Mathematical Models of Life Cycle Evolution

by

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Abstract

In this thesis, I investigate several aspects of life cycle evolution using mathematical models.

We expect natural selection to favour organisms that reproduce as often and as quickly as possible. However, many species delay development unless particular environments or rare disturbance events occur. I use models to ask when delayed development (e.g., seed dormancy) in long-lived species can be favoured by selection. I find that long-lived plants experience 'immaturity risk': the risk of death due to a population-scale disturbance, such as a fire, before reproducing. This risk can be sufficient to favour germination in the disturbance years only. I show how demographic models can be constructed in order to estimate the contribution of this mechanism (and two other mechanisms) to the evolution of dormancy in a particular environment.

All sexually reproducing eukaryotes alternate between haploid and diploid phases. However, selection may not occur in both phases to the same extent. I use models to investigate the evolution of the time spent in haploid versus diploid phases. The presence of a homologous gene copy in diploids has important population genetic effects because it can mask the other gene copy from selection. A key innovation of my investigation is to allow haploids and homozygous diploids to have different fitnesses (intrinsic fitness differences). This reveals a novel hypothesis for the evolution of haploid-diploid strategies (where selection occurs in both phases), where the genetic effects of ploidy are balanced against intrinsic fitness differences.

Many sex chromosome systems are characterized by a lack of recombination between sex chromosome types. The predominant explanation for this phenomenon involves differences in selection between diploid sexes. I develop a model for the evolution of recombination between the sex chromosomes in which there is a period of selection among haploid genotypes in pollen or sperm. I find that a period of haploid selection can also drive the evolution of suppressed recombination between sex chromosomes, which should become enriched for genes selected in the haploid phase. This model predicts that the tempo of sex chromosome evolution can depend on the degree of competition among haploids.

Preface

Chapter 2 of this thesis has been published. Chapter 3 has been submitted and Chapter 4 is in preparation for publication. The contributions of the candidate are as follows:

Scott, M.F. and Otto, S.P. (2014) Why wait? Three mechanisms selecting for environment-dependent developmental delays. *Journal of Evolutionary Biology* 27:2219-2232.

- The candidate designed and analyzed the models and wrote the manuscript. S.P. Otto guided and assisted with model analysis and edited the manuscript.

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- The candidate designed and analyzed the majority of models and wrote the manuscript. M. Rescan designed and performed the explicit multi-locus simulations, wrote this portion of the manuscript and edited the manuscript. S.P. Otto supervised the project, edited the manuscript and provided helpful input. D. Roze supervised the contributions of M. Rescan and edited the manuscript.

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- The candidate designed and analyzed the models and wrote the manuscript. S.P. Otto assisted with model analysis and edited the manuscript.

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Chapter 1

Mathematical Modelling of Life Cycle Evolution

1.1 Summary

A central aim of evolutionary biology is to understand how and why the diversity of organisms that we observe came to be. A prominent aspect of biodiversity is that organisms exhibit a large number of adaptations to different environments and interactions; remarkably, organisms also display a large amount of structural variation in their life cycles. That is, there is significant variation among species in the number and nature of life cycle stages between birth and reproduction, in the mode of reproduction, in the number of copies of genetic material, and in the way genetic material is inherited between generations. Evolutionary theory should give us insight into how this life cycle variation arose and why.

In this Chapter, I first briefly discuss the role of mathematical models in investigating evolutionary problems and describe the logic of the techniques and methodology that I will use. Then, I outline the features of life cycle evolution that are investigated in this thesis, using the example of two organisms that demonstrate variation in these life cycle aspects.

1.2 The Utility of Mathematical Models in Evolutionary Theory

Most theories in evolutionary biology are given in verbal form. The most famous is the theory of evolution by natural selection itself, which Darwin expressed as follows:

> Owing to this struggle for life, any variation, however slight and from whatever cause proceeding, if it be in any degree profitable to an individual of any species, in its infinitely complex relations

> to other organic beings and to external nature, will tend to the preservation of that individual, and will generally be inherited by its offspring. The offspring, also, will thus have a better chance of surviving, for, of the many individuals of any species which are periodically born, but a small number can survive. I have called this principle, by which each slight variation, if useful, is preserved, by the term of Natural Selection.
> —Charles Darwin, *On The Origin of Species* Chapter III

This verbal theory constitutes a model about how the world works, consisting of some form of verbal 'if...then...' statement through which we describe the logical consequences of some initial conditions. The same principle can be applied to simpler theories, such as 'if you heat water to 100°C, then it will boil'. In this view, our representation of the world, or schema, consists of a series of theories and so theories are extremely common.

It is often useful to make our theories formal using mathematical models. This approach has a long and successful tradition in evolutionary biology. Mathematical models played a key role in the modern synthesis, when the ideas of natural selection and Mendelian genetics were reconciled (among other advances, Mayr and Provine 1998). For example, in the first of a series of papers, Haldane (1924) used formal mathematical models to describe the change in frequency of a trait under selection when traits are controlled by a single Mendelian locus. Indeed, Haldane's "Mathematical theory of natural and artificial selection" forms the basis of the models of selection that I use in Chapters 3 and 4.

Theories provide the framework into which observations can be placed. Empirical observation is ultimately the only way to determine whether a process occurs in nature. The role of mathematical models is often to formalize the logic of our theories, providing an argument of the form 'if A, then B' or 'A would promote B' (Sober 2011). Some authors have argued that models themselves are often constructed and analyzed as a logical test of an idea, analogous to an experimental test (Caswell 1988, Servedio et al. 2014). The questions addressed by these models may take the form 'Am I correct in thinking that A would promote B due to an interaction with C that causes ...?' (Kokko 2007, Chapter 1).

Models can reveal features that might otherwise not be evident. For example, Haldane (1964) argues that his attempts to model natural selection led him to the concept of mutation load and a method for estimating the mutation rate in humans. Anecdotally, I believe the role of mathematical modelling in generating previously unexpected and unknown results is

under-appreciated. This is probably because models are usually presented in a way that makes the conclusions most logical; this presentation is generally decided upon after the results have been obtained. In Chapter 5, I will highlight results from this thesis that were unexpected at the outset.

The eventual success of a model is typically assessed by its usefulness. However, it can be difficult to evaluate the usefulness of a model directly, particularly when a model aims to advance our understanding of some process in a heuristic way. In many cases, models can appear to be caricatures of reality, and yet still be extremely useful for advancing our understanding. This idea is well explicated in this satirical analogy by Lewis Carroll:

> "What a useful thing a pocket-map is!" I remarked.
> "That's another thing we've learned from your Nation," said Mein Herr, "map-making. But we've carried it much further than you. What do you consider the largest map that would be really useful?"
> "About six inches to the mile."
> "Only six inches!" exclaimed Mein Herr. "We very soon got to six yards to the mile. Then we tried a hundred yards to the mile. And then came the grandest idea of all! We actually made a map of the country, on the scale of a mile to the mile!"
> "Have you used it much?" I enquired.
> "It has never been spread out, yet," said Mein Herr: "the farmers objected: they said it would cover the whole country, and shut out the sunlight! So we now use the country itself, as its own map, and I assure you it does nearly as well."
> –Lewis Carroll, *Sylvie and Bruno Concluded*, Chapter XI

Of course, the vastly detailed map created by Mein Herr will provide little insight. However, our experience will probably tell us that abstracted maps can help us to understand the structure of the world by including only the key details. The key details that are included in a model (or a map) depend on its purpose (Levins 1966). Like maps, models can provide us with very useful representations of the world and reveal features that might otherwise not be evident (Hillis 1993).

