Selection in two-sex structured populations

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Publication date
2019
Document Version
Other version
License
Other

[Link to publication](#)

Citation for published version (APA):
de Vries, C. (2019). *Selection in two-sex structured populations*. [Thesis, fully internal, Universiteit van Amsterdam].

6

General Discussion

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"The fusion of population genetics with population ecology can be compared to a prearranged marriage between partners who speak different languages. Although both families agree that the marriage is advantageous, it is somewhat difficult to achieve because of cultural difference between geneticists and ecologists."

— Joan Roughgarden, *Roughgarden (1979)*

6.1 Introduction

Variation is the raw material of evolution. Without genetic variation and associated phenotypic variation, evolution would come to a screeching halt. Understanding how genetic variation is maintained in the face of selective pressures is therefore one of the most fundamental challenges in evolutionary biology.

Explanations for the maintenance of genetic variation fall roughly into two categories: neutral theories and theories of balancing selection. The neutral theory of evolution posits that genetic variation is maintained through genetic drift of neutral mutant alleles. Balancing selection, on the other hand, posits that genetic variation is maintained through selective forces that promote genetic variation, such as heterozygote advantage, or variation in selection in space, in time, between the sexes, between different life stages or between fitness components. Of course, balancing selection and neutral processes both play a role in the maintenance of genetic diversity.

Among the core results of population genetics are the criteria that determine whether selection leads to fixation of one gene at a locus, or to coexistence of multiple types in a polymorphism. Heterozygote advantage in fitness is well known to be a necessary and sufficient condition for a stable polymorphism, for viability selection, at a single locus, in diploid populations with discrete generations and random mating. However, it is equally well known that the world is populated by species with complex age- or stage-structured life cycles, subject to selection not only on viability but also on sex- and stage-specific survival, growth, development, and fertility rates throughout those life cycles.

Summary of this thesis

In this thesis we have explored how life cycle complexity affects the maintenance of genetic polymorphisms in Mendelian populations. Table 6.1 gives an overview of the conditions for a protected polymorphism under the different assumptions considered.

In Chapters 2 and 3, we derived conditions for a protected genetic polymorphism for species with linear demography. Chapter 2 introduced a females-only model, where all adult males have the same mating success, i.e., there is no sexual selection acting on males. Chapter 3 introduced a two-sex model with sexual dimorphism in any demographic rate. The two-sex coexistence conditions reduce to heterozygote superiority in population growth rate when there is no sexual dimorphism in demographic rates and the primary sex ratio is equal to one (second row of Table 6.1, sexually monomorphic population). The construction and analysis of

Table 6.1: Conditions for a protected polymorphism under different assumptions. The vector $\mathbf{c}$ is an indicator vector, with entries that show which stages take part in mating. That is, the ith entry of $\mathbf{c}$ is 1 if stage i reproduces, and 0 otherwise. The $\mathbf{D}_{AA}$ and $\mathbf{D}_{aa}$ matrices are given by equation (3.19) and (3.20).

