Selection in two-sex structured populations

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General Introduction

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"For hundreds and millions of years life came into being by way of unicellular animals which reproduced through fission. Fission can't be called birth, since it's the same cell, which suddenly splits into two identical specimens. And it's difficult to say that these twins are the originals cell's children, since you can't be your own child, you can't be transformed without a trace into your own offspring. Literally without a trace. In dividing, the initial cell simply vanishes. But it's not what other, more complicated animals know as death. As in every decent criminal investigation, a corpse is required to establish a death. [...] For reasons known only to itself, the evolution of living beings began to abandon its original concept and move on to the production of mortals, whose lives divide into distinctive phases that always occur in the same order: birth, childhood, maturity, old age, and death. I don't know why it turned out this way and not otherwise, and those who do know also don't actually know."

— Wisława Szymborska*, Non-required reading*

1.1 Sex, death, and evolution

Individuals die, develop, degenerate, shrink, shrivel, mature, move, grow, and procreate at rates that depend on their age, stage, size, genotype, environment, mood, or marital status. As a consequence, the distribution of individuals over different stages, genotypes, size-classes, moods, or environments changes. That is, populations evolve, shrink, or grow as a consequence of individuals moving through their life-cycles.

This interindividual variation means that a trait that is beneficial for one individual can be disadvantageous for another. In our humanoid ancestors, for example, a hypothetical gene that led to larger hips would have become very beneficial for females when the humanoid brains, and therefore the heads of their babies, started growing. Males with larger hips, on the other hand, couldn't run as fast, so the same gene increased female survival but might have decrease male survival.

Classical population genetics ignores interindividual variation except for genotype and treats all individuals in a population as equal in all other regards. Although there have been many interesting extensions of population genetics (e.g., Levene 1953; MacArthur 1962; Roughgarden 1971; Giesel 1972; Lewontin and Krakauer 1973; Waples 1989), very few extensions have included individual differences within a population. Noteworthy exceptions are a series of papers by Charlesworth on age-structured population genetics (Charlesworth 1970, 1971, 1972; Charlesworth and Giesel 1972*a*,*b*), and two more recent papers exploring structured population genetics under equilibrium conditions (Diekmann et al. 2003), and combined with an integral projection model (Coulson et al. 2011).

A general, mathematically tractable framework that can include density-dependent selection, sexual reproduction, and *any* relevant individual differences has been lacking, however. The primary goal of this thesis is to provide such a framework in which ecological and genetic processes operate simultaneously. The framework can be coupled to demographic data and by incorporating sex it can improve our understanding of life-history evolution in the context of sexual reproduction.

Some of the most extravagant interindividual differences in sexually reproducing populations are those between males and females. Perhaps unsurprisingly, males become impossible to ignore when modeling sexual reproduction. Including males lead to some important and unexpected consequences of sexual dimorphism for life-history evolution. The broader implications of these findings are explored in the Discussion (Chapter 6).

This introduction chapter is organized into sections that consecutively answer the following questions: 1. *Why* bother combining sexual reproduction, genetics, and demography? Short answer: because life history matters and because sex matters. 2. *How to combine sexual reproduction, genetics, and demography?* Short answer: with matrix models and population genetics. 3. *What's in the rest of the thesis?* Short answer: Density-independent models without and with males (Chapters 1 and 2), density-dependent models without and with males (Chapter 3 and 4).

Box 1: Some definitions

Ecology (The study of) changes in population numbers due to intra- and inter-specific (biotic) interactions and interactions with abiotic environmental factors.

Evolution (The study of) changes in the genetic composition of a population.

Demography The analysis of ecological processes in terms of internal population structure, distinguishing individuals on the basis of age, developmental stage, size, etc. Demographic models, in our terminology, are a subset of ecological models.

Development The process of growth and differentiation through the life-cycle of an organism, including acquiring the ability to reproduce.

Life history The pattern of survival, development, and reproduction events typical for a member of the species.

Reproduction The creation of new individuals.

Sex Mixing of genetic materials of two (or more) individuals. In this thesis, we restrict ourselves to species with two sexes, and to species that combine reproduction and sex.

1.2 Motivation

Life history matters

Development is fundamental to all forms of life. Even unicellular organisms have to develop before they can reproduce, since biological reproduction always involves some material overlap between parent and offspring (in contrast to non-biological multiplication by copying, (Griesemer 2000, 2001)). Biological entities therefore need to grow before they can reproduce (otherwise each generation of individuals would be smaller than the previous). Since growing takes time, no organism can reproduce immediately after they are born[1].

