Joint effects of selective mating and ecology on the persistence of polymorphism
and the divergence of sexually selected traits

by

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Abstract

Classical theory suggests that sexually selected traits should have low variance, yet many species show polymorphism in these traits. To what extent does the combination of mate choice and variable ecological conditions contribute to the maintenance and persistence of sexual trait polymorphism? This is not well understood. Here I identify specific combinations of mate choice parameters and ecological factors which determine whether polymorphism in sexual traits is likely to persist or likely to result in divergent fixation and loss. For that, I build upon the null model of intersexual selection. I show that strong assortative mating makes sexual trait polymorphisms significantly more robust even against large perturbations in male-female gene frequencies. Consequently, polymorphic populations with strong assortative mating are more likely to remain polymorphic for a long time even in the face of large perturbations in gene frequencies. I show that ecologically-driven negative frequency-dependent selection interacts with selective mating to either maintain sexual trait polymorphisms or cause fixed divergence, even when mate preferences are under direct selection, classically thought to promote the loss of sexual trait polymorphism. My results explicitly show that mate choice parameters and ecological factors can interact in ways not predictable from considering either alone. The persistence of polymorphism and divergence of sexually selected traits are contingent on the details of interaction and not just on their individual effects.
Chapter 1: Thesis Overview

The aim of my thesis is to understand how mate choice interacts with ecological processes and affect the persistence of polymorphism and divergence of sexually selected traits. By ecological factors, I mean natural selection not related to mate choice. For that, I use the null model of intersexual selection (Kirkpatrick 1982; Prum 2010, 2012, 2013) as a foundation.

Sexual selection theory has produced rich diversity of mathematical models of trait and mate preference evolution. These models normally search for equilibrium values of male ornaments and female preferences and examine which conditions promote or constrain the exaggeration of sexual ornaments [see (Gavrilets 2004; Mead and Arnold 2004; Fuller et al. 2005b; Kokko et al. 2006b; Kuijper et al. 2012) for detailed review on sexual selection models]. Sexual selection models also analyse the role of mate choice in local adaptation and speciation. These models identify conditions that constrain or promote speciation [see (Kirkpatrick and Ravigné 2002; Gavrilets 2004; Servedio 2016) for detailed discussion]. However, these models do not explicitly examine how female preferences constrain or promote the persistence of polymorphic sexual traits under variable environments in various ecological contexts. Here I develop and analyse a series of haploid population genetic models that examine the robustness of sexual trait polymorphism in populations.

I use the null model of intersexual selection (Prum 2010) as a foundation for my models. Null model is based upon models by Fisher (Fisher 1915, 1958), Lande (Lande 1981) and Kirkpatrick (Kirkpatrick 1982). It shows that mate choice can exert direct sexual selection on male traits, can set up a genetic correlation between male
traits and female preferences, and can generate indirect selection on mating preferences which can then drive evolution of both traits and preferences, and produce rich evolutionary dynamics. The Fisher process (the genetic correlation between trait and preference that arises from mate choice) is an engine of trait-preference co-evolution and remains at the heart of intersexual selection models. I use the haploid version of null model (Kirkpatrick 1982) as a foundation in all models and identify conditions which determine whether polymorphism in sexual traits is likely to persist or likely to result in the divergent fixation or loss.

Firstly, I introduce known biological processes which can produce variation in the sensory perception within populations and consequently contributes to the maintenance of sexual trait polymorphisms via selective mating (Chapter 2). For simplicity, I focus only on the photoreceptors of visual systems. In Chapter 3, I examine the effects of mate choice parameters on the robustness of sexual trait polymorphisms in the face of temporary and potentially large perturbations in male-female allele frequencies. Chapter 4 and Chapter 5 examine the joint effects of ecologically-driven negative frequency-dependent selection and selective mating on the persistence of polymorphism and divergence of sexually selected traits under variable selective conditions. In Chapter 6, I study how initial conditions and ecology bias the evolutionary direction of aposematic traits in variable environments. Lastly, in Chapter 7, I discuss the implications of my results for the persistence of polymorphism, population divergence and speciation and present a general conclusion.

I will now summarize each chapter and highlight important fundamental questions.
Chapter 2: Variable sensory bias within populations and its implications for the maintenance of sexual trait polymorphisms.

Fundamental Question: Does variation in sensory systems within populations maintain sexual trait polymorphisms, and if so, how?

Mate choice can be viewed as a communication process between males and females. The sensory drive hypothesis suggests that any bias in chooser’s (usually females) sensory system can produce variation in mate preferences and can determine the direction of sexual trait preferences and evolution. Sensory bias can also contribute to the maintenance of sexual trait polymorphism within populations. However, sensory drive studies normally ignore the maintenance of variation. In this chapter, I show that known biological processes can produce variation in sensory perception and variation in sensory behaviour within populations. These biological processes can, therefore, produce variation in mate preferences which can maintain sexual trait polymorphisms within populations via selective mating. For simplicity, I focus only on the photoreceptors of visual systems.

Chapter 3: Resilience of sexual trait polymorphisms to gene frequency perturbations in variable environments.

Fundamental Question: How do mate preferences, selective parameters independent of mate preferences, and their interactions, affect the robustness of sexual trait polymorphisms in the face of large perturbations in male-female gene frequencies within populations?
This chapter examines the robustness of sexual trait diversity in the face of large random changes in male and female gene frequencies within populations. Substantial diversity is reported in sexually selected traits. To what extent mate preferences contribute to the maintenance and persistence of this diversity remains a critical question. Recent theory suggests that on their own, mate preferences can maintain sexual trait diversity for a long time (M’Gonigle et al. 2012). In this chapter, I use the null model of intersexual selection and show that the strong mate preferences and strong selective mating make sexual trait polymorphisms significantly more robust even against potentially large perturbations in male-female gene frequencies. Consequently, even in the face of large perturbations in gene frequencies, populations can remain polymorphic for a long time when assortative mating is strong within populations but not when it is weak.

I examine two models in this chapter. The first (model 3.1) is a true null model of selective mating where sexual traits are affected only by selective mating and neither sexual traits nor mate preferences are under any direct selection independent of mate choice. However, a standard null model of sexual selection usually assumes directional selection on sexual traits (Kirkpatrick 1982; Prum 2010). Consequently, in the second model (model 3.2) I add the directional selection on male traits. In both models, I examine how variable mate preferences affect the robustness of sexual trait diversity in the face of large perturbations in male-female gene frequencies.
Chapter 4: Persistence and divergence of sexually selected traits affected by frequency-dependent predation and consistent mating advantage.

Fundamental Question: How do ecologically-driven negative frequency-dependent selection (NFD) and selective mating interact and jointly affect the persistence of polymorphism and divergence of sexually selected traits?

In this chapter, I explore the joint effects of frequency-dependent predation and selective mating and identify conditions that enhance or reduce the persistence of polymorphism and divergence in sexual traits. In many cases, sexually selected traits are affected by negative frequency-dependent selection (NFD). For example, colour polymorphisms can be under frequency-dependent predation and can also show selective mating. Standard sexual selection model normally assumes directional viability selection on the sexual trait. In this chapter, I use the framework of a null model of selective mating but I assume that sexual traits are under NFD instead of directional selection. I identify threshold combinations of mate preferences in variable NFD environments that separate the maintenance and loss of sexual trait polymorphisms and set the conditions for fixed divergence in sexual traits. I also examine how NFD parameters, mate preferences, and their interactions affect the resilience of sexual trait polymorphisms. I show that the persistence of polymorphism and divergence in sexually selected traits is contingent on the relative values of mate choice parameters and NFD factors and not on their individual effects.

I examine two models in this chapter. In the first model (model 4.1), I assume that sexual traits are male-limited and therefore only males are affected by NFD. In the second model (model 4.2) I remove this assumption and consider a scenario where
expression of sexually selected traits is not sex-limited and both males and females are affected by NFD.

Chapter 5: Polymorphism maintenance and divergence of sexual traits affected by frequency-dependent predation when mate preferences are under directional selection.

Fundamental Question: How do NFD parameters and selective mating interact and maintain sexual trait polymorphisms even when mate preferences are under directional natural selection (DNS), classically thought to promote the loss of sexual trait polymorphism?

All NFD models in chapter 4 assume that mate preferences are not under any direct natural (non-sexual) selection. However, in many cases, mate preferences can be under direct selection caused by physical environment, independent of mate choice. Therefore, in this chapter, I remove this assumption and include directional selection on mate preferences in NFD models. Following on from chapter 4, I will identify threshold combinations of mate preferences that separate the maintenance and loss of polymorphisms in variable selective regimes. I examine three models in this chapter. In the first model (model 5.1) I assume that sexual traits are male-limited, only males are affected by NFD and females are affected by DNS. Later, in model 5.3, I remove this assumption and examine the scenario where the expression of sexual traits is not sex-limited, both males and females are affected by NFD and females are also under DNS. Then, I consider a scenario where the machinery underlying mate preferences is not sex-limited (model 5.2) e.g. scenarios where mate preferences are co-opted due to sensory bias. I show that when the machinery underlying mate preferences is not sex-
limited and under the selection then polymorphic populations cannot diverge in opposite directions even if polymorphic populations show extreme differences in mate preferences.

Chapter 6: Joint effects of ecology and initial conditions on the evolutionary direction of aposematic traits in variable environments.

**Fundamental Question:** How do initial conditions bias the subsequent evolutionary direction of aposematic traits in variable environments?

In this chapter, I examine the joint effects of ecologically-driven positive frequency-dependent selection (PFD), selective mating and initial conditions on the evolutionary direction of aposematic traits. For this, I use the basic framework of the null model of intersexual selection. Aposematic traits can be under PFD due to frequency-dependent predation and can also show non-random mating. As described earlier, the null model of selective mating normally assumes directional natural selection on sexual traits (model 3.2); instead, here I assume sexually selected traits are affected by PFD and consistent mating advantage. I will examine two models in this chapter. In the first model (model 6.1), I assume that aposematic traits are male-limited and therefore only males are affected by PFD. In the second model (model 6.2) I remove this assumption and consider a scenario where aposematic traits are expressed in both sexes and both males and females are affected by PFD. In this chapter, I show that in environments where assortative mating is weaker than PFD, perturbations in male frequencies are more likely to influence the evolutionary direction of polymorphic populations than perturbations in female frequencies. On the contrary, in the environments where assortative mating is stronger than PFD,
perturbations in female frequencies affect the evolutionary direction of polymorphic populations more than perturbations in male frequencies. I also show that the differences between PFD parameters cannot eliminate the effects of initial conditions in environments where assortative mating is stronger than PFD. These results are true irrespective of difference between effects of aposematic trait expression in one or both sexes.

Chapter 7: Discussion and Conclusion

In this chapter, I highlight the important results of my research and discuss their implications for the persistence of sexual trait polymorphism, population divergence and speciation. My results explicitly show that mate choice parameters and ecological factors can interact in ways not predictable from considering either alone. The persistence of polymorphism and divergence of sexually selected traits are contingent on the details of interaction and not just on their individual effects.
Chapter 2 : Variable sensory bias within populations and its implications for the maintenance of sexual trait polymorphism.

2.1 Abstract

Mate choice can be viewed as a communication process between signallers (usually males) and choosers or evaluators (usually females). Any bias in the chooser’s sensory and/or cognitive systems can bias the direction of signal evolution and determine the fate of sexual trait polymorphism within populations. Usually, previous studies explore the link between variation in sensory systems and variation in sexual traits between populations and/or between closely related species. Sensory bias is poorly explored within populations especially relative to the maintenance of sexual trait polymorphism. I hypothesize that variation in sensory systems can produce variation in sensory perception which can then produce variation in mate preferences within populations (Endler 1992). Consequently, variation in mate preferences can maintain sexual trait polymorphisms within populations via selective mating. Here I summarize biological processes which can produce variation in sensory perception and mate preferences within populations. For simplicity, I will focus only on photoreceptors.
2.2 Introduction

Variation among individuals within populations is fundamental for evolution. Therefore, it is crucial to understand mechanisms underlying the origin and maintenance of trait variation within populations. Secondary sexual traits include body size, weapons and ornamental traits like bright colours, calls, elaborate behaviours used in courtship displays for attracting mates (Andersson 1994). Secondary sexual traits are present in animals ranging from invertebrates to vertebrates (Andersson 1994) and show high genetic and phenotypic variation (Pomiankowski and Moller 1995). How does intersexual selection contribute to the maintenance of sexual trait polymorphism within populations remain a critical question.

Mate choice can be viewed as a communication process between signallers (usually males) and choosers or evaluators (usually females) (Bradbury and Vehrencamp 1998; Stevens 2013). Once we view mate choice as a communication process, it becomes obvious that the chooser’s detection and perception of signals via sensory and cognitive systems is important in influencing mating decisions. Sensory system properties and female preferences are intimately linked. Sensory systems which are biased in any physiological, ecological or evolutionary contexts other than mate choice can also favour particular female preferences and can bias the direction of signal evolution. This has been discussed repeatedly by many authors in the past which includes theories like pre-existing bias (Basolo 1990), sensory exploitation (Ryan 1990), sensory drive (Endler 1992; Boughman 2002) and sensory traps (Christy 1995). These theories demonstrate that any bias in the chooser’s sensory systems can influence mate choice, sexual selection dynamics which can determine the direction of signal evolution. Although the link between variation in sensory systems, mating
preferences, and sexual traits is established, it has only been explored between populations and/or between closely related species. We know little about this link within populations, especially relative to the maintenance of sexual trait polymorphism.

Variation in sensory systems can produce variation in mate preferences within populations and can, therefore, cause selective mating within populations. Selective mating can contribute to the maintenance of sexual trait diversity when mate preferences vary within populations (Kirkpatrick 1982; Jennions and Petrie 1997; Brooks 2002; Kingston et al. 2003; M’Gonigle et al. 2012), when choosers (generally females) show homogenous but shifting mate preferences, or when choosers show negative frequency-dependent mate choice (Eakley and Houde 2004; Kokko et al. 2007; Hughes et al. 2013). Therefore, it is important to establish a definite link between variation in sensory systems and variation in mate preferences within populations of species and specifically to ask how sensory bias would affect the maintenance of sexual trait polymorphisms within populations.

Here I summarize biological processes that can produce variation in the peripheral sensory receptors within populations which can consequently produce variation in mate preferences. For brevity, I will focus on visual systems. I will explore different ways in which photoreceptors can vary within populations. Now I will show how sensory bias can play a crucial role in the maintenance of sexual trait polymorphisms within populations.
2.3 A process deciding the fate of sexual trait polymorphism within population

Polymorphism in sensory systems can produce polymorphism in mating preferences within populations. I hypothesize that variation in the sensory system within populations can create variation in perception and thus variation in mate preferences within populations. Selective mating resulting from this process can maintain variation in sexual traits within populations as explained in the previous section. Figure 2.1 summarizes the mechanism.

![Figure 2.1](image)

Figure 2.1: A proposed process which relates intraspecific sensory system variation to the maintenance of sexual trait polymorphism within populations. Here variation should be taken as the variation among individuals within populations. Arrows indicate cause leading to effect (after Endler 1992).

In order to explore the complete dynamics of proposed hypothesis, it is important to understand the causes of sensory system variation within populations. The origin of variation will determine the heritability of variation and decide its impact over evolutionary time. For simplicity, I will focus only on colour polymorphisms and visual systems.
2.4 Colour polymorphism and variation in photoreceptors

Colour polymorphisms are ideal systems to examine sensory bias in the context of the maintenance of sexual trait polymorphisms within populations. Colour polymorphisms are common (Gray and McKinnon 2007; Wellenreuther et al. 2014). Colour polymorphic species can show individual variation in mate preferences within populations (Brooks and Endler 2001b; Brooks 2002; Morris et al. 2003) and non-random mating based on colour components (Wellenreuther et al. 2014). In such cases, variation in visual systems can potentially produce variation in colour perception, variation in mate preferences and maintain colour polymorphisms within populations. Below, I explain briefly about photoreceptors and then discuss biological processes which can produce variation in them within populations.

Photoreceptors

Vertebrate eyes carry rod and cone photoreceptor cells in the retina. Cones are responsible for colour vision in vertebrates. The visual process starts as soon as photoreceptors take in photons. Different Photoreceptor types are sensitive to different regions of the electromagnetic spectrum. Visual pigments in the photoreceptors determine its sensitivity towards the specific wavelengths. Visual pigments are composed of the protein opsin and the light absorbing chromophore. Cone opsins are categorized into different classes depending on the characteristic wavelength where they show the highest absorption ($\lambda_{\text{max}}$). Each opsin type exhibits a different $\lambda_{\text{max}}$ value which allows us to describe the entire absorption spectrum because the shape of the absorbance curve does not change with $\lambda_{\text{max}}$. In many vertebrates and some Salticid spiders, there are four classes of opsins. The opsin’s $\lambda_{\text{max}}$ vary among vertebrate
species, and one or more may be missing, for example, RH2 is absent in mammals. Bird, reptiles and some fish eyes carry coloured oil droplets in addition to visual pigments which also determine the colour sensitivity (Yokoyama 2000).

Variation in photoreceptor sensitivity can be genetic and/or environmental in origin (Fuller et al. 2005a; Horth 2007). Photoreceptors can vary in opsin genes, chromophores and/or in their relative numbers present in the retina. Figure 2.2 summarizes the biological processes which can lead to variation in photoreceptors, colour perception, and mate choice. I will now discuss how these processes can be functional within populations.

Figure 2.2: Biological processes leading to variation in photoreceptors and thereby variation in colour perception, mate preferences and maintenance of sexual trait polymorphisms. Arrows indicate cause leading to effect.
2.5 Biological processes leading to variation in photoreceptors

2.5.1 Variation in opsin genes

Variation in opsin genes can lead to individual variation in behaviour (Endler 1991; Winderickx et al. 1992). Independent studies on primate photoreceptors and behaviour support this fact (Mollon et al. 1984). Thus, molecular variation in opsins can influence individual behaviour and can also influence individual mating decisions within populations. Consequently, in such cases, polymorphism in mate preferences can maintain colour polymorphisms via selective mating.

There is MWS/LWS pigment polymorphism in the populations of new world monkeys and humans (Jacobs 1996; Kawamura et al. 2012). Most of the work explaining the link between opsin genes and behaviour has been done in vertebrates, particularly in primates (Jacobs 1996; Yokoyama and Radlwimmer 2001; Yokoyama et al. 2008; Kawamura et al. 2012). Variation in the amino acid sequence of opsin genes often controls shifts in the absorption sensitivity of the opsin. Replacement of amino acids at certain positions in the opsin can bring up to a 30nm shift in its $\lambda_{\text{max}}$ (Neitz et al. 1991). Thus, specific non-synonymous single point mutations in opsin genes can potentially create different colour vision morphs within populations. Non-synonymous mutations are also found to be more frequent than synonymous mutations in LWS opsins from cichlids of Lake Victoria (Terai et al. 2002).

Apart from primates, an extensive LWS pigment polymorphism is known in guppies Poecilia reticulata, where there are three different LWS forms in a single population with $\lambda_{\text{max}}$ ranging from 520nm to 580nm (Archer et al. 1987). Molecular analysis confirms the high genetic variation in LWS opsins within populations.
Cichlids of Lake Victoria also show high variation in LWS opsin genes within populations (Terai et al. 2002).

**Visual pigment and sexual trait polymorphism within Guppy populations**

*Poecilia reticulata* have been extensively used to study the effects of natural and sexual selection on the evolution of male colour patterns (Endler 1978, 1980). What maintains extreme colour polymorphism in guppy males is not fully understood. Polymorphism in colour patterns and vision (Archer et al. 1987) within populations in guppies provides an excellent opportunity to explore the effects of variation in visual systems on the evolution of colour patterns within populations (Endler 1991).