1.3 Evolutionary Invasion Analysis

In this thesis, assorted evolutionary problems are addressed primarily using the same technique: evolutionary invasion analysis (described in Kokko

2007, Chapter 7, and Otto and Day 2007, Chapter 12). This general approach has a long history in population genetics (Fisher 1928, Nei 1969), life history theory (Cohen 1966, Metz et al. 1992), and the evolution of social interactions (Hamilton 1964). Invasion analyses are typically used to address long-term evolutionary questions in which we wish to consider the fate of large number of possible alleles, each corresponding to a different trait, and evaluate the expected direction of evolution.

An evolutionary invasion analysis considers whether a population that is initially fixed for a particular allele can be invaded by a mutant allele that specifies a different trait value. We then infer how the trait is expected to evolve by determining which alleles can invade which populations. Thus, evolutionary invasion analyses proceed by considering a large number of pairwise interactions between 'resident' and 'mutant' types. The 'resident' is the allele that is initially fixed in the population and 'mutant' is the allele whose invasion into the resident population will be evaluated.

It is generally assumed that new alleles arise rarely; this assumption can greatly simplify analysis, allowing us to examine more complex phenomena. Because new mutants rarely occur, it is generally assumed that the resident population first reaches some long-term dynamical state (e.g., an equilibrium) without the presence of mutant alleles in the population. Invasion of a mutant allele is then evaluated in the context of this resident population. A mutant allele invades successfully if it increases in frequency from an initially low level.

While there might seem to be a prohibitively large number of pairwise interactions between residents and mutants to consider, types of successful or unsuccessful mutants can often be categorized. In a simple example of categorization, mutants might always be able to invade residents if they confer a higher trait value. In other cases, mutants that increase the trait value may only be successful under certain conditions. Therefore, categorization can divide parameter space into regions under which one evolutionary outcome or another is expected. Categorizations are often used to make predictions about what trait values we expect to evolve in species with particular attributes.

1.4 Life Cycle Variation

1.4.1 Two Example Life Cycles

To illustrate variation in life cycles, we can compare the life cycle stages of an angiosperm, *Silene latifolia*, and a green alga, *Ulva lactuca*. While these

organisms are simply examples, they demonstrate many of the key life cycle features examined in this thesis.

White campion (*S. latifolia*) can often be found along roadsides across Europe and North America, growing to approximately waist height and bearing white flowers. Diploid *S. latifolia* plants are either male or female; each mature individual bears flowers with only male or female sexual organs. Meiosis occurs within each flower type, which halves the number of genomic copies and yields haploid microspores (in males) or megaspores (in females). In male flowers, microspores mature into pollen grains and are presented to pollinators, who may transfer them to a female flower on a different individual. Once found on the receptive stigma of a female flower, these haploid pollen grains germinate and begin to grow as pollen tubes through the style towards the mature female megaspores (female gametophytes). Many pollen tubes can grow at the same time, each competing to fertilize the egg cells of female gametophytes. A fertilized egg cell (zygote) will thus inherit one nuclear genome from the father and one from the mother. If the successful pollen tube had an X chromosome, the zygote will eventually develop into a female adult (with one maternal and one paternal X chromosome), whereas males develop from egg cells fertilized by pollen tubes that contain Y-bearing nuclei (diploid males have a maternally inherited X and a paternally inherited Y chromosome).

Diploid *Ulva lactuca* green algae grow in rock pools and shallow subtidal areas and predominantly consist of green sheet-like thalli. Their overall appearance gives the species its common name, Sea Lettuce. Some cells in the leaf-like thalli become reproductive and undergo meiosis to produce four spores, each bearing half the number of genomic copies (haploid). These spores are motile and, if successful, will settle on a rock and begin to grow into another lettuce-sized adult, this time a haploid. Haploid and diploid adults are difficult to distinguish morphologically. Reproductive cells of haploids produce motile gametes (also haploid) via mitosis. To form a new diploid zygote, these gametes must fuse with a gamete released by another individual of opposite 'mating type', where mating types are determined by the haploid genotype. After fusion, a zygote will also settle on a suitable substrate and grow into a diploid adult, completing the sexual life cycle (Raven et al. 2005).

Even in these highly simplified descriptions, *S. latifolia* and *U. lactuca* exhibit qualitative differences in their life cycles. Firstly, when zygotes of *S. latifolia* are dispersed in seeds, a fraction of seeds delay germination (remain dormant) for a short period (Purrington and Schmitt 1995). However, in *U. lactuca*, growth and development of zygotes is not delayed by environmental

conditions or time (Hoek et al. 1995). Secondly, the haploid phase of *U. lactuca* is as large and independent as the diploid phase and presumably experiences similar selection pressures; whereas the haploid phase of *S. latifolia* is physically small and grows primarily within diploid tissue. Finally, *S. latifolia* has separate sexes in the diploid phase (and sex is determined by the X and Y chromosomes), whereas *U. lactuca* does not.

1.4.2 Aspects of Life Cycle Evolution Investigated

Life cycles are highly evolutionarily significant; we expect most of the structural differences between life cycles to be important for individual survival and/or reproduction (Roff 1992, Stearns 1992). In addition, the variation in the way genetic material is exposed to natural selection (e.g., how many copies are present) and inherited (e.g., the asymmetrical inheritance pattern of XY sex chromosomes through males and females) will affect the way selection manifests changes in the hereditary material through time (Altenberg and Feldman 1987). Thus, it is perhaps surprising that organisms display such diverse life cycles. The evolutionary forces affecting some of the structural aspects of the life cycle are explored theoretically in this thesis. I present investigations into three aspects of life cycle evolution: developmental delays, selection in both ploidy phases, and sex chromosome evolution, which are all evident in the life cycles of *Silene latifolia* and *Ulva lactuca* described above.

Developmental Delays: Typically, we expect natural selection to favour organisms that reproduce as often and as quickly as possible (Rees 1996). However, many organisms delay development and subsequently reproduction for long periods (Tuljapurkar and Wiener 2000); a classic example is seed dormancy, as displayed by *S. latifolia*. In Chapter 2, we develop mathematical models that reveal three mechanisms via which developmental delays can be selectively favoured. One key novelty is that, unlike most previous models, we allow adults to be long-lived (e.g., a perennial plant rather than an annual). This yields the insight that dormancy can be favoured in order to minimize 'immaturity risk', that is, death in a large-scale environmental disturbance such as a fire before reproductive maturity is reached (mechanism 3 in Chapter 2), something that is not possible in a model of a short-lived, annual plant.

Haploid-Diploid Life Cycles: While sexual reproduction in eukaryotes necessitates an alternation between haploid and diploid phases, it is not

necessary for both haploid and diploid phases to experience selection to the same extent. For example, while *U. lactuca* appears to experience selection similarly in the haploid and diploid phases, the diploid phase of *S. latifolia* is physically much larger and very different from the haploid phase. The ploidy level (diploidy or haploidy) affects how alleles are exposed to selection because the presence of an extra genomic copy can 'mask' the fitness effects of an allele (Fisher 1930). Thus, masking can alter individual fitness directly and also alter the response to selection, affecting the frequency of alleles in future generations (Crow and Kimura 1965, Otto and Goldstein 1992). In Chapter 3, we evaluate whether life cycles evolve to expose either the haploid or diploid phase to selection. A key innovation in our model is that we fully explore fitness differences between haploids and homozygous diploids ('intrinsic fitness differences'). This reveals that the balance between intrinsic fitness differences and masking effects can favour haploid-diploid life cycles (growth and development in both phases).