Including development in population models by distinguishing between reproducing (adults) and non-reproducing (juvenile) individuals can have a number of surprising consequences. For example, competition between adults and juveniles for resources can result in an increase in juvenile biomass when mortality rates increase, or even in an increase in total population biomass (biomass overcompensation, see De Roos et al. (2007); de Roos and Persson (2013)). Juvenile-adult competition has consequences for fisheries management (Van Leeuwen et al. 2008; Ohlberger et al. 2011), and for the ecology and evolution of cannibalism (Toscano et al. 2017), metamorphosis (ten Brink et al. 2018), and disease dynamics (Boerlijst and de Roos 2015).

As individuals move through their life cycle, the effect of biotic and abiotic factors such as population density, predation, or climatic conditions on individuals changes. And vice versa, individuals affect population density, predators, competitors, or prey, and their abiotic environment differently as they move through their life cycle. Changing climatic conditions affect developmental stages differently therefore and life-history strategies impact a species' ability to adapt to changing climatic conditions. Since long-lived species with relatively slow life histories (slow growth, late maturation, few offspring) are slower to respond to environmental change, it has been suggested that they are at greater risk than short-lived species with high potential rates of increase (Martínez-Ruiz and Knell 2017; Webb et al. 2002).

Despite the obvious importance of development, many eco-evolutionary models treat all individuals in a population as equal regardless of their life history stage (see for example, Abrams and Matsuda 1997), and models of sexual selection or sexual conflict often treat all individuals of the same sex as equal, regardless of their developmental stage (see for example, Rankin and Kokko 2007). Resource

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[1]Even aphids, despite being born pregnant, do not reproduce immediately after being born (Stern 2008).

competition between juveniles and adults as well as males and females might alter the effect of sexual selection on sexual size dimorphism, for example.

Individuals in unstructured population models are capable of reproducing as soon as they are born, ignoring the time lag necessary to acquire the ability to reproduce. In wild populations, however, many individuals do not make it across the time lag to a reproductive stage, leading to strongly skewed distributions of lifetime reproductive output. Disentangling how much of this variation is due to genuine genetic differences, and therefore can be operated on by evolution, and how much of the variation is due to luck, requires a framework that includes genetic differences, developmental differences, and differences due to chance (often termed stochasticity).

Sex matters

Imagine having a copulatory apparatus which takes up one-third of your total body volume. Admittedly, you will also have to imagine being a seed shrimp (ostracod) with no heart and one eye. But even under those circumstances, it might strike you as an excessive investment of resources into reproduction[2]. To accomodate these giant genitals, male ostracods need larger shells than females (Fernandes Martins et al. 2017).

In contrast, female triplewart seadevils (*Cryptopsaras couesii*) can be up to 30 times larger than males, see Figure 1.1. Male triplewart seadevils attach themselves to females, after which their circulatory systems combine, upon which the male slowly looses its organs and eyes, transforming into nothing more than a bag of sperm.

Sexual dimorphism in physical characteristics like size, morphology, pigmentation, immune system, or behaviour are widespread, and have been studied since Darwin (1888). These differences in physical characteristics between males and females lead to differences in survival, maturation rate, reproductive success, and other demographic rates. Despite sexual dimorphism being so common and evidently impacting demographic rates, it is often ignored in demographic models.

Darwin distinguished between natural and sexual selection, arguing that natural selection arises from variance in individual survival and fecundity, whereas sexual selection results from variance in mating success. Sexual selection has led to some of the most extravagant displays of sex differences in nature, e.g. birds of paradise, peacocks and some argue even the human brain. However, there are also examples of sexual dimorphism driven by ecological factors (Shine 1989). Sexual dimorphism in bill size and shape in a hummingbird, *Eulampis jugularis*,

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[2]On the other hand, seed shrimp do get to boast the Guinness World Record for the oldest fossilised penis. `http://www.guinnessworldrecords.com/world-records/oldest-penis`

has evolved to match the flowers of two different *Heliconia* species (Temeles et al. 2000); mouthparts in male and female mosquitos have evolved to suck nectar and blood, respectively (Wahid et al. 2003). Ultimately the distinction between sexual and natural selection is blurry and not necessarily well defined. Sexually selected traits often result in concomitant viability changes, and size dimorphism due to sexual selection might initiate ecological resource specialization if bigger individuals have access to different food sources.