Male colour patterns and female preference for colour patterns both vary between populations as well as within populations (Endler 1978, 1980; Endler and Houde 1995; Brooks and Endler 2001a, b) Visual sensitivity is known to be a heritable trait in guppies (Endler et al. 2001). As mentioned earlier, individuals show significant variation in the LWS pigments. In addition, females differ in their preference for the orange spots on males which vary amongst individuals in size and chroma (Houde and Endler 1990; Brooks and Endler 2001b)

Given this scenario, it is quite possible that different females prefer males based on the visual sensitivity-colour match (Endler 1991). The extent of this matching would depend on the individual female’s LWS pigment sensitivity. Though individual females vary in their preferences within population we do not know whether it is due to individual variation in visual systems or not. Polymorphism in mate preferences
within populations can produce selective mating and thus, can maintain colour polymorphisms.

2.5.2 Environmental variability can lead to variation in photoreceptors and can produce variation in mate preferences

The sensory drive hypothesis explicitly includes the interaction between sensory systems and signaling environments (Endler 1992). Environmental conditions can vary over time and space within populations. Theory predicts a match between visual system properties and environmental variables in such a way that visual systems would be able to maximize their effectiveness in that given environment. Among species variation in visual systems is often correlated with the variation in environmental conditions (Lythgoe 1979; Partridge and Cummings 1999). Given this scenario, alternative visual microhabitats can produce disruptive sexual selection which can favour different colour morphs in different microhabitats, and consequently can maintain colour polymorphisms within populations.

In such cases, visual systems are expected to match alternative environments and shift mate preferences by shifting the visual sensitivity. In the face of environmental variability, visual spectral sensitivity can be tuned to the environment either by changing the relative frequency of cone types in the retina, or by changing the relative opsin expression in the retina. However, it is important to remember that environmental variability, along with the plasticity in opsin gene expression can maintain sexual trait polymorphisms within populations (Jennions and Petrie 1997).
Variation in spectral sensitivity by changing the relative cone frequency of cones

Spectral sensitivity can be altered by changing the relative frequency of cones in the retina. This can be achieved either by changing the opsins or by changing the chromophores. Changing the A1 chromophore to A2 derived chromophore (3-dehydroretinal) results in a higher $\lambda_{\text{max}}$ than with the A1 derived chromophore (retinal). Thus, the relative proportions of cones carrying A1 and A2 derived chromophores in the retina become important in yielding spectral sensitivity and colour perception.

Seasonal variation in the chromophore use is reported in some species like *Scardinius erythrophthalmus* (rudd) (Partridge and Cummings 1999). Migration between different environments is shown to be correlated with the shifts in the A1: A2 ratios in the retina of *Anguilla anguilla* (European eel) (Partridge and Cummings 1999). *Gasterosteous aculeatus* (threespine stickleback) individuals vary within populations in the relative proportion of cones carrying A1 and A2 chromophores. Sticklebacks living in red shifted water carry significantly more LWS/LWS double cones than individuals living in any other lighting environments (Flamarique et al. 2013). Sensory drive has been demonstrated in threespine stickleback (Boughman 2001). Thus, any variation which potentially creates variation in visual sensitivity within populations of this species becomes important in the context of signal evolution. Relative abundance of cone types is correlated with lighting conditions in *Lucania goodei* (bluefin killifish) where individuals living in more UV transmitting habitats possess more UV and blue cones than individuals living in less UV transmitted habitat (Fuller et al. 2003).

The examples listed above suggest that variable environments can produce variation in cone frequencies and variation in colour perception. In such cases,
variation in colour perception can produce variation in mate preferences which can maintain colour polymorphisms within populations.

Variation in retinal oil droplets can contribute to the variation in colour discrimination

Bird, reptiles and some fish eyes carry carotenoid based coloured oil droplets in addition to visual pigments which also determine colour sensitivity (Yokoyama 2000) and colour discrimination (Toomey et al. 2015). Dietary availability of carotenoids can influence the colour discrimination ability (Partridge 1989; Knott et al. 2010; Lim and Pike 2016).

Given this scenario, it is possible that variation in diet content and/or metabolism within species can affect oil droplet content and thus can produce variation in colour discrimination within populations. Though dietary content affects the concentration of carotenoids in oil droplets and can produce variation in colour discrimination, we do not know whether it produces variation in mate preferences.

Variation in spectral sensitivity by changing the relative opsin expression in the retina

Spectral sensitivity can also be tuned to the local light environment by adjusting the relative opsin expression in the retina. Relative opsin expression is known to vary as a function of environmental variations. Populations of Lucania goodei (Bluefin killifish) living in different environments vary in their relative opsin expression. Individuals raised under different lighting conditions show significant
variation in the relative opsin expression (Fuller et al. 2005a). Lucania goodei males are polymorphic for their colour patterns within populations where relative abundance of colour morphs is correlated with the lighting environment of habitats (Fuller 2002). Individuals of Lucania goodei from the same population show significant genetic as well as environmental variation in the relative opsin expression (Fuller et al. 2005a). The presence of polymorphic colour patterns in males and profound environmental, as well as genetic variation in visual systems within populations, suggest that there is a need to explore the link between visual systems and colour morphs within populations.

Lighting conditions can alter an individual's visual sensitivity independently from its genetics as opsin show phenotypic plasticity. Some studies demonstrate opsin plasticity (Fuller et al. 2005a; Fuller and Claricoates 2011) while others fail to find it (Flamarique et al. 2013). Plasticity in opsin expression means that environmental heterogeneity within populations could create variation in visual systems during the mating season, but this is entirely unexplored. It is important to note that variation in opsin expression is one of the potential contributors to the variation in colour discrimination. However, it is entirely possible that organisms vary their opsin expression in order to maintain a constant capacity to discriminate colours within environments. Whether variation in opsin expression explains variation in perception or it is compensatory in nature is entirely unexplored and needs to be studied further. The effects of environmental heterogeneity on sexual selection dynamics within populations will depend on the extent of plasticity in opsin expression and the heritability of variation.
2.6 Conclusion

Variation in genes affecting the sensory system can produce polymorphism in sensory behaviour and mate preferences within populations. In such cases, it is quite possible that different females mate selectively. Polymorphism in sensory systems can maintain sexual trait polymorphisms within populations via selective mating. Thus, genetic variation in sensory genes can produce polymorphism in mate preferences and can contribute to the maintenance of sexual trait polymorphism within populations.

Sensory bias can also contribute to the maintenance of sexual trait polymorphism independent of genetic variation in sensory genes. Environmental variation is often correlated with the variation in the sensory receptors which can alter sensory perception and mate preferences within populations for example variation in cone frequency or the relative opsin expression. Homogenous, but shifting mate preferences can maintain sexual trait variation within populations in the face of environmental variation. Environmental variation in photoreceptors described here can be plastic and can create variation in vision during the mating season. This is entirely unexplored and we do not know about the heritability of this variation. Furthermore, we need to analyse the perceptual consequences of genetic and physiological variation in vertebrate visual systems.

In summary, I have summarized biological processes which can alter sensory systems within populations and therefore can change mate preferences within populations. Consequently, mate choice can maintain sexual trait variation within populations. For simplicity, I have concentrated only on the variation in photoreceptors of visual systems. However, mechanism presented here are equally applicable to the olfactory and auditory peripheral receptors.
Chapter 3: Resilience of sexual trait polymorphisms to gene frequency perturbations in variable environments

3.1 Abstract

Substantial diversity is recorded in sexually selected traits. To what extent does selective mating contribute to the maintenance and persistence of this diversity is a critical question. Recent theory suggests that mate preferences can promote the maintenance of sexual trait diversity. Here I examine how mate preference affects the resilience or robustness of sexual trait polymorphisms in the face of potentially large male-female gene frequency perturbations. I define resilience of sexual trait polymorphisms as the capacity of polymorphic populations to resist large gene frequency perturbations which can shift stable polymorphic populations into a monomorphic state and lose sexual trait polymorphism. I show that strong assortative mating makes sexual trait polymorphisms significantly more robust even in the face of large perturbations in male-female gene frequencies. Consequently, in the face of large perturbations in gene frequencies, nearly stable polymorphic populations are less likely to shift to the monomorphic state and persist sexual trait polymorphism when mate preferences are strong than when they are weak. I show that, in the presence of large gene frequency perturbation, sets of nearly stable polymorphic populations with weaker assortative mating are more likely to show accidental divergence in sexual traits than sets of populations with strong assortative mating. This is because strong mate preferences make sexual trait diversity significantly more robust against large perturbations in gene frequencies.
3.2 Introduction

An important goal of evolutionary ecology is to understand how deterministic and stochastic processes interact and maintain diversity. Substantial diversity is recorded in sexually selected traits. Selective mating can maintain this diversity (Kirkpatrick 1982; Brooks 2002; Kingston et al. 2003; M’Gonigle et al. 2012). Sexual selection models exploring the maintenance of sexual trait diversity usually describe whether or not selective mating and/or other given mechanisms produce polymorphic stable states, and, if present examines the conditions for stable equilibria (Gavrilets 2004; Chunco et al. 2007). However, these models do not focus on the resilience or the robustness of sexual trait diversity in the face of perturbations. Here I examine the resilience of sexual trait polymorphisms to male-female gene frequency perturbations in variable environments.

Resilience or robustness is defined as the capacity of a system to persist and maintain its properties despite the presence of disturbance (Jen 2003; Deffuant and Gilbert 2011; Hodgson et al. 2015). Polymorphic populations can face perturbations in gene frequencies and environmental parameters. Consequently, the resilience of sexual trait polymorphisms can be defined as the capacity of a population to remain polymorphic in sexual traits despite the presence of male-female gene frequency perturbations in variable environments. There are two important aspects of resilience: “engineering resilience” and “ecological resilience” (Holling 1973; Holling 1996; Beisner et al. 2003; Levin and Lubchenco 2008). Engineering resilience measures the system recovery followed by the small, repeated disturbance whereas ecological resilience focuses on the system resistance against temporary, potentially large disturbance that can shift the stable state into alternative attraction basins (Holling
1973; Holling 1996; Levin and Lubchenco 2008). Here I focus particularly on the “ecological resilience” aspect of sexual trait polymorphisms. This is because I am particularly interested in the resilience of sexual trait polymorphisms against large gene frequency perturbations which can potentially shift nearly stable polymorphic populations into the monomorphic state and lose polymorphism.

I explore the resilience of sexual trait polymorphism in the context of intersexual selection where one sex has traits which result in its being chosen by the other sex. Sexual selection occurs in environments where mate choice parameters interact with other selective parameters and parameter values vary over time and space. Changes in mate choice parameters can increase the risk of loss of sexual trait diversity (Seehausen et al. 1997). However, how does the risk changes as a function of sexual selection still remains unknown. I use the dynamical systems theory approach (Meyer 2015) and examine how mate choice parameters, other selective forces independent of mate choice, and their interactions, influence the resilience of sexual trait polymorphisms in the face of potentially large male-female allele frequency perturbations. I use the size and shape of attraction basins as measures of resilience (Beisner et al. 2003; Meyer 2015); an attraction basin is the set of all starting male-female allele frequency combinations from which populations in a given environment evolve to a set of polymorphic equilibria within the basin. I use the null model of sexual selection (Kirkpatrick 1982; Prum 2010) as a starting point and examine two groups of models for resilience. In model 3.1 the sexual trait is only affected by selective mating whereas in model 3.2 directional viability selection also affects the sexual trait.
3.3 Models and Results

3.3.1 Model 3.1: Selective mating only

The standard model of intersexual selection (Kirkpatrick 1982; Prum 2010) assumes directional viability selection on male traits in addition to mate choice. Here I first work with a true null model with no viability selection. Consider a haploid population exhibiting polymorphism in both a sexual trait and in mating preferences for the sexual traits. Assume locus T controls male traits and an unlinked locus P controls female preferences for the male traits. Let each locus have two alleles which correspond to different phenotypes, T1, T2 for different sexual traits in males and P1, P2 for different female preferences. Let the relative preference of a P1 female for T1 males be 1 and her preference for T2 males be 1-α1. Similarly, let the preference of P2 females for T2 males be 1 and her preference for T1 males be 1-α2, where α1 and α2 are mate choice parameters (discrimination coefficients) measuring the strength of preference. If α1 = α2 = 0 there is no choice with respect to male traits and α1 = α2 = 1 means both females only chose their preferred traits (complete positive assortative mating).

The model consists of recurrence equations for zygote frequencies (see the 3.6 Supplementary Information for modeling details). Recurrence equations for genotype frequencies were solved and equilibrium T1 frequencies were computed numerically by iterating the equations for 30000 generations for all combinations of male-female starting frequencies and α1 and α2 using MATLAB 2015b. Generations are assumed to be discrete and non-overlapping.

For a given constant α1 and α2, joint initial male-female allele frequencies that will maintain sexual trait polymorphisms in future form a zone in the joint frequency
state space with two distinct boundaries (Figure 3.1A). I refer to these as the upper (U) and lower (L) boundaries by where they intersect the P axis (Figure 3.1A).

In order to determine the polymorphic zone and boundaries, I first computed the equilibrium T\textsubscript{1} frequency (T\textsubscript{1} frequency after 30000 generations) for all possible combinations of starting frequencies of T\textsubscript{1} and P\textsubscript{1} for a given constant \(\alpha_1\) and \(\alpha_2\). The polymorphic zone is an attraction basin for polymorphic equilibria; it represents all joint T\textsubscript{1} and P\textsubscript{1} starting frequencies that eventually produce the equilibrium T\textsubscript{1} frequency between 0.001 and 0.999 (0.001 \(\leq\) T\textsubscript{1} (equilibrium frequency) \(\leq\) 0.999). To compute U (the boundary separating the polymorphic zone and the attraction basin for T\textsubscript{1} fixation), I identified the threshold starting frequencies of P\textsubscript{1} for the entire range of T\textsubscript{1} starting frequencies such that any change in starting frequency of P\textsubscript{1} above the threshold will result in T\textsubscript{1} fixation, i.e. T\textsubscript{1} (equilibrium frequency) > 0.999. Similarly, to compute L (the boundary separating the polymorphic zone and the attraction basin for T\textsubscript{2} fixation), I identified the threshold starting frequencies of P\textsubscript{1} such that any change in the starting frequency of P\textsubscript{1} below this threshold will result in T\textsubscript{1} loss i.e. T\textsubscript{1} (equilibrium frequency) < 0.001.

U and L separate very different evolutionary outcomes. Populations with joint male-female allele frequencies starting anywhere inside the central zone (within U and L) retain sexual trait polymorphisms. Populations with joint frequencies starting anywhere outside the central zone lose sexual trait polymorphism (either T\textsubscript{1} is fixed or it is lost i.e. T\textsubscript{2} is fixed).

In the dynamical systems framework, the size and shape of the attraction basin in state space describes the resilience of the system to a single (temporary) and potentially large perturbation in state variables (Beisner et al. 2003; Meyer 2015).
Following this, the area of the polymorphic zone indicates the resilience of sexual trait polymorphisms in the face of single-time large, transient perturbation in male-female allele frequencies. Each pair of frequencies represents a perturbation from polymorphic equilibria. Processes such as significant fluctuations in climatic or other environmental parameters, any other process resulting in fluctuating selection, and genetic drift (including 'bottlenecks'), as well as sporadic immigration or emigration, can produce transient, large perturbations in male-female gene frequencies.

For a given magnitude of male-female allele frequency perturbations, the resilience of sexual trait polymorphisms to allele frequency perturbations is proportional to the area of the polymorphic zone. If the area of the polymorphic zone is small then even small transient perturbations in allele frequencies can easily cross the boundary and result in a loss of polymorphisms, whereas if the area is large then boundary crossing is less likely. Moreover, populations on parts of the line of equilibria that are closer to the boundaries are less resilient than those far away.

It is important to note that the area of attraction basin or its width are good predictors of resilience even in stochastic systems containing continuous random fluctuations (Nolting and Abbott 2016). Changes in mate choice parameters ($\alpha_1$ and $\alpha_2$) alter the size and shape of the polymorphic zone, affecting resilience among populations at different positions on the equilibrium line. I explored the effects of $\alpha_1$ and $\alpha_2$ on the area of the polymorphic zone.
Figure 3.1: Effects of selection parameters ($\alpha_1$, $\alpha_2$ and $s$) on polymorphic zones. (A) Phase map showing attraction basin of polymorphic equilibria (polymorphic zone), delimited by two thresholds, U and L (thick black curves) for $\alpha_i = \alpha = 0.8$ for model 3.1. The thin black line is the theoretical stable line of polymorphic equilibria. (B) Changes in the polymorphic zone when mate preferences are equal in both female types ($\alpha = \alpha = \alpha$), varying $\alpha$. (C) Changes in the polymorphic zone for unequal mating preferences: varying $\alpha_1$ and holding $\alpha_2$ constant and strong ($\alpha_2 = 0.8$). (D) Changes in the polymorphic zone as a function of viability selection strength $s$ for $\alpha_1 = \alpha_2 = 0.6$. Each combination of $\alpha_1$ and $\alpha_2$ or $s$ (vertical axis) in B, C and D corresponds with one upper and one lower boundary and forms one polymorphic zone. Dark black lines on the light gray surface (U) are upper boundaries and those on the dark gray surface (L) are lower boundaries. Note the differences in shape, size of polymorphic zones. Starting frequencies of $P_1$ and $T_1$ alleles anywhere inside U and L boundaries maintain polymorphism in T in future. Starting points outside U and L surfaces lose T polymorphism in future.
**Effects of choice parameters on the resilience of sexual trait polymorphisms to gene frequency perturbations**

Different combinations of \( \alpha_1 \) and \( \alpha_2 \) alter the position, shape, and size of the polymorphic zone (Figure 3.1B and C). When preferences are nearly equal and weak, the polymorphic zone remains narrow; the system has low resilience. As \( \alpha_1=\alpha_2=\alpha \) increases, the zone boundaries (U and L) gradually move apart and the polymorphic zone becomes broad (note the gradual increase with \( \alpha \) in Figure 3.1B). In a broad zone, polymorphic populations near equilibrium are much less likely to be sensitive to perturbations in male-female allele frequencies than narrow zones because the zone boundaries are less likely to crossed by a temporary perturbation; polymorphisms within broad zones are relatively more resilient than those in narrow zones. This is generally true even if the population equilibrium shifts along the line of potential polymorphic equilibria. Large allele frequency perturbations are necessary to push stable polymorphic populations outside of broad boundaries when mating preferences are strong in both females unless they sit on the equilibrium line which is very close to a boundary.