Sex Chromosome Evolution: Finally, we consider the asymmetrical inheritance patterns of sex chromosomes, such as the X and Y chromosomes of *S. latifolia*. The presence of the Y sex-determining region specifies maleness and so the Y is always found in males, whereas the X is sometimes present in males and in females but more often in females. One consequence of this inheritance pattern is that associations can build up between male-beneficial alleles and the Y and between female-beneficial alleles and the X (Fisher 1931, Bull 1983, Rice 1987). Suppressed recombination between X and Y chromosomes is thought to evolve in order to strengthen these associations (Charlesworth and Charlesworth 1980, Lenormand 2003, Otto et al. 2011, Charlesworth 2015). In Chapter 4, we investigate the spread of large effect modifiers of recombination (such as fusions or inversions) that link haploid-expressed genes with the sex-determining region. We find that a period of haploid selection (e.g., pollen or sperm competition) can drive the evolution of suppressed recombination between sex chromosomes.

The studies in this thesis use mathematical models to investigate several components of life cycle evolution. The larger theory of life cycle evolution includes various other aspects, including the evolution of iteroparity (Cole 1954, Charnov and Schaffer 1973, Tuljapurkar and Wiener 2000), age at first reproduction (Stearns 1992, Roff 1992, Charlesworth 1994), senescence (Medawar 1952, Partridge and Barton 1993, Rose 1994), mating systems (Emlen and Oring 1977, Barrett and Eckert 2012), sexual systems (Barrett

2002, Otto 2009), the number of sexes (Hurst and Hamilton 1992, Togashi and Cox 2011), and dispersal (Hamilton and May 1977, McPeek and Holt 1992, Doebeli and Ruxton 1997). The overall aim of examining these problems is that, by combining the theory developed for the evolution of different aspects, we can better understand how and why complex life cycles (like those described above) evolved.

Chapter 2

Why Wait? Three Mechanisms Selecting for Environment-Dependent Developmental Delays[1]

2.1 Summary

Many species delay development unless particular environments or rare disturbance events occur. How can such a strategy be favoured over continued development? Typically, it is assumed that continued development (e.g., germination) is not advantageous in environments that have low juvenile/seedling survival (mechanism 1), either due to abiotic or competitive effects. However, it has not previously been shown how low early survival must be in order to favour environment-specific developmental delays for long-lived species. Using seed dormancy as an example of developmental delays, we identify a threshold level of seedling survival in 'bad' environments below which selection can favour germination that is limited to 'good' environments. This can be used to evaluate whether observed differences in seedling survival are sufficient to favour conditional germination. We also present mathematical models that demonstrate two other, often overlooked, mechanisms that can favour conditional germination in the absence of differences in seedling survival. Specifically, physiological trade-offs can make it difficult to have germination rates that are equally high in all environments (mechanism 2). We show that such trade-offs can either favour conditional germination or intermediate (mixed) strategies, depending on the trade-off shape. Finally, germination in every year increases the likelihood that some individuals are killed in population-scale disturbances before reproducing; it can thus be favourable to only germinate immediately after a disturbance

[1] A version of this chapter has been published. Michael F Scott and Sarah P Otto (2014) Journal of Evolutionary Biology, 27: 2219-2232.

(mechanism 3). We demonstrate how demographic data can be used to evaluate these selection pressures. By presenting these three mechanisms and the conditions that favour conditional germination in each case, we provide three hypotheses that can be tested as explanations for the evolution of environment-dependent developmental delays.

2.2 Introduction

One might expect organisms to reproduce as early as possible, yet many organisms delay development such that their eventual reproduction is also delayed, a strategy that should typically lead to a slower growth rate (Rees 1996). This is the classic evolutionary problem posed by developmental delays (Tuljapurkar and Wiener 2000), such as seed, spore, and cyst dormancy in plants, fungi and bacteria (Cohen 1967, Ellner 1985*a*, Rees 1996), non-seed ('prolonged' or 'vegetative') dormancy in plants (Roerdink 1988, Gremer et al. 2012), and diapause in insects, crustaceans, sponges and fish (Tuljapurkar and Istock 1993, Evans and Dennehy 2005, Venable 2007). In this paper we consider the evolution of strategies that delay development in a manner that depends on environmental state in a demographically structured population. First, we briefly review previous studies that explore the evolution of developmental delays and then place our work in this context.

Two classic studies of seed dormancy in annual plants are the influential theoretical papers by Cohen (1966; 1967). Cohen (1966) constrained germination rate to be the same in all years (constant germination strategy) but allowed the seed yield produced per germinating seed to vary across years. The optimal germination strategy was found to depend on the variation in yield across years. If, in some years, yield is lower than survival in the soil, partial seed dormancy can evolve. Cohen (1967) considered a different scenario, in which germination strategy can vary according to the environment at the time of germination (state-dependent germination strategy, sometimes called 'predictive germination', Venable and Lawlor 1980). If seeds are able to perfectly predict eventual yield based on the environment they experience, germination should occur in 'good' years and dormancy in 'bad' years. If the yield cannot be accurately predicted at the time of germination, then the optimal germination rate in a particular perceived environment depends on the distribution of yields that might actually occur; this set can include some 'good' and some 'bad' yields, in which case intermediate germination rates can again evolve. See the Model Background section for some mathematical details of these models.

Related studies have modelled the timing of diapause in insects and crustaceans in which the diapausing fraction can vary over a year in response to temperature and day length cues (Cohen 1970, Taylor 1980, Hairston and Munns 1984, Taylor and Spalding 1989, Spencer and Colegrave 2001). This is equivalent to an extremely plastic germination strategy, and these studies similarly find that populations should switch from non-diapausing to diapausing when the reproductive yield from breaking diapause is lower than the survival of a diapausing individual. For example, Taylor (1980) found that diapause should begin when the time until catastrophe (frost) is less than the time required to reach maturation and produce one offspring of diapausing age. This result assumes that the date of the first frost is predictable. In reality, catastrophes do not reliably occur on the same date each year. Consequently, there is variation in reproductive yield on each day, which can favour a mixed diapause strategy (Cohen 1970, Hairston and Munns 1984, Taylor and Spalding 1989, Spencer and Colegrave 2001).

The above models correspond to annual plant and diapausing insect life-cycles in which only individuals of a single age class persist between years. This allows the demographic dynamics to be described by a single equation: the number of seeds, diapausing eggs, lavae, pupae or adults that overwinter. However, developmental delays are also common in species with overlapping generations. For example, while not explicitly comparing germination rates in annuals and perennials, Baskin and Baskin (2014) find that the percentage of tree or shrub species with some form of seed dormancy is generally similar to the percentage of herbaceous species with dormancy (figures 12.3 and 12.4) in a review of over 13,000 species. With overlapping generations, demographic modelling becomes more complex because survival and reproduction of each age (or stage) class must be considered. Conceptually, a key difference is that lifetime reproductive output must be calculated over several time steps and so may include several environments and the particular order of those environments.