The distinction between survival, fecundity, and sexual selection become even more unclear when development is included. Increased juvenile predation might select for faster maturation, for example (Bell et al. 2011). But increased maturation also leads individuals to start reproducing earlier, which could both increase or decrease total lifetime reproductive output depending on the organism, the sex of the individual, and on resource availability (Auer et al. 2010). Earlier maturation might affect the expression of secondary sexual characteristics, which would in turn affect mating success. Sexual and natural selection are tightly interwoven, and selection acts on the entire life-cycle of both males and females.

There is a large body of theoretical and empirical work that attempts to establish whether sexual selection increases or decreases population extinction risk (Kokko and Brooks 2003; Møller 2003; Morrow and Fricke 2004; Bonduriansky and Chenoweth 2009; Martínez-Ruiz and Knell 2017; Hasegawa and Arai 2018). If mate choice makes it harder for individuals with deleterious mutations to reproduce, then a population with sexual selection could purge such mutations faster than a population without sexual selection. This implies that the mutational load is lower on populations with sexual selection (Lorch et al. 2003; Whitlock and Agrawal 2009; Lumley et al. 2015). On the other hand, the effective population size is reduced as a consequence of the skewed reproductive success which could lead to inbreeding depression (Kokko and Brooks 2003).

Sexually selected traits often correlate with reduced male survival either due to costly ornaments or due to male-male aggression (Promislow et al. 1992; Brooks 2000), and it has been suggested that this could have led to population extinctions, for example in the Irish elk (Moen et al. 1999; Van Geel et al. 2018). However, as the number of males decreases due to their low survival, mate competition decreases and males with less elaborate ornaments and higher survival would be at a competitive advantage again (Kokko and Brooks 2003). It therefore seems impossible for this mechanism to lead a population to extinction deterministically, but it could still reduce population size and therefore increase the probability of extinction as a consequence of demographic stochasticity, as argued by Kokko and Brooks (2003).
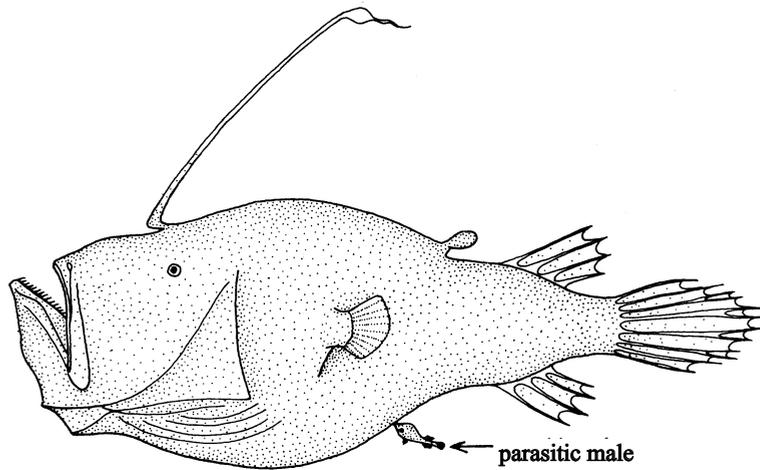
Figure 1.1: Triplewart seadevil with parasitic male attached. Source: Commons sibi via Wikipedia

Sexual reproduction has more ways of making trouble than just sexual selection. Mean fitness in a population does not necessarily increase when there is selection for viability and fertility in both sexes, which has been known since the early days of population genetics (Bodmer 1965; Hadeler and Liberman 1975; Pollak 1978). The problem is that the optimal trait value in males and females often differs, so genes can invade that benefit one sex at the expense of the other sex (intralocus sexual conflict). Such conflict would result in high fitness fathers siring low fitness daughters, and high fitness mothers having low fitness sons. Negative correlations between parents and offspring of the opposite sex have been shown in a natural population of red deer (*Cervus elaphus*, Foerster et al. (2007)), and in a laboratory population of *Drosophila* (Chippindale et al. 2001). It is expected that population fitness is reduced when females are the 'losing' sex in this conflict (Kokko and Jennions 2014).

Despite the many empirical and theoretical reasons to believe that sexual conflict and sexual selection will impact population viability, and will impact the evolutionary response of a population to different selection pressures, sex is generally not included in structured population models. For example, (stochastic) matrix population models, one of the most commonly used tools for assessing population viability (e.g., Alvarez-Buylla et al. 1996; Caswell 2001, Chap. 18), almost always

ignore males. The population growth rate or the stochastic growth rate are calculated from female demographic rates in these models, and are used to estimate species extinction risk in the face of anthropogenic stressors, and to estimate the relative importance of different demographic rates using selection gradients.