	Linear demography	**Nonlinear demography**
Sexually dimorphic population $\mathbf{U}_i, \mathbf{U}_i'$ $\mathbf{F}_i, \mathbf{F}_i', \alpha$	$\rho\begin{pmatrix} \mathbf{U}_{Aa}+\frac{1}{2}\alpha\mathbf{F}_{Aa} & \frac{1}{2}\alpha\mathbf{D}_{AA} \\ (1-\alpha)\frac{1}{2}\mathbf{F}_{Aa} & \mathbf{U}_{Aa}'+\frac{1}{2}(1-\alpha)\mathbf{D}_{AA} \end{pmatrix} > \lambda_{AA}$	$\rho\begin{pmatrix} \mathbf{U}_{Aa}+\frac{1}{2}\alpha\mathbf{F}_{Aa} & \frac{1}{2}\alpha\mathbf{D}_{AA} \\ (1-\alpha)\frac{1}{2}\mathbf{F}_{Aa} & \mathbf{U}_{Aa}'+\frac{1}{2}(1-\alpha)\mathbf{D}_{AA} \end{pmatrix}\bigg|_{\hat{\mathbf{m}}_{AA}} > 1$
	$\rho\begin{pmatrix} \mathbf{U}_{Aa}+\frac{1}{2}\alpha\mathbf{F}_{Aa} & \frac{1}{2}\alpha\mathbf{D}_{aa} \\ (1-\alpha)\frac{1}{2}\mathbf{F}_{Aa} & \mathbf{U}_{Aa}'+\frac{1}{2}(1-\alpha)\mathbf{D}_{aa} \end{pmatrix} > \lambda_{aa}$	$\rho\begin{pmatrix} \mathbf{U}_{Aa}+\frac{1}{2}\alpha\mathbf{F}_{Aa} & \frac{1}{2}\alpha\mathbf{D}_{aa} \\ (1-\alpha)\frac{1}{2}\mathbf{F}_{Aa} & \mathbf{U}_{Aa}'+\frac{1}{2}(1-\alpha)\mathbf{D}_{aa} \end{pmatrix}\bigg|_{\hat{\mathbf{m}}_{aa}} > 1$
Sexually monomorphic population $\mathbf{U}_i = \mathbf{U}_i' \quad \forall i$ $\mathbf{F}_i = \beta\mathbf{F}_i' \quad \forall i$ $\alpha = 0.5$	$\rho\left(\mathbf{U}_{Aa}+\alpha\mathbf{F}_{Aa}\right) > \lambda_{AA}$	$\rho\left(\mathbf{U}_{Aa}(\hat{\mathbf{m}}_{AA})+\alpha\mathbf{F}_{Aa}(\hat{\mathbf{m}}_{AA})\right) > 1$
	$\rho\left(\mathbf{U}_{Aa}+\alpha\mathbf{F}_{Aa}\right) > \lambda_{aa}$	$\rho\left(\mathbf{U}_{Aa}(\hat{\mathbf{m}}_{aa})+\alpha\mathbf{F}_{Aa}(\hat{\mathbf{m}}_{aa})\right) > 1$
No differential male mating succes, no dimorphism in viability $\mathbf{U}_i = \mathbf{U}_i' \quad \forall i$ $\mathbf{F}_i' = \mathbf{e}_1 \otimes \mathbf{c}^\top \quad \forall i$	$\rho\left(\mathbf{U}_{Aa}+\frac{1}{2}\alpha\mathbf{F}_{Aa}+\frac{1}{2\mathrm{p}_b}\alpha(\mathbf{F}_{AA}\hat{\mathbf{p}}_{AA})\otimes\mathbf{c}^\top\right) > \lambda_{AA}$	$\rho\left(\mathbf{U}_{Aa}+\frac{1}{2}\alpha\mathbf{F}_{Aa}+\frac{1}{2N_b}\alpha(\mathbf{F}_{AA}\hat{\mathbf{n}}_{AA})\otimes\mathbf{c}^\top\right)\bigg|_{\hat{\mathbf{m}}_{AA}} > 1$
	$\rho\left(\mathbf{U}_{Aa}+\frac{1}{2}\alpha\mathbf{F}_{Aa}+\frac{1}{2\mathrm{p}_b}\alpha(\mathbf{F}_{aa}\hat{\mathbf{p}}_{aa})\otimes\mathbf{c}^\top\right) > \lambda_{aa}$	$\rho\left(\mathbf{U}_{Aa}+\frac{1}{2}\alpha\mathbf{F}_{Aa}+\frac{1}{2N_b}\alpha(\mathbf{F}_{aa}\hat{\mathbf{n}}_{aa})\otimes\mathbf{c}^\top\right)\bigg|_{\hat{\mathbf{m}}_{aa}} > 1$

the model were demonstrated using data on a color polymorphism in the common buzzard, *Buteo buteo*.

In Chapters 4 and 5, we construct and analyze two density-dependent models. Chapter 4 considers a case where males and females have the same stage-specific survival and transition rates, and all adult males have the same mating success. Chapter 5 introduces a two-sex model with sexual dimorphism in any linear or nonlinear demographic rate. We find that sexually dimorphic populations can evolve towards lower equilibrium densities in all stages for both sexes, making them more vulnerable to extinction as a consequence of demographic stochasticity. In the absence of sexual dimorphism, when the sex-ratio is even, and when the demographic rates are a function of one weighted sum of stages, we find that a heterozygote can only invade if its genotype-specific growth rate is larger than 1 when evaluated at the equilibrium stage abundance distribution of the resident homozygote.

In this paragraph I will attempt to give some biological intuition for the mathematical terms in Table 6.1. For all the coexistence conditions in Table 6.1, the left-hand side of the inequality is the invasion growth rate of the heterozygote, which is compared to the growth rate of the resident homozygote on the right-hand side of the inequality. During invasion of the AA boundary only two processes contribute to an increase in heterozygotes: AA females mating with Aa males (or, more accurately, randomly picking an a allele out of the gamete pool), and Aa females mating with AA males. Because AA males are much more prevalent during invasion, the probability of an Aa female picking an A allele out of the gamete pool is 1 to first order. The term $\frac{1}{2}\alpha\mathbf{F}_{Aa}$ in every condition in Table 6.1 thus represents production of new heterozygote females through Aa females mating with AA males. The term representing AA females picking an a allele out of the gamete pool *is* a function of the proportion of a alleles in the gamete pool. This term changes as our assumptions about male mating success change. It is $\frac{1}{2}\alpha\mathbf{D}_{AA}$ in the most general case (top row), it reduces to $\frac{1}{2}\alpha\mathbf{F}_{Aa}$ in the absence of sexual dimorphism, and to $\frac{1}{2\mathrm{p}_b}\alpha(\mathbf{F}_{AA}\hat{\mathbf{p}}_{AA}) \otimes \mathbf{c}^{\mathsf{T}}$ when all males have equal breeding success. Note that in a sexually monomorphic population with linear demographic rates, the invasion growth rate of the heterozygote (second column of Table 6.1) becomes independent of the population structure on the boundary, and independent of the demographic rates of the resident homozygote.