Nevertheless, there have been some studies that have considered the evolution of developmental delays in age- or stage- structured populations experiencing temporally varying environments. These studies generally consider environmental variation that affects fertility (seed yield) only (but see Koons et al. 2008, discussed below) and assume that strategies do not depend on the environment. Roerdink (1988; 1989) modelled the evolution of delayed reproduction in a predominantly biennial species that dies after reproducing. Similarly, Tuljapurkar (1990*a*) presented a model for the evolution of delayed reproduction in semelparous organisms and organisms with a very short adult life-span. Additionally, Tuljapurkar and Istock (1993)

considered the evolution of a short developmental delay, e.g., diapause in insects that can delay maturation for one year only. These studies have shown that delays can evolve in a demographically structured population to buffer against environmental variability in fertility, as in the unstructured model considered by Cohen (1966).

Developmental delays spread the reproductive effort from a seed/juvenile cohort over time, providing an 'escape in time' from environmental variation (Venable and Lawlor 1980). Iteroparity also spreads reproductive effort over time, buffering against environmental variation even in the absence of developmental delays (Tuljapurkar and Istock 1993, Tuljapurkar and Wiener 2000). Developmental delays can evolve in an iteroparous population, providing both forms of buffering, but only if mean seed (juvenile) survival is higher than mean adult survival and thus seeds (juveniles) are able to 'spread the risk' more than iteroparity alone (Koons et al. 2008). Tuljapurkar and Wiener (2000) also explored the evolution of both iteroparity and developmental delays, assuming a linear trade-off between adult survival and yearly reproductive effort. They tended to find either the evolution of iteroparity or developmental delays, but other trade-off functions might generate simultaneous selection for a mixture of iteroparity and developmental delay (as suggested by Wilbur and Rudolf 2006).

The above studies for demographically structured populations all assume a constant strategy in all years, as in Cohen (1966). Here, we model the evolution of a state-dependent strategy in a demographically structured population, that is, germination rate can be different in different environments. The case where cues allow the strategy to depend on the time of year has been considered in models for the timing of diapause (Taylor 1980, Hairston and Munns 1984, Spencer and Colegrave 2001). However, particular environments can provide cues that allow germination rates to vary in a state-dependent (not time-dependent) manner. Examples of state-dependent developmental delays include seed germination responses to light and rainfall (Pake and Venable 1996, Evans et al. 2007) or spore germination responses to heatshock (Perkins and Turner 1988), amino acid concentrations or host-specific substances (Cohen 1967). In a particularly clear example, smoke or temperature cues from fires cause increased germination rates or release of seeds from fruiting structures ('serotiny') in many species (including many perennials, Keeley 1995). Treatment with smoke is estimated to increase germination rates in over 2,500 species (Bradshaw et al. 2011) and up to 1,200 perennials exhibit serotiny (Lamont et al. 1991, Lamont and Enright 2000).

For simplicity, we will use botanical terms (seeds, germination, etc.), al-

though the models themselves can apply to other developmental delays that depend on environmental state. As discussed above and elsewhere (Rees 1996, Evans et al. 2007) the evolutionary problem posed by dormancy is that delaying development eventually delays reproduction and so reproductive opportunities seem to be passed up. In this context, the problem of conditional germination strategies is not 'Why germinate in environment 1?' but 'Why forgo germination in environment 2?'.

In this work, we investigate this problem and present three mechanisms generating selection that favours organisms that pass up germination opportunities: (1) Avoiding germination in 'bad' environments that have low seedling survival. (2) Avoiding costly physiological trade-offs between the germination rates in different environments (in addition to the fundamental 'trade-off' that seeds that germinate are no longer available to germinate in the future). (3) Minimizing the risk of experiencing a severe disturbance before reproducing (note that this requires state-dependent germination and perenniality).

This provides a framework for researchers wishing to investigate the evolution of environment-dependent developmental delays. We provide a threshold level of seedling survival in 'bad' environments below which conditional germination should evolve. Thus providing a quantitative means to test whether the most commonly envisaged mechanism can explain the evolution of conditional germination in a particular organism. If not (or if there are also physiological trade-offs or large-scale disturbances), we point out that the other, less commonly discussed, mechanisms should be considered. With demographic data for a particular species in different environments, one can investigate whether these selective mechanisms should act by manipulating the relevant parameters separately as we do here. For example, setting seedling survival in all environments to be equivalent eliminates mechanism 1 and reducing the number of years required to reach maturity can eliminate mechanism 3. We discuss some specific empirical data for these mechanisms in more detail in the discussion section.

2.3 Model Background

To connect our model with previous results, we first provide a brief overview of some key mathematical results. In the model by Cohen (1966), the num-

ber of seeds (S) at time t is given by

$$S[t] = S[0]\left(\prod_i ((1-g)s_S + gy_i)^{p_i}\right)^t, \tag{2.1}$$

where g is the germination rate (assumed constant), s_S is the survival of seeds in the soil and p_i is the proportion of the t years that has environment i in which the environment-specific seed yield is y_i. Increasing germination rate will increase (decrease) growth rate if the derivative of the parenthetical term with respect to g is positive (negative), where the sign of this derivative depends on $\sum_i \frac{p_i(y_i - s_S)}{(1-g)s_S + gy_i}$. Dormancy may evolve if some years yield fewer seeds than would survive in the soil ($y_i < s_S$). For example, a population that germinates 100% of its seeds would go extinct if ever an extremely 'bad' year (no seed set) were encountered, favouring the evolution of seed dormancy.

Where environments vary over space, however, lineages can escape extinction by surviving in 'good' environments and recolonizing. This has been called 'escape in space' via dispersal in contrast to 'escape in time' via dormancy (Venable and Lawlor 1980). MacArthur (1972, p.165-168) introduced a model with many patches and global dispersal among them, finding that the optimal strategy is the one that has the highest growth rate averaged over all patches. In this model, a proportion of the population experiences each environment in each year and so

$$S[t] = S[0]\left(\sum_i p_i((1-g)s_S + gy_i)\right)^t, \tag{2.2}$$

where p_i is the proportion of the population that experiences environment i with yield y_i. In this model, changes in germination rate affect growth rate according to $\sum_i p_i(y_i - s_S)$, which must be positive in a population capable of growth, therefore seed dormancy should not evolve. These two models, with variability entirely temporal or spatial are extreme cases and intermediate scenarios have been considered by others (Levin et al. 1984, Cohen and Levin 1987, Klinkhamer et al. 1987, Wiener and Tuljapurkar 1994), who also find that 'escape in space' via dispersal lessens the need for 'escape in time' via dormancy.

Closer to the models we consider, Cohen (1967) includes environment-specific germination into equation (2.1):

$$S[t] = S[0]\left(\prod_i ((1-g_i)s_S + g_i y_j)^{p_{ij}}\right)^t, \tag{2.3}$$

where g_i is the germination rate in a particular seed environment and p_{ij} is the proportion of years that seeds are in environment i and yield y_j seeds if germinated. Selection on the germination rate in a particular environment (g_i) then has the same sign as $\sum_{ij} \frac{p_{ij}(y_j - s_S)}{(1-g_i)s_S + g_i y_j}$. Evolution of germination rate in each environment therefore evolves in a similar manner to the overall germination rate in the Cohen (1966) model. However, each seed environment can have a different optimum. In a special case (termed 'complete information'), the yield is reliably given by the seed environment (i), such that p_{ij} and y_j can be replaced by p_i and y_i. In this case, the pure strategies of complete germination ($g_i = 1$) and complete dormancy ($g_i = 0$) are favoured in 'good' ($y_i > s_S$) and 'bad' ($y_i < s_S$) environments respectively.