Recap and link to this thesis

In summary: Despite sexual dimorphism being linked to extinction risk, it is a widely ignored complication in demographic models. Despite life history strategy affecting extinction risk, it is a widely ignored complication in the literature on the ecological and evolutionary consequences of sex. Despite the many interesting and counterintuitive effects of both sex and development on population dynamics and evolution, they are rarely combined. In this thesis I combine demography, genetics, and sexual dimorphism into one mathematical framework by using the powerful demographic tool of matrix population models.

Linear matrix population models are one of the most commonly used tools to to assess species extinction risk (e.g., Alvarez-Buylla et al. 1996; Fieberg and Ellner 2001; Caswell 2001, Chap. 18). The population growth rate or the stochastic growth rate are used to assess whether the species is exponentially growing or declining. These growth rates are assumed to be proxies for fitness, and derivatives of the growth rate with respect to individual vital rates are assumed to represent selection gradients (Hamilton 1966; Silvertown et al. 1993; van Tienderen 2000; Caswell and Salguero-Gómez 2013; Caswell and Shyu 2017). However, generally these models ignore sexual dimorphism and genetics. In this thesis we investigate the population level consequences of including evolution and sex-specific demography acting on the same time-scale and assess whether above mentioned assumptions still hold.

1.3 Modeling biological evolution

A mutation in the DNA of an organism leads to evolutionary change if its effects ripple through several levels of organisation, eventually leading to a population level change in the allele frequencies at that locus. To do so, allelic effects have to cascade through several mappings: from genotype to enzymes and their regulation, from enzymes to the development of physical characteristics like plumage colour or immune competency, from physical characteristics to individual demographic rates, and finally from demographic rates to population level characteristics. A complete understanding of evolution would therefore require knowledge of this entire cascade of mappings (Rueffler et al. 2006; Coulson et al. 2006).

Although there is a growing field of academics studying the map from genotype to enzymes and their regulation (see Pigliucci (2010); Wagner and Zhang (2011)), traditionally theoretical biologists have either ignored the genotype-phenotype map altogether (quantitative genetics, adaptive dynamics, game theory, and optimization models) or assumed an extremely simplified genotype-phenotype map by assigning fixed fitness values to alleles (population genetics). This thesis fits squarely in the tradition of population genetics, but expands the phenotype from a single scalar fitness value to the entire life cycle of the organism. It can therefore incorporate genes with arbitrarily many pleiotropic and sex-specific effects.

Extending the framework in this thesis to include more than two alleles at one locus is fairly straightforward. Extending the framework to include more than one locus is more difficult, which is the main limitation of the framework. With the increasing number of genomic scale data sets, one-locus and a-few-allele models might seem a little last century.

There are many examples of single loci with large pleiotropic effects, however. In humans, it is believed that the inactivation of one gene (Cmah) led to increased running endurance and decreased muscle fatigability (Okerblom et al. 2018). A plumage colour polymorphism in Eleonora's falcon (*Falco eleonorae*) segregates as a simple one locus, two allele Mendelian trait (Gangoso et al. 2011). The gene affects survival and fertility in a highly pleitropic and sex-specific fashion, through concealment during hunting, resistance to plumage abrasion, and immune responses (Gangoso et al. 2011). The same gene is correlated with plumage or coat colour in jaguars (*Panthera onca*, Eizirik et al. (2003)), lesser snow goose (*Anser c. caerulescens*) and artic skua (*Stercorarius parasiticus*, Mundy et al. (2004)), bananaquit (*Coereba flaveola*, Theron et al. (2001)), and red-footed booby (*Sula sula*, Baião et al. (2007)).

1.4 Structured population models

Individual differences in age, size, sex, environment, history, or genotype lead individuals to move through their life-cycle at different speeds, to respond to changing environments differently, and to experience different selection pressures. Predicting how a population will respond to a change in environmental conditions therefore requires knowing how its individuals will respond, which in turn requires knowing the age, size, or stage distribution of individuals. Structured population models describe populations in terms of the distributions of individuals in these categories, and provide a map from individual vital rates to population level characteristics (Tuljapurkar and Caswell 1996).

Individuals are also the biological entities that empirical biologists tend to interact with, and measure properties of. This makes structured population models relatively easy to connect to data, since data tends to be about individuals.