Figure 3.2A shows how the area of polymorphic zone changes over the entire range of combinations of \( \alpha_1 \) and \( \alpha_2 \). It is clear that different combinations of \( \alpha_1 \) and \( \alpha_2 \) alter the polymorphic zone area in different ways. The rate of change of resilience as a function of \( \alpha_1 \) and \( \alpha_2 \) is smaller when \( \alpha_1 \) and \( \alpha_2 \) are weak and it increases as \( \alpha_1 \) and \( \alpha_2 \) become stronger. Figure 3.3A shows the relationship between the polymorphic zone area (the resilience of polymorphisms in the face of gene frequency perturbations) and the mean strength of mate choice (\( \alpha_{\text{mean}} \)).
The polymorphic zone area (degree of resilience) disproportionately increases as a function of the mean strength of mate choice ($\alpha_{\text{mean}}$) (Figure 3.3A). There are unique maximum and minimum values of resilience for a constant $\alpha_{\text{mean}}$ (Figure 3.3A). The maximum and minimum values of resilience are biologically important because they indicate the uppermost and lowermost limits of resilience that polymorphic populations can achieve in the given environment. For a given mean strength of mate choice and for the same magnitude of gene frequency fluctuations, polymorphism resilience cannot exceed the maximum value of resilience and/or cannot go below the minimum value of resilience. Polymorphism resilience can vary within these limits depending on the relative and absolute values of $\alpha_1$ and $\alpha_2$. The maximum and minimum resilience values remain very low (close to zero) when $\alpha_{\text{mean}}$ is weak. Maximum and minimum resilience values increase disproportionately as $\alpha_{\text{mean}}$ increases and both remain high for stronger $\alpha_{\text{mean}}$. Minimum resilience remains close to zero for the range of $\alpha_{\text{mean}}$ ranging from $\alpha_{\text{mean}}=0.05$ to $\alpha_{\text{mean}}=0.51$ and increases rapidly when $\alpha_{\text{mean}}$ exceeds 0.5 (Figure 3.3A). For a given range of perturbations in male-female allele frequencies, small differences in $\alpha_1$ and/or $\alpha_2$ among populations can cause disproportionately large differences in the resilience of polymorphisms; even if the populations are identical in their $\alpha_{\text{mean}}$. 
Figure 3.2: Relationship between the area of the polymorphic zone and choice parameters $\alpha_1$, $\alpha_2$. (A) Changes in the area of polymorphic zone showed as a surface over different combinations of $\alpha_1$ and $\alpha_2$ in the absence of viability selection ($s=0$). (B) Changes in the area of polymorphic zone showed as a surface over different combinations of $\alpha_1$ and $\alpha_2$ in the presence of viability selection ($s=0.3$).
Figure 3.3 Relationship between the area of the polymorphic zone and the mean strength of mate choice ($\alpha_{\text{mean}}$) under different viability selection ($s$) regimes. Each dot in the panels A to I represents the area of the polymorphic zone for a unique combination of $\alpha_1$ and $\alpha_2$.

3.3.2 Model 3.2: Trait (T) under directional viability selection

The standard model of intersexual selection (Kirkpatrick 1982; Prum 2010) normally assumes directional viability selection on male traits apart from the mate choice. For example, viability selection could be caused by the physical environment.
I explored the range of resilience when male traits are under directional viability selection independent of the preference trait P. Let T₂ males have a disadvantage such that the T₂ trait viability is 1-s relative to T₁ males; s is the viability selection coefficient (0 ≤ s ≤ 1). Let viability selection on males occur before selective mating; this alters the frequencies of males available for mating (see section 3.6 Supplementary Information). Aside from this modification, the model is the same as model 3.1. T₁ frequencies were computed numerically by iterating the equations for 5000 generations (for s=0.1, 10,000 generations) for all combinations of male-female starting frequencies and α₁ and α₂ using MATLAB 2015b. 5000 generations were more than sufficient to reach a stable equilibrium.

**Joint effects of mate choice and natural selection parameters on sexual trait polymorphism resilience against gene frequency perturbations**

The strengths of s, α₁, and α₂ have interacting effects and this determines the size, shape, and position of the polymorphic zone. Figure 3.1D shows changes in the zone as a function of s for α₁=α₂=0.6. Figure 3.3 shows how the polymorphic zone area changes as a function of α_mean under different viability selection regimes. Overall, the polymorphic zone area increases as a function of α_mean under different strengths of s. For a constant α_mean, increasing s increases the maximum resilience (compare the maximum polymorphic zone area in each plot in Figure 3.3). The rate of change in the maximum value of resilience when α_mean < 0.5 is smaller than the rate of change when α_mean > 0.5. For a constant α_mean, s also decreases the minimum resilience value (compare the minimum area of polymorphic zone across all plots in Figure 3.3A). As s increases, overall resilience remains high for stronger α_mean.
For a constant $\alpha_{\text{mean}}$, $s$ increases the difference between the maximum and minimum resilience and thus, effectively increases the resilience range (this effect is most prominent when the strength of $\alpha_{\text{mean}}$ is moderate). This suggests that for a given change in $\alpha_1$ and/or $\alpha_2$, the resilience of polymorphisms to gene frequency perturbations changes more if $s$ is stronger than if it is weak (each dot in Figure 3.3A to Figure 3.3I represent the area of polymorphic zone for the unique combination of $\alpha_1$ and $\alpha_2$). Differences in mate preferences among populations can produce disproportionately large differences in the resilience of sexual trait diversity, even if populations are identical in their mean strengths of mate choice and natural selection.

3.4 Discussion

Classical theory suggests that sexually selected traits should have low variance (Kirkpatrick and Ryan 1991; Pomiankowski and Moller 1995) yet many species show variation in these traits (Pomiankowski and Moller 1995; Brooks and Endler 2001a; Gray and McKinnon 2007; Wellenreuther et al. 2014). To what extent does mate preferences maintain this variation remains a critical question. Recent theory suggests that mate preferences can promote the maintenance of sexual trait diversity (M’Gonigle et al. 2012), classically thought to prevent it. My study clearly shows that strong mate preferences make sexual trait polymorphisms significantly more robust even in the face of potentially large perturbations in gene frequencies. Consequently, stable or nearly stable polymorphic populations with strong assortative mating are less likely to shift to monomorphic state and are more likely to persist sexual trait diversity for a long time even in the face of large perturbations in male-female gene frequencies. Now, I discuss the potential mechanism which can produce these results.
Selective mating produces an overall negative frequency-dependent effect which makes the line of polymorphic equilibria a stable attractor (Seger 1985). For a constant frequency of the preference allele in populations, male traits exhibit higher fitness relative to the other morph when lower in frequency (see figure 1b in Seger 1985). Thus, populations starting with a higher frequency of male alleles require a higher frequency of corresponding female alleles to continue the Fisher process and lead populations to fixation (this is the reason why U and L in Figure 3.1A are curved and not horizontal straight lines). Strong assortative mating amplifies the negative frequency-dependent effect (see the Figure 3.1B that U and L remain straight horizontal lines when $\alpha$ is weak and become more curved as $\alpha$ becomes strong). This can potentially make polymorphic attractors more robust when assortative mating is strong than when it is weak.

These results have strong implications for population divergence because excursions both above the upper (U) and below the lower (L) polymorphic boundaries will result in fixation of different sexual trait alleles. Selective conditions that enhance the polymorphism resilience actually reduce the potential for accidental divergence among polymorphic populations that sit on or near the line of polymorphic equilibria, compared to conditions that reduce the resilience of polymorphisms. When the assortative mating is strong then the polymorphic zone remains broad (greater polymorphism resilience) and hence a relatively larger perturbation in male-female gene frequencies is required to cause accidental divergence in sexual traits among polymorphic populations (i.e. to throw stable polymorphic populations across the zone boundaries in opposite directions). Thus, for the same magnitude of large gene frequency fluctuations, population divergence is less likely in conditions that enhance the polymorphism resilience (i.e. strong assortative mating). In contrast, when the
assortative mating is weak then polymorphic zone remains narrow (lower polymorphism resilience) and relatively small perturbations in allele frequencies can easily push populations beyond the polymorphic zone boundaries. Consequently, my results suggest that in the face of potentially large male-female gene frequency perturbations, sets of isolated and nearly stable polymorphic populations (populations sitting at different positions on or near the line of equilibria in Figure 3.1A) with strong (but not complete) assortative mating are less likely to show accidental divergence than sets of populations with weaker assortative mating. In other words, sets of isolated polymorphic populations near the line of equilibria are less likely to cross the zone boundaries in opposite directions when assortative mating is strong than when it is weak.

Servedio and Bürger (Servedio and Bürger 2014) showed that the Fisher process (the null process of intersexual selection), on its own, reduces the likelihood of divergence and speciation in the face of gene flow between populations with different natural selection parameters. My results suggest that in the absence of gene flow, for a given viability selection regime, strong mate preferences can reduce the likelihood of accidental divergence among isolated polymorphic populations (which sit near the polymorphic equilibria) in the face of temporary and large gene frequency perturbations. Sets of isolated polymorphic populations near the line of equilibria are less likely to cross the zone boundaries in opposite directions and are less likely to diverge accidentally when assortative mating is strong than when it is weak. Gene flow is a chronic or continuous perturbation by immigrant alleles. Gene flow between polymorphic populations can reduce the size of random fluctuations, hence can lead to greater resilience of polymorphisms and lower probability of fixation or loss outside the polymorphic zone. Gene flow can also reduce the effect of random perturbations
because gene frequency perturbations in different populations connected by gene flow are likely to average out as much smaller values than a single large population, and the more populations exchanging genes, the less the effect of perturbations in gene frequencies and/or environments. The relationship between gene flow and the robustness of sexual trait polymorphisms is entirely unexplored. For example, there is a possibility that a combination of isolation-by-distance gene flow and very strong preferences may permit some polymorphism under certain circumstances. One interesting possibility is very low gene flow where the alleles coming into the population of interest fluctuate at random such that directional bias changes in time.

Gavrilets (Gavrilets 2004) used a hybrid deficiency index (I) to measure the potential for reproductive isolation in a model similar to my model 3.1. He found that hybrids are maintained in populations even if both females show strong mating preferences. Hybrids are eliminated only when mating preferences are extremely strong (but not completely assortative). For example when $\alpha_1=\alpha_2=\alpha > 0.9$; see figure 9.5 in (Gavrilets 2004). My results show that strong mating preferences make sexual trait polymorphisms more resilient even in the face of larger gene frequency perturbations. Thus, for strong $\alpha$, polymorphic populations sitting on or close to the stable line may not necessarily develop reproductive isolation and can remain polymorphic for long periods in spite of large allele frequency perturbation. In summary, populations can remain polymorphic in sexual traits for a long time even in the face of large gene frequency perturbation if the assortative mating is strong, but not if it is weak. These results suggest that early stages of speciation could stall in the face of large transient gene frequency perturbations, especially with strong mating preferences, and further parametric changes may need to occur before speciation completes.
3.5 Acknowledgements

I would like to thank Ben Fanson, Mark Kirkpatrick and Hanna Kokko for helpful discussions in the early stages of this project. I would also like to thank Sergey Gavrilets for useful discussion in the final stages of this project. I also thank my friends Swanand Khare and Priyavrat Deshpande for quickly replying to some of my mathematics and programming related queries.

3.6 Supplementary Information

Model 3.1

Mating is followed by full recombination and then zygotes are formed for the next generation. Generations are assumed to be discrete and non-overlapping. The model consists of recurrence equations for zygote frequencies. Mating frequencies for different genotypes and recurrence equations for zygote frequencies are provided in Table 3.1.

Model 3.2

For model II, new mating frequencies after the viability selection can be obtained by replacing $m_1$, $m_2$, $m_3$ and $m_4$ respectively with $m_1'$, $m_2'$, $m_3'$ and $m_4'$ in Table 3.1. Similarly, zygote frequencies in the next generation can be obtained by substituting $m_1'$, $m_2'$, $m_3'$ and $m_4'$ for $m_1$, $m_2$, $m_3$ and $m_4$ in equations A, B, C and D. New gamete frequencies available for mating in males are $m_1' = \frac{m_1}{W}$; $m_2' = \frac{m_2}{W}$; $m_3' = \frac{m_3(1-s)}{W}$; $m_4' = \frac{m_4(1-s)}{W}$; $W = 1 - st_2$ and $t_2 = m_3 + m_4$. 


Table 3.1: Frequencies of different mating types. \( m_1, m_2, m_3 \) and \( m_4 \) and are frequencies of \( T_1P_1, T_1P_2, T_2P_1 \) and \( T_2P_2 \) genotypes in males and \( f_1, f_2, f_3 \) and \( f_4 \) are their frequencies in females.

<table>
<thead>
<tr>
<th></th>
<th>( T_1P_1 )</th>
<th>( T_1P_2 )</th>
<th>( T_2P_1 )</th>
<th>( T_2P_2 )</th>
<th>Total</th>
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<tbody>
<tr>
<td>( T_1P_1 )</td>
<td>( \frac{f_1 m_1}{z_1} )</td>
<td>( \frac{f_1 m_2}{z_1} )</td>
<td>( \frac{f_1 (1 - \alpha_1) m_3}{z_1} )</td>
<td>( \frac{f_1 (1 - \alpha_1) m_4}{z_1} )</td>
<td>( f_1 )</td>
</tr>
<tr>
<td>( T_1P_2 )</td>
<td>( \frac{f_2 (1 - \alpha_2) m_1}{z_2} )</td>
<td>( \frac{f_2 (1 - \alpha_2) m_2}{z_2} )</td>
<td>( \frac{f_2 m_3}{z_2} )</td>
<td>( \frac{f_2 m_4}{z_2} )</td>
<td>( f_2 )</td>
</tr>
<tr>
<td>( T_2P_1 )</td>
<td>( \frac{f_3 m_1}{z_1} )</td>
<td>( \frac{f_3 m_2}{z_1} )</td>
<td>( \frac{f_3 (1 - \alpha_1) m_3}{z_1} )</td>
<td>( \frac{f_3 (1 - \alpha_1) m_4}{z_1} )</td>
<td>( f_3 )</td>
</tr>
<tr>
<td>( T_2P_2 )</td>
<td>( \frac{f_4 (1 - \alpha_2) m_1}{z_2} )</td>
<td>( \frac{f_4 (1 - \alpha_2) m_2}{z_2} )</td>
<td>( \frac{f_4 m_3}{z_2} )</td>
<td>( \frac{f_4 m_4}{z_2} )</td>
<td>( f_4 )</td>
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\[
\begin{align*}
\text{Total} & = m_1 \left[ \frac{P_1}{z_1} + \frac{(1 - \alpha_1)P_2}{z_2} \right] + m_2 \left[ \frac{P_1}{z_1} + \frac{(1 - \alpha_2)P_2}{z_2} \right] + m_3 \left[ \frac{(1 - \alpha_1)P_1}{z_1} + \frac{P_2}{z_2} \right] + m_4 \left[ \frac{(1 - \alpha_1)P_1}{z_1} + \frac{P_2}{z_2} \right] \\
& = \frac{1}{z_1} + \frac{1}{z_2} \\
\end{align*}
\]

\( z_1 = m_1 + m_2 + (1 - \alpha_1)(m_3 + m_4) \) ; \( z_2 = (1 - \alpha_2)(m_1 + m_2) + m_3 + m_4 \) ; \( P_1 = f_1 + f_3 \) ; \( P_2 = f_2 + f_4 \) here \( m_i = f_j \)
Recurrence equations for zygote frequencies in the next generation are

\[
T_1 P'_1 = f_1 \left[ \frac{m_1}{z_1} + \frac{m_2}{2z_1} + \frac{m_3(1-\alpha_1)}{2z_1} + \frac{m_4(1-\alpha_2)}{4z_1} \right] + f_3 \left[ \frac{m_1}{2z_1} + \frac{m_2}{4z_1} \right] \\
+ f_2 \left[ \frac{m_1(1-\alpha_2)}{2z_2} + \frac{m_3}{4z_2} \right] + f_4 \left[ \frac{m_1(1-\alpha_2)}{4z_2} \right] 
\] (3.1)

\[
T_1 P'_2 = f_2 \left[ \frac{m_1(1-\alpha_2)}{2z_2} + \frac{m_2(1-\alpha_2)}{z_2} + \frac{m_3}{4z_2} + \frac{m_4}{2z_2} \right] + f_1 \left[ \frac{m_2}{2z_1} + \frac{m_4(1-\alpha_1)}{4z_1} \right] + f_3 \left[ \frac{m_2}{4z_1} \right] 
\] (3.2)

\[
T_2 P'_1 = f_1 \left[ \frac{m_3(1-\alpha_1)}{2z_1} + \frac{m_4(1-\alpha_1)}{4z_1} \right] + f_3 \left[ \frac{m_1}{2z_1} + \frac{m_2}{4z_1} + \frac{m_3(1-\alpha_1)}{z_1} + \frac{m_4(1-\alpha_1)}{2z_1} \right] \\
+ f_2 \left[ \frac{m_3}{4z_2} \right] + f_4 \left[ \frac{m_1(1-\alpha_2)}{4z_2} + \frac{m_3}{2z_2} \right] 
\] (3.3)

\[
T_2 P'_2 = f_2 \left[ \frac{m_3}{4z_2} + \frac{m_4}{2z_2} \right] + f_4 \left[ \frac{m_3(1-\alpha_2)}{4z_2} + \frac{m_2(1-\alpha_2)}{2z_2} + \frac{m_3}{2z_2} + \frac{m_4}{z_2} \right] \\
+ f_1 \left[ \frac{m_4(1-\alpha_1)}{4z_1} \right] + f_3 \left[ \frac{m_2}{4z_1} + \frac{m_4(1-\alpha_1)}{2z_1} \right] 
\] (3.4)

Here,

\[ z_1 = m_1 + m_2 + (1 - \alpha_1)(m_3 + m_4) \]
\[ z_2 = (1 - \alpha_2)(m_1 + m_2) + m_3 + m_4 \]
Chapter 4: Persistence and divergence of sexually selected traits affected by frequency-dependent predation and a consistent mating advantage

4.1 Abstract

Ecologically-driven negative frequency-dependent natural selection (NFD) can interact with mate choice to either maintain sexual trait polymorphisms or cause their fixed divergence. Combinations of mate choice parameters define thresholds between the maintenance and loss of polymorphisms. Same thresholds also set the conditions for the divergent fixation or loss of sexual traits among populations. Populations diverge in allele frequencies but remain polymorphic if they are within a zone defined by two thresholds in the mate choice parametric space. However, sexual traits diverge completely and can run away in opposite directions if populations fall outside the zone. If NFD and mate preferences are weak or moderate, then small differences in mate preferences are enough to cause fixed divergence in sexual traits among populations. However, if mate preferences of different female types are strong within populations then populations can remain polymorphic for a long time, even if NFD is weak. Early stages of speciation could stall under such circumstances, and further parametric changes may need to evolve before speciation completes.
4.2 Introduction

Sexually selected traits evolve in an ecological context where non-random mating is often based on traits which are also used in other ecological functions (Gavrilets 2004; Servedio et al. 2011). For example, animal colour patterns are used as sexual signals during mate choice as well as for the protection against predators. Here I explore interactions between the negative frequency-dependent natural selection (NFD) and selective mating and examine how their interactions affect to the polymorphism persistence and divergence in sexually selected traits.

NFD is important in the maintenance of polymorphisms within populations (Clarke and O'Donald 1964; Ayala and Campbell 1974). It can arise from various ecological processes such as frequency-dependent predation (Clarke 1962; Allen and Greenwood 1988) and/or competition for resources (Ayala and Campbell 1974). If sexually selected traits are simultaneously under ecologically-driven NFD, then mate choice parameters can interact with NFD and this will dictate the maintenance of polymorphism or divergence of sexually selected traits.

Colour polymorphisms are ideal systems to look for such interactions. Colour polymorphisms can be under NFD due to frequency-dependent predation (Allen 1972; Cooper 1984; Endler 1988; Bond and Kamil 1998; Punzalan et al. 2005; Bond 2007; Ishii and Shimada 2010). Additionally, colour polymorphic species can show variation in mate preferences and selective mating based on colour pattern components (Morris et al. 2003). For example, colour morphs in male guppies are under NFD (Farr 1977; Olendorf et al. 2006; Hughes et al. 2013). Additionally, female preferences for colour patterns vary
within (Brooks and Endler 2001b), as well as among (Endler and Houde 1995), guppy populations. In this case, NFD parameters can interact with mate choice parameters of individual female types and affect the maintenance of polymorphism of, or divergence in, sexually selected traits.

Here, I examine two models. In the first model, only males are affected by NFD whereas, in the second model both males and females are affected by NFD. In both models, I will identify threshold conditions that separate the maintenance and loss of polymorphism and favour the strong or fixed divergence in sexually selected traits. I will also examine how mate choice parameters, NFD parameters, and their interactions influence the resilience of polymorphic populations in variable environments. I define polymorphic resilience as the capacity of populations to remain polymorphic in sexual traits despite perturbations in mate choice and/or NFD parameters.

