis. *Behavioral Ecology and Sociobiology* 7 12, 107–112.
- Jones AG, SJ Arnold, and R Bürger (2003). Stability of the G-matrix in a population experiencing pleiotropic mutation, stabilizing selection, and genetic drift. *Evolution* 57, 1747–1760.
- Jones AG, R Bürger, and SJ Arnold (2014). Epistasis and natural selection shape the mutational architecture of complex traits. *Nature communications* 5, 1–10.



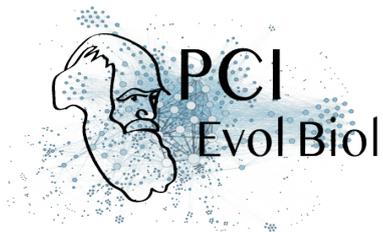
- Jones AG and NL Ratterman (2009). Mate choice and sexual selection: what have we learned since Darwin? *Proceedings of the National Academy of Sciences* 106, 10001–10008.
- Jones AG, SJ Arnold, and R Bürger (2007). The mutation matrix and the evolution of evolvability. *Evolution* 61, 727–745. issn: 00143820. doi: 10.1111/j.1558-5646.2007.00071.x.
- Kashtan N and U Alon (2005). Spontaneous evolution of modularity and network motifs. *Proceedings of the National Academy of Sciences* 102, 13773–13778.
- Kent C, R Azanchi, B Smith, A Formosa, and JD Levine (2008). Social context influences chemical communication in *D. melanogaster* males. *Current Biology* 18, 1384–1389.
- Kirkpatrick M and NH Barton (1997). Evolution of a species' range. *The American Naturalist* 150, 1–23.
- Kokko H, I Booksmythe, and MD Jennions (2015). Mate-sampling costs and sexy sons. *Journal of evolutionary biology* 28, 259–266.
- Kokko H and MD Jennions (2008). Parental investment, sexual selection and sex ratios. *Journal of Evolutionary Biology* 21, 919–948. issn: 1010061X. doi: 10.1111/j.1420-9101.2008.01540.x.
- Kokko H and DJ Rankin (2006). Lonely hearts or sex in the city? Density-dependent effects in mating systems. *Philosophical Transactions of the Royal Society B: Biological Sciences* 361, 319–334.
- Krupp JJ, C Kent, JC Billeter, R Azanchi, AKC So, JA Schonfeld, BP Smith, C Lucas, and JD Levine (2008). Social experience modifies pheromone expression and mating behavior in male *Drosophila melanogaster*. *Current Biology* 18, 1373–1383.
- Labonne J and AP Hendry (2010). Natural and sexual selection giveth and taketh away reproductive barriers: models of population divergence in guppies. *The American Naturalist* 176, 26–39.
- Labonne J, V Ravigné, B Parisi, and C Gaucherel (2008). Linking dendritic network structures to population demogenetics: the downside of connectivity. *Oikos* 117, 1479–1490.
- Lamichhaney S, G Fan, F Widemo, U Gunnarsson, DS Thalmann, MP Hoepfner, S Kerje, U Gustafson, C Shi, H Zhang, et al. (2016). Structural genomic changes underlie alternative reproductive strategies in the ruff (*Philomachus pugnax*). *Nature Genetics* 48, 84–88.
- Lande R (1976). Natural selection and random genetic drift in phenotypic evolution. *Evolution* 30, 314–334.
- (1980). The genetic covariance between characters maintained by pleiotropic mutations. *Genetics* 94, 203–215.
- (1981). Models of speciation by sexual selection on polygenic traits. *Proceedings of the National Academy of Sciences* 78, 3721–3725.
- Lande R and SJ Arnold (1985). Evolution of mating preferences and sexual dimorphism. *Journal of Theoretical Biology* 117, 651–664. issn: 00225193. doi: 10.1016/S0022-5193(85)80245-9.
- Lehtonen J, MD Jennions, and H Kokko (2012). The many costs of sex. *Trends in ecology & evolution* 27, 172–178.