The first step in developing a structured population model is to decide which individual characteristics (i-state variables) determine the fate of an individual, such that the fate depends only on an individuals current i-state and the environment (Metz 1977; Caswell and John 1992; Metz and Diekmann 1986). This requires the state variable to capture all the aspects of the individual's history that are relevant to its future fate (Caswell et al. 1972; Caswell 2001; de Vries and Caswell 2017). The task of the population modeler is to find an i-state variable that successfully captures the most important information about an individual.

There are many different kinds of structured population models. They can be divided along two main axes: 1. Treating time as discrete (matrix population models, integral projection models), or continuous (delay-differential equation models and partial differential equation models). 2. Classifying individuals by discrete stages (e.g. life history stages in matrix models and delay-differential equation models), or continuous traits (eg. size and/or age in physiologically structured population models and integral projection models). One of the major benefits of discrete time models is the relative ease of constructing stochastic versions of the models. Other benefits of matrix models are their mathematical tractibility and their straightforward connection to individual survival and fertility data. For an introduction to structured population models that nicely compares and contrasts these different models, see Tuljapurkar and Caswell (1996).

Both an advantage and a disadvantage of matrix population models is the requirement for a discrete i-state. For species with distinct developmental stages, like instars in arthropods, this is a feature; for species with continuous growth in size or bodymass, like fish or frogs, this can be a bug (which was solved by the introduction of integral projection models, see Rees and Ellner (2016)).

A further feature of matrix models is that the survival and transition matrix can be turned into an absorbing Markov Chain by adding an absorbing state for dead individuals. By attaching rewards to transitions, all the statistical moments of the distributions of demographic output variables, such as longevity, life-time reproductive output, healthy years, can be calculated (Caswell 2011; Hartemink et al. 2017; van Daalen and Caswell 2017). In addition, the variance in any of these demographic quantities can be decomposed into variance due to genuine differences between individuals (e.g. due to frailty, environment, or genotype), and variance due to stochasticity.

In this thesis I will be using matrix population models, and I will take advantage of the mathematical machinery of matrix calculus to derive invasion condi-

tions for a new allele invading a resident homozygous population. We define a protected polymorphism by the stability of the boundaries to invasion, i.e. if both alleles are unstable to invasion by the other, then the population can never return to the boundary once both alleles are present. In classical population genetics, with viability selection, at a single locus, in discrete generation, random mating, diploid populations, heterozygote advantage in fitness is a necessary and sufficient condition for a stable polymorphism. However, the world is populated by species with complex age- or stage-structured life cycles, subject to selection not only on viability but on sex- and stage-specific survival, growth, development, and fertility rates throughout those life cycles. In this thesis I will calculate conditions for a protected polymorphism for species with complex life cycles.

A matter of time

Ecology and evolution were traditionally believed to operate on different timescales, but empirical evidence against this idea has been accumulating. Examples are the rapid evolution of resistance to antibiotics and pesticides (Neve et al. 2014), and rapid life-history responses to environmental changes in urban environments (Schilthuizen 2018). The study of eco-evolutionary dynamics is predicated on the idea that ecological and evolutionary processes may operate on the same, or very similar, time scales. If this is true, feedbacks between ecological and evolutionary processes may have important consequences for both sides of the eco-evolutionary dividing line. There exists a long history of attempts to incorporate ecology and evolution into theories to explore this interaction (Fussmann et al. 2007; Pelletier et al. 2009).

Structured population models have been combined with models of evolution in numerous ways. Quantitative genetic models for changes in phenotype means have been combined with age-classified (Lande 1982b), stage-classified (Barfield et al. 2011) demographic models, and integral projection model for trait distributions (Coulson and Tuljapurkar 2008). Childs et al. (2016) extended this framework to include both sexes and develop an extension of the age-structured Price equation for two-sex populations. Integral projection models have also been combined with adaptive dynamics to study the evolution of function-valued traits (Metcalf et al. 2008; Rees and Ellner 2016).

Charlesworth developed a theory for age-classified population genetics using discrete difference equations (Charlesworth 1970; Charlesworth and Giesel 1972a; Charlesworth 1972; Charlesworth and Giesel 1972b; Orive 1995). Orive (1995) extended Charlesworth's framework to study the effect of clonal reproduction on the evolution of senescence. Tuljapurkar (1982) added variable environments and derived invasion conditions for a new allele into a homozygous, age-structured

population with no demographic differences between the sexes. The framework presented in this thesis expands this well-established body of work by going beyond age-structure to populations classified by any i-state variable, and by using the mathematical machinery of matrix population models.