4.3 Models and Results

4.3.1 Model 4.1: Trait expression is male-limited (only males are affected by NFD)

Consider a haploid population showing polymorphism in both a mating traits and in mating preferences. Assume locus T controls sexual traits in males and the unlinked locus P controls female preferences for traits. Let each locus have two alleles which correspond to different mating traits T₁, T₂ and female preferences P₁, P₂ respectively. The standard or null model of selective mating (Kirkpatrick 1982; Prum 2010) normally assumes directional viability selection on sexual traits. Instead, here I assume sexual traits
are affected by ecologically-driven NFD such as frequency-dependent predation. Aside from this modification, the model is the same as the standard model of selective mating.

Let $m_1, m_2, m_3$ and $m_4$ be the frequencies of $T_1P_1$, $T_1P_2$, $T_2P_1$ and $T_2P_2$ zygotes in males and $f_1, f_2, f_3$ and $f_4$ be their frequencies in females. Let $\beta$ be the strength of NFD. Consequently, the fitness measures of male genotypes are $W_{T_1P_1} = 1 - \beta m_1 ; W_{T_1P_2} = 1 - \beta m_2 ; W_{T_2P_1} = 1 - \beta m_3 ; W_{T_2P_2} = 1 - \beta m_4$. Let NFD on mating traits occur before mating; this alters the frequencies of males available for selective mating. Frequencies of male genotypes available for selective mating after NFD are $m_1' = m_1(1 - \beta m_1) / \overline{W} ; m_2' = m_2(1 - \beta m_2) / \overline{W} ; m_3' = m_3(1 - \beta m_3) / \overline{W} ; m_4' = m_4(1 - \beta m_4) / \overline{W}$, where $\overline{W} = \left[ (m_1(1 - \beta m_1)) + (m_2(1 - \beta m_2)) + (m_3(1 - \beta m_3)) + (m_4(1 - \beta m_4)) \right]$. Note that in this model, since there is no NFD on females, the female genotype frequency available for selective mating after NFD is the same as males before NFD or $f_i' = f_i$.

Let the relative preference of a $P_1$ female for $T_1$ males be 1 and her preference for $T_2$ males be $1 - \alpha_1$. Similarly, let the preference of $P_2$ females for $T_2$ males be 1 and her preference for $T_1$ males be $1 - \alpha_2$. $\alpha_1$ and $\alpha_2$ are sexual selection coefficients. New mating frequencies after NFD can be obtained by replacing $m_1, m_2, m_3, m_4$ respectively with $m_1', m_2', m_3', m_4'$ and by replacing $f_1, f_2, f_3, f_4$ with $f_1', f_2', f_3', f_4'$ in Table 3.1 from chapter 3. Similarly, zygote frequencies in the next generation after selective mating can be obtained by substituting $m_1', m_2', m_3'$ and $m_4'$ for $m_1, m_2, m_3$ and $m_4$ and $f_1', f_2', f_3', f_4'$ for $f_1, f_2, f_3$ and $f_4$ in equations 3.1, 3.2, 3.3 and 3.4 (refer section 3.6 Supplementary Information from chapter 3). Generations are assumed to be discrete and non-overlapping.
Recurrence equations for genotype frequencies were solved and equilibrium $T_1$ frequencies were computed numerically by iterating the equations for 1000 generations using MATLAB version 2015b. I found this to be more than sufficient time for populations to attain stable equilibria.

For a given NFD strength $\beta$, combinations of $\alpha_1$ and $\alpha_2$ that maintain sexual trait polymorphisms form a zone in the $\alpha_1$-$\alpha_2$ state space with two distinct threshold boundaries (Figure 4.1B). I will refer to the central zone as the polymorphic zone and the threshold boundaries as upper (U) and lower (L) boundaries by where they intersect the $\alpha_1$ axis (Figure 4.1B).

In order to determine the polymorphic zone and boundaries, I first computed the equilibrium $T_1$ frequency ($T_1$ frequency after 1000 generations) for all possible combinations of $\alpha_1$ and $\alpha_2$ for a constant set of starting $P_1$ and $T_1$ frequencies. The polymorphic zone is an attraction basin for polymorphic equilibria; it represents all combinations of $\alpha_1$ and $\alpha_2$ that eventually produce equilibrium $T_1$ frequency between 0.001 and 0.999 ($0.001 < T_1 \text{ (equilibrium frequency)} < 0.999$). To compute U (the boundary separating the polymorphic zone and the attraction basin for $T_1$ fixation), I identified threshold $\alpha_1$ for the entire range of $\alpha_2$ such that any change in $\alpha_1$ above the threshold will result in $T_1$ fixation, i.e. $T_1 \text{ (equilibrium frequency)} > 0.999$. Similarly, to compute L (the boundary separating the polymorphic zone and the attraction basin for $T_2$ fixation), I identified threshold $\alpha_1$ such that any change in the starting frequency of $\alpha_1$ below L will result in $T_1$ loss, i.e. $T_1 \text{ (equilibrium frequency)} < 0.001$. 
Results

In a given NFD strength ($\beta$), and for a given set of starting male-female allele frequencies, any change in the relative strengths of $\alpha_1$, $\alpha_2$ alters the $T_1$ equilibrium frequency. Figure 4.1 shows this general result for the entire range of $\alpha_1$ and $\alpha_2$ when $\beta = 0.1$.

U and L separate very different evolutionary outcomes. For a given $\alpha_2$, any value of $\alpha_1$ above U results in the fixation of $T_1$ whereas any value of $\alpha_1$ below the L results in the loss of $T_1$ i.e. fixation of $T_2$ (Figure 4.1B). Note that in Figure 4.1B, U ranges from $\alpha_2 = 0.05$ to $\alpha_2=0.48$. If $\alpha_2 > 0.48$ then there is no upper threshold value of $\alpha_1$ that separates the polymorphism and monomorphism and populations will remain polymorphic. Similarly, if $\alpha_2 < 0.1$ then there is no lower limit of $\alpha_1$ that separates the polymorphism and monomorphism.

Populations with $\alpha_1$-$\alpha_2$ combinations anywhere in the polymorphic zone (within U and L) maintain sexual trait polymorphisms within populations. It is important to note that if populations fall inside the polymorphic zone, they remain polymorphic but diverge in allele frequencies even if not going to fixation and loss. If populations happen to fall on the opposite sides of the polymorphic zone in figure 4.1B then populations diverge completely and frequencies go to fixation and loss. I refer this divergence (fixation or loss of sexual trait allele) as complete divergence. Note that even though Figure 4.1 shows the result for a constant set of $P_1$ and $T_1$ starting frequencies (starting $T_1$ frequency = 0.5 and starting $P_1$ frequency = 0.5), same result holds true for the entire range of starting frequencies of $T_1$ and $P_1$. 
Changes in $\beta$ alter the positions of U and L and hence the shape and size of the polymorphic zone (Figure 4.2). The polymorphic zone gradually increases in size as $\beta$ increases (note the systematic expansion with a gradual increase in $\beta$ in Figure 4.2A). In order to describe changes in U and L as a function of $\beta$, I used the quadratic function ($y = p_1 x^2 + p_2 x + p_3$) which consistently describes both U and L for the entire range of $\beta$ (Figure 4.3). In this function, $y$ represents either U or L and $x$ represents the strength of $\alpha_2$. For a given $\alpha_2$, any value of $\alpha_1$ above U will result in the fixation of $T_1$. Any value of $\alpha_1$ below L will result in the loss of $T_1$ i.e. fixation of $T_2$. Therefore, if populations fall above U and below L then they will strongly diverge in sexual traits. Figure 4.4 shows a systematic change in model parameters over $\beta$ estimated by the quadratic approximation. Using the estimated parameters (Table 4.1 and Table 4.2), U and L can be estimated for the given intensity of $\beta$. 
Figure 4.1: Consequences of interaction between the NFD and mate choice parameters. (A) Surface plot of $T_1$ equilibrium frequency as a function of $\alpha_1$ and $\alpha_2$. For this particular case $\beta=0.1$, starting $T_1$ frequency = 0.5 and starting $P_1$ frequency = 0.5. Note that same result holds true for the entire range of $P_1$ and $T_1$ starting frequencies. Solid black lines on the surface indicate threshold boundaries that separate maintenance and loss of polymorphism (either fixation or loss of $T_1$). (B) Phase map showing polymorphic zone and upper (U) and lower (L) threshold boundaries when $\beta=0.1$. Populations sitting anywhere between U and L maintain polymorphisms in sexually selected traits whereas populations outside U and L lose polymorphisms within populations. For a given $\alpha_2$, any value of $\alpha_1$ above U will result in the fixation of $T_1$. Any value of $\alpha_1$ below the L will result in the loss of $T_1$ i.e. fixation of $T_2$. 
Figure 4.2: Changes in the upper (U) and lower (L) threshold boundaries as a function of NFD strength ($\beta$). (A) Changes in U and L when only males are affected by the NFD (model 4.1). (B) Changes in U and L when both sexes are affected by the NFD (model 4.2).
Figure 4.3: Fitted quadratic function (red curves) to the upper (U) and lower (L) boundaries (dots) for different strengths of $\beta$ when only males are affected by NFD (model 4.1). See that the fit of the quadratic function is excellent.
Figure 4.4: Changes in the parameters estimated by the quadratic model \( y=p_1 x^2+p_2 x+p_3 \) as a function of \( \beta \). (A) Changes in parameters for the upper threshold boundary (U). (B) Changes in parameters for the lower threshold boundary (L). Each black dot in A and B shows a combination of parameters for a given strength of \( \beta \). Note that in this model \( y \) represents threshold \( \alpha_1 \) (upper (U) and lower (L)) that separate the maintenance and loss of polymorphisms whereas \( x \) is \( \alpha_2 \).
Table 4.1: Parameters estimated by the quadratic model \( y = p_1 x^2 + p_2 x + p_3 \) for the upper threshold boundary (U) for different \( \beta \). 95% confidence intervals are given in brackets.

<table>
<thead>
<tr>
<th>( \beta )</th>
<th>( p_1 )</th>
<th>( p_2 )</th>
<th>( p_3 )</th>
<th>( r^2 )</th>
<th>Value of ( \alpha_1 )</th>
</tr>
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<td>0.05</td>
<td>2.347</td>
<td>0.6484</td>
<td>0.08243</td>
<td>0.9995</td>
<td>0.05 – 0.505</td>
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<td>(0.5991, 0.6977)</td>
<td>(0.07699, 0.08787)</td>
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<td>0.1</td>
<td>2.27</td>
<td>0.7107</td>
<td>0.1229</td>
<td>0.9994</td>
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<tr>
<td></td>
<td>(2.167, 2.373)</td>
<td>(0.6584, 0.7629)</td>
<td>(0.1174, 0.1284)</td>
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<tr>
<td>0.2</td>
<td>2.172</td>
<td>0.7472</td>
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<td>(0.7015, 0.7929)</td>
<td>(0.2135, 0.2226)</td>
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<tr>
<td>0.3</td>
<td>1.886</td>
<td>0.8543</td>
<td>0.3099</td>
<td>0.9995</td>
<td>0.05 – 0.425</td>
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<td></td>
<td>(1.773, 1.998)</td>
<td>(0.8043, 0.9043)</td>
<td>(0.3052, 0.3145)</td>
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<td>0.4</td>
<td>1.718</td>
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<td>0.9995</td>
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<td>(0.4002, 0.4079)</td>
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<td>0.5</td>
<td>1.431</td>
<td>0.969</td>
<td>0.5004</td>
<td>0.9995</td>
<td>0.05 – 0.355</td>
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<tr>
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<td>(1.3, 1.563)</td>
<td>(0.9215, 1.017)</td>
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<td>0.6</td>
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<td>1.014</td>
<td>0.598</td>
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<td>(0.5948, 0.6013)</td>
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</tr>
<tr>
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<td>0.9984</td>
<td>0.05 – 0.245</td>
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<td>0.7981</td>
<td>0.9982</td>
<td>0.05 – 0.175</td>
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<td>(0.9243, 1.141)</td>
<td>(0.7939, 0.8023)</td>
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Table 4.2: Parameters estimated by the quadratic model ($y = p_1 x^2 + p_2 x + p_3$) for the lower threshold boundary (L) for different $\beta$. 95% confidence intervals are given in brackets.

Here I use the dynamical systems theory approach (Meyer 2015) to describe the resilience of stable polymorphic populations against perturbations in mate choice.
parameters. Analysis of resilience becomes particularly important when selection parameters vary erratically over time and space. Recent sexual selection studies show that fluctuations in mate choice parameters are more common than previously thought [reviewed in (Miller and Svensson 2014)].

In the dynamical systems theory framework, the distance from any given point in x-y state space to the threshold boundaries is used as a measure of the resilience of that point against single and potentially large perturbations in state parameters (Meyer 2015). Here I focus on the resilience of sexual trait polymorphisms against perturbations in mate choice parameters which can potentially shift nearly stable polymorphic populations into the monomorphic state and lose sexual trait polymorphism. Significant sporadic perturbations in environmental parameters can produce transient, large perturbations in mate choice parameters. For example, for mate choice based on vision, any change in the light environment or visual backgrounds can result in either temporary or long-term changes in mate choice parameters.

Following this, for a given range of perturbations in mate choice parameters, the polymorphism resilience is proportional to the distance from that point in the $\alpha_1-\alpha_2$ state space to the nearest threshold boundary. The minimum distance indicates the minimum perturbation in mate choice parameters needed to lose polymorphisms. If a stable polymorphic population is near the threshold boundary, then even small perturbations in mate choice parameters can make polymorphic population cross the boundary and result in the loss of polymorphisms, whereas if a population is farther away from boundary then the boundary crossing is less likely for the same magnitude of parameter fluctuations.
Figure 4.5 shows how NFD and mate choice parameters interact non-linearly and alter the resilience of polymorphic populations on the diagonal line $D$ (see $D$ in the inset of Figure 4.5). Note that $P_1$ and $P_2$ females show equal mate preferences ($\alpha_1=\alpha_2$) in populations on $D$. When NFD is weak ($\beta=0.1$, curve I), the resilience of polymorphic populations increases disproportionately as a function of $\alpha$ (curve I in Figure 4.5). The resilience of polymorphic populations is low when mate preferences are weak (see that the width of the polymorphic zone in remains narrow when $\alpha_1=\alpha_2=$ weak in Figure 4.1B). In such cases, even small perturbation in mate choice parameters can result in the loss of polymorphisms in populations on $D$. Resilience increases when both types of females show strong mate preferences ($U$ and $L$ expand more when $\alpha_1$ and $\alpha_2$ are strong). Under strong NFD ($\beta=0.8$), the overall resilience of polymorphic populations remains high irrespective of mate choice parameters within populations. In contrast, resilience is low when both types of females show moderate mate preferences relative to weak or strong mate choice parameters (curve IV in Figure 4.5).
Figure 4.5: Resilience of stable polymorphic populations to temporary, large perturbations in mate choice parameters under different NFD regimes. This particular example shows the resilience of stable polymorphic populations on the diagonal $D$ (see the inset). Resilience is measured as the nearest distance ‘$q$’ (showed in the inset small box) from each point on the diagonal to either threshold boundary. Each curve in the figure shows changes in the resilience of diagonal as a function of $\alpha$ under different NFD regimes.
4.3.2 Model 4.2: Trait expression is not sex-limited (both males and females are affected by NFD)

In this model, I examined the dynamics of threshold boundaries (U and L) when both sexes are affected by NFD. This can occur when the expression of sexually selected traits is not sex-limited.

Let $m_1, m_2, m_3$ and $m_4$ be the founding frequencies of $T_1P_1$, $T_1P_2$, $T_2P_1$ and $T_2P_2$ zygotes respectively in males and $f_1, f_2, f_3$ and $f_4$ be the founding frequencies in females within the population. The only difference between this model and model 4.1 is that in this model, females are also affected by NFD and therefore, $f'_i = m'_i$ instead of $f'_i = m_i$. Now that $f'_i$ and $m'_i$ are the female and male genotypes frequencies available for selective mating after NFD. Aside from this change, the model is identical to model 4.1. Note that the computation procedure to estimate polymorphic zone and basin boundaries (U and L) remains same as explained in the model 4.1.

For a given strength of NFD ($\beta$), threshold boundaries separate more, yielding a larger polymorphic zone, when both sexes are affected by NFD. The rate of zone area expansion is approximately doubled when both sexes are affected by NFD compared to model 4.1; For example, the position of U and L for $\beta=0.3$ in Figure 4.2B (NFD on both sexes) is the same as the position of U and L for $\beta=0.6$ in the Figure 4.2A (NFD only on males). When both sexes are affected by NFD, NFD intensity is doubled and thus the rate of expansion of U and L is also doubled. Note that even though Figure 4.2 shows the result for a constant set of $P_1$ and $T_1$ starting frequencies (starting $T_1$ frequency = 0.5 and starting $P_1$ frequency = 0.5), same result holds true for the entire range of starting frequencies of $T_1$ and $P_1$. 

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When both sexes are under NFD, U and L disappear only if $\beta > 0.48$ and polymorphism in T is maintained irrespective of the mate choice parameters of females. Differences in mate choice parameters of females cannot cause strong or fixed divergence in sexually selected traits if $\beta > 0.48$. In such cases, sexual traits can completely diverge only if $\beta$ is weak and below 0.48.

### 4.4 Discussion

Negative frequency-dependent natural selection (NFD) can interact with mate choice to either maintain polymorphism or cause strong divergence in sexually selected traits. Whether populations stay polymorphic or diverge completely depends on the relative values of NFD and individual mate choice parameters. For a given frequency dependent selection intensity ($\beta$), mate choice parameters of different female types interact in a systematic non-linear way and determine thresholds that separate the maintenance and loss of sexual trait polymorphisms. These same thresholds also determine the strong or fixed divergence of mating traits.

These results have strong implications for population divergence and speciation because excursions both above U and below L will result in fixation of different sexual trait alleles. If a polymorphic species shows geographical variation in mate choice parameters, then sexual traits can diverge in opposite directions if polymorphic populations happen to fall on the opposite sides of the polymorphic zone in figure 4.1B. However, populations are likely to remain polymorphic if they are anywhere inside the polymorphic zone.
Colour polymorphisms can be under NFD due to frequency-dependent predation. Colour polymorphic species can also show geographical differences in mate preferences. For example, female preferences for colour patterns vary among guppy populations (Endler and Houde 1995; Brooks 2002). In such cases, when NFD is weak and when different female types show weak or moderate mate preferences within populations, then small differences in mate choice parameters among populations are enough to cause complete divergence in mating traits. However, as NFD becomes stronger, threshold boundaries gradually move apart (Figure 4.2). In such cases, strong differences in mate choice parameters are necessary to cause complete divergence in sexual traits.

When both sexes are affected by NFD, geographical differences in mate choice parameters can cause strong divergence in sexual traits as long as NFD is below a certain threshold value (NFD threshold strength is approximately 0.48). If NFD strength is beyond this threshold, then sexual traits cannot diverge. In such cases, populations remain polymorphic irrespective of differences in mate choice parameters.

Interactions between NFD and selective mating can significantly alter the resilience of polymorphic populations in the face of fluctuations in mate choice parameters. For a given NFD regime, polymorphic populations are more likely to lose polymorphisms in the face of fluctuations in mate choice parameters when NFD is weak. When different types of females show weak mating preferences then resilience of polymorphisms is low.

For a given NFD value, polymorphic populations are less likely to lose polymorphisms due to perturbations in mate preferences if different types of females show
strong mate preferences within populations. In such cases, polymorphic populations sit far away from threshold boundaries (higher resilience) and therefore larger perturbations in mate choice parameters is required to cross these threshold boundaries. Therefore, polymorphic populations can remain polymorphic for a long time even if NFD is weak but only if different types of females show strong mate preferences. Early stages of speciation could stall under such scenarios, and further parametric changes may need to evolve before speciation completes.