- Lehtonen J, GA Parker, and L Schärer (2016). Why anisogamy drives ancestral sex roles. *Evolution* 70, 1129–1135.
- Lipson H, JB Pollack, and NP Suh (2002). On the origin of modular variation. *Evolution* 56, 1549–1556.
- Lorch PD, S Proulx, L Rowe, and T Day (2003). Condition-dependent sexual selection can accelerate adaptation. *Evolutionary Ecology Research* 5, 867–881.
- Lynch M (2010). Evolution of the mutation rate. *TRENDS in Genetics* 26, 345–352.
- Matessi C and C Di Pasquale (1996). Long-term evolution of multilocus traits. *Journal of Mathematical Biology* 34, 613–653.
- Matuszewski S, J Hermisson, and M Kopp (2014). Fisher's geometric model with a moving optimum. *Evolution* 68, 2571–2588.
- Mead LS and SJ Arnold (2004). Quantitative genetic models of sexual selection. *Trends in ecology & evolution* 19, 264–271.
- Misevic D, C Ofria, and RE Lenski (2006). Sexual reproduction reshapes the genetic architecture of digital organisms. *Proceedings of the Royal Society B: Biological Sciences* 273, 457–464.
- Moore AJ, ED Brodie III, and JB Wolf (1997). Interacting phenotypes and the evolutionary process: I. Direct and indirect genetic effects of social interactions. *Evolution* 51, 1352–1362.
- Muniz DG and G Machado (2018). Mate sampling influences the intensity of sexual selection and the evolution of costly sexual ornaments. *Journal of theoretical biology* 447, 74–83.
- Nachman MW and SL Crowell (2000). Estimate of the mutation rate per nucleotide in humans. *Genetics* 156, 297–304.
- Neuenschwander S, F Hospital, F Guillaume, and J Goudet (2008). quantiNemo: an individual-based program to simulate quantitative traits with explicit genetic architecture in a dynamic metapopulation. *Bioinformatics* 24, 1552–1553.
- Nichols RA and RK Butlin (1989). Does runaway sexual selection work in finite populations? *Journal of Evolutionary Biology* 2, 299–313.
- Oddou-Muratorio S and H Davi (2014). Simulating local adaptation to climate of forest trees with a Physio-Demo-Genetics model. *Evolutionary applications* 7, 453–467.
- Oke KB, G Rolshausen, C LeBlond, and AP Hendry (2017). How parallel is parallel evolution? A comparative analysis in fishes. *The American Naturalist* 190, 1–16.
- Otto SP, MR Servedio, and SL Nuismer (2008). Frequency-dependent selection and the evolution of assortative mating. *Genetics* 179, 2091–2112.
- Paaby AB and MV Rockman (2013). The many faces of pleiotropy. *Trends in Genetics* 29, 66–73.
- Parker GA, RR Baker, and VGF Smith (1972). The origin and evolution of gamete dimorphism and the male-female phenomenon. *Journal of Theoretical Biology* 36, 529–553. issn: 10958541. doi: 10.1016/0022-5193(72)90007-0.
- Parter M, N Kashtan, and U Alon (2007). Environmental variability and modularity of bacterial metabolic networks. *BMC evolutionary biology* 7, 169.



- Peng B and M Kimmel (2005). simuPOP: a forward-time population genetics simulation environment. *Bioinformatics* 21, 3686–3687.
- Pigliucci M (2008). Is evolvability evolvable? *Nature Reviews Genetics* 9, 75–82.
- Piou C and E Prévost (2012). A demo-genetic individual-based model for Atlantic salmon populations: Model structure, parameterization and sensitivity. *Ecological Modelling* 231, 37–52.
- Plomion C, JM Aury, J Amselem, T Leroy, F Murat, S Duplessis, S Faye, N Francillonne, K Labadie, G Le Provost, et al. (2018). Oak genome reveals facets of long lifespan. *Nature Plants* 4, 440–452.
- Pomiankowski A and Y Iwasa (1998). Runaway ornament diversity caused by Fisherian sexual selection. *Proceedings of the National Academy of Sciences* 95, 5106–5111.
- Ramsey DM (2011). Mutual mate choice with multiple criteria. In: *Advances in dynamic games*. Springer, pp. 337–355.
- Rintamäki PT, RV Alatalo, J Höglund, and A Lundberg (1995). Mate sampling behaviour of black grouse females (*Tetrao tetrix*). *Behavioral Ecology and Sociobiology* 37, 209–215.
- Ritchie MG (2007). Sexual Selection and Speciation. *Annual Review of Ecology, Evolution, and Systematics* 38, 79–102. issn: 1543-592X. doi: 10.1146/annurev.ecolsys.38.091206.095733.
- Rittschof CC and GE Robinson (2014). Genomics: Moving behavioural ecology beyond the phenotypic gambit. *Animal Behaviour* 92, 263–270. issn: 00033472. doi: 10.1016/j.anbehav.2014.02.028.
- Roff D and T Mousseau (1999). Does natural selection alter genetic architecture? An evaluation of quantitative genetic variation among populations of *Allenomobius socius* and *A. fasciatus*. *Journal of Evolutionary Biology* 12, 361–369.
- Romero-Mujalli D, FJeltsch, and R Tiedemann (2019). Individual-based modeling of eco-evolutionary dynamics: state of the art and future directions. *Regional Environmental Change* 19, 1–12.
- Sasaki A and U Dieckmann (2011). Oligomorphic dynamics for analyzing the quantitative genetics of adaptive speciation. *Journal of mathematical biology* 63, 601–635.
- Schwartz AK and AP Hendry (2007). A test for the parallel co-evolution of male colour and female preference in Trinidadian guppies (*Poecilia reticulata*). *Evolutionary Ecology Research* 9, 71–90.
- Shaw FH, RG Shaw, GS Wilkinson, and M Turelli (1995). Changes in genetic variances and covariances: G whiz! *Evolution* 49, 1260–1267.
- Sinervo B and E Svensson (2002). Correlational selection and the evolution of genomic architecture. *Heredity* 89, 329–338.
- Smith M (1976). the theory of games: In situations characterized by conflict of interest, the best strategy to adopt depends on what others are doing. *Am. Sci* 64, 41–45.
- Steppan SJ, PC Phillips, and D Houle (2002). Comparative quantitative genetics: evolution of the G matrix. *Trends in Ecology & Evolution* 17, 320–327.