MacArthur (1972) did not include environment-specific germination rates into his model with purely spatial environmental variation. However, one can modify equation (2.2) to allow germination rate to vary along with the environment that affects seed yield, such that g becomes g_i. This modification may seem equivalent to the 'complete information' case in Cohen (1967), but it also applies with uncertain assessment of yield if y_i is defined as the average yield from seeds across environments – correctly or incorrectly – assessed as being in state i. Although the yield in each patch is uncertain, this uncertainty can be averaged across the patches in each year to give a particular yield for each seed environment. This model also predicts complete germination in 'good' ($y_i < s_S$) patches and dormancy in 'bad' ($y_i < s_S$) patches.

In this study, we consider perennial species and assume that a fixed proportion of the population experiences each environment in each time step in sections 1 and 2 (mechanisms 1 and 2), as in the annual plant model by MacArthur (1972, p165-168). We use the approach explained above to include environment-specific germination rates. In the final section, we include temporal variation where the whole population experiences the same environment in each time step, as in Cohen (1966; 1967). In order to deal with temporal variation in a demographically structured population we first consider strictly periodic disturbances to obtain some approximate analytical results and then use numerical simulations based on the demography of *Banksia hookeriana* (following Enright et al. 1998) to investigate the evolution of environment-dependent developmental delays with non-periodic disturbances. For this section we consider the 'complete information' case because we focus on the effects of disturbance risk rather than uncertain assessment of yield. That said, when disturbances are non-periodic, we incorporate uncertainty in the ordering of environments even though the

demographic parameters in each environment are constant.

2.4 Model and Results

We evaluate the evolution of environment-dependent germination (conditional germination) with a variety of stage-structured models. All analyses were conducted using *Mathematica* (Wolfram Research Inc. 2010), a file for replicating our analyses is available on request. We considered environmental variation that can affect all life-history parameters. In our notation for environment i, the survival of adult plants is s_{Ai}, seed survival is s_{Si}, germination rate is g_i, post-germination seedling survival is s_{Yi} and each adult produces b_i seeds in each time step. We allow both seeds (S) and adults (A) to survive between time steps.

2.4.1 Mechanism 1: Low Seedling Survival in Some Environments

It is commonly thought that conditional germination evolves to avoid germination in environments with low seedling survival (e.g., Lamont et al. 1991, Lamont and Enright 2000, Midgley 2000, Keeley et al. 2011). To test this mechanism we first modelled a population in which a random proportion of the population (p_i) experience each environment in each time step ($\sum_{i=1}^{n} p_i = 1$, where n is the total number of environments), with no temporal autocorrelations in patch type (either because migration is global or patches change randomly at each time step). Initially, we examine a density-independent growth model, but we then show that similar conditions arise with a density-dependent model. The change in seed and adult population sizes from time step t to time $t+1$ are described by the following recursion equations written in matrix form:

$$\begin{pmatrix} S[t+1] \\ A[t+1] \end{pmatrix} = \mathbf{T_A} \begin{pmatrix} S[t] \\ A[t] \end{pmatrix}, \tag{2.4}$$

where

$$\mathbf{T_A} = \begin{pmatrix} \sum_{i=1}^{n} p_i s_{Si}(1-g_i) & \sum_{i=1}^{n} p_i b_i \\ \sum_{i=1}^{n} p_i s_{Yi} g_i & \sum_{i=1}^{n} p_i s_{Ai} \end{pmatrix}. \tag{2.5}$$

We used the leading eigenvalue (λ) of the transition matrix, $\mathbf{T_A}$, to approximate the long-term growth rate of the population of seeds and adults. Then we examined whether mutants that alter the germination parameters (g_i) have an increased or decreased long-term growth rate. A small change in the germination rate in environment j, g_j, will affect the long-term growth rate, λ, according to

$$\frac{\partial \lambda}{\partial g_j} = \frac{p_j s_{Sj} \sum_{i=1}^{n} p_i s_{Ai} + p_j s_{Yj} \sum_{i=1}^{n} p_i b_i - p_j s_{Sj} \lambda}{2\lambda - \sum_{i=1}^{n} p_i s_{Ai} - \sum_{i=1}^{n} p_i s_{Si}(1 - g_i)}. \tag{2.6}$$

Unlike an annual plant version of the same model, equation (2.6) has terms from all n environments. That is, optimal germination rate in environment j depends on the quality of the other environments that adults might subsequently experience when demographic structure is included. If equation (2.6) is positive for some environments (j) and negative for others, then conditional germination is expected to evolve. From this point on we will focus on the case where environments can be classified into two groups. Two is the minimum number of environments required for conditional germination, in which dormancy is favoured in one environment but not another.

In this section we demonstrate that differences in seedling survival can favour conditional germination. For this purpose we define a 'good' ($i = 1$) patch as one in which seedling survival is higher than in the 'bad' ($i = 2$) patches ($s_{Y1} > s_{Y2}$). Assuming that the population is capable of growth ($\lambda > 1$), germination rates in the 'good' environment should always be maximized (mutants with higher g_1 values always have high higher long-term growth rates). By contrast, germination rates in the 'bad' environments (g_2) should sometimes evolve to be as high as possible and sometimes as low as possible, with the transition occurring when the following condition holds:

$$\frac{s_{Y2}(b_2 s_{Y2} + s_{A2} - s_{S2})}{g_1 s_{S1}(s_{Y1} - s_{Y2}) + s_{Y2}(s_{Y2}(b_2 - b_1) + s_{A2} - s_{A1} + s_{S1} - s_{S2})} - p = 0, \tag{2.7}$$

where we have specified that the 'good' ($i = 1$) environment is experience by p proportion of the population and the 'bad' ($i = 2$) environment by $(1-p)$. See appendix A.1 for more details of our analysis. An example of how the long-term growth rate (λ) changes on either side of this point is shown in figure 2.1A.

Figure 2.1B illustrates the region in which conditional germination is

expected to evolve, with germination only occurring in 'good' patches. The proportion of 'good' patches (p) must be high enough and seedling survival in 'bad' patches (s_{Y2}) must be sufficiently low. When seedling survival in both environments is equivalent (dashed line in figure 2.1B), conditional germination should never evolve in populations capable of growth ($\lambda > 1$). It has previously been noted that conditional germination should evolve when establishment ability in 'bad' environments is negligible ($s_{Y2} = 0$, Lamont et al. 1991). Equation (2.7) echoes this result but also shows a more general case, in which we indicate exactly how low seedling survival in 'bad' patches (s_{Y2}) must be.