4.5 Summary

- In many cases, ecologically-driven frequency-dependent selection (NFD) can affect sexually selected traits. In such cases, NFD interacts with mate choice parameters to either maintain polymorphism or cause fixed divergence in sexually selected traits.

- For a given NFD intensity, mate choice parameters interact with each other in a systematic non-linear way and determine thresholds that separate the maintenance and loss of sexual trait polymorphisms. These thresholds can be determined approximately by the quadratic model.

- Polymorphic populations diverge in frequencies but remain polymorphic if they are within the two thresholds. However, polymorphic populations strongly diverge in opposite directions if they fall outside the zone and sexual traits show fixed divergence in such cases.

- Sexual trait polymorphisms show greater resilience to potentially large perturbations in mate choice parameters when NFD and mate preferences are strong. Consequently, in the presence of large perturbations in mate choice parameters, sets of polymorphic
populations with strong NFD and strong mate preferences are less likely to show fixed divergence in sexually selected traits than populations with weak NFD and weak mate preferences.

- When both sexes are affected by ecologically-driven NFD, differences in mate preferences among isolated populations can cause fixed divergence in sexual traits if and only if NFD is below a certain threshold value (NFD threshold strength is approximately 0.48). Once NFD crosses this threshold value, difference in mate preferences cannot cause complete divergence in sexually selected traits anymore. In such cases, polymorphic populations with the strong difference in mate choice parameters and NFD less than approximately 0.48 to 0.5 are more likely to diverge in sexually selected traits than sets of polymorphic populations with NFD more than 0.48 to 0.5.

- For a given NFD strength, strong differences in mate preferences are more likely to produce complete divergence in sexual traits among polymorphic populations in species in which trait expression is sex-limited than species in which trait is expressed in both males and females.

4.6 Conclusion

Mate choice parameters and ecological factors determining negative frequency dependence interact in ways not predictable from considering either alone, and the persistence of polymorphism and divergence in sexual traits are contingent on the details of the interaction between NFD factors and mate choice parameters and not just on their individual effects.
Chapter 5: Polymorphism maintenance and divergence of sexual traits affected by frequency-dependent predation when mate preferences are under directional selection

5.1 Abstract

The maintenance of genetic variation for sexually selected traits is something of a puzzle especially when mate preferences are under directional selection. Here I show that ecologically-driven negative frequency-dependent selection (NFD) and selective mating can interact to maintain sexual trait polymorphism even though direct selection on mate preferences (DNS) is classically thought to promote the loss of sexual trait polymorphism. When DNS is weak, and NFD is weak or moderate, then NFD interacts with mate choice to either maintain sexual trait polymorphisms or cause their fixed divergence. In such cases, combinations of mate choice parameters define thresholds between the maintenance and loss of polymorphisms. When DNS is moderate or strong then interactions between NFD and mate choice cannot produce a fixed divergence in sexual traits. In such cases, populations can diverge in allele frequencies but either remain polymorphic or can become monomorphic. Additionally, I show that polymorphic populations cannot diverge in opposite directions when machinery underlying mate preferences is not sex-limited (for example when mate preferences are co-opted due to sensory bias). In such cases, interactions between NFD and mate choice parameters cannot produce fixed divergence in sexually selected traits among isolated polymorphic populations irrespective of DNS strength and difference in mate preferences.
5.2 Introduction

Substantial genetic variation has been reported for sexual traits (Pomiankowski and Moller 1995); but its maintenance is somewhat a puzzle (Kirkpatrick and Ryan 1991; Kotiaho et al. 2008) because even weak directional selection on mate preferences is expected to deplete variation in sexually selected traits (Kokko et al. 2006a; Prum 2010). Although sexually selected traits evolve in an ecological context, selective mating is often considered in isolation from other ecological processes. Here, I examine how ecologically-driven negative frequency-dependent selection (NFD) and selective mating interact and maintain sexual trait polymorphisms when mate preferences are under directional natural selection (DNS).

In all NFD models considered in chapter 4, mate preferences are not affected by any direct selection. However, in nature, sexual traits can be under ecologically-driven NFD and mate preferences can be simultaneously under DNS, independent of mate choice, caused by the physical environment. For example, in a given environment, colour polymorphisms can be under frequency-dependent predation (Clarke 1962; Allen 1972; Cooper 1984; Endler 1988; Bond and Kamil 1998; Punzalan et al. 2005; Ishii and Shimada 2010) and machinery responsible for mate choice can be under DNS, independent of mate choice. For example, polymorphic guppy males show frequency-dependent survivorship (Olendorf et al. 2006) and visual system that affects guppy mate choice can be under selection caused by the environment, independent of the mate choice (Endler 1991; Sandkam et al. 2016). Though it is hypothesized that ecologically-driven NFD can retain sexual trait polymorphisms when mate preferences are under DNS (Maan and Seehausen 2011), its evolutionary dynamics remain unexplored.
I will explore this using three different models. In the first model, only males are affected by ecologically-driven NFD whereas females are under the DNS independent of mate choice. In the second model, males are also affected by DNS in addition to NFD. Finally, in the third model, both males and females are affected by ecologically-driven NFD and additionally, females are affected by DNS. In all models, I will identify thresholds that separate the maintenance and loss of sexual trait polymorphisms in variable environments, and conditions for loss also indicate the possibility of very strong divergence in sexually selected traits.

5.3 Models and Results

5.3.1 Model 5.1: Males are affected by NFD and females are affected by DNS

As in previous chapters, consider a haploid population showing polymorphism in both sexual traits and in mating preferences. Assume locus T controls mating traits and the unlinked locus P controls female preferences for traits. Let each locus have two alleles which correspond to different mating traits T₁, T₂ and female preferences P₁, P₂ respectively. I assume sexual traits are affected by ecologically-driven negative frequency-dependence (NFD) such as frequency-dependent predation. Additionally, I assume female preference is under the directional natural selection (DNS) caused by the environment independent of mate choice. Aside from this modification, the model is identical to model 4.1 in chapter 4.

Let $m₁, m₂, m₃$ and $m₄$ be the frequencies of T₁P₁, T₁P₂, T₂P₁ and T₂P₂ zygotes in males and $f₁, f₂, f₃$ and $f₄$ be their frequencies in females. Let $β$ be the strength of NFD.
Consequently, the fitness measures of male genotypes are $W_{T_1P_1} = 1 - \beta m_1$; $W_{T_2P_1} = 1 - \beta m_2$; $W_{T_2P_2} = 1 - \beta m_3$; $W_{T_2P_2} = 1 - \beta m_4$. Let NFD on mating traits occur before mating; this alters the frequencies of males available for selective mating. Frequencies of males available for mating after NFD are $m_1' = \frac{m_1(1-\beta m_1)}{W_{\text{males}}}$; $m_2' = \frac{m_2(1-\beta m_2)}{W_{\text{males}}}$; $m_3' = \frac{m_3(1-\beta m_3)}{W_{\text{males}}}$; $m_4' = \frac{m_4(1-\beta m_4)}{W_{\text{males}}}$. where $W_{\text{males}} = [(m_1(1 - \beta m_1)) + (m_2(1 - \beta m_2)) + (m_3(1 - \beta m_3)) + (m_4(1 - \beta m_4))]$.

Let $P_2$ females have a disadvantage such that the $P_2$ viability is $1 - \gamma$ relative to $P_1$ females; $\gamma$ is the viability selection coefficient ($0 \leq \gamma \leq 1$). Let DNS on females occur before mating. Frequencies of females available for mating after the DNS are $f_1' = \frac{f_1}{W_{\text{females}}}$; $f_2' = \frac{f_2(1 - \gamma)}{W_{\text{females}}}$; $f_3' = \frac{f_3}{W_{\text{females}}}$; $f_4' = \frac{f_4(1 - \gamma)}{W_{\text{females}}}$; where $W_{\text{females}} = [f_1 + f_2(1 - \gamma) + f_3 + f_4(1 - \gamma)]$.

As in previous models, let the relative preference of $P_1$ females for $T_1$ males be 1 and her preference for $T_2$ males be $1 - \alpha_1$. Similarly, let the preference of $P_2$ females for $T_2$ males be 1 and her preference for $T_1$ males be $1 - \alpha_2$. $\alpha_1$ and $\alpha_2$ are sexual selection coefficients. New mating frequencies after NFD and DNS can be obtained by replacing $m_1, m_2, m_3, m_4$ respectively with $m_1', m_2', m_3', m_4'$ and by replacing $f_1, f_2, f_3, f_4$ with $f_1', f_2', f_3', f_4'$ in Table 3.1 (chapter 3). Similarly, zygote frequencies in the next generation can be obtained by substituting $m_1', m_2', m_3', m_4'$ for $m_1, m_2, m_3, m_4$ and $f_1, f_2, f_3, f_4$ with $f_1', f_2', f_3', f_4'$ in equations 3.1, 3.2, 3.3 and 3.4 (see section 3.6 Supplementary Information from chapter 3). Generations are assumed to be discrete and non-overlapping.

Recurrence equations for genotype frequencies were solved and equilibrium $T_1$ frequencies were computed numerically by iterating the equations for 1000 generations.
using MATLAB version 2015b. I found this to be more than sufficient time for populations to attain stable equilibria.

As in previous models from chapter 4, to determine the polymorphic zone and boundaries (U and L), I first computed the equilibrium $T_1$ frequency ($T_1$ frequency after 1000 generations) for all possible combinations of $\alpha_1$ and $\alpha_2$ for constant set of starting $P_1$ and $T_1$ frequencies. Polymorphic zone is an attraction basin for polymorphic equilibria; it represents all combinations of $\alpha_1$ and $\alpha_2$ that produce equilibrium $T_1$ frequencies between 0.001 and 0.999 ($0.001 < T_1$ (equilibrium frequency) $< 0.999$). To compute U (the boundary separating the polymorphic zone and the attraction basin for $T_1$ fixation), I identified the threshold $\alpha_1$ for the entire range of $\alpha_2$ such that any change in $\alpha_1$ above the threshold will result in $T_1$ fixation i.e. $T_1$ (equilibrium frequency) $> 0.999$. Similarly, to compute L (the boundary separating the polymorphic zone and the attraction basin for $T_2$ fixation), I identified the threshold $\alpha_1$ for the entire range of $\alpha_2$ such that any change in the $\alpha_1$ below this threshold will result in $T_1$ loss i.e. $T_1$ (equilibrium frequency) $< 0.001$.

I will illustrate results of this model under weak (Figure 5.2A), moderate (Figure 5.3A) and strong (Figure 5.4A) DNS. For weak DNS, I illustrate the results with DNS $\gamma=0.1$, for moderate DNS $\gamma=0.3$ and for strong DNS $\gamma=0.7$.

**Weak DNS**

If DNS is weak ($\gamma=0.1$), then for a given strength of NFD ($\beta$), combinations of $\alpha_1$ and $\alpha_2$ that maintain sexual trait polymorphisms form a zone in the $\alpha_1$-$\alpha_2$ state space with two distinct threshold boundaries (like model 4.1 in chapter 4). Figure 5.1A and B show
this general result when $\gamma=0.1$ and $\beta=0.2$. Note that even though Figure 5.1 shows the result for a constant set of $P_1$ and $T_1$ starting frequencies (starting $T_1$ frequency = 0.5 and starting $P_1$ frequency = 0.5), same result holds true for the entire range of starting frequencies of $T_1$ and $P_1$.

As in previous models, I refer to the upper (U) and lower (L) threshold boundaries by where they intersect the $\alpha_1$ axis (Figure 5.1B). These boundaries separate very different evolutionary outcomes. Populations above U fix $T_1$, whereas populations below L lose $T_1$ (i.e. $T_2$ is fixed). Populations with $\alpha_1$-$\alpha_2$ combinations anywhere between U and L maintain sexual trait polymorphisms. It is important to note that if populations fall inside the polymorphic zone, they remain polymorphic but diverge in frequencies even if not going to fixation and loss. If populations happen to fall off the opposite boundaries of the polymorphic zone in Figure 5.1B then populations diverge completely and frequencies go to fixation and loss, and since this is related to mate preferences, speciation could result. I refer to this divergence (fixation or loss of sexual trait allele) as strong or fixed divergence.

The strengths of NFD ($\beta$) and DNS ($\gamma$) have interacting effects and this determines changes in the shape of U and L hence the extent and shape of the polymorphic zone. When DNS ($\gamma$) is weak, U and L gradually expand as the strength of NFD ($\beta$) increases (see the expansion of U and L in Figure 5.2A), resulting in an expansion of the polymorphic zone. L disappears when NFD ($\beta$) < DNS ($\gamma$). This is because when NFD < DNS, the disadvantageous preference allele ($P_2$) cannot fix its matching trait allele ($T_2$) and L disappears.
In order to describe changes in U and L as a function of \( \beta \), I used exponential \((y=a e^{bx} + ce^{dx})\) and quadratic \((y=p_1 x^2 + p_2 x + p_3)\) functions to consistently describe non-linear U and L boundaries, respectively (Figure 5.5). In these functions, \( y \) represents either U or L and \( x \) represents the strength of \( \alpha_2 \). If these results are compared with the model 4.1 (see Figure 4.3 in chapter 4) then it is clear that the weak DNS on mate preferences alters the shape of U (from quadratic function to exponential function) but it has little effect on the shape of L. For a given \( \alpha_2 \), any value of \( \alpha_1 \) above the upper threshold value of \( \alpha_1 \) will result in the fixation of \( T_1 \). Any value of \( \alpha_1 \) below the lower threshold value of \( \alpha_1 \) will result in the loss of \( T_1 \) i.e. fixation of \( T_2 \). Estimated parameters for the fitted models are given in supplementary information (SI) (Table 5.1 and Table 5.2). Using the parameters estimated by the exponential and quadratic models (Table 5.1 and Table 5.2), we can approximately estimate the form of U and L when DNS is weak.
Figure 5.1: Joint effects of NFD, DNS and mate choice parameters on the maintenance of polymorphisms when males are under NFD and females are under DNS (i.e. model 5.1). (A) Surface plot of $T_1$ equilibrium frequency as a function of $\alpha_1$ and $\alpha_2$ when DNS is weak ($\gamma=0.1$) and NFD ($\beta=0.2$). (B) Phase map showing polymorphic zone, upper (U) and lower (L) boundaries when DNS is weak ($\gamma=0.1$) and $\beta=0.2$. (C) Surface plot of $T_1$ equilibrium frequency as a function of $\alpha_1$ and $\alpha_2$ for moderate DNS ($\gamma=0.3$) when $\beta=0.7$. (D) Phase map showing polymorphic zone and upper (U) polymorphic boundary when $\gamma=0.3$ and $\beta=0.7$. Note that these results are when starting $T_1$ frequency=0.5 and starting $P_1$ frequency=0.5. However, same result holds true for the entire range of $P_1$ and $T_1$ starting frequencies. Solid black lines on surface plots are thresholds that separate the maintenance and loss of polymorphism (either fixation or loss of $T_1$ allele).
Figure 5.2: Changes in the upper (U) and lower (L) threshold boundaries as a function of $\beta$ (NFD strength) when DNS is weak ($\gamma=0.1$). (A) Changes in U and L as a function of $\beta$, when only males are affected by NFD and females, are affected by DNS (model 5.1). (B) Changes in U and L as a function of $\beta$, when males are affected by NFD and females, are affected by both NFD and DNS (model 5.3). Each colour represents a pair of U (solid line) and L (dashed line) for a unique combination of $\beta$ and $\gamma$. Populations anywhere between U and L maintain polymorphisms in sexually selected traits whereas populations outside U and L lose polymorphisms. For a given $\alpha_2$, any value of $\alpha_1$ above U will result in the fixation of $T_1$. Any value of $\alpha_1$ below the L will result in the loss of $T_1$ i.e. fixation of $T_2$. 
**Moderate and strong DNS**

When DNS is moderate or strong, combinations of $\alpha_1$ and $\alpha_2$ that maintain sexual trait polymorphisms do not form a zone in the $\alpha_1$-$\alpha_2$ state space with two distinct boundaries. If DNS is moderate or strong, then, for a given strength of DNS ($\gamma$), the lower threshold ($L$) is lost irrespective of the strength of NFD ($\beta$). Consequently, when DNS is moderate or strong then there are only two possible outcomes: fixation of advantageous sexual trait allele (fixation of $T_1$) or the maintenance of polymorphism in $T$. Figure 5.1 C and D describe this general result when $\gamma=0.3$ and $\beta=0.7$. Note that even though Figure 5.1 shows the result for a constant set of $P_1$ and $T_1$ starting frequencies (starting $T_1$ frequency $=0.5$ and starting $P_1$ frequency $=0.5$), same result holds true for the entire range of starting frequencies of $T_1$ and $P_1$. When directional selection on females is strong, the disadvantageous female allele ($P_2$ females) cannot fix its matching male allele ($T_2$ males), irrespective of mate choice strengths ($\alpha_1$, $\alpha_2$) and NFD ($\beta$) parameters and, $L$ is always lost.

For a given strength of DNS ($\gamma$), the shape of $U$ changes systematically as a function of NFD strength ($\beta$) (Figure 5.6). $U$ remains linear when NFD ($\beta$) << DNS ($\gamma$) (see the horizontal black line in Figure 5.3A, Figure 5.4A and blue line in the Figure 5.4A). This suggests that when NFD ($\beta$) << DNS ($\gamma$), polymorphism in $T$ is maintained if and only if NFD is stronger than the mate preference strength of advantageous $P_1$ females (i.e. when $\beta > \alpha_1$). If $\beta < \alpha_1$ then polymorphism in $T$ is lost and $T_1$ (advantageous trait allele) is fixed irrespective of $\alpha_1$ and $\alpha_2$. 
U becomes non-linear when NFD ($\beta$) $\gg$ DNS ($\gamma$) (see red and blue curves in the Figure 5.3A and red curve in Figure 5.4A). For a given DNS, Table 5.3 shows the approximate strength of NFD ($\beta$) where U flips from a line to a curve. Note that U always follow the exponential function ($y=ae^{hx} + ce^{dx}$) when non-linear (Table 5.2 in the supplementary materials gives the parameters estimated by the exponential model when DNS is moderate and strong).

U becomes non-linear when NFD ($\beta$) $\gg$ DNS ($\gamma$) because, for a given strength of DNS, strong balancing selection (NFD) can maintain polymorphism in T despite strong $\alpha_1$ (mate preference of advantageous P$_1$ females which is expected to fix T$_1$). In such cases, NFD balances the directional selection on sexual traits. Thus, for a given $\alpha_2$ (mate preference of disadvantageous P$_2$ females), stronger $\alpha_1$ (mate choice strength of advantageous P$_1$ females) is needed to lead populations to T$_1$ fixation when balancing selection on T is weaker compared to directional selection on P. As a result, U becomes non-linear when NFD $\gg$ DNS. Whether U is linear or not is biologically important because the linear U implies that $\alpha_2$ does not affect the maintenance of polymorphism. Polymorphism is maintained if and only if $\beta > \alpha_1$, irrespective of $\alpha_2$ (i.e. when NFD is stronger than mate preference strength of advantageous P$_1$ females irrespective of mate preference strength of P$_2$ females). However, non-linear U implies that the maintenance of polymorphism in T is contingent on the relative values of $\alpha_1$ and $\alpha_2$. 