- Tazzyman SJ and Y Iwasa (2010). Sexual selection can increase the effect of random genetic drift—A quantitative genetic model of polymorphism in *Oophaga pumilio*, the strawberry poison-dart frog. *Evolution: International Journal of Organic Evolution* 64, 1719–1728.
- Wakano JY and Y Iwasa (2013). Evolutionary branching in a finite population: deterministic branching vs. stochastic branching. *Genetics* 193, 229–241.
- Wakano JY and L Lehmann (2014). Evolutionary branching in deme-structured populations. *Journal of theoretical biology* 351, 83–95.
- Walsh B and MW Blows (2009). Abundant Genetic Variation + Strong Selection = Multivariate Genetic Constraints: A Geometric View of Adaptation. *Annual Review of Ecology, Evolution, and Systematics* 40, 41–59. issn: 1543-592X. doi: 10.1146/annurev.ecolsys.110308.120232. arXiv: 1132.
- West-Eberhard MJ (1983). Sexual selection, social competition, and speciation. *The Quarterly Review of Biology* 58, 155–183.
- Wilbur HM and VH Rudolf (2006). Life-history evolution in uncertain environments: bet hedging in time. *The American Naturalist* 168, 398–411.
- Wilkinson GS, F Breden, JE Mank, MG Ritchie, AD Higginson, J Radwan, J Jaquiere, W Salzburger, E Arriero, S Barribeau, et al. (2015). The locus of sexual selection: moving sexual selection studies into the post-genomics era. *Journal of evolutionary biology* 28, 739–755.
- Wolf JB, ED Brodie III, and AJ Moore (1999). Interacting phenotypes and the evolutionary process. II. Selection resulting from social interactions. *The American Naturalist* 153, 254–266.

Appendix

A1. The model

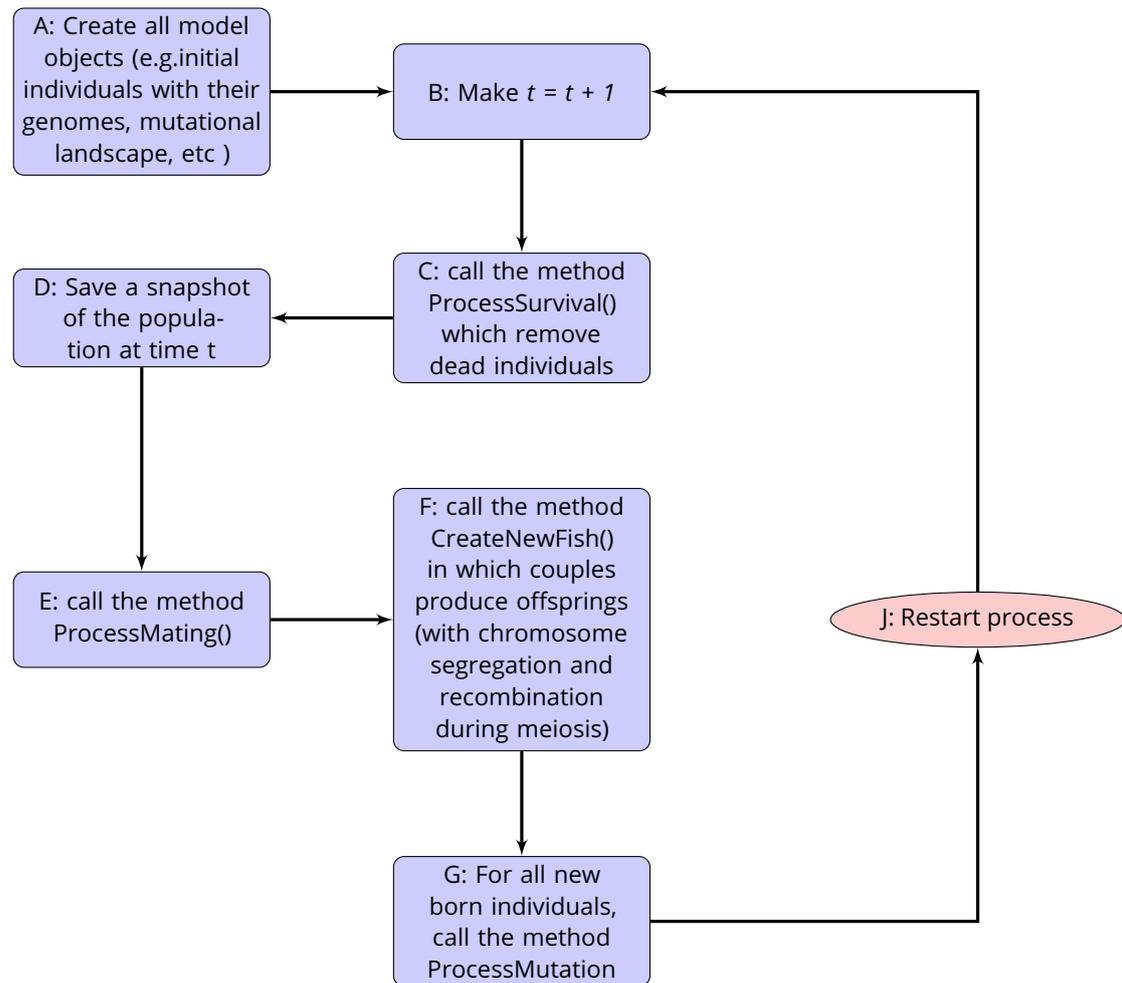
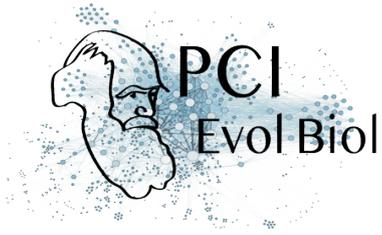


Figure A1. Flowchart showing the order of the processes implemented in the model.