This Chapter

In this chapter I will consecutively try to answer the following questions

1. *So what? What are the implications of this thesis?*
   Sneak preview of Section 6.2: a) One scalar fitness proxy does not fit all purposes. b) Demographers should worry more about sex. c) There are many ways to go extinct when you have sex.

2. *How do these results fit into the broader context of things biologists already knew about evolutionary transitions, sex, and cooperation?*
   Sneak preview: conflict is risky, and some other wild speculations, see Section 6.3).

3. *What's next?* Sneak preview: marriage functions and assortative mating, see Section 6.4.

4. *What didn't we do?* In section 6.5 I will make two conjectures that we could not prove but that common sense and simulations suggest are true[1]. And finally we end with a brief conclusion in Section 6.6.

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[1]But we are very hopeful that some clever committee member might be able to prove our conjectures.

6.2 Implications

Fitness proxies

In classical population genetics, assuming viability selection, at a single locus, in discrete generation, random mating, diploid populations with no density-dependence, fitness, usually denoted by w, is tasked with the following three jobs:

1. to describe the growth of a population in terms of the ability of genotypes to project themselves forward in time, $\bar{w} = p^2 w_{AA} + 2pq w_{Aa} + q^2 w_{aa}$,

2. to capture all the phenotypic differences between genotypes in survival, reproduction, and development in one scalar parameter,

3. to determine the outcome of selection (heterozygote superiority in w leads to a balanced polymorphism).

It is very tempting to look for a scalar parameter in structured populations that can perform all three functions as well. However, the existence of such a general scalar measure of fitness in the context of complex life cycles and sexual reproduction seems unlikely. In our model these three different tasks are performed by three different mathematical operators:

1. $\tilde{\mathbf{A}}$ projects the population forward in time. If the population converges to a stable distribution, then the growth rate of the population per time step is $\lambda = \rho(\tilde{\mathbf{A}}(\hat{\mathbf{n}}))$. Or alternatively, $R_0(\hat{\mathbf{n}})$, describes the population growth rate per generation.

2. $\mathbf{U}_i, \mathbf{F}_i, \mathbf{U}_i', \mathbf{F}_i'$ capture the phenotypic differences between genotypes in survival, reproduction, development, and mating success.

3. ζ_{AA} and ζ_{aa} determine the outcome of selection, i.e. the invasion fitness of the heterozygote at both boundaries determines the outcome of selection.

It is regularly stated that evolution maximizes growth rates in populations without density regulation, and population density in populations that are density regulated (see for example, Engen and Sæther (2017); Lande (1982b)). We have found that both statements are wrong in the context of a Mendelian population with sexual dimorphism in demographic rates. Mylius and Metz (2004) show that evolution will only maximize anything when the environment set by the resident phenotype is one-dimensional. However, Mylius and Metz (2004) consider phenotypic evolution and do not take into account the mode of inheritance, so the connection to our results is not immediately obvious.

Outside of the classical population genetics context, fitness is generally defined as some measure of the expected number of offspring that an organism will contribute to the population, often relative to individuals of a different genotype or phenotype. In the presence of Mendelian inheritance, individuals of a genotype do not only produce offspring of their own genotype, so it is unclear how an individual's reproductive success should be measured. More generally, individual fitness always depends on the environment of an individual, e.g. on the number of competitors or predators, on resource levels, and on the availability of mates. Invasion fitness therefore appears to be the only fitness proxy that will reliably predict the outcome of selection, independent of model assumptions about density- or frequency dependence, or sexual dimorphism (see Metz (2008) for a more eloquent discussion of fitness proxies).

Life-history evolution

> *A young gardener said to his prince, "Save me! I met Death in the garden this morning and he made a menacing gesture. Tonight I wish by some miracle I could be far away, in Isfahan."*
> *The prince lent him his swiftest horse.*
> *That afternoon, walking in the garden, the prince came face to face with Death. "Why", he asked, "did you make a threatening gesture at my gardener this morning?"*
> *"It was not a threatening gesture, answered Death. "It was a gesture of surprise. I saw him far from Isfahan this morning and I knew that I must take him in Isfahan tonight."*

> — Jean Cocteau, an interpretation of a story by Jalal ad-Din Rumi.

There is no escaping death, as Rumi already knew. Theories of ageing have largely focused on how sensitive the population growth rate, λ, is to changes in female fertility and mortality schedules (see for example, Caswell and Salguero-Gómez (2013)). In doing so, these papers assume that λ is a valid fitness proxy, and that the sensitivity of λ with respect to individual parameters represent selection gradients. For a notable exception to these female-focused papers, see Tuljapurkar et al. (2007).

As discussed in the last section, the results in this thesis show that λ is only a valid fitness proxy in sexually reproducing populations without any sexual dimorphism in demographic rates, similar to results by Charlesworth (1994) using discrete differences equations for age-structured populations with no sexual dimorphism. The invasion coefficients derived in this thesis (Table 6.1) provide a more robust alternative for studying life-history evolution. If, and how much, our

understanding of life-history evolution will be impacted by moving from genotype-specific growth rates, λ_{Aa}, to invasion fitness of Aa at the AA and aa boundaries, ζ_{AA} and ζ_{aa}, remains to be seen.