Figure 5.3: Changes in the upper threshold boundary (U) as a function of $\beta$ (NFD strength) when DNS is moderate ($\gamma=0.3$). (A) Changes in U for model 5.1. (B) Changes in U for model 5.2. (C) Changes in U for model 5.3. Note the changes in U within a model as well between models. Also, note that any combination of $\alpha_1$ and $\alpha_2$ below U maintain sexual trait polymorphisms whereas any $\alpha_1-\alpha_2$ combination above U leads to the fixation of $T_1$ (advantageous trait allele).
Figure 5.4: Changes in the upper threshold boundary (U) as a function of $\beta$ (NFD strength) when DNS is strong ($\gamma=0.7$). (A) Changes in U for model 5.1. (B) Changes in U for model 5.2. (C) Changes in U for model 5.3. Note the changes in U within a model as well between models. Also, note that any combination of $\alpha_1$ and $\alpha_2$ below U maintain sexual trait polymorphisms whereas any $\alpha_1$-$\alpha_2$ combination above U leads to fixation of $T_1$ (advantageous trait allele).
5.3.2 Model 5.2: Females are affected by DNS and males are affected by NFD and DNS

In many cases, the factors affecting mate preference are not sex limited. For example, when mate preferences are co-opted and sensory system properties determine mate preferences (Ryan 1990; Endler 1992; Endler and Basolo 1998). In such cases, if sensory systems are under DNS due to the physical environment then both males and females can be under DNS independent of mate choice. In this section, I model this scenario. Aside from this modification, the model is identical to the model 5.1.

Let $m_1$, $m_2$, $m_3$ and $m_4$ be the frequencies of $T_1P_1$, $T_1P_2$, $T_2P_1$ and $T_2P_2$ zygotes respectively in males and $f_1$, $f_2$, $f_3$ and $f_4$ be their frequencies in females within the population. Let $\beta$ be the strength of NFD and $\gamma$ be the strength of DNS which is frequency-independent selection acting on males carrying $P_2$ allele. Let males carrying $P_2$ allele have a disadvantage such that the viability of males with $P_2$ alleles is $1-\gamma$ relative to males carrying $P_1$ allele; $\gamma$ is the frequency-independent viability selection coefficient ($0 \leq \gamma \leq 1$).

Consequently, the fitness measures of male genotypes are $W_{T_1P_1} = 1 - \beta m_1$; $W_{T_1P_2} = 1 - \beta m_2 - \gamma$; $W_{T_2P_1} = 1 - \beta m_3$; $W_{T_2P_2} = 1 - \beta m_4 - \gamma$. Let NFD and DNS occur before selective mating; this alters the frequencies of males available for mating.

Frequencies of males available for mating after NFD and DNS are $m_1' = \frac{m_1(1-\beta m_1)}{W_{mates}}$; $m_2' = \frac{m_2(1-\beta m_2 - \gamma)}{W_{mates}}$; $m_3' = \frac{m_3(1-\beta m_3)}{W_{mates}}$; $m_4' = \frac{m_4(1-\beta m_4 - \gamma)}{W_{mates}}$; where $W_{mates} = [(m_1 (1 - \beta m_1)) + (m_2 (1 - \beta m_2 - \gamma)) + (m_3 (1 - \beta m_3)) + (m_4 (1 - \beta m_4 - \gamma))]$.
Let \( P_2 \) females have a disadvantage such that the \( P_2 \) viability is \( 1 - \gamma \) relative to \( P_1 \) females; \( \gamma \) is the viability selection coefficient \((0 \leq \gamma \leq 1)\). Let DNS on females occur before mating. Frequencies of females available for mating after the DNS are:

\[
\begin{align*}
\frac{f_1'}{W_{\text{females}}} &= \frac{f_2(1-\gamma)}{W_{\text{females}}} ; \\
\frac{f_3'}{W_{\text{females}}} &= \frac{f_4(1-\gamma)}{W_{\text{females}}}
\end{align*}
\]

where \( W_{\text{females}} = [f_1 + f_2(1 - \gamma) + f_3 + f_4(1 - \gamma)] \).

New mating frequencies after NFD and DNS can be obtained by replacing \( m_1, m_2, m_3 \) and \( m_4 \) respectively with \( m_1', m_2', m_3', m_4' \) and by replacing \( f_1, f_2, f_3, f_4 \) with \( f_1', f_2', f_3', f_4' \) in the Table 3.1 (refer chapter 3). Similarly, zygote frequencies in the next generation after the selective mating can be obtained by substituting \( m_1', m_2', m_3', m_4' \) for \( m_1, m_2, m_3 \) and \( m_4 \) and \( f_1, f_2, f_3 \) and \( f_4 \) with \( f_1', f_2', f_3', f_4' \) in equations 3.1, 3.2, 3.3 and 3.4 (refer section 3.6 Supplementary Information from chapter 3). Note that the computation procedure to estimate attraction basins and basin boundary remains same as explained in the model 5.1.

When both sexes are affected by DNS, the effective DNS intensity is doubled. In such cases, the disadvantageous females \((P_2 \) females\) can’t fix its matching males \((T_2 \) males\), irrespective of mate choice strengths \((\alpha_1, \alpha_2)\) and NFD \((\beta)\) parameters. Therefore, L is lost for the entire range of DNS.

When DNS \((\gamma) \gg \) NFD \((\beta)\), polymorphism in T is maintained if and only if NFD is stronger than the preference strength of advantageous females \((P_1)\) \((i.e. \beta > \alpha_1)\). If \( \beta < \alpha_1 \) then advantageous preference allele fixes its preferred trait allele \((T_1)\) irrespective of \( \alpha_1-\alpha_2 \) combinations. This result is consistent with the model 5.1 except that DNS is doubled. However, when DNS intensity is doubled, the same combinations of \( \beta \) and \( \gamma \) that
produce non-linear U in model 5.1, now produce linear U in this model (compare blue and red curves in Figure 5.3A and Figure 5.3B). The difference between effects of expression in one or both sexes is simply a factor of 2. Note that even though Figure 5.3 shows the result for a constant set of P₁ and T₁ starting frequencies (starting T₁ frequency = 0.5 and starting P₁ frequency = 0.5), same result holds true for the entire range of starting frequencies of T₁ and P₁.

5.3.3 Model 5.3: Females are affected by NFD and DNS whereas males are affected only by NFD.

In this model, I will examine the dynamics of threshold boundaries when both sexes are affected by NFD and additionally, females are affected by DNS. Note the difference between previous models and this model. Only males were affected by NFD in all the previous models. However, in this model, I assume that the expression of a sexually selected trait is not sex limited and consequently both sexes are affected by NFD. This scenario can occur when the expression of sexually selected traits is not sex limited. For example, many colour polymorphic species show expression of colour in both sexes and also show colour based assortative mating within populations (Wellenreuther et al. 2014). Consequently, in such cases, ecologically-driven colour-based NFD can affect both sexes.

Let m₁, m₂, m₃ and m₄ be the frequencies of T₁P₁, T₁P₂, T₂P₁ and T₂P₂ zygotes respectively in males and f₁, f₂, f₃ and f₄ be their frequencies in females within the population. The fitness measures of male genotypes are \( W_{T₁P₁} = 1 - \beta m₁ \); \( W_{T₁P₂} = 1 - \beta m₂ \); \( W_{T₂P₁} = 1 - \beta m₃ \); \( W_{T₂P₂} = 1 - \beta m₄ \). Let NFD on mating traits occur before
mating; this alters the frequencies of males available for selective mating. Frequencies of males available for mating after NFD are
\[ m_1' = \frac{m_1(1-\beta m_1)}{\bar{W}_{\text{males}}}; \quad m_2' = \frac{m_2(1-\beta m_2)}{\bar{W}_{\text{males}}}; \quad m_3' = \frac{m_3(1-\beta m_3)}{\bar{W}_{\text{males}}}; \quad m_4' = \frac{m_4(1-\beta m_4)}{\bar{W}_{\text{males}}}; \quad \text{where} \quad \bar{W}_{\text{males}} = \left[ (m_1(1 - \beta m_1)) + (m_2(1 - \beta m_2)) + (m_3(1 - \beta m_3)) + (m_4(1 - \beta m_4)) \right]. \]

Females are affected by NFD and DNS. Let \( P_2 \) females have a disadvantage such that the \( P_2 \) viability is \( 1 - \gamma \) relative to \( P_1 \) females; \( \gamma \) is the DNS coefficient \( (0 \leq \gamma \leq 1) \). Let NFD and DNS occur before mating. The fitness measures of female genotypes are
\[ W_{T_1P_1} = (1 - \beta f_1), \quad W_{T_2P_2} = (1 - \beta f_2 - \gamma), \quad W_{T_2P_1} = (1 - \beta f_3) \quad \text{and} \quad W_{T_2P_2} = (1 - \beta f_4 - \gamma). \]
Frequencies of females available for mating after NFD and DNS are
\[ f_1' = \frac{f_1(1-\beta f_1)}{\bar{W}_{\text{females}}}; \quad f_2' = \frac{f_2(1-\beta f_2-\gamma)}{\bar{W}_{\text{females}}}; \quad f_3' = \frac{f_3(1-\beta f_3)}{\bar{W}_{\text{females}}}; \quad f_4' = \frac{f_4(1-\beta f_4-\gamma)}{\bar{W}_{\text{females}}}; \quad \text{where} \quad \bar{W}_{\text{females}} = [f_1(1 - \beta f_1) + f_2(1 - \beta f_2 - \gamma) + f_3(1 - \beta f_3) + f_4(1 - \beta f_4 - \gamma)]. \]

New mating frequencies after viability selection can be obtained by replacing \( m_1, m_2, m_3 \) and \( m_4 \) respectively with \( m_1', m_2', m_3', m_4' \) and by replacing \( f_1, f_2, f_3, f_4 \) with \( f_1', f_2', f_3', f_4' \) in Table 3.1 from chapter 3. Similarly, zygote frequencies in the next generation after the selective mating can be obtained by substituting \( m_1', m_2', m_3', m_4' \) for \( m_1, m_2, m_3 \) and \( m_4 \) and \( f_1, f_2, f_3 \) and \( f_4 \) with \( f_1', f_2', f_3' \) and \( f_4' \) in equations 3.1, 3.2, 3.3 and 3.4 (refer section 3.6 Supplementary Information from chapter 3). Note that the computation procedure to estimate attraction basins and basin boundary remains same as explained in the model 5.1.

When DNS is weak and when both sexes are affected by NFD, the rate at which U and L move apart is approximately doubled (compare blue curves of U and L in Figure
5.2 A and Figure 5.2B). When DNS is moderate or strong, L disappears irrespective of NFD strength. In such cases, the rate of expansion in U is doubled (compare the black horizontal line in Figure 5.2B, C and between Figure 5.3B and C).

I will now summarize all the results of three models.

5.4 Summary

- Negative frequency-dependent natural selection (NFD) and selective mating can interact to either maintain polymorphism or cause divergence in sexually selected traits, even when mate preferences are under directional selection (DNS), classically thought to promote the loss of sexual trait polymorphism.

- If NFD is weak or moderate and DNS is weak then differences in mate choice parameters can either maintain sexual trait polymorphisms or cause their complete divergence. Populations remain polymorphic if they fall within thresholds (Figure 5.1A). In contrast, sexual traits can strongly diverge in opposite directions if populations are outside and on the opposite sides of the polymorphic zone. This result has strong implications for divergence in sexual traits because excursions both above the upper and below the lower thresholds will result in fixation of different sexual trait alleles.

- For a given NFD intensity, differences in mate choice parameters can produce a complete divergence in sexual traits as long as DNS on mate preferences is weak. However, it is important to note that, when NFD is strong, then differences in mate choice parameters cannot produce fixed divergence among isolated populations even if DNS is weak. Therefore, for a given combination of mate choice
parameters, sets of populations with weak NFD and weak DNS are more likely to diverge in sexual traits than sets of populations with any other NFD-DNS combination.

- When DNS is moderate or strong, then differences in mate choice parameters cannot produce complete divergence in sexual traits among populations irrespective of NFD strength.
- Therefore, moderate or strong DNS on female preference prevents complete population divergence and subsequent speciation when sexual traits are under balancing natural selection (NFD). Populations either remain polymorphic in sexual traits or the advantageous trait (trait matching with advantageous female preference) is fixed within populations. Whether polymorphic populations maintain or lose sexual trait polymorphisms in a given DNS regime entirely depends on the relative magnitudes of NFD and mate choice parameters. Thus, for a given constant environment, when DNS is moderate or strong and when sexual traits are under balancing ecological selection, isolated polymorphic populations cannot diverge in opposite directions even if populations show strong differences in mate preferences.
- If NFD \ll DNS, then sexual trait polymorphism is maintained if and only if \( \beta > \alpha_1 \) i.e. if NFD > mate preference strength of advantageous female type. This is true for all three models.
- If the machinery underlying mate preference is not sex-limited (model 5.2) then, the differences in mate choice parameters cannot produce fixed divergence in sexual traits even if DNS on preference is very weak. In such cases, polymorphic
populations cannot diverge in opposite directions irrespective of differences in mate preferences. Such scenarios can arise in various contexts; for example when mate preferences are co-opted due to sensory bias. In such cases, if sexual traits are under ecologically-driven NFD, then NFD and mate choice interactions cannot produce a complete divergence in sexual traits among populations even if mate preferences are under weak DNS and even if mate choice parameters vary greatly among isolated populations. Therefore, population divergence is less likely in such cases.

5.5 Conclusion

Ecologically-driven negative frequency-dependent natural selection can interact with selective mating to either maintain sexual trait polymorphism or cause fixed divergence even when mate preferences are under directional natural selection which is expected to destroy sexual trait polymorphisms. However, whether populations remain polymorphic or not depends on the relative strengths of NFD, DNS and mate choice parameters. Moderate and strong directional selection on mate preferences decreases the chances of strong population divergence. When sexual traits are under NFD, population divergence under moderate and/or strong DNS is impossible even if mate choice parameters dramatically vary among isolated populations. When DNS is strong, populations cannot fix disadvantageous trait for any combinations of mate preferences even if balancing selection is strong within populations. As a result, populations showing strong differences in mate preferences cannot diverge in opposite directions when DNS is
strong and hence chances of strong populations divergence decrease when DNS is strong. In such cases, populations can either remain polymorphic or advantageous trait is fixed within populations.
5.6 Supplementary Information (SI)

Figure 5.5: Fitted exponential \(y = ae^{bx} + ce^{dx}\) and quadratic \(y = p_1x^2 + p_2x + p_3\) functions to the upper (U) and lower (L) boundaries respectively for different strengths of \(\beta\) when males are affected by NFD and females are affected by weak DNS (model 5.1). The fit of the exponential and quadratic functions is excellent.
Table 5.1: Parameters estimated by the exponential model ($y=ae^{bx} + ce^{dx}$) for the upper threshold boundary (U) as a function of NFD strength ($\beta$) when DNS is weak $\gamma=0.1$. The estimated parameters are for model 5.1 where males are affected by NFD and females are affected by DNS. 95% confidence intervals are given in brackets.

<table>
<thead>
<tr>
<th>$\beta$</th>
<th>$a$</th>
<th>$b$</th>
<th>$c$</th>
<th>$d$</th>
<th>$r^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.05</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
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<tr>
<td>0.1</td>
<td>0.09286</td>
<td>0.6349</td>
<td>2.41e-07</td>
<td>15.45</td>
<td>0.9965</td>
</tr>
<tr>
<td></td>
<td>(0.08862, 0.09711)</td>
<td>(0.5359, 0.7339)</td>
<td>(1.029e-07, 3.791e-07)</td>
<td>(14.86, 16.04)</td>
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<tr>
<td>0.2</td>
<td>0.159</td>
<td>1.589</td>
<td>0.007142</td>
<td>5.791</td>
<td>0.9996</td>
</tr>
<tr>
<td></td>
<td>(0.1559, 0.1621)</td>
<td>(1.395, 1.783)</td>
<td>(0.002748, 0.01154)</td>
<td>(5.158, 6.424)</td>
<td></td>
</tr>
<tr>
<td>0.3</td>
<td>0.173</td>
<td>0.9218</td>
<td>0.07242</td>
<td>3.593</td>
<td>0.9995</td>
</tr>
<tr>
<td></td>
<td>(0.1012, 0.2448)</td>
<td>(-0.07563, 1.919)</td>
<td>(-0.002251, 0.1471)</td>
<td>(2.682, 4.505)</td>
<td></td>
</tr>
<tr>
<td>0.4</td>
<td>0.1866</td>
<td>0.3564</td>
<td>0.1451</td>
<td>3.025</td>
<td>0.9997</td>
</tr>
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<td>(0.08634, 0.2869)</td>
<td>(-0.7939, 1.507)</td>
<td>(0.04269, 0.2476)</td>
<td>(2.366, 3.684)</td>
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</tr>
<tr>
<td>0.5</td>
<td>0.1044</td>
<td>-1.719</td>
<td>0.317</td>
<td>2.243</td>
<td>0.9996</td>
</tr>
<tr>
<td></td>
<td>(0.03533, 0.1735)</td>
<td>(-4.112, 0.6736)</td>
<td>(0.2455, 0.3886)</td>
<td>(1.954, 2.533)</td>
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</tr>
<tr>
<td>0.6</td>
<td>0.07131</td>
<td>-3.307</td>
<td>0.4395</td>
<td>1.864</td>
<td>0.9995</td>
</tr>
<tr>
<td></td>
<td>(0.009893, 0.1327)</td>
<td>(-7.671, 1.057)</td>
<td>(0.3753, 0.5036)</td>
<td>(1.63, 2.098)</td>
<td></td>
</tr>
<tr>
<td>0.7</td>
<td>0.04376</td>
<td>-6.272</td>
<td>0.5597</td>
<td>1.552</td>
<td>0.9993</td>
</tr>
<tr>
<td></td>
<td>(0.007747, 0.07978)</td>
<td>(-13.93, 1.382)</td>
<td>(0.5209, 0.5985)</td>
<td>(1.401, 1.704)</td>
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</tr>
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<td>0.8</td>
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<td>0.6649</td>
<td>1.334</td>
<td>0.9991</td>
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<td>(-0.01875, 0.07885)</td>
<td>(-22.37, 7.712)</td>
<td>(0.6134, 0.7164)</td>
<td>(1.137, 1.532)</td>
<td></td>
</tr>
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</table>
Table 5.2: Parameters estimated by the quadratic model \( y=p_1 x^2 + p_2 x + p_3 \) for the lower threshold boundary (L) as a function of NFD strength (\( \beta \)) when DNS is weak \( \gamma=0.1 \). The estimated parameters are for model 5.1 where males are affected by NFD and females are affected by DNS. 95% confidence intervals are given in brackets.

<table>
<thead>
<tr>
<th>( \beta )</th>
<th>( p_1 )</th>
<th>( p_2 )</th>
<th>( p_3 )</th>
<th>( r^2 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.05</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
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<td>-</td>
</tr>
<tr>
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<td>-0.2089</td>
<td>0.9995</td>
</tr>
<tr>
<td></td>
<td>(-0.1298, -0.09608)</td>
<td>(0.4348, 0.4867)</td>
<td>(-0.2186, -0.1992)</td>
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<td>0.6162</td>
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</tr>
<tr>
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<td>(-0.1659, -0.1398)</td>
<td>(0.5963, 0.6361)</td>
<td>(-0.2844, -0.2697)</td>
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<td>0.4</td>
<td>-0.204</td>
<td>0.7802</td>
<td>-0.3849</td>
<td>0.9993</td>
</tr>
<tr>
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<td>(-0.2402, -0.1677)</td>
<td>(0.7228, 0.8377)</td>
<td>(-0.4072, -0.3625)</td>
<td></td>
</tr>
<tr>
<td>0.5</td>
<td>-0.2546</td>
<td>0.9478</td>
<td>-0.5188</td>
<td>0.9991</td>
</tr>
<tr>
<td></td>
<td>(-0.3206, -0.1885)</td>
<td>(0.8374, 1.058)</td>
<td>(-0.5645, -0.4731)</td>
<td></td>
</tr>
<tr>
<td>0.6</td>
<td>-0.1356</td>
<td>0.8059</td>
<td>-0.5295</td>
<td>0.9981</td>
</tr>
<tr>
<td></td>
<td>(-0.308, 0.03673)</td>
<td>(0.5041, 1.108)</td>
<td>(-0.661, -0.3981)</td>
<td></td>
</tr>
<tr>
<td>0.7</td>
<td>0.3782</td>
<td>-0.06815</td>
<td>-0.213</td>
<td>0.9981</td>
</tr>
<tr>
<td></td>
<td>(-0.06893, 0.8252)</td>
<td>(-0.8954, 0.7591)</td>
<td>(-0.5951, 0.169)</td>
<td></td>
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</table>
Figure 5.6: Fitted exponential ($y=a e^{b x} + c e^{d x}$) function to the upper boundary (U) for different strengths of $\beta$ when males are affected by NFD and females are affected by moderate and strong DNS (model 5.1). (A) Moderate DNS ($\gamma=0.3$). (B) Strong DNS ($\gamma=0.7$). The fit of the exponential function is excellent.
Table 5.3: Parameters estimated by the exponential model \(y=ae^{bx}+ce^{dx}\) for the upper threshold boundary (U) as a function of NFD strength \((\beta)\) when DNS is moderate \((\gamma=0.3)\) and strong \((\gamma=0.7)\). The estimated parameters are for model 5.1 where males are affected by NFD and females are affected by DNS. 95% confidence intervals are given in brackets.