A2. calculation of the allelic correlation index by bootstrap

$$A2SSD = \sum_{n=l_0}^{n=l_x} [(G_n - P_n)^2]$$

with G_n and P_n the mean allelic values at loci n for G and P and x the number of loci. SSD is the sum of square differences between allelic values for the traits at every loci. The pleiotropy index corresponds to the rank of the sum of the squared difference in the distribution of the sum of the squared differences calculated by bootstrap.

A3. Evolutionary rate in Haldanes

We measure evolutionary rate in Haldanes using the same formula as Hendry and Kinnison (1999):

$$A3h = \frac{\frac{x_2 - x_1}{s_p}}{g}$$

where x_2 and x_1 represent mean trait values for the single population at two different times, s_p is the pooled standard deviation $s_p = \frac{(n1-1)*(sd_{log(x1)})^2 + (n2-1)*(sd_{log(x2)})^2}{(n1-1)+(n2-1)}$, and g is the number of generations between the two different times.

A4: supplementary figures

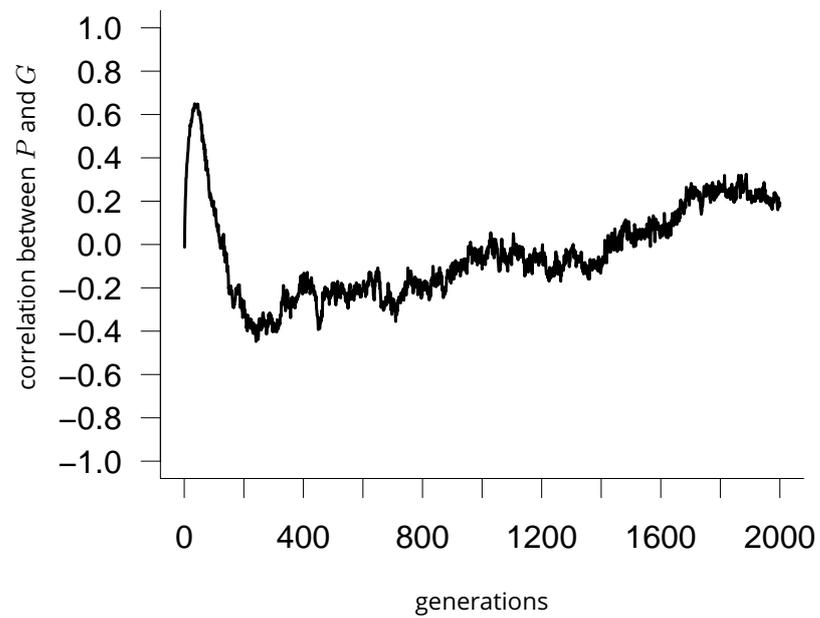


Figure A4. Individual genetic correlation between G and P values in the population, for one simulation of the preference driven mating system ($G + P$).

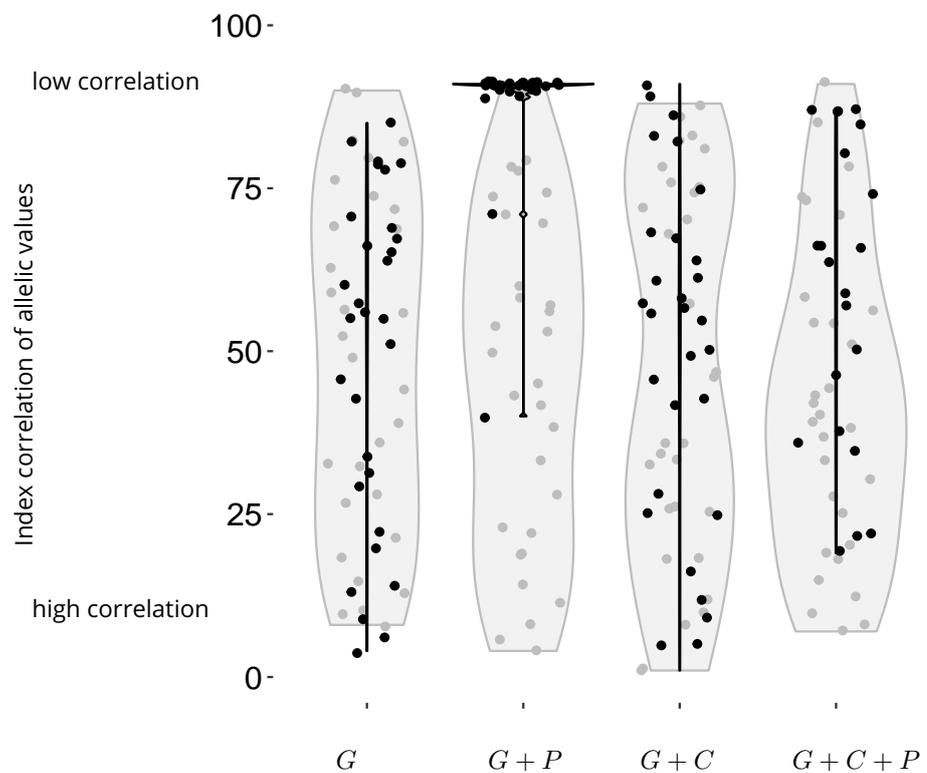


Figure A5. Index correlation of allelic values for *G* and *P* with a uniform distribution of allelic values. The measures are showed initially (in grey) and after evolution over 5000 time step (in black) for 30 replications for each mating system (random encounter without preference, random encounter and preference, competitive encounter without preference, competitive encounter and preference). Greyed areas indicate density probability for the distribution of points in the variation range. The mutational landscape follows an uniform distribution.