One way to assess the importance of sexual dimorphism for life-history evolution is to introduce a parameter that measures the level of sexual dimorphism in the model, ϵ, and to expand the invasion coefficients, ζ_{AA} and ζ_{aa}, as a function of this parameter. For sufficiently small ϵ, the invasion conditions will be approximately equal to heterozygote superiority in genotype-specific λ_i. Investigating how fast the invasion conditions deviate from heterozygote superiority in growth rate as a function of ϵ will give an indication of how strong the effect of sexual dimorphism on life-history evolution is. However, performing a Taylor expansion on a spectral radius is likely to be a nontrivial mathematical exercise.

Life-history theory based on λ concludes that increased external mortality independent of age does not change selection gradients, and therefore cannot explain senescence, since it does not impact the relative importance of older individuals (Caswell 2007a). Increased external mortality independent of age does not effect our invasion criteria either. It would be interesting to see how ζ_{AA} and ζ_{aa} are impacted by increased external mortality on, for example, males, females, or juveniles only. To properly assess the importance of males and sexual conflict for life-history evolution in species with biparental care (like humans) or in species with female skewed adult sex ratios, we might need to think more carefully about the males, either by including a marriage function or by using an individual based modelling approach to keep track of mating pairs explicitly.

Sexual conflict

In two-sex populations, genes can live in two different types of individuals: males or females. Genes can increase their frequency in the next generation by benefitting males, females, or both. The presence of sexual dimorphism therefore allows traits to invade that benefit one of the sexes at the expense of the other sex (intralocus sexual conflict), reducing the total population growth rate or population density (for thorough reviews, see Kokko and Brooks 2003; Bonduriansky and Chenoweth 2009). [2]

Kokko and Jennions (2014) define sexual conflict as the existence of a hypothetical tool that allows individuals of sex A to alter what individuals of sex B do at a cost to sex B, and with a selective benefit to sex A. The cases of evolutionary suicide in this thesis (Section 2.2 and Section 3.5), or reduced equilibrium abundance (section 5.6), fall squarely within this definition of sexual conflict. That is,

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[2]There is a tempting analogy between dispersal models of individuals living in two different patches, and genes living in two different sexes.

the autosomal gene can be thought of as a tool used by males to the detriment of females in these examples.

We found a plethora of mechanisms that could lead to deterministic extinction, although not all of these made it into a paper, and therefore into the thesis yet. Examples are intralocus sexual conflict through a trade-off between increased maturation rate in both sexes and female fertility, a trade-off between male mating success and female fertility, between survival (of all stages or of specific stages) in both sexes and female fertility. These trade-offs all have in common that both sexes get a benefit, but only one of the sexes pays a price for that benefit, namely the females. The same trade-offs seem to lead to evolutionary suicide if only males get the benefit, and females pay the price without receiving the benefit, which is a more traditional example of intralocus sexual conflict.

Trade-offs between male survival or maturation and male mating success cannot lead to evolutionary suicide in our model, unless the gene also differentially affects females. Male parameters only affect the population growth rate indirectly in our model, i.e., male parameters affect the genotype frequencies in females, which affects the population growth rate.

We have not been able to find regions of parameter space where a trade off between female survival and female fertility leads to evolutionary suicide either. If the gene only affects females, then the outcome of invasion is determined by the performance of the invading females relative to resident females. Succesful invasion therefore implies the invading females genuinely produce more offspring per time unit than the resident females during invasion, either due to their higher survival, or due to higher reproductive rates. This verbal argument might provide some intuition as to why trade-offs within one sex cannot lead to evolutionary suicide.

The cure and the ailment

Sexual dimorphism is both the cause of, and the solution to, intralocus sexual conflict. If the sexes are somehow locked into the exact same life-history strategy, then intralocus sexual conflict cannot happen. If this symmetry is broken, it opens up the playing field for genetic elements that benefit one sex over the other. As a consequence, mechanisms to stop the expression of genes in one of the sexes have an evolutionary advantage, thereby enhancing sexual dimorphism, and so the aforementioned intraspecific Red Queen race starts (Rice and Holland 1997; McDaniel 2005; Harano et al. 2010).

The impact of life-cycle complexity on this Red Queen race has received little attention so far (Bonduriansky et al. 2008). For example, how does increased external mortality impact the strength of sexual selection? Bonduriansky et al.

(2008) hypothesize that increased external mortality will lead to increased sexual selection. This hypothesis could be tested in our framework by testing whether increased external mortality increases the sensitivity of the invasion coefficients, ζ_{AA} and ζ_{aa}, to male mating success. It seems like external mortality will not affect the sensitivity of the invasion conditions in the case of linear demographic rates, but it might affect the sensitivity when the demography is nonlinear. Furthermore, it would be interesting to investigate how an external mortality that only affects some subset of the population, e.g., only females, or only reproducing individuals, affects the strength of sexual selection as defined by the sensitivity of the invasion conditions to changes in male mating success.