<table>
<thead>
<tr>
<th>NFD strength</th>
<th>0.3</th>
<th>0.4</th>
<th>0.5</th>
<th>0.6</th>
<th>0.7</th>
<th>0.8</th>
<th>0.9</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.3</td>
<td>(a=0.2563) (0.2533, 0.2593)</td>
<td>(a=0.3122) (0.3104, 0.3139)</td>
<td>(a=0.3651) (0.3623, 0.3679)</td>
<td>(a=0.42) (0.4123, 0.4277)</td>
<td>(a=0.4724) (0.4559, 0.4889)</td>
<td>(a=0.5255) (0.4949, 0.5562)</td>
<td>(a=0.3575) (0.1581, 0.5568)</td>
</tr>
<tr>
<td>0.4</td>
<td>(b=0.4696) (0.4327, 0.5065)</td>
<td>(b=0.5497) (0.5127, 0.5867)</td>
<td>(b=0.5457) (0.3702, 0.5413)</td>
<td>(b=0.394) (0.2813, 0.5068)</td>
<td>(b=0.3328) (0.195, 0.4706)</td>
<td>(b=0.2752) (0.1061, 0.4444)</td>
<td>(b=-0.5519) (-1.486, 0.3824)</td>
</tr>
<tr>
<td>0.5</td>
<td>(c=7.283e-05) (4.969e-05, 9.597e-05)</td>
<td>(c=0.02283) (0.01367, 0.032)</td>
<td>(c=0.03884) (0.02092, 0.05675)</td>
<td>(c=0.0577) (0.02588, 0.08951)</td>
<td>(c=0.3003) (0.09938, 0.5011)</td>
<td>(d=9.773) (9.436, 10.11)</td>
<td>(d=6.688) (6.458, 6.919)</td>
</tr>
<tr>
<td>0.6</td>
<td>(d=5.051) (4.691, 5.411)</td>
<td>(d=4.387) (3.957, 4.818)</td>
<td>(d=3.885) (3.393, 4.378)</td>
<td>(d=3.501) (2.915, 4.088)</td>
<td>(d=1.857) (1.243, 2.472)</td>
<td>(a=0.4353) (0.4327, 0.438)</td>
<td>(a=0.4768) (0.4741, 0.4795)</td>
</tr>
<tr>
<td>0.7</td>
<td>(a=0.5181) (0.5158, 0.5203)</td>
<td>(a=0.5595) (0.5574, 0.5617)</td>
<td>(a=0.602) (0.6007, 0.6034)</td>
<td>(a=0.624) (0.6217, 0.6268)</td>
<td>(a=0.6597) (0.6574, 0.6620)</td>
<td>(a=0.701) (0.698, 0.704)</td>
<td>(a=0.744) (0.741, 0.747)</td>
</tr>
<tr>
<td>0.8</td>
<td>(b=0.1314) (0.1177, 0.1451)</td>
<td>(b=0.1509) (0.1346, 0.1672)</td>
<td>(b=0.1579) (0.1421, 0.1737)</td>
<td>(b=0.1606) (0.1431, 0.1781)</td>
<td>(b=0.1646) (0.1513, 0.1779)</td>
<td>(b=0.174) (0.1607, 0.1873)</td>
<td>(b=0.184) (0.1706, 0.1974)</td>
</tr>
<tr>
<td>0.9</td>
<td>(c=1.491e-08) (4.879e-09, 2.494e-08)</td>
<td>(c=1.301e-05) (7.978e-06, 1.803e-05)</td>
<td>(c=0.0001407) (0.0000859, 0.0001955)</td>
<td>(c=0.0005115) (0.0003655, 0.0006575)</td>
<td>(c=0.000997) (0.0001778, 0.001216)</td>
<td>(d=17.65) (16.95, 18.34)</td>
<td>(d=10.97) (10.56, 11.37)</td>
</tr>
</tbody>
</table>
|              | \(d=8.629\) (8.322, 8.936) | \(d=7.346\) (7.042, 7.651) | \(r^2 = 0.9997\) | \(r^2 = 0.9997\) | \(r^2 = 0.9997\) | \(r^2 = 0.9997\) | \(r^2 = 0.9997\) | \(r^2 = 0.9997\)
Table 5.4: The Approximate strength of NFD ($\beta$) that changes the shape of upper boundary (U) from a line to the exponential curve. I will call this strength of NFD as ‘critical $\beta$’. For a given DNS strength ($\gamma$), U becomes non-linear if NFD strength ($\beta$) > critical $\beta$. Note that when $\beta < \text{critical } \beta$, U remains a horizontal line. This is important because the horizontal line in the $\alpha_1$-$\alpha_2$ state space implies that when $\beta < \text{critical } \beta$ then $\alpha_2$ (mate preference strength of disadvantageous $P_2$ females) does not affect the polymorphism maintenance. Polymorphism is maintained if and only if $\beta > \alpha_1$ (mate preference strength of advantageous $P_1$ females), irrespective of $\alpha_2$. Also, note that critical $\beta$ is approximately doubled when DNS intensity is doubled (model 5.2).

<table>
<thead>
<tr>
<th>Strength of DNS ($\gamma$)</th>
<th>Model 5.1</th>
<th>Model 5.2</th>
<th>Model 5.3</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.1</td>
<td>0.1</td>
<td>0.2</td>
<td>0.06</td>
</tr>
<tr>
<td>0.2</td>
<td>0.18</td>
<td>0.36</td>
<td>0.11</td>
</tr>
<tr>
<td>0.3</td>
<td>0.25</td>
<td>0.48</td>
<td>0.16</td>
</tr>
<tr>
<td>0.4</td>
<td>0.3</td>
<td>0.6</td>
<td>0.22</td>
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<tr>
<td>0.5</td>
<td>0.35</td>
<td>0.7</td>
<td>0.27</td>
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<td>0.78</td>
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<td>0.43</td>
<td>0.85</td>
<td>0.37</td>
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<tr>
<td>0.8</td>
<td>0.46</td>
<td>0.92</td>
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Chapter 6: Joint effects of ecology and initial conditions on the evolutionary direction of aposematic traits in variable environments

6.1 Abstract

Current selective conditions and evolutionary history (initial conditions) can influence evolutionary change, yet are rarely considered together, especially when environments are variable. Here I use mate choice models involving traits affected by ecologically-driven positive frequency-dependent selection (PFD) and explore how PFD and sexual selection parameters interact with initial conditions and how their interactions bias the evolutionary outcomes in variable environments. I show that in the absence of selective mating, starting male frequencies determine the subsequent evolutionary direction of aposematic traits irrespective of starting female frequencies. When assortative mating is weaker than PFD, starting male frequencies affect the evolutionary change more than starting female frequencies. Female frequencies begin to affect the evolutionary change as the strength of assortative mating increases. When assortative mating is stronger than PFD, starting female frequencies determine the subsequent evolutionary direction irrespective of starting male frequencies. Consequently, in such environments, perturbations in female gene frequencies are more likely to affect evolutionary change in the polymorphic populations than perturbations in male frequencies. I show that the differences between PFD parameters cannot eliminate the effects of initial conditions in environments where assortative mating is stronger than PFD. These results are true irrespective of differences between effects of aposematic trait expression in one or both sexes.
6.2 Introduction

Current ecology (natural and sexual selection parameters) and initial conditions (historical contingency) can have a profound effect on the subsequent direction of evolution. Relative contribution of ecology and historical contingency to the origin of biological diversity has been extensively discussed (Gould and Lewontin 1979; Mayr 1983; Gould 1989; Travisano et al. 1995; Losos 2010), but their interaction has not explicitly modeled, especially when environments are variable. Here I examine how natural and sexual selection parameters interact with initial conditions and how their interactions bias the direction of evolution in variable environments. I will examine this using models involving traits which are affected by ecologically-driven positive frequency-dependent selection (PFD) and selective mating.

In PFD, fitness increases with the phenotype's frequency or density, and this is characteristic of species with aposematic traits (Allen and Greenwood 1988; Endler 1988; Mallet and Joron 1999; Endler and Mappes 2004; Noonan and Comeault 2009). If a trait’s fitness is positive frequency-dependent then populations typically evolve to one of the alternative stable states depending on where they start in the allele frequency space (Lehtonen and Kokko 2012). Consequently, in such cases, initial conditions (history) significantly affects the subsequent direction of trait evolution (Endler 1988; Endler and Mappes 2004; Lehtonen and Kokko 2012).

Sexual selection can also bias the evolutionary direction of aposematic traits along with frequency-dependent predation (Maan and Cummings 2009). Species with aposematic traits can show selective mating based on the aposematic signal components. For example, Poison dart frogs (*Oophaga pumilio*) use visual cues during
mate choice (Summers et al. 1999; Siddiqi et al. 2004; Maan and Cummings 2008) and females prefer males with matching phenotype (Reynolds and Fitzpatrick 2007).

Classical theory suggests that aposematic traits should have low variance (Endler 1988; Endler and Mappes 2004) yet many species show intraspecific variation in these traits. For example, ladybirds (O'Donald and Majerus 1984), moths (Nokelainen et al. 2011; Gordon et al. 2015) and frogs (Siddiqi et al. 2004; Rojas and Endler 2013). The interactions between deterministic (PFD and sexual selection) and stochastic parameters can jointly maintain intraspecific variation in aposematic traits among populations (Gordon et al. 2015). However, how their interactions bias the evolutionary direction of aposematic traits and maintain variance in these traits, especially in variable environments, remains unclear.

Here, I examine the joint effects of PFD, sexual selection and initial conditions using two models. In the first model, I let aposematic traits be sex limited and only males be affected by PFD. In the second model, I let both males and females express aposematic traits and both are affected by PFD. In both models, I will examine the interactions between PFD, selective mating and initial conditions, and examine how these interactions bias the direction of evolution in variable environments. I will also examine the contribution of selective mating to the maintenance of aposematic trait polymorphisms within populations.

6.3 Models and Results

6.3.1 Model 6.1: Trait expression is male-limited

Consider a haploid population in which both mate preferences and aposematic traits are polymorphic. Let the expression of aposematic traits be male-limited.
Assume locus T controls aposematic traits in males and the unlinked locus P controls female preferences for the T traits. Let each locus have two alleles which correspond to traits \( T_1, T_2 \) and female preferences \( P_1, P_2 \) respectively. I use the standard model of selective mating (Kirkpatrick 1982; Prum 2010) which normally assumes directional viability selection on sexual traits. Instead, here I assume traits are affected by ecologically-driven PFD such as frequency-dependent predation.

Let \( m_1, m_2, m_3 \) and \( m_4 \) be the frequencies of \( T_1 P_1, T_1 P_2, T_2 P_1 \) and \( T_2 P_2 \) zygotes in males and \( f_1, f_2, f_3 \) and \( f_4 \) be their frequencies in females. Let \( \beta_1 \) and \( \beta_2 \) are the PFD coefficients for \( T_1 \) and \( T_2 \) males respectively; the fitnesses of male genotypes are

\[
W_{T_1 P_1} = 1 + \beta_1 m_1; W_{T_1 P_2} = 1 + \beta_1 m_2; W_{T_2 P_1} = 1 + \beta_2 m_3; W_{T_2 P_2} = 1 + \beta_2 m_4.
\]

Let PFD occur before mate choice; this alters the frequencies available for mating. Consequently, the frequencies of male genotypes available for selective mating after NFD are

\[
m_1' = \frac{m_1(1+\beta_1 m_1)}{\bar{W}}, \quad m_2' = \frac{m_2(1+\beta_2 m_2)}{\bar{W}}, \quad m_3' = \frac{m_3(1+\beta_2 m_3)}{\bar{W}}, \quad m_4' = \frac{m_4(1+\beta_4 m_4)}{\bar{W}},
\]

where

\[
\bar{W} = \left[ (m_1(1 + \beta m_1)) + (m_2(1 + \beta m_2)) + (m_3(1 + \beta m_3)) + (m_4(1 + \beta m_4)) \right].
\]

Note that in this model, since there is no PFD on females, the female genotype frequency available for selective mating after PFD is the same as males before PFD or \( f_i' = m_i \).

Let the relative preference of \( P_1 \) females for \( T_1 \) males be 1 and her preference for \( T_2 \) males be \( 1-\alpha_1 \). Similarly, let the preference of \( P_2 \) females for \( T_2 \) males be 1 and her preference for \( T_1 \) males be \( 1-\alpha_2 \). \( \alpha_1 \) and \( \alpha_2 \) are the sexual selection coefficients.

New mating frequencies after PFD can be obtained by replacing \( m_1, m_2, m_3, m_4 \) respectively with \( m_1', m_2', m_3', m_4' \) and by replacing \( f_1, f_2, f_3, f_4 \) with \( f_1', f_2', f_3', f_4' \) in chapter 3, Table 3.1. Similarly, zygote frequencies in the next generation can be obtained by substituting \( m_1', m_2', m_3', m_4' \) for \( m_1, m_2, m_3, m_4 \) and \( f_1, f_2, f_3, f_4 \) with \( f_1' \),
Recurrence equations for genotype frequencies were solved and equilibrium 
T_1 frequencies were computed numerically by iterating equations for 1000 generations 
for all combinations of male-female starting frequencies for a given \( \alpha \) and \( \beta \), using 
MATLAB 2015b. For a given constant \( \alpha \) and \( \beta \), joint initial male-female allele 
frequencies evolve to one of the alternative stable state i.e. T_1 fixation or T_2 fixation 
(Figure 6.1). The distinct boundary separates the regions of initial conditions that lead 
to different evolutionary outcomes i.e. T_1 fixation or T_1 loss i.e. T_2 fixation (see the 
thick black boundary in the Figure 6.1).

To determine the attraction basins for T_1 and T_2 fixation, I first computed 
equilibrium T_1 frequencies (T_1 frequency after 1000 generations) for all possible 
combinations of starting frequencies of T_1 and P_1 for a given constant \( \alpha \) and \( \beta \). 
Attraction basin for T_1 fixation represents the joint T_1, P_1 starting frequencies that 
eventually produce T_1 (equilibrium frequency) > 0.999. Similarly, attraction basin for T_2 
fixation (see the area below L in the Figure 6.1A) represents all possible combinations 
of T_1 and P_1 starting frequencies that eventually produce T_1 (equilibrium frequency) < 0.001. 
To compute basin boundary (the boundary separating two attraction basins) I identified 
the threshold starting frequency of P_1 for the entire range of starting frequencies of T_1 
where T_1 (equilibrium frequency) flips from 0 to 1.

**Results**

When sexually selected traits are affected by ecologically-driven PFD and have 
the consistent mating advantage, then the evolutionary outcome is determined by the 
direction and magnitude of differences between the strength of PFD (\( \beta \)) and the
strength of selective mating ($\alpha$). The relative values of $\alpha$ and $\beta$ affect the position and shape of threshold boundary in T₁-P₁ allele frequency space (Figure 6.1).

In the absence of assortative mating ($\alpha_1=\alpha_2=\alpha=0$), the boundary between attraction basins remains at the T₁ starting frequency=0.5. In such cases starting male frequencies determine the subsequent direction of evolution of T irrespective of starting female frequencies; if the starting T₁ frequency > 0.5 then T₁ is fixed and if starting T₁ frequency < 0.5 then T₁ is lost irrespective of starting female frequencies (Figure 6.1A).

However, as soon as assortative mating $\alpha > 0$, starting female frequencies begin to affect the subsequent direction of trait evolution (compare Figure 6.1A and B noting that the shape of threshold boundary changes from a straight vertical line to a curve). If $\alpha > \beta$ then the threshold boundary collapses to a horizontal line (Figure 6.1C). In such cases starting female frequencies determine the subsequent direction of evolution irrespective of starting male frequencies. If the starting P₁ frequency > 0.5 then T₁ (matching male trait) is fixed and if the starting P₁ frequency < 0.5 then T₁ is lost irrespective of starting male frequencies (Figure 6.1C).

Therefore, it is clear that in environments where the strength of selective mating is stronger than the strength of PFD, perturbations in female frequencies affect the subsequent evolutionary direction of polymorphic populations more than the perturbations in male frequencies. Processes such as significant fluctuations in climatic or other environmental parameters, any other process resulting in fluctuating selection, and genetic drift (including 'bottlenecks'), as well as sporadic immigration or emigration, can produce transient, large perturbations in male-female frequencies.
The threshold line tilts as $\alpha$ increases further and becomes very strong (i.e. when $\alpha \gg \beta$) (compare Figure 6.1C and D). In the standard model of selective mating, for a constant frequency of the preference allele in the population, male traits exhibit higher fitness relative to the other morph when lower in frequency (Seger 1985). Consequently, selective mating produces an overall negative frequency-dependent effect. The negative frequency dependent effect is amplified when selective mating is very strong compared to ecologically-driven PFD. Consequently, populations starting with a higher frequency of male alleles require a higher frequency of corresponding female alleles to continue the Fisher process and lead populations to fixation. Therefore, threshold boundary tilts when $\alpha \gg \beta$ (Figure 6.1D).
Figure 6.1: Changes in the position and shape of threshold boundary (solid black line) between attraction basis under different selective regimes when only males are under PFD (model 6.1). Note that here $\alpha_1=\alpha_2=\alpha$ and $\beta_1=\beta_2=\beta$. The threshold boundary separates very different outcomes ($T_1$ fixation or $T_1$ lost). (A) No assortative mating. (B) Strength of PFD $>$ strength of assortative mating. (C) Strength of PFD $<$ strength of assortative mating. Note how the boundary collapses to a horizontal line in this case. (D) Strength of PFD $<<$ strength of assortative mating.
Now I consider the effects of selective mating on the maintenance of variation in aposematic traits within populations.

### 6.3.1.1 Polymorphism maintenance in aposematic traits within populations

Non-random mating can maintain sexual trait diversity (M'Gonigle et al. 2012). Selective mating can create an overall negative frequency-dependent selection and can, therefore, maintain sexual trait polymorphisms within populations. If mate preferences are not under directional selection, selective mating is likely to maintain aposematic trait variation within populations.

However, my results suggest that selective mating is not sufficient to maintain polymorphisms in aposematic traits even when non-random mating and PFD act in opposite directions (Figure 6.5A); here $a_1<<a_2$ but $\beta_1>>\beta_2$. It is clear that no starting condition can maintain polymorphism in $T$ in such cases. However, when PFD is weak and mate preferences are asymmetric (Figure 6.5) then populations sitting on the threshold boundary can remain polymorphic. However, the boundary is so narrow (Figure 6.5A) that slightest random change in allele frequencies will cause one allele to be fixed. These results suggest that the indirect negative frequency-dependence induced by non-random mating is not enough to maintain aposematic trait variation within populations.