Additional method

C1. The model

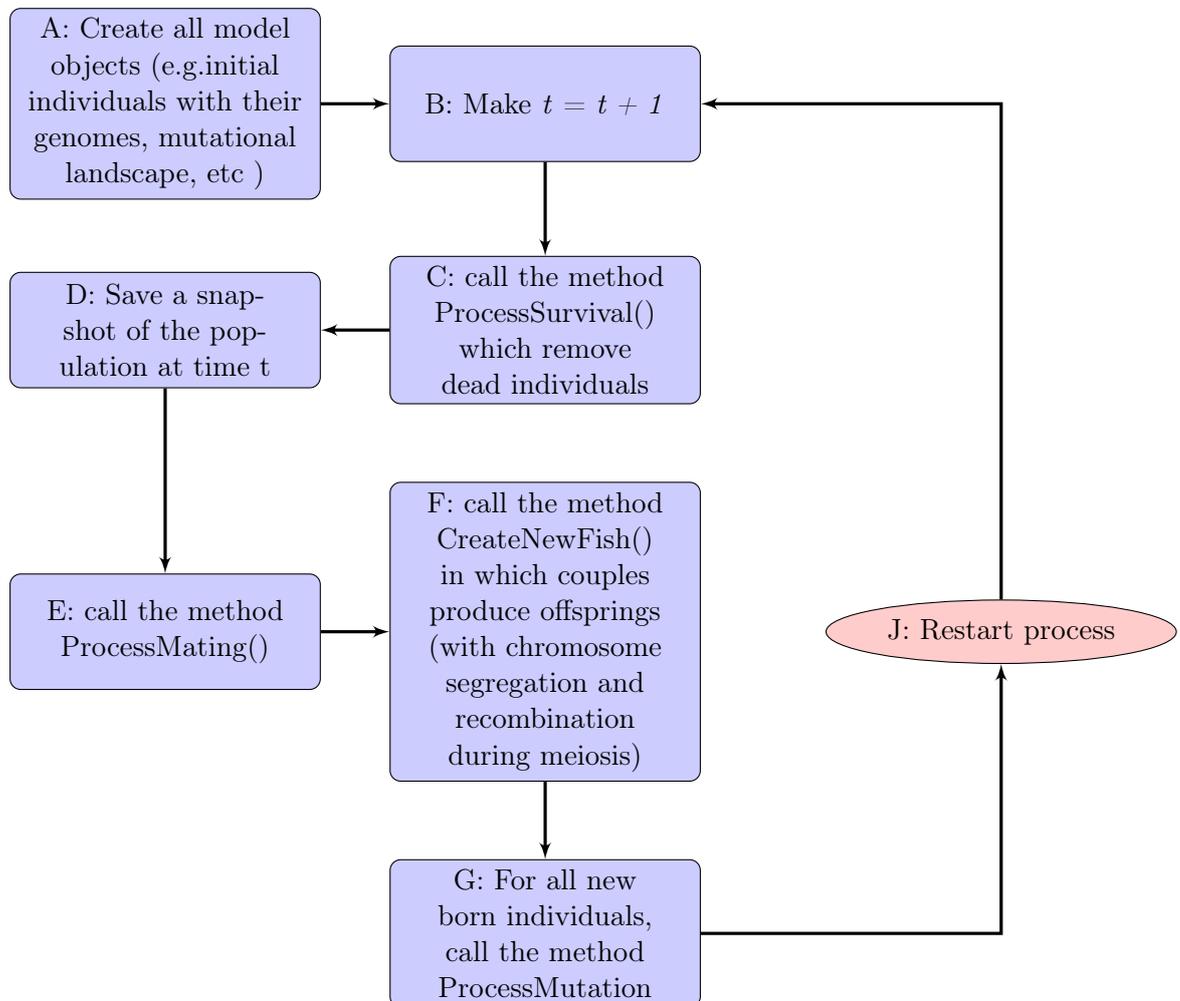


Figure C1: Flowchart showing the order of the processes implemented in the model.

C2. calculation of the allelic correlation index by bootstrap

$$SSD = \sum_{n=l_0}^{n=l_x} [(G_n - P_n)^2] \quad (\text{B2})$$

with G_n and P_n the mean allelic values at loci n for G and P and x the number of loci. SSD is the sum of square differences between allelic values for the traits at every loci. The pleiotropy index corresponds to the rank of the sum of the squared difference in the distribution of the sum of the squared differences calculated by bootstrap.

C3. Evolutionary rate in Haldanes

We measure evolutionary rate in Haldanes using the same formula as Hendry and Kinnison (1999):

$$h = \frac{\frac{x_2 - x_1}{s_p}}{g} \quad (\text{B3})$$

where x_2 and x_1 represent mean trait values for the single population at two different times, s_p is the pooled standard deviation $s_p = \frac{(n1-1)*(sd_{log(x1)})^2 + (n2-1)*(sd_{log(x2)})^2}{(n1-1)+(n2-1)}$, and g is the number of generations separating the two different times.