1.5 Outline of this thesis

In Chapter 2 the mathematical framework on which the rest of the thesis is based is introduced. The framework combines basic Mendelian genetics with matrix population models. Genotypes may affect, in fully pleiotropic fashion, any mixture of demographic traits (viability, fertility, development) at any points in the life cycle. The dynamics of the stage×genotype structure of the population are given by a nonlinear population projection matrix. We show how to construct this matrix and use it to derive sufficient conditions for a protected genetic polymorphism for the case of linear, time-independent demography. These conditions demonstrate that genotype-specific population growth rates (λ) do not determine the outcome of selection. Except in restrictive special cases, heterozygote superiority in λ is neither necessary nor sufficient for a genetic polymorphism. As a consequence, population growth rate does not always increase and populations can be driven to extinction due to evolutionary suicide. We demonstrate the construction and analysis of the model using data on a color polymorphism in the common buzzard, *Buteo buteo*. The model exhibits a stable genetic polymorphism and declining growth rate, consistent with field data and previous models.

In Chapter 3 we present a general matrix model that incorporates both nonlinearity due to sexual reproduction and nonlinearity due to ecological interactions. We apply the model to the case of pesticide resistance in the flour beetle *Tribolium castaneum*. Our model permits arbitrary life-cycle complexity and nonlinearity. The stage-classified model of *Tribolium* is nonlinear due to cannibalism of eggs by larvae, and of eggs and pupae by adults. *Tribolium* has developed resistance to the pesticide malathion due to a dominant allele at a single autosomal locus, so the genetic dynamics are Mendelian recombination with one locus and two alleles. Using parameters from laboratory experiments, we analyze the stability of the boundary (homozygous) attractors and find that the resistant allele excludes the susceptible allele, even in the absence of malathion, agreeing with previously reported results.

In Chapter 4 we extend the framework to explicitly include males as well as females. The model can incorporate genes with arbitrarily many pleiotropic and sex-specific effects. Mean fitness in the population, as defined by the one-step growth rate, does not necessarily increase and evolutionary suicide can happen

through many different kinds of sexual conflict. We derive a set of analytical conditions for a protected polymorphism by analyzing the stability of the homozygote boundaries to invasion. We show that these conditions reduce to heterozygote superiority in population growth rate in the absence of sexual dimorphism and when the primary sex ratio is one.

Chapter 5 combines the two-sex stage-structured matrix population model from Chapter 3 with the density-dependence introduced in a one-sex model in Chapter 4. The model permits arbitrary life-cycle complexity and nonlinearity, as well as sexual dimorphism in both of these. Evolution can lead to a lower equilibrium stage abundance for all stages of both sexes in this model, which shows that evolution does not optimize any of the stage abundances, or any weighted sum of the stage abundances in the presence of sexual dimorphism in life-history characteristics. We derive a set of conditions for a protected polymorphism by analyzing the stability of the homozygote equilibria to invasion. We show that these conditions reduce to a simple equation, which resembles heterozygote superiority in growth rate, in the absence of sexual dimorphism and when the primary sex ratio is one.

Finally, in Chapter 6 I will briefly summarize the results and then discuss the following questions: 1. *So what? What are the implications of this thesis?* Brief answer: a) One scalar fitness measure can not be the longed-for panacea. b) There are many ways to go extinct when you have sex. c) Demographers should worry more about sex, see Section 6.2. 2. *How do these results fit into the broader context of things biologists already knew about evolutionary transitions, sex, and cooperation?* Brief answer: conflict is risky, and some other wild speculations, see Section 6.3). 3. *What's next?* Brief answer: marriage functions and (Oedipean) assortative mating, see Section 6.4. 4. *What didn't we do?* Brief answer: We did not prove two conjectures that common sense and simulations suggest are true[3]. And finally we close with a brief conclusion in Section 6.5.

A lengthy appendix follows all four Chapters, in which the conditions for a stable polymorphism are derived, and in which simplifications of the model are considered. The derivations in these appendices follow a similar sequence of steps with the necessary modifications for each chapter. The appendices are included for completeness, and for the very keen reader. Readers that do not enjoy wading through pages of matrix calculus should feel free to skip them, and the thesis should make as much, or as little, sense without wading through the lengthy exercises in matrix calculus.

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