Sexual conflict and genetic diversity

The case study in Section 3.5 of Chapter 3 suggests that sexual conflict leads to stable polymorphisms for wide ranges of parameter space, thereby increasing genetic diversity. Experimental studies in red deer (Foerster et al. 2007), and in *Drosophila* provide empirical support for this idea (Chippindale et al. 2001). Rice and Chippindale (2001) suggest this is more likely to happen for alleles with large effect, basing their argument on heterozygote advantage. When the effect of the allele on males and females is identical ($a = 1$ in Section 3.5), we found that there is a protected polymorphism for any strength of the allelic effect (that is, for any value of β). When the allelic effect is stronger on females than on males ($a < 1$), we find that indeed coexistence only happens above a certain minimum value of β, see Figure 3.2.

6.3 Speculations on survival of the fittest or of the most cooperative?

A recent estimate of the number of eukaryotic species on earth put it around ~ 8.7 million ($\pm$ 1.3 million SE, Mora et al. (2011)). All of these eukaryotes are hypothesized to have evolved from a symbiotic association between prokaryotic cells with the ancestor of eukaryotes (endosymbiosis). As Hartman (1984) writes, "The origin of the eukaryotic cell may tell us why *it* rather than its prokaryotic relative evolved into the metazoans who are reading this paper."

The origin of eukaryotes, sex, multicellularity, and social groups all involve a transition from entities that were capable of independent replication before the transition to entities that can only reproduce as part of a larger whole after the transition (Szathmáry and Smith 1995; Griesemer 2001). Evolutionary transitions therefore all involve cooperation between these lower level entities. But of course the component entities of the larger whole have different interests, and so the new
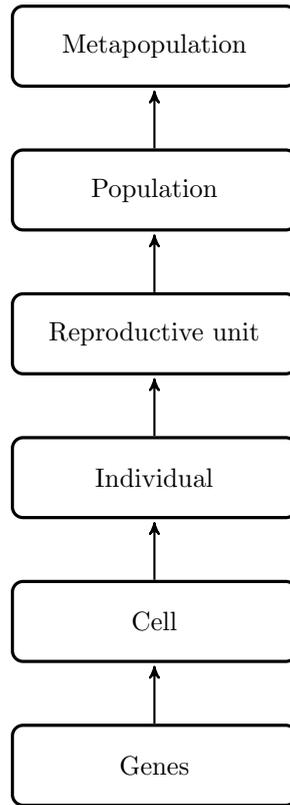
Figure 6.1: Biological levels of organisation. The term reproductive unit refers to the number of individuals required for reproduction, e.g. the mating pair in sexually reproducing species, the colony in eusocial species, and the individual in asexually reproducing organisms. Of course the levels that get incorporated in such a figure are a bit arbitrary and depend on the specific study system.

cooperative whole is susceptible to cheating unless mechanisms evolve that alter the genetic interests of component entities in the direction of cooperation. From this perspective, cancer can be seen as a breakdown of multicellular cooperation (Díaz-Muñoz et al. 2016). And sexual conflict can be seen as the breakdown of cooperation between the sexes.

Risky strategies

> *"Gentle Citizens,*
> *nature does not err, but it loves its little joke:*
> *please note the laughably small head –*
>
> *Ladies, Gentlemen,*
> *a head this size does not have room for foresight,*
> *and that is why its owner is extinct –"*
>
> — From "Dinosaur Skeleton" by Wisława Szymborska

Every major evolutionary transition adds a new level of biological organization to the existing order, see Figure 6.1. Although it is a highly debated subject, many biologists argue that selection can operate at these different levels: from genes to individuals to populations (Okasha 2006). Selection at a lower level can promote genes that do not benefit higher levels (or in view of the previous section, cooperation between the lower units can break down). The most striking example of this are selfish genetic elements, which encode traits beneficial to their own transmission at the expense of fitness costs to the organisms carrying them (Werren et al. 1988; Hatcher 2000).

I define a selfish strategy as a strategy that benefits a lower level at the expense of one or more higher levels. Selfish strategies are risky strategies, since lower levels can no longer reproduce independently. Reducing the fitness of the level above you could therefore eventually lead to your own extinction. For example, cancer cells increase rapidly initially but risk dying if they kill their host. Similarly, the examples of sexual conflict in this thesis allow a gene to increase in frequency, but eventually the gene goes extinct if it leads to evolutionary suicide. Similarly, in the density-dependent case, selfish strategies invade, but as these strategies lower population size, or metapopulation size, they increase the risk of extinction as a consequence of demographic stochasticity. Constable et al. (2016) give a nice demonstration of a similar mechanism in yeast. The burden imposed by selfish strategies on population size or growth rate can be considered as a kind of "conflict load", analogously to the genetic load imposed by migration.

Evolutionary whack-a-mole or Red Queen?

Game theory teaches us that there is nothing to stop selfish strategies from arising and invading regularly. However, a (meta)population that has been invaded by a selfish strategy will have a lower growth rate or equilibrium size than before the selfish invasion, and is therefore more likely to go extinct. Maybe selfish genotypes pop up like moles in a whack-a-mole game as evolution random walks through the

space of all possible genotypes, but eventually these selfish strategies get pushed down again by stochastic fluctuations? This "whack-a-mole hypothesis" predicts that risky or selfish strategies should be more common in regions with relatively little stochastic environmental fluctuations, since the frequency with which the risky strategies are whacked over the head is lower in more stable regions.