B4.index of allelic correlation with a uniform mutational landscape

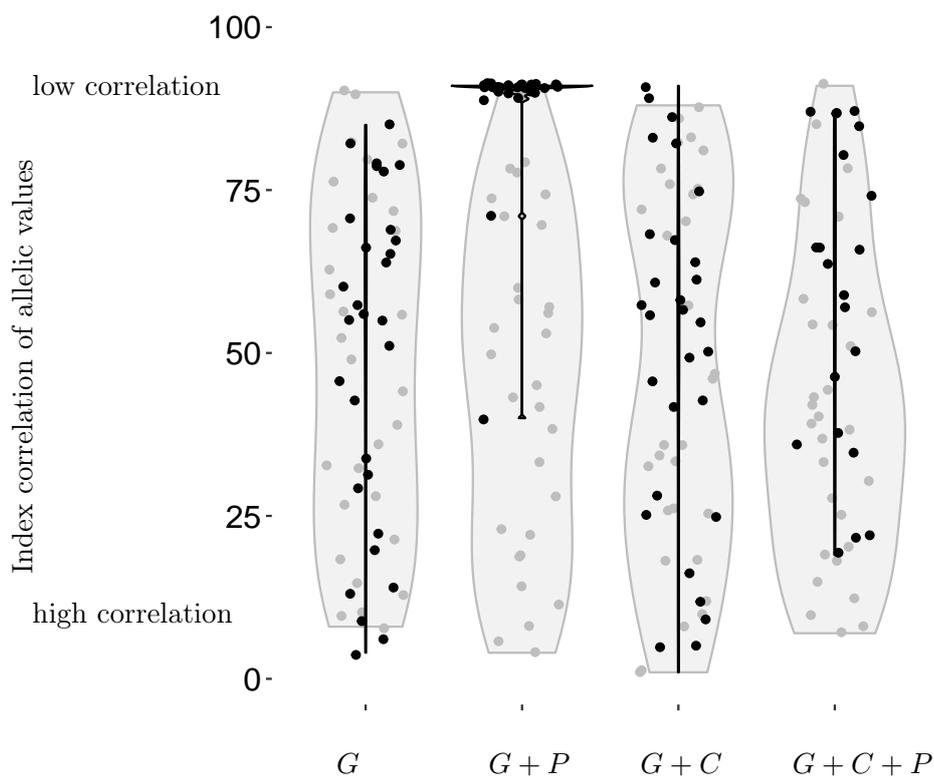


Figure C4: Index correlation of allelic values for G and P of the mean genome in the population. The measures are showed initially (in grey) and after evolution over 5000 time step (in black) for 30 replications for each mating system (random encounter without preference, random encounter and preference, competitive encounter without preference, competitive encounter and preference). Greyed areas indicate density probability for the distribution of points in the variation range. The mutational landscape follows an uniform distribution.

List of Figures

1.1	: The different descriptors of the preference function. Left panels show an unimodal shape of the preference function (intermediaire traits values being the most attractive). Right panels show a directional shape of the preference function (maximum trait values being the most attractive). The tolerance, strength and responsiveness are sometimes called "choosiness". From [Kilmer <i>et al.</i> 2017]	6
2.1	Example of hypothetical curves representing the trade-offs between current reproductive effort and expectation of future offspring at any particular instant (or age). Concave curve (in red) leads to only one reproductive event (semelparity), convex one (blue) result in repeated reproduction (iteroparity). Dots mark the reproductive tactics that maximize the lifetime reproductive success. Modified from [Pianka 1976].	26
2.2	An illustration of the optimization approach used to calculate the optimal choosiness. The newly calculated choosiness strategies are used to calculate the new distributions of partner qualities across the season that, in turn, would lead to new adjustments of the choosiness strategies.	32

- 2.3 Optimal choosiness for females and males as functions of time and individual quality, in three mating systems: monogamy ($\tau_{\sigma} \gg T$, and $\tau_{\varphi} \gg T$), polygynandry ($\tau_{\varphi} \ll T$, and $\tau_{\sigma} \ll T$, polygyny ($\tau_{\sigma} \ll T$, and $\tau_{\varphi} \gg T$). Optimal choosiness is measured as the partner quality that an individual is ready to accept with at least a probability of 0.5. Thick black lines indicates High Quality (HQ, $q = 75$), Intermediate Quality (IQ, $q = 50$) and Low Quality (LQ, $q = 25$) individuals. 34
- 2.4 Final mating pattern. For a given quality of male and female, the size of the points is proportional to the frequency of pairs formed during the breeding season. r indicates the degree of assortative mating measured as the pearson coefficient correlation. 35
- 2.5 The multivariate breeder's equation. 39
- 2.6 The joint evolution of female preferences and male trait. Male trait is subject to an ordinary stabilizing natural selection and to sexual selection. The population will evolve along the dotted lines (following the direction of the arrows). Depending on the genetic variance parameters, the equilibrium line (solid line) can be stable (A) or unstable (B). Unstable equilibrium is triggered when the genetic covariance exceeds a critical value determined by the intensity of female preference and the strength of stabilizing natural selection on male phenotype [Lande 1981, Arnold 1983]. 41
- 2.7 The demogenetic conceptual model, integrating population dynamic, behavioral interactions and the evolution of genetic architecture. . . 47