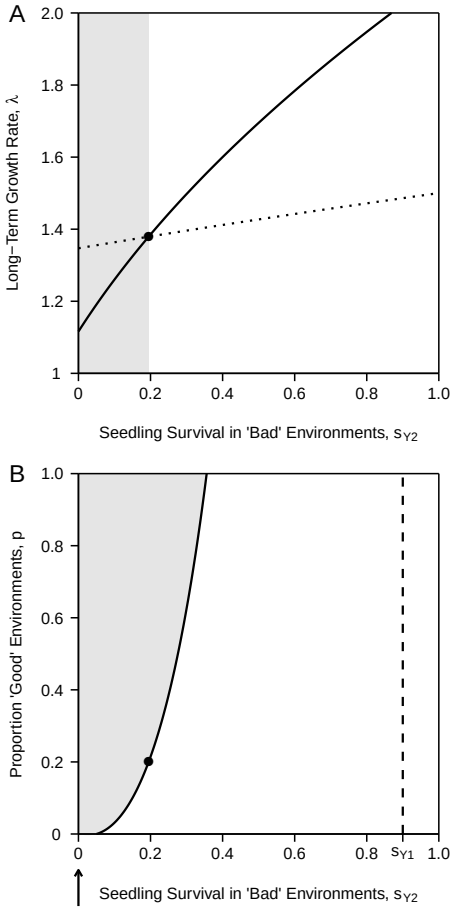

Figure 2.1: Life-history parameters for which conditional germination strategies have a higher long-term growth rate. A) The long-term growth rate (λ) for a plant with a conditional germination strategy (dotted line, $g_2 = 0.1$) and a plant without one (black line, $g_2 = 1$). Conditional germination confers a higher growth rate when seedling survival is below the transition point specified by equation (2.7) (circle). The grey region shows where conditional germination is expected to evolve. B) When the frequency of 'good' environments (p) is high enough and the seedling survivorship in bad years (s_{Y2}) is low enough, conditional germination should evolve (grey). If seedlings never establish in 'bad' years ($s_{Y2} = 0$, see arrow), a plant always falls in the region favouring conditional germination (grey). The dashed line indicates where seedling survival is equal across 'good' and 'bad' years ($s_{Y1} = s_{Y2}$), in which case conditional germination never evolves (see appendix A.1). Note that, even where selection would favour germination if only the 'bad' environment were experienced (see white region along x-axis, $p = 0$), conditional germination can evolve. The other parameters used are $g_1 = 1$, $p = 0.2$, $s_{Y1} = 0.9$, $s_{S1} = 0.8$, $s_{S2} = 0.8$, $b_1 = b_2 = 4$, $s_{A1} = 0.6$, $s_{A2} = 0.6$.

In appendix A.1 we also show that the region in which conditional germination should evolve expands when the seed bank is more persistent (s_{Si} is larger), the proportion of the population experiencing 'good' conditions

(p) is larger and when germination rate (g_1) and seedling survival (s_{Y1}) in good patches is higher. In contrast, this region will contract when adult survival (s_{Ai}) is higher, number of seeds produced (b_i) is higher, or when seedling survival in 'bad' years (s_{Y2}) is higher.

2.4.2 Mechanism 2: Trade-Offs

In the above model and MacArthur (1972), germination rates evolve to be either maximized or minimized. We next include physiological trade-offs, which can allow intermediate germination rates to evolve even with purely spatial variation in environments. This is true for both annual and perennial plant models (see appendix A.2 for a version of the MacArthur 1972 annual plant model with trade-offs, which produces very similar results to the perennial model presented here). Trade-offs could exist between any of the demographic parameters, see the discussion section for some examples, but to demonstrate the qualitative effects of trade-offs on germination rate, we incorporated a direct trade-off between germination rates in different environments (g_1 and g_2) using a generic function ($g_1[g_2]$). For two types of patches and global migration, the transition matrix describing changes in seed and adult populations then becomes:

$$\mathbf{T_B} = \begin{pmatrix} ps_{S1}(1-g_1[g_2]) + (1-p)s_{S2}(1-g_2) & pb_1 + (1-p)b_2 \\ ps_{S1}s_{Y1}g_1[g_2] + (1-p)s_{S2}s_{Y2}g_2 & ps_{A1} + (1-p)s_{A2} \end{pmatrix}. \tag{2.8}$$

Here, we are particularly interested in cases where conditional germination is expected to evolve where it wouldn't without the trade-off. Therefore, we start by presenting the special case of (2.8) where seedling survival is constant ($s_{Y1} = s_{Y2}$), which never yielded conditional germination strategies in the previous section.

Our approach (details in appendix A.2) was to identify evolutionarily stable strategies (ESS) for germination rates (g_1 and g_2) where no mutant would have a higher growth rate, λ. For model (2.8) with $s_{Y1} = s_{Y2}$, a singular point occurs when:

$$\frac{s_{S2}}{s_{S2} - s_{S1}g_1'[g_2]} - p = 0 \tag{2.9}$$

where $g_1'[g_2]$ is the first derivative of the trade-off function. In some cases, traits that maximize germination rate in one environment could also increase germination rates in other environments (e.g., Simons 2014). If germination rates are positively coupled in this manner ($g_1'[g_2] > 0$), the singular point

in (2.9) cannot be satisfied and germination rates should evolve to be high. However, in figure 2.2B, we plot equation (2.9) for a negative trade-off (where physiological constraints make it difficult to have simultaneously high germination rates in all environments, $g_1'[g_2] < 0$). We next determined whether this singular point is a maximum or a minimum growth rate in order to assess whether germination rates are expected to evolve towards this point or away (whether it is an ESS or evolutionary repeller). We found that singular point (2.9) changes from an ESS to an evolutionary repeller when the shape of the trade-off function transitions between concave ($g_1''[g_2] < 0$) and convex ($g_1''[g_2] > 0$), see figure 2.2.

When trade-offs are concave (solid line in figure 2.2), seeds are able to germinate reasonably well in both environments, and the germination strategy is expected to reach an intermediate ESS germination rate in both environments, where the two germination rates satisfy equation (2.9). Observing intermediate germination rates could then suggest the presence of a trade-off (e.g., Tonnabel et al. 2012, discussed below) or temporal variation (see next section).

With a convex (dashed line in figure 2.2) trade-off, plants are expected to specialize on germination in either environment 1 or 2. Thus, conditional germination can evolve as a means to specialize and avoid a costly trade-off. The germination strategy predicted to evolve with a convex trade-off depends on seed survival rates (s_{Si}), the proportion of patch types 1 versus 2 and any initial specialization. Importantly, though, even if survival and fertility are equal in all environments, conditional germination can still evolve, simply because the traits that allow good germination in one environment prevent it in the other. Empirically then, trade-offs are likely present in cases where little difference in demographic parameters can be detected.

We next combine the effects of a trade-off with differences in seedling survival (mechanisms 1 and 2). As in the previous section we arbitrarily assume that environment 1 has superior seedling survival ($s_{Y1} > s_{Y2}$). When $s_{Y2} \neq s_{Y1}$ the simple solution (2.9) no longer applies. We obtained a more complicated expression for the singular point (ESS or repeller) and plotted an example in figure 2.2C. What is apparent is that, decreasing seedling survival in 'bad' environments (s_{Y2}) increases the region of parameter space over which germination rates in the 'bad' environments should evolve to be low.

The above models ignore competition and assess growth rates of different life-history strategies. We next incorporated density dependence into this model by including a competition function that limits population size. For example, competition-related mortality might affect seedling survival such