However, selfish strategies make victims, which means there is selection pressure on the losing side to evolve mechanisms that prevent or reduce the impact of selfish strategies, leading us back to the Red Queen hypothesis. For example, when males evolve to harrass females, which lowers female survival and fecundity, females evolve to look like males to avoid being harrassed, e.g., in damselflies Cordero et al. (1998). Similarly, in response to alleles that cheat by changing the meiotic process to favor the transmission of themselves over other alleles (meiotic drive), driver suppressor alleles evolved to stop this (Núñez et al. 2018). The question then becomes a matter of time scales: does the Red Queen run fast enough to prevent selfish strategies from lowering population sizes, or does she get whacked over the head by a stochastic event whilst trying to catch up?

Implications for the demography of eusociality

A neat example where conflict might have influenced life-history evolution are eusocial organisms. Eusocial species tend to live almost an order of magnitude longer than closely related solitary species. Naked mole rats (*Heterocephalus glaber*) can live up to 30 years, for example. Most of the research into the demography of eusociality has focused on the life-cycle of the reproductive caste, e.g. the queen bee. The results in this thesis emphasize that when reproduction requires cooperation, conflict is always looming in the background. Genes in sexually reproducing species with two sexes live in two different kinds of hosts/environments (except for the y-chromosome in species with xy sex-determination, and the w in zw sex-determination). Genes in eusocial animals live in as many different hosts as there are specialized behavorial groups or casts (depending a bit on the specifics of the genetic system of the species). Genes can invade that benefit one type of host at the expense of the other type(s) of host. To understand the life-history evolution of eusocial species therefore requires modeling the ecology and evolution of all the different castes together.

6.4   Extensions

Extension: periodic environments

Selection pressures can change with the seasons, for both sexes or for one of the sexes. A nice example of this occurs in the leopard frogs in the United States,

which have a polymorphism at one locus with a dominant allele that leads to unspotted frogs. Merrell and Rodell (1968) found that the unspotted type was better at surviving the winter, and they suggest that the polymorphism might be maintained through seasonal differences in the direction of selection. Similarly, Barrett et al. (2008) found that an allele affecting the number of armor plates in threespine sticklebacks had increased growth rates and higher overwinter survival, but decreased in frequency over summer. This type of periodic selection could easily be modeled in our framework by using periodic matrix models, see Chapter 13 in Caswell (2001).

Extension: marriage functions

We have assumed female demographic dominance throughout this thesis, which means we have assumed that there are always enough males to mate with, so that female fertility is not limited by mate availability, and that the number of offspring produced in a mating is not affected by the stage or genotype of the male. The first assumption is violated if male mortality is much higher than female mortality, and males become scarce. But mate preference can also lead to a violation of this assumption. In humans, for example, local marriage squeezes occur because males and females generally prefer to marry a partner with a similar education level, but cities often have a surplus of one sex. In the Netherlands, for example, the city of Utrecht has a surplus of educated women, whereas the city of Eindhoven has a surplus of educated men.

Extension: state-dependent female preference

Female mating preference was assumed to be independent of female age, stage, or genotype in this thesis. That is, all females were assumed to pick an allele out of the same gamete pool, independent of their i-state. For example, equation (2.11), calculates the allele frequencies in the gamete pool,

$$\mathbf{q}_b = \mathbf{W}\mathbf{p}_b, \tag{6.1}$$

which are then used to calculate the genotype distribution of the offspring of a mother with genotype i, equation (2.16). The vector of allele frequencies in the breeding pool, $\mathbf{q}_b$, is the same for females of any genotype i. Similarly, equation (3.5) in chapter 3, calculates the allele frequencies in the gamete pool,

$$\left(\begin{array}{c} q'_A \\ q'_a \end{array} \right) = \frac{\mathbf{W}'\mathbb{F}'\mathbf{p}'}{\|\mathbf{W}'\mathbb{F}'\mathbf{p}'\|} = \frac{\mathbf{W}'\mathbb{F}'\mathbf{n}'}{\|\mathbf{W}'\mathbb{F}'\mathbf{n}'\|}, \tag{6.2}$$

and all females are assumed to interact with this same gamete pool. These allele frequencies are then used to calculate the distribution of offspring of females. The

matrices $\mathbf{H}_j(\tilde{\mathbf{n}})$ for $j = 1, \ldots, \omega$, of dimension $g \times g$, assign the offspring of a mother in stage j to the genotypes. The (k, l) entry of $\mathbf{H}_j$ is the probability that the offspring of a genotype l mother, of stage i, has genotype k. In the main text, we assumed that mating is random with respect to stage and genotype of the mother, and hence that the parent-offspring map is the same for females of all stages, i.e. $\mathbf{H}_i(\tilde{\mathbf{n}}) = \mathbf{H}(\tilde{\mathbf{n}})$.

$$\mathbf{H}(\tilde{\mathbf{n}}) = \begin{pmatrix} q'_A & \frac{1}{2}q'_A & 0 \\ q'_a & \frac{1}{2} & q'_A \\ 0 & \frac{1}{2}q'_a & q'_a \end{pmatrix}. \tag{6.3}$$

But what if females of different genotypes or ages or stages differ in their mate preferences? We will discuss these two cases in the next sections.