-
- 3.1 survival probability as a function of population size for different values of R . with resource limitation parameter $K=10000$ 56
- 3.2 Probability that a potential partner will be accepted, as a function of the values of gametic investment of this partner for two different values of preference ($G_{pref} = 0.5$ dotted line, and $G_{pref} = 0.8$ plain line). Individuals differ in the value of G of their most preferred mate, but all individuals share the same tolerance ($\nu = 0.2$). 59
- 3.3 Example of allelic values distribution for the gametic investment (A) and consequent distribution of gametic investment trait values in the population (B). This distribution is obtained by drawing allelic values on the Beta distribution of shape parameters [0.65, 24.5]. 62
- 3.4 Schematic representation of the indexes measured to describe the genetic architecture, A) inter-loci RSD, B) index of allelic correlation. The blue and the red bars represent the mean allelic value of each locus for G and for P respectively in the population. 64
- 3.5 Evolution of G (in blue) and P (in red) in the population over 5000 time step, for one simulation of either (A) random mating , (B) preference driven mating. Mean values of traits are showed in thick lines and standard deviation values are represented in transparency. Also appear in the figure the evolution rates in Haldanes for the two traits (dashed lines) and the relationship between lifetime reproductive success (LRS) and G values at different time step during the simulation. 70
- 3.6 Genetic correlation between G and P over generations, for two mating scenarios: random mating in grey, mating preference in black. 71

-
- 3.7 Number of mated (top grey and black lines) and unmated individuals (bottom lines) after each breeding event for two mating scenarios: random mating in grey, mating preference in black 72
- 3.8 Mean number of different alleles per locus in the population for two mating scenarios: random mating in grey, mating preference in black 74
- 3.9 The mean genome in the population, at initialization ($t=0$) and after evolution over 5000 generation. The height of bars at each locus represents the mean effect of alleles at each locus for the gametic investment (bar in blue) and preference (bar in red). The mean effect of alleles at a locus being calculated by subtracting the mean allelic value of the loci for the trait in the genome from the mean allelic value at the given locus. Grey circles give the graduations for allelic effects. At the initialization, all loci have average allelic values that do not deviate from the mean. After evolution some locus have strong (positives or negatives) effect for one or both of the traits . . . 75
- 3.10 The Lifetime reproductive success of individuals as a function of their allelic correlation for G and P at time step $t=50$, for two mating scenarios: A) Random mating, B) Preference driven mating. 76
- 3.11 Mean traits values in the population for G and P for 30 simulations with two mating scenarios, random mating (P is not expressed) and mating preference. Left panels show the evolution thought time, right panel show boxplots of the mean traits values in the simulations initilly (in grey) and at the final states (in colors). 79

- 3.12 Mean values of gametic investment, preference and competitiveness, initially (in grey) after evolution over 5000 time step (in color). Four mating systems simulated : random mating (G), random encounter and preference ($G + P$), competitive encounter without preference ($G + C$), competitive encounter and preference ($G + C + P$). Each dot represents the main trait value in a simulation. A boxplot indicates the 2.5, 25, 50, 75 and 97.5 quantiles of the distribution among simulations. 80
- 3.13 Mean population size in the different simulations after evolution over 5000 time step. Boxplots indicate the 2.5, 25, 50, 75 and 97.5 quantiles of the distribution among replications. Four mating systems are simulated : random mating (G), random encounter and preference ($G + P$), competitive encounter without preference ($G+C$), competitive encounter and preference ($G + C + P$). 81
- 3.14 The relative standard deviation of the mean allelic values between loci for the different traits present in the scenarios (G , P , C , in the first scenario the preference is not expressed), initially (in grey) and after evolution over 5000 generations (in color). The boxplots are drawn with 30 simulation of the four mating routines (random encounter, preference driven mating, competitive encounter, competitive encounter and preference driven mating) and report the mean and 95% interquartile range interval. 84
- 3.15 Index correlation of allelic values for the pairs of traits. The measures are showed initially (in grey) and after evolution over 5000 time step (in color) for 30 replications for each mating system (random mating, random encounter and preference, competitive encounter without preference, competitive encounter and preference). The boxplots report the mean and 95% interquartile range interval. 84

-
- 3.16 The effect of the parameters influencing the trade-off reproduction-survival on evolution. The boxplots show values of representative variable at the pseudo equilibrium for 30 simulations. the variables shown are the mean trait values in the population, their standard deviation, the correlation between G and P , the populations size and the mean probability of survival in the population. 89
- 3.17 The effect of genetic parameters on evolution. The boxplots show values of representative variable at the pseudo equilibrium for 30 simulations. the variables shown are the mean trait values in the population, their standard deviation, the correlation between G and P , the populations size and the mean probability of survival in the population. 90
- 3.18 The effect of preference shape parameters on evolution. The boxplots show values of representative variable at the pseudo equilibrium for 30 simulations. the variables shown are the mean trait values in the population, their standard deviation, the correlation between G and P , the populations size and the mean probability of survival in the population. 91