In order to understand how starting male-female allele frequencies bias the direction of evolution in variable environments, I calculated the area of attraction basin of $T_1$ fixation under different combinations of selective parameters. An attraction basin is the set of male-female frequency combinations from which populations in a given environment will evolve to one of the alternative stable state i.e. either $T_1$ fixation or
T₁ loss (i.e. T₂ fixation). For a given combination of selective parameters, the size of attraction basin in the male-female allele frequency space describes the degree of bias the starting male-female frequencies produce in that given environmental regime; a larger area means a stronger bias towards that outcome.

6.3.1.2 Effects of initial conditions on the evolutionary direction under variable environments.

Relative values of PFD (β₁ and β₂) and mate choice parameters (α₁ and α₂) alter the size and shape of attraction basins. I explored the effects of varying β₁ and β₂ independently on the area of attraction basin of T₁ fixation for variable assortative mating strengths (Figure 6.2). Similarly, I also explored the effects of varying α₁ and α₂ independently on the area of attraction basin of T₁ fixation under different PFD strengths (Figure 6.3).

(A) Effects of PFD parameters (β₁ and β₂) on the area of attraction basin of T₁ fixation under the different strength of assortative mating.

In the absence of assortative mating, relatively small differences between β₁ and β₂ can erase the effects of initial conditions. In such cases, populations evolve towards the direction of stronger βᵢ irrespective of initial conditions (see the red and blue areas in the Figure 6.2A). As soon as assortative mating becomes strong, stronger differences between β₁ and β₂ are necessary to erase the effects of initial conditions. When assortative mating is moderate then populations evolve towards one of the alternative stable states (T₁ fixation or T₁ loss i.e. T₂ fixation) irrespective of initial conditions only if the differences between β₁ and β₂ is extremely strong (Figure 6.2B and C). When assortative mating becomes very strong then differences between PFD
parameters cannot erase the effects of initial conditions (Figure 6.2D). In other words, in the environments where assortative mating is very strong than PFD (i.e. $a >> \beta$), initial conditions always determine the direction of subsequent evolution irrespective of differences between the $\beta$.

When assortative mating ($a$) is strong than PFD ($\beta$), then the threshold boundary collapses to a horizontal line irrespective of differences between $\beta_1$ and $\beta_2$. This arises because, when assortative mating is strong, starting male frequencies have very little effect on the direction of subsequent evolution of aposematic traits (Figure 6.1D). Consequently, for the same degree of difference between $\beta_1$ and $\beta_2$, a greater diversity of starting frequencies can maintain the advantageous trait when assortative mating is strong than when it is weak.

Figure 6.3 shows this general result when assortative mating is weak versus when assortative mating is strong. Note that the threshold boundary is curved when assortative mating is weak (Figure 6.3B) but collapses to a horizontal line when assortative mating is strong (Figure 6.3D). Consequently, more starting frequencies can lead to the fixation of the advantageous trait ($T_1$) when assortative mating is strong, the area of the $T_1$ fixation attraction basin increases in such cases.
Figure 6.2: Effects of varying PFD parameters ($\beta_1$ and $\beta_2$) on the area of attraction basin of T1 fixation under the different strength of assortative mating ($\alpha_1 = \alpha_2 = \alpha$).
Figure 6.3: Changes in the shape and position of the threshold boundary between attraction basins (solid black line) for a given ratio of PFD parameters ($\beta_1=0.8, \beta_2=0.1$), under different assortative mating regimes. (A) Weak assortative mating ($\alpha_1=\alpha_2=\alpha=0.1$). (B) Strong assortative mating ($\alpha_1=\alpha_2=\alpha=0.7$).
(B) Effects of mate choice parameters \((a_1 \text{ and } a_2)\) on the area of attraction basin of \(T_1\) fixation under variable PFD strengths \((\beta_1=\beta_2=\beta)\).

When PFD is very weak compared to mate choice strength \((a_1 \text{ and } a_2)\) this removes the effects of initial conditions. When PFD is weak and \(a_1 \gg a_2\) then populations evolve to the advantageous trait \(T_1\) irrespective of starting frequencies (here \(T_1\) is advantageous because \(a_1 \gg a_2\)). Very few starting frequencies can lead to loss of \(T_1\) in such cases (see the red colour in Figure 6.4A).

Weak differences between \(a_1\) and \(a_2\) can eliminate the effects of initial conditions more when PFD is strong than when it is weak. Environments with strong PFD and strong differences between mate choice parameters bias the direction of aposematic trait evolution towards the advantageous trait relatively more than environments with weak PFD and weak differences between mate choice parameters. For a given difference between \(a_1\) and \(a_2\), more starting conditions lead to the fixation of an advantageous trait when PFD is strong than when it is weak (Figure 6.4).

I will now describe the results of a model where the aposematic trait expression is not sex limited and both males and females are affected by ecologically-driven PFD.
Figure 6.4: Effects of variable mate preferences ($\alpha_1$ and $\alpha_2$) on the area of attraction basin of $T_1$ fixation under different strengths of PFD ($\beta_1=\beta_2=\beta$).
Figure 6.5: Changes in the shape and position of threshold boundary (solid black line) for the constant degree of differences between mate preferences ($\alpha_1=0.8$, $\alpha_2=0.1$), under different PFD regimes. (A) Weak PFD ($\beta_1=\beta_2=\beta=0.1$). (B) Strong PFD ($\beta_1=\beta_2=\beta=0.7$).
6.3.2 Model 6.2: Trait expression is not sex-limited

In many species, aposematic traits are expressed in both sexes, for example, poison dart frogs. In this case, ecologically-driven PFD should affect the viability of both males and females. Here I describe a model where both males and females are affected by PFD and assortative mating based on aposematic traits.

Consider a haploid population showing polymorphism in aposematic traits and in mating preferences. Assume locus T controls aposematic traits in males and the unlinked locus P controls female preferences for traits. Let each locus have two alleles which correspond to different traits T₁, T₂ and female preferences P₁, P₂ respectively. The only difference between this model and model 6.1 is that in this model females are also affected by PFD.

Let \( f_1, f_2, f_3 \) and \( f_4 \) be the frequencies of \( T_1P_1, T_1P_2, T_2P_1 \) and \( T_2P_2 \) zygotes in females. Let \( \beta_1 \) and \( \beta_2 \) are the PFD coefficients for females carrying \( T_1 \) allele and \( T_2 \) allele respectively. Consequently, the fitness of female genotypes is 
\[
W_{T_1P_1} = 1 + \beta_1 f_1; \quad W_{T_1P_2} = 1 + \beta_1 f_2; \quad W_{T_2P_1} = 1 + \beta_2 f_3; \quad W_{T_2P_2} = 1 + \beta_2 f_4.
\]
Let PFD occur before mate choice; this alters the frequencies of males and females available for mating. Frequencies of female genotypes available for selective mating after NFD are 
\[
f_1' = \frac{f_1(1+\beta f_1)}{W_{\text{females}}}; \quad f_2' = \frac{f_2(1+\beta f_2)}{W_{\text{females}}}; \quad f_3' = \frac{f_3(1+\beta f_3)}{W_{\text{females}}}; \quad f_4' = \frac{f_4(1+\beta f_4)}{W_{\text{females}}}; \quad \text{where} \quad W_{\text{females}} = [(f_1(1 + \beta f_1)) + (f_2(1 + \beta f_2)) + (f_3(1 + \beta f_3)) + (f_4(1 + \beta f_4))].
\]
Note that the fitness of male genotypes remains same as explained in the model 6.1. Also, the computation procedure to estimate attraction basins and basin boundary remains same as explained in the model 6.1.
The difference between effects of expression in one or both sexes does not alter the evolutionary dynamics. When PFD is doubled, it compensates the effect of assortative mating which produces the overall negative frequency-dependent selection. Figure 6.6 shows the general result when PFD affects both sexes when $\beta_1=\beta_2=\beta=0.2$ and $\alpha$ are variable. The difference between effects of expression in one or both sexes is simply a factor of 2. For example, the shape and position of threshold boundary for $\alpha=0.2$ in Figure 6.6 (PFD on both sexes) is the same as the shape and position of threshold boundary for $\alpha=0.1$ in Figure 6.1 (PFD only on males).
Figure 6.6: Changes in the position and shape of threshold boundary (solid black line) under different selective regimes when both males and females are under PFD (model 6.2). Note that here $\alpha_1 = \alpha_2 = \alpha$ and $\beta_1 = \beta_2 = \beta$. Threshold boundary separates very different outcomes ($T_1$ fixation and $T_1$ loss). (A) Strength of PFD > strength of assortative mating. (B) Strength of PFD = strength of assortative mating. (C) Strength of PFD < strength of assortative mating. See that in such selective regimes, boundary collapses to the horizontal line. (D) Strength of PFD $\ll$ strength of assortative mating.
I will now summarize all the results of both models.

6.4 Summary

- In many cases, sexually selected traits are affected by ecologically-driven positive frequency dependent selection (PFD) and a consistent mating advantage. Aposematic traits are ideal systems to look for such examples. In such cases, the relative strengths of assortative mating and PFD determine whether male or female starting frequencies influence the subsequent direction of evolution.

- If assortative mating is stronger than PFD, then starting male frequencies do not affect the subsequent evolutionary direction of aposematic traits irrespective of difference between effects of expression in one or both sexes. In such cases starting female frequencies determine the subsequent evolutionary direction irrespective of male starting male frequencies within populations.

- Consequently, in such environments, perturbations in female frequencies affect the subsequent direction of trait evolution more than perturbations in male frequencies. In such cases, perturbations in male frequencies have less impact on the evolutionary direction than perturbations in female frequencies. This result is true irrespective of difference between effects of trait expression in one or both sexes.

- When selective mating is stronger than PFD, differences between PFD parameters cannot eliminate the effects of starting male-female frequencies. In such cases, differences between PFD parameters cannot completely bias the direction of evolution irrespective of starting male-female frequencies.
Consequently, perturbations PFD parameters cannot determine the evolution of aposmatic traits in such cases. Instead, perturbations in female gene frequencies are more important to determine the subsequent evolutionary direction of aposmatic traits.

6.5 Conclusion

The relative strengths of ecologically-driven positive frequency-dependent selection (PFD) and selective mating determine whether male or female starting frequencies influence the subsequent direction of evolution. Perturbations in male frequencies are more likely to alter evolutionary change than perturbations in female frequencies in the environments where PFD is stronger than selective mating. However, in environments where selective mating is stronger than PFD, perturbations in female frequencies are more likely to alter evolutionary change of polymorphic populations. I found that selective mating in itself is not sufficient to maintain variation in aposmatic traits within populations. As a result, variation in mate choice cannot maintain variation in aposmatic traits within populations.
Chapter 7 : Discussion and Conclusion

An important aim of evolutionary ecology is to understand how evolutionary and ecological processes interact and promote the maintenance of diversity within and among species. To what extent does selective mating contribute to the maintenance and persistence of sexual trait polymorphism, especially in variable ecological conditions? This is not well understood. In this thesis, I have identified specific combinations of mate choice parameters and ecological factors which promote or reduce the persistence of polymorphisms or divergence in sexually selected traits in variable environments. For that, I have used the null model of intersexual selection (Kirkpatrick 1982; Prum 2010, 2012) as a foundation. Here I summarize the main results of my research and discuss their implications for the persistence of polymorphism, population divergence, and speciation.

7.1 Strong assortative mating makes polymorphic populations significantly more robust in the face of large perturbations in male and female allele frequencies.

Sexual selection models exploring the maintenance of sexual trait polymorphisms usually focus on equilibrium conditions but do not focus on the robustness of sexual trait polymorphisms in the face of gene frequency and/or environmental perturbations. I used the null model of sexual selection to examine the robustness of sexual trait polymorphisms in the face of temporary perturbations in male-female gene frequencies in variable environments. My results clearly show that strong assortative mating significantly increases the robustness of sexual trait diversity even in the presence of large gene frequency perturbations. Consequently, stable or
nearly stable polymorphic populations with strong mate preferences are more robust and hence are less likely to shift to monomorphic state and are more likely to remain polymorphic for a long time in the face of temporary perturbations in gene frequencies.

These results have implications for divergence among isolated polymorphic populations. Selective conditions that enhance the polymorphism resilience actually reduce the potential for strong accidental divergence among polymorphic populations that sit on or near the line of polymorphic equilibria compared to conditions that reduce the resilience of polymorphisms. When assortative mating is strong then the polymorphic zone remains broad (greater polymorphism resilience) and hence a relatively larger perturbation in male-female gene frequencies is required to cause accidental divergence in sexual traits among polymorphic populations that sit on or very close to the line of equilibria (i.e. to throw stable polymorphic populations across the zone boundaries in opposite directions). Therefore, for the same large magnitude of gene frequency fluctuations, sets of polymorphic populations (populations sitting on the equilibrium line in figure 3.1A) with strong but not complete assortative mating are more likely to show accidental divergence than sets of populations with weak assortative mating.

In summary, sexual trait polymorphisms can persist for a long time even in the face of potentially large gene frequency perturbation when assortative mating is strong, but not when it is weak. These results suggest that early stages of speciation could stall in the face of large gene frequency perturbations, especially with strong mating preferences, and further parametric changes may need to occur before speciation completes.
Future work: In this thesis, I have particularly focused on the resilience of sexual trait polymorphisms to temporary and potentially large gene frequency perturbations. This is because I was more interested in the resilience against perturbations which can potentially shift polymorphic populations into the monomorphic state and completely lose sexual trait polymorphisms in future. However, polymorphic populations also face small, repeated random perturbations in male-female gene frequencies which may not shift polymorphic populations into monomorphic states but can displace frequencies while maintaining polymorphism in the future. For example, gene flow is a chronic or continuous perturbation by immigrant alleles. In such cases, it will be interesting to examine how mate choice parameters affect the resilience of sexual trait diversity against small, repeated perturbations in gene frequencies (measured by the “recovery time”) and how that differs from the resilience to temporary and large gene frequency perturbations. My models assume unlinked trait and preference loci. It will be interesting to examine how linkage might affect the results because linkage can be an important facilitator or inhibitor on the process of polymorphism maintenance and divergence in sexually selected traits.

7.2 Frequency-dependent predation interacts with selective mating to either maintain sexual trait polymorphisms or cause strong divergence.

In many cases, sexually selected traits are affected by negative frequency-dependent selection (NFD), in which fitness decreases with phenotype frequency. For example, colour polymorphisms can be under frequency-dependent predation and can also be under consistent mating advantage. In such cases, my results show that for a given intensity of ecologically-driven negative frequency-dependent selection (NFD),
mate choice parameters interact with each other in a systematic non-linear way and determine thresholds that separate the maintenance and loss of polymorphisms, and set conditions for divergence in sexual traits. Populations diverge in frequencies but remain polymorphic if they are within a zone defined by two thresholds (figure 4.1). However, sexual traits diverge completely and diverge in opposite directions if populations fall outside the zone.

7.3 In the face of large perturbations in mate choice parameters, sets of polymorphic populations with strong NFD and strong mate preferences are less likely to diverge than those with weak NFD and weak mate preferences.

Sexual trait polymorphisms show greater resilience to large perturbations in mate choice parameters when NFD and mate preferences are strong (figure 4.5). Consequently, sets of polymorphic populations these properties are more likely to remain polymorphic for a long time. This result suggests that environments with strong frequency-dependent predation and strong assortative mating can stall early stages of population divergence and speciation.

7.4. The presence or absence of sex-limited trait expression can significantly affect the divergence of sexually selected traits.

When both sexes are affected by frequency-dependent predation (no sex-limited expression), NFD intensity is doubled, compared to cases of sex limitation. This more strongly promotes the maintenance of polymorphisms more than if only males are affected by the frequency-dependent predation.
This result suggests that for a given NFD strength, geographical differences in mate preferences are more likely to produce fixed/strong divergence in sexually selected traits in species which express sexual traits only in males than species which express traits in both males and females.

7.5 Frequency-dependent predation and selective mating can interact and maintain sexual trait polymorphisms even when mate preferences are under the strong directional natural selection, classically thought to promote the loss of polymorphism.

Classical theory suggests that even weak directional selection on mate preferences can deplete sexual trait diversity (Kokko et al. 2006a; Prum 2010). Given this scenario, it is hypothesized that ecologically-driven frequency-dependent selection can potentially maintain variation in sexually selected traits when mate preferences are under directional selection independent of mate choice (Maan and Seehausen 2011).

My results show that strong differences in mate preferences among populations can cause divergence in sexual traits if and only if the directional selection is weak. When mate preferences are under moderate or strong natural selection, even strong differences in mate choice parameters cannot produce a strong or fixed divergence in sexual traits among populations irrespective of the NFD strength. In such cases, populations can either remain polymorphic or the relatively advantageous trait allele is fixed within populations. In other words, polymorphic populations showing strong differences in mate preferences cannot diverge in different directions if the directional selection is strong, but can if it is weak.
These results suggest that directional selection on mate preferences can significantly reduce the divergence in sexual traits among populations when sexual traits are affected by the frequency-dependent predation or other selection.

### 7.6 Sex-limitation of mate preferences can significantly affect the divergence in sexual traits among populations.

If the machinery underlying mate choice is sex-limited then polymorphic populations with strong differences in mate preferences can diverge in different directions when the directional selection on mate preferences is weak. However, polymorphic populations cannot diverge when mate preferences are not sex-limited (for example when mate preferences are co-opted due to sensory bias). This is true irrespective of differences in mate preferences, even when the strength of directional selection is weak.

This result suggests that when frequency-dependent predation occurs, directional selection on sensory systems (or any other mate choice machinery which is not sex specific) caused by the physical environment can significantly reduce the potential for divergence in sexually selected traits among populations even if populations differ remarkably in mate preferences.


7.7 Ecologically-driven positive frequency dependent selection (PFD) interacts with selective mating and their relative strengths to determine whether perturbations in male or female frequencies influences the subsequent direction of evolution of aposematic traits.

My results show that when assortative mating is weaker than PFD, male starting frequencies affect the subsequent evolutionary direction of aposematic traits more than female starting frequencies. Consequently, perturbations in male frequencies are more likely to alter evolutionary change than perturbations in female frequencies in such environments. In contrast, when assortative mating is stronger than PFD, female frequencies affect the evolutionary direction more than male starting frequencies. Thus, perturbations in female frequencies are more likely to alter evolutionary change of polymorphic populations in such environments.

7.8 Differences in PFD parameters cannot eliminate the effects of initial conditions in environments where assortative mating is stronger than PFD.

If assortative mating is strong in polymorphic populations then even strong differences in PFD parameters cannot bias the evolutionary direction of aposematic traits. It is important to note that sex-limitation of an aposematic trait does not affect this result.

This result suggests that any ecological parameters which can produce differences in the strength of PFD can only weakly bias the evolutionary outcome of the sets of polymorphic populations (populations consisting of different aposematic morphs) with strong assortative mating.
7.9 General Conclusion

Strong assortative mating makes sexual trait diversity significantly more robust against large perturbations in male-female frequencies and promotes the persistence of polymorphisms in the face of large fluctuations in populations in variable environments.

Selective mating can interact with ecological processes which promote the maintenance of genetic variation, such as frequency-dependent predation (negative frequency dependence). Their interactions can promote the maintenance of sexual trait polymorphisms even when mate preferences are under strong direct selection, classically thought to promote the loss of sexual trait polymorphism. In such cases, it is important to examine the dynamics of threshold conditions that separate the maintenance and loss of polymorphisms. Consequently, I have explicitly identified threshold combinations of mate choice parameters and survivorship factors that separate the maintenance and loss of sexual trait polymorphisms. I show that same thresholds also set conditions for strong divergence in sexually selected traits.

My results show that mate choice parameters and ecological factors can interact in ways not predictable from considering either alone. The persistence of polymorphism and divergence of sexually selected traits are contingent on the details of interaction and not just on their individual effects.
References


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