Case 1: Assortative mating by stage

Let's assume for the moment that genotype does not affect female preference, but stage does. Let's furthermore assume that male mating success is solely determined by female choice from now on, so we assume there is no sperm competition or any other process that would lead to differential mating succes. If females of different stages differ in their mate preference, then the mother-offspring matrices are different for each stage, that is, $\mathbf{H}_j(\tilde{\mathbf{n}}) \neq \mathbf{H}(\tilde{\mathbf{n}})$ for $j = 1, \ldots, \omega$. To avoid confusion, for the rest of this section the index i will run over all i-states ($i = 1, \ldots, 2g\omega$), the indices k and l run over all genotypes ($1, \ldots, g$), and the index j runs over all stages ($1, \ldots, \omega$).

Since each female stage has a different mating preference, we need to create an effective male gamete pool for each female stage, i.e. an $\mathbb{F}'_j$ matrix for each stage j and hence effective allele frequencies in the gamete pool for each female stage j,

$$\begin{pmatrix} q'_A \\ q'_a \end{pmatrix}_j = \frac{\mathbf{W}'\mathbb{F}'_j\mathbf{p}'}{\|\mathbf{W}'\mathbb{F}'_j\mathbf{p}'\|}. \tag{6.4}$$

These effective allele frequencies then become the entries of the $\mathbf{H}_j$ matrices.

Case 2: assortative mating by genotype

Female preference may also be affected by female genotype rather than age or stage. To prevent the number of superscripts and subscripts per variable from becoming unbearable, let's assume there is no assortative mating by age as considered above. The first type of assortative mating is defined as preferentially mating with partners that we share some phenotypic characteristic with. For example, in the case of a colour polymorphism, individuals have a preference for

mating partners of the same colour morph. The second type of non-random mating behaviour considered here is a preference for mating partners that share a physical characteristic with a parent or family member.

Case 2.1: Assortative mating, "you look like me"

Assortative mating, sometimes referred to as positive assortative mating, means that individuals with a similar phenotype mate with each other more frequently than they would if mating was random. In disassortative mating, or negative assortative mating, individuals with a similar phenotype are less likely to mate with each other than they would if mating was random. Incorporating negative or positive assortative mating is mathematically very similar. If females of different genotypes have different mating preferences, they also interact with different effective mating pools. This means that the allele frequencies in each column of $\mathbf{H}$ are different.

We denote the effective frequency of allele A in the male gamete pool for a female of genotype k with q_A^k. The $\mathbf{H}$ matrix then becomes,

$$\mathbf{H}(\tilde{\mathbf{n}}) = \begin{pmatrix} q_A^1 & \frac{1}{2}q_A^2 & 0 \\ q_a^1 & \frac{1}{2} & q_A^3 \\ 0 & \frac{1}{2}q_a^2 & q_a^3 \end{pmatrix}, \tag{6.5}$$

where for each female genotype k the allele frequencies are calculated as follows,

$$\begin{pmatrix} q_A^k \\ q_a^k \end{pmatrix}_i = \frac{\mathbf{W}'\mathbb{F}'_k\mathbf{p}'}{\|\mathbf{W}'\mathbb{F}'_k\mathbf{p}'\|}. \tag{6.6}$$

Case 2.2: pedigree-based mating preferences, "you look like my mother"

Pedigree-based nonrandom mating means that an individuals' mating preferences are a function of their pedigree. This type of mating behaviour has been reported in a number of bird species. For example, *Buteo buteo* individuals generally prefer to mate with individuals of the same colour morph as their mother, apparently because they imprint on the mother who stays at the nest and distributes food delivered by the father (Krüger et al. 2001; Boerner et al. 2013). Another example of assortative mating based on familial appearances was found in the lesser snow geese, *Anser caerulescens*, by Cooke et al. (1976). Cooke et al. (1976) found that both parental color and sibling color appeared to influence mate choice in the lesser snow geese. The bird's own color, on the other hand, did not appear to be important in mate choice in either field or experimental conditions.

To model this behaviour requires incorporating an individuals' pedigree into the i-state of the demographic model. For example, in the *Buteo buteo* form of assortative mating, we have to incorporate mother's genotype into the i-state. This is a maternal effect, which is a kind of historical effect since the model has to keep a memory of who your mother was, and can therefore be incorporated similarly, see (de Vries and Caswell 2017).

Dispersal

Polymorphic dispersal strategies are found in many plant and animal species and the maintenance of such genetic variation is subject of much debate (Fronhofer et al. 2011; Saastamoinen et al. 2018). By adding a dispersal kernel to the (two-sex) structured genetic model, it might be possible to calculate (analytical) conditions for the spread of an allele which affects dispersal rate (Neubert and Caswell 2000; Miller et al. 2011).