- 4.1 Percentage of unmated individuals (i.e., that fail to reproduce during a breeding event) for different mating scenarios and mating group size. The plot show the mean (dark line) and SD values (grey filling around the line) calculated over the 30 replication of simulations conducted for each mating scenario and each mating group size. The different panels correspond to different mating routines: (A) random mating , (B) preference driven mating, (C) competitiveness driven mating, (D) competitiveness and preference driven mating; and the shades of grey correspond to the with mating group sizes (from light gray to dark gray: $MG = N$, $MG = 300$, $MG = 100$, $MG = 30$) . In the scenario of competitive encounter with preference and $MG = 30$, in all the simulation the population goes extinct after 4500 generations. 104
- 4.2 Mean values of reproductive traits at the pseudoequilibrium, for the 4 mating routines and for different mating group sizes. 30 replications are conducted for each scenario. 108
- 4.3 Genetic correlation between G and P in simulations where mating is driven by preference with different mating group size. The boxplots show the correlation in the 30 different simulations at different times step, the lower panel show the evolution of the correlation for 3 simulations each with a different MG 109
- 4.4 The evolution of the mean value of competitiveness (and sd) over generations. 110

- 4.5 Lifetime Reproductive success of individuals as a function of their competitiveness at two different time steps. At $t = 10$, higher values of competitiveness clearly provides higher reproductive success (directional selection), which explains why the distribution of the trait in the population is shifted from the higher values at time $t = 100$. From this time, a stabilizing selection is observed on the Competitiveness. 110
- 4.6 Index correlation of allelic values for the traits taken two by two. The measures are showed initially (in grey) and after evolution over 5000 time step (in color). The boxplot are drawn with the indexes calculated for 30 simulations of the four mating routines (random encounter, preference driven mating, competitive encounter, competitive encounter and preference driven mating) and for different mating group sizes ($MG = N$, $MG = 300$, $MG = 100$, $MG = 30$). A high index (100) signals a mean allelic correlation that is more negative than what could be observed by chance in the population (i.e. modularity). Conversely, an index close to 0 signals a mean allelic correlation more positive than what could be observed by chance in the population. 30 replications are conducted for each scenario. In the scenarios with some extinction risk (but which do not crash all the time), I conduct more simulations in order to get 30 simulations that do not crash. 113

-
- 4.7 The relative standard deviation of the mean allelic values between loci for the different traits present in the scenarios (G, P, C, in the first scenario the preference is not expressed), initially (in grey) and after evolution over 5000 generations (in color). The boxplots are drawn with 30 simulation for the 4 mating systems scenarios (random encounter, preference driven mating, competitive encounter, competitive encounter and preference driven mating) and for different mating group sizes. The boxplots report the mean and 95% interquartile range interval. 114
- 4.8 Relation between Lifetime reproductive success and individuals' allelic correlation, for the preference driven mating scenario and two mating group size, at two time step of the simulation ($t=10$, $t= 50$, and $t=100$). negative correlation correspond to modular architecture 115
- 5.1 Mean values of reproductive traits for the 4 mating routines (random mating G , preference driven mating $G - P$, competitiveness driven mating $G - C$, and competitiveness and preference driven mating $G - P - C$) and for different genetic maps. 125
- 5.2 Mean genetic correlations between gametic Investment G and preference P over 5000 generations (black line, and standard deviation around the mean, grey area) for 30 replications of the three genetic maps (10 loci on 10 chromosomes, 10 loci on 2 chromosomes and 100 loci on 100 chromosomes). 127

- 5.3 The relative standard deviation of the mean allelic values between loci for the different traits present in the different scenarios, initially (in grey) and after evolution over 5000 generations (in color). The boxplots are drawn with 30 simulations of the four mating routines (random encounter, preference driven mating, competitive encounter, competitive encounter and preference driven mating) and for the different genetic maps, and report the mean and 95 % interquartile range interval. 128
- 5.4 Index correlation of allelic values (calculated with a bootstrap approach) for traits for the different genetic maps. The boxplot are drawn with the indexes calculated for 30 simulations of the four mating routines (random encounter, preference driven mating, competitive encounter, competitive encounter and preference driven mating) and for the different genetic maps. A high index (10000 or 100) signals that the mean allelic correlation is more negative than what could be observed by chance in the population (i.e. modularity). Conversely, an index close to 0 signals a mean allelic correlation more positive than what could be observed by chance in the population. 129
- 5.5 Evolution of G for males (in dark blue) and females (in blue) and P for males (in dark red) and females (in red) in the population over 5000 time steps, for one simulation of either (A) random mating, (B) preference driven mating. Mean values of traits are showed in thick lines and standard deviation values are represented in transparency. 132

-
- 5.6 The mean allelic effects per loci in the population, on the autosomes and on the sexual chromosomes (wherein loci are linked). The height of bars at each locus represents the mean effect of alleles at each locus for the gametic investment (bar in blue) and preference (bar in red). Grey circles give the graduations for allelic effects. Alleles on sexual chromosomes are only expressed on either males or females. 133
- 6.1 A schematic illustration of feedbacks between mating behaviours, demography and genetic architecture with the mating scenario of preference driven mating 140
- C1 Flowchart showing the order of the processes implemented in the model. 245
- C4 Index correlation of allelic values for G and P of the mean genome in the population. The measures are showed initially (in grey) and after evolution over 5000 time step (in black) for 30 replications for each mating system (random encounter without preference, random encounter and preference, competitive encounter without preference, competitive encounter and preference). Greyed areas indicate density probability for the distribution of points in the variation range. The mutational landscape follows an uniform distribution. 247

List of Tables

2.1	Summary of the main phenotypic assumptions of various evolutionary modelling approaches.	48
2.2	Summary of the main genetic assumptions of various evolutionary modelling approaches.	49