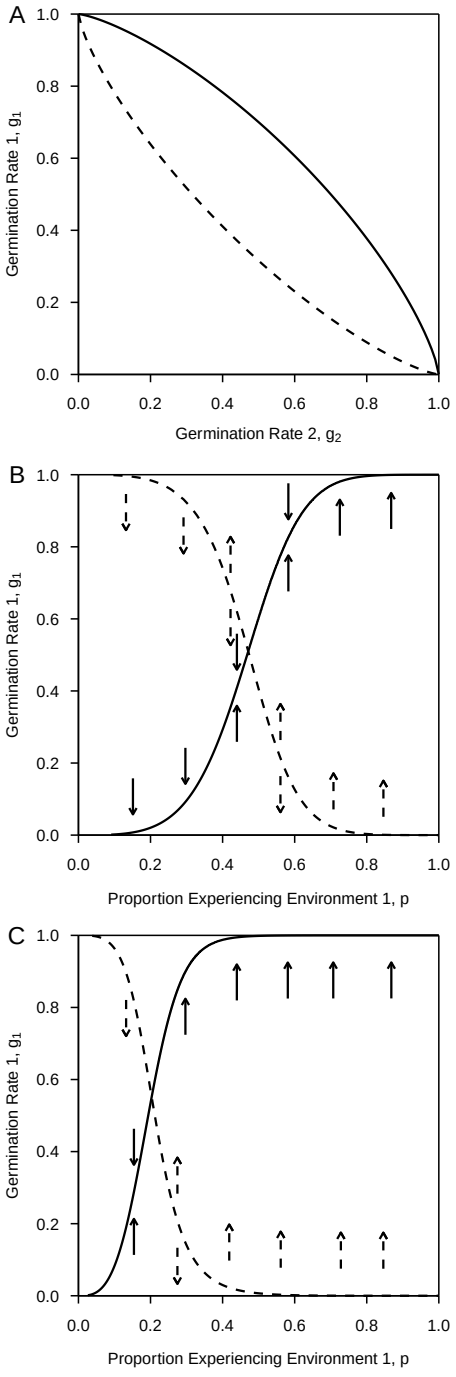

Figure 2.2: Trade-offs between germination rates (g_1 and g_2) can favour intermediate strategies or specialization. Panel A) shows a concave (solid, $s = 4/3$) and convex (dashed, $s = 3/4$) example for the trade-off function using $g_1[g_2] = 1-(1-(1-g_2)^{\frac{1}{s}})^s$. Panels B) and C) show the evolutionarily stable germination strategy (solid) or the repelling strategy (dashed), with arrows representing the expected evolutionary trajectory for germination rate for a given frequency of environments 1 (p) versus 2 ($1-p$). Panel B) assumes $s_{Y2} = s_{Y1}$ (corresponding to the dashed line in figure 1B), in which case the curves are given by equation (2.9). Panel C) shows an example where $s_{Y2} \neq s_{Y1}$ ($s_{Y1} = 0.9$, $s_{Y2} = 0.4$). Other parameters in B) and C) are as in figure 1.

that the lower left element in matrix $\mathbf{T_B}$ is multiplied by the logistic density-dependent function $(1 - \frac{A[t]}{K})$ where K is the population carrying capacity of adults. More generally, we multiplied seedling survival by an arbitrary competition function ($comp[A[t]]$) to re-write the transition matrix as:

$$\mathbf{T_C} = \begin{pmatrix} ps_{S1}(1 - g_1[g_2]) + (1-p)s_{S2}(1-g_2) & pb_1 + (1-p)b_2 \\ (ps_{S1}s_{Y1}g_1[g_2] + (1-p)s_{S2}s_{Y2}g_2)comp[A[t]] & ps_{A1} + (1-p)s_{A2} \end{pmatrix}. \tag{2.10}$$

We then conducted an evolutionary invasion analysis, in which a resident population was allowed to reach an equilibrium size (assuming this to be stable) and then the invasion ability of a mutant with a different germination rate was evaluated, as measured by the leading eigenvalue of $\mathbf{T_C}$ for a rare mutant (details in appendix A.2). If germination rates affect the number of seedlings but not the nature of competition (i.e., $comp[A[t]]$ is not a function of g_1 or g_2), the results remain the same as above (for mechanisms 1 and 2), but with birth rates now multiplied by $comp[A[t]]$.

2.4.3 Mechanism 3: Effects of Synchronization With Disturbances

Here we focus on a particular type of temporal variation in environment, such as large-scale disturbances like fire, which affect adult survival and potentially germination rates across the entire population at the same time. Synchronizing germination to occur immediately after a disturbance then maximizes the number of years as an adult before experiencing the next disturbance. By contrast, plants that germinate in non-disturbance years 1) have fewer chances to produce seeds before experiencing a disturbance and 2) are more likely to die in a disturbance before producing seeds at all. We show that these costs of poor synchronization can be strong enough to cause plants to forgo germination in years without disturbances, even in the absence of differential seedling survival or trade-offs.

In this section, the notation for environment 1 ($i = 1$) is used for years with population-scale disturbances and environment 2 ($i = 2$) specifies life-history parameters in non-disturbance years. We assume that the population census is such that germination rate in disturbance years (disturbance-induced germination rate, g_1) is measured for the first germination event after the disturbance (so that it can be affected by disturbances). With fire, for example, fire years ($i = 1$) would be associated with low adult survival (s_{A1}) but potentially high seedling survival (s_{Y1}) because seeds emerging after the fire experience a low competition and high nutrient environment.

The transition matrices describing changes in seed and adult population sizes in non-disturbance and disturbance years are as follows:

$$\mathbf{T_1} = \begin{pmatrix} s_{S1}(1-g_1) & b_1 \\ s_{S1}s_{Y1}g_1 & s_{A1} \end{pmatrix}, \tag{2.11a}$$

$$\mathbf{T_2} = \begin{pmatrix} s_{S2}(1-g_2) & b_2 \\ s_{S2}s_{Y2}g_2 & s_{A2} \end{pmatrix}. \tag{2.11b}$$

Firstly, we consider a disturbance cycle, in which disturbances occur every τ years. That is, we include a number of non-disturbance years $(\tau-1)$ followed by a disturbance year. To describe population size changes over the entire cycle we apply the disturbance year transition matrix ($\mathbf{T_1}$) and then iterate the transition matrix $\tau - 1$ times for non-disturbance years ($\mathbf{T_2}$). Using standard rules of matrix algebra,

$$\mathbf{T_2}^{\tau-1} \cdot \mathbf{T_1} = \mathbf{A} \cdot \mathbf{D}^{\tau-1} \cdot \mathbf{A}^{-1} \cdot \mathbf{T_1} \tag{2.12}$$

where $\mathbf{A}$ is a matrix in which the columns are the eigenvectors of $\mathbf{T_2}$ and $\mathbf{D}$ is a matrix in which the diagonal elements are the eigenvalues of $\mathbf{T_2}$.

The logic of our analysis is similar to above. We evaluate whether modifying the germination parameters increases or decreases the long-term growth rate, λ, given by the leading eigenvalue of the entire cycle matrix (equation 2.12). We provide the details of our approach in appendix A.3.

While (2.12) accurately describes changes in the long-term growth rate over the entire cycle, it is quite complex to analyse. We thus used an approximation to simplify the analysis. Specifically, we assume that $\mathbf{D}$ can be approximated by omitting the smaller eigenvalue. This approximation is most accurate when the difference between eigenvalues is large and/or when the number of years between disturbances is large (over time, the effects of the larger eigenvalue dominate, e.g., Otto and Day 2007, box 9.1). Care must therefore be taken in interpreting the results when the cycle length is short, which is also when we find that conditional germination strategies tend to be favoured. Thus, this approximation only serves as a guide to conditions that favour conditional germination; the accuracy of the approximation is discussed in appendix A.3.

To distinguish synchronization effects from those already explored, we focus on the case where there are no direct trade-offs between germination rates (g_1 and g_2) and where seedling survival rates in disturbance and non-disturbance environments are equal ($s_{Y1} = s_{Y2}$). We found that mutants

with higher disturbance-induced germination rates (g_1) are expected to have higher long-term growth rates, given that the population is able to grow in normal years (as assumed throughout this section). We therefore assumed that disturbance-induced germination rate is high ($g_1 = 1$) when analyzing the evolution of the germination rate in non-disturbance years, g_2. For very long disturbance cycles (high τ), higher germination rates in non-disturbance years should also give higher long-term growth rates. However, when the disturbance cycle is short enough (less than the critical value τ_c, see equation A.46), conditional germination, $g_2 < 1$, is favoured.

We took the derivative of τ_c with respect to life-history parameters in the disturbance year to see the effect that the parameters have on the length of the disturbance cycles over which conditional germination is expected to evolve. We found that increasing seed bank persistence through disturbances (s_{S1}) and increasing disturbance-induced germination (g_1) increases the parameter space over which selection favours conditional germination strategies. However, increasing seeds produced in the disturbance year (b_1) and adult survival through disturbances (s_{A1}) decreases the range of disturbance intervals for which conditional germination should evolve.

Our results indicate that, conditional germination ($g_2 < 1$) should evolve when adults that germinate in non-disturbance years risk death in a disturbance before producing a significant number of seeds. By contrast, conditional germination should not generally evolve when disturbances have little effect on adult survival (s_{A1} is high) and when adults are guaranteed to produce a large number of seeds even if they mature for the first time in the disturbance year (b_1 is high), see figure 2.3.

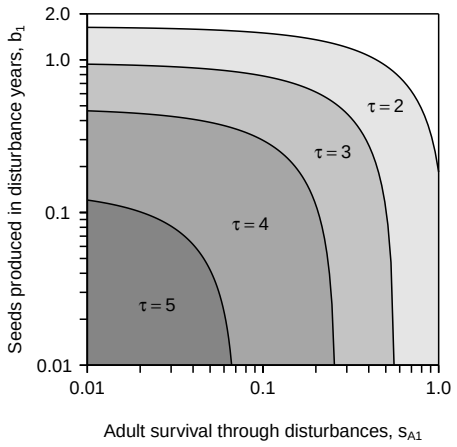

Figure 2.3: The parameters for which conditional germination is expected to evolve for various different disturbance cycle lengths (τ) based on our approximation, assuming adults reach reproductive maturity immediately (in the time step after germination). See figure A.2 for comparison with a non-approximated model. Increasingly dark grey areas indicate where the germination rate in non-disturbance years (g_2) is expected evolve to be below one (conditional germination) for cycle lengths of 2, 3, 4 and 5 (lighter regions overlap darker regions). In the white region, conditional germination is not expected to evolve for any cycle length, τ. Other parameters are $g_1 = 1$, $s_Y = 0.6$, $s_{S1} = s_{S2} = 0.9$, $b_2 = 2$ and $s_{A2} = 0.7$.

Figure 2.3 suggests that conditional germination should only evolve for relatively short disturbance cycles. However, in the above models, organisms become reproductively mature after one year and so the advantages of synchronization are necessarily weak. We expand on these analytical results using some numerical simulations that include more complex demography.

Table 2.1: Default parameters used in numerical simulations

Parameter	Symbol	Default Value
fire-induced germination rate	g_1	1
normal germination rate	g_2	0-1*
seed survival	$s_{S2} = s_{S1}$	0.94
adult survival	s_{A2}	$p[age]^\dagger$
adult survival (fire)	s_{A1}	0.005
seedling survival	s_Y	0.042
seed production	$b_2 = b_1$	$m[age]^\ddagger$
age at first reproduction	$A2$	5
age at max reproduction	$A3$	15
max seed age	Vm	15
max adult age	$A4$	40

* Varied between 0 and 1 in steps of 0.05, the value yielding the highest long-term growth rate (λ, leading eigenvalue of the transition matrix) was recorded.

† For $1 < age < 25$, $p[age] = (1/f[age])/(1/f[age-1])$ where $f[age] = 69.03 \log_{10}[age] + 23.60$. For $25 \leq age$, $p[age] = (1/f[age])/(1/f[age-1])(1 - 0.01(age - 24))$.

‡ For $age < A2$, $m[age] = 0$. $m[age] = 200$ when $A3 \leq age$. For $A2 \leq age < A3$, $m[age] = \frac{200(age+1-A2)}{1+A3-A2}$.

We based our simulations on those of Enright et al. (1998), using parameters that approximately correspond to the demography of *Banksia hookeriana*, an Australian shrub in the Proeaceae that retains almost all seeds on the plant until immediately after a fire. The parameters are given in Table 2.1. The major technical difference between our simulations and those of Enright et al. (1998) is that we assume seeds remain in the seed bank after plant death, whereas seeds died with the parent (but not in fires) in the original model. This change allowed us to simulate the entire population by multiplying by the appropriate matrix in (2.11) rather than tracking individuals. We also allow a small fraction of adults to escape disturbances in microclimates ($s_{A1} = 0.005$), this prevents complete population extinction if ever two disturbance events occur in a row. We made two important biological modifications to expand on our analytical results: 1) we varied

the number of years before maturity is reached to show that conditional germination should only evolve when there is a significant risk of death before producing seeds, 2) we explored non-periodic disturbances (fires in this model) to show that the 'synchronization effect' continues to favour conditional germination. In all our simulations, there is no difference in seedling survival (s_Y) between environments (mechanism 1 absent).

For particular fixed disturbance (fire) cycle lengths, we varied the number of years to first reproduction ($A2$), from 1 to 3 to 5 years and recorded the optimal germination rate in normal years (the g_2 that yielded the highest long-term growth rate, λ). The results are plotted in figure 2.4, which shows that the advantage of conditional germination is increased when the number of years to reproductive maturity is increased. This demonstrates that 'synchronization advantages' favour conditional germination in this model, which was not originally made explicit in Enright et al. (1998).

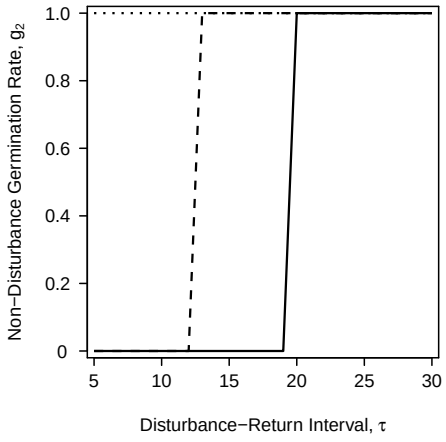

Figure 2.4: The germination rate in non-disturbance (fire) years (g_2) that yields the highest long-term growth rate in our numerical analysis of a life history akin to *Banksia hookeriana* (Enright et al. 1998) for different disturbance-return intervals. The solid line is for the default parameters with an age of reproductive maturity of 5 years, whereas the dashed and dotted lines are where age at first reproduction ($A2$) was reduced to 3 and 1 years, respectively. Notice that when adults become reproductively mature immediately environment-dependent germination never evolves (dotted line).

For variable disturbance cycles, we next drew integer disturbance intervals from a Weibull distribution, see figure 2.5A. We varied the regularity of disturbances by using a shape parameter (β) of 1, 2 or 4, which represent increasing regularity of disturbances, starting from the exponential distribution ($\beta = 1$, constant disturbance risk, $\beta = \infty$ corresponds to the periodic case considered above). In figure 2.5B we plotted the germination strategy in non-fire years that gave the highest growth rate (averaged across replicate 100 draws of 20 disturbances) for various mean disturbance intervals. Figure 2.5B shows that, even when disturbance intervals are highly variable ($\beta = 1$), conditional germination (low g_2) can be advantageous. We also note from figure 2.5B that variability tends to favour mixed strategies, with

g_2 values between zero and one, representing bet hedging between the long and short intervals.