6.5   Two conjectures

Notational warning: In this section, we follow the notation introduced in Chapter 5. That is, $\tilde{\mathbf{m}}$ denotes the entire $2\omega g \times 1$ two-sex population vector, whereas $\mathbf{n}$ denotes the female vector and $\mathbf{n}'$ denotes the male vector. The entries of $\tilde{\mathbf{m}}$ are denoted by m_i when we want to consider a general entry of the population vector, i.e. we do not want to specify the genotype or sex of the entry.

If the demographic rates in a nonlinear model are a monotonically decreasing function of one weighted sum of stages, denoted by m, then heterozygote superiority in growth rate is equivalent to heterozygote superiority in equilibrium stage abundance,

$$\bar{m}_{Aa} > \bar{m}_{AA}, \tag{6.7}$$

$$\bar{m}_{Aa} > \bar{m}_{aa}, \tag{6.8}$$

as was shown in Chapter 5. Therefore a heterozygote can only invade if its hypothetical equilibrium density, $\bar{m}_{Aa}$, is larger than both actual equlibrium densities of the two homozygotes.

This does not prove that the asymptotic equilibrium density after invasion will be larger than the two boundary equilibrium densities, however. To prove that the weighted density $\bar{m}$ will increase after an invasion requires proving that the weighted density at the coexistence equilibrium, $\bar{m}_c$, is larger than both $\bar{m}_{AA}$ and $\bar{m}_{aa}$. To the best of our knowledge, it is impossible to get a general analytical expression for the internal equilibrium. Therefore we have been unable to prove

that $\bar{m}_c > \bar{m}_{AA}$ and $\bar{m}_c > \bar{m}_{aa}$ when there is no sexual dimorphism, although both intuition and simulations suggest it is true.

Similarly, in the linear model in the absence of sexual dimorphism and when the primary sex ratio is a half, heterozygote superiority in population growth rate leads to a protected polymorphism,

$$\lambda_{Aa} \quad > \quad \lambda_{AA}, \tag{6.9}$$

$$\lambda_{Aa} \quad > \quad \lambda_{aa}. \tag{6.10}$$

The protected polymorphism converges to a stable distribution with an exponential growth or decline rate, denoted by λ_c. Since the polymorphic state contains a mixture of the three genotypes, I would expect that λ_c is bounded by the genotype-specific growth rates, i.e. $\lambda_{AA}, \lambda_{aa} < \lambda_c < \lambda_{Aa}$. Simulations suggest this is correct, but proving it is difficult because it requires relating the spectral radius of a block structured matrix to the spectral radii of its blocks. Such a relationship has only been found for a few special cases (for example, when all the blocks commute, or when they are real and symmetric).

In summary, I propose the following two conjectures:

Conjecture 1 *A protected polymorphism in a Mendelian matrix model with linear demography specified by the equations in section 3.2, with no sexual dimorphism in demographic rates and a primary sex ratio of one, will converge to a population growth rate, λ_c, which is bounded by the genotype-specific population growth rates, i.e.*

$$\lambda_{AA} < \lambda_c < \lambda_{Aa},$$

$$\lambda_{aa} < \lambda_c < \lambda_{Aa}.$$

Conjecture 2 *A protected polymorphism in a Mendelian matrix model with non-linear demography specified by the equations in section 5.5, with no sexual dimorphism in demographic rates, a primary sex ratio of one, and with demographic rates that are a decreasing function of one stage, m_i, or of one weighted sum of several stages,*

$$\bar{m} = \sum_i \beta_i m_i. \tag{6.11}$$

will have an equilibrium density of that stage, m_c, which is bounded by the genotype-specific equilibrium densities of that stage, i.e.

$$m_{AA} < m_c < m_{Aa},$$

$$m_{aa} < m_c < m_{Aa}.$$

The first conjecture implies that a succesful invasion will always lead to a larger asymptotic growth rate (in a model with density-independent demographic rates). The second conjecture implies that a succesful invasion will always lead to a larger equilibrium abundance of the stage or sum of stages that exert a density-dependent pressure.

6.6 Conclusions

Ignoring sexual dimorphism in demographic rates can lead to erroneous predictions of the trajectory of an invading allele, and potentially even of the viability of an entire population. The importance of sex for (eco-)evolutionary dynamics and extinction risk is not new (Møller 2003; Kokko and Brooks 2003), but it has rarely been considered in the context of structured population models, see (Lindström and Kokko 1998; Harts et al. 2014) for exceptions. Including population structure means including development, growth, and maturation; these are fundamental biological processes that affect both population dynamics and evolution, and are affected by both population dynamics and evolution.

The framework presented in this thesis makes genotype frequencies just one more type of demographic structure, differing from age, size, or stage structure only in the details of the reproduction process and the nonlinearity this creates. This approach to incorporating population genetics into matrix models has the potential to contribute to evolutionary demography through several routes. The detailed derivation of the model lays bare the assumptions required, and therefore simplifies the task of extending the model to relax those assumptions, for example, by including more than two alleles, nonrandom mating, mutations, or by allowing males to matter a bit more by including a marriage function. Second, the model is relatively straightforward to couple to individual data on demographic rates per sex, genotype, and age or stage. This close connection to data might allow us to investigate the following fundamental question in future: how much has intralocus sexual conflict affected life-history evolution, and how much is it likely to affect life-history evolution and population viability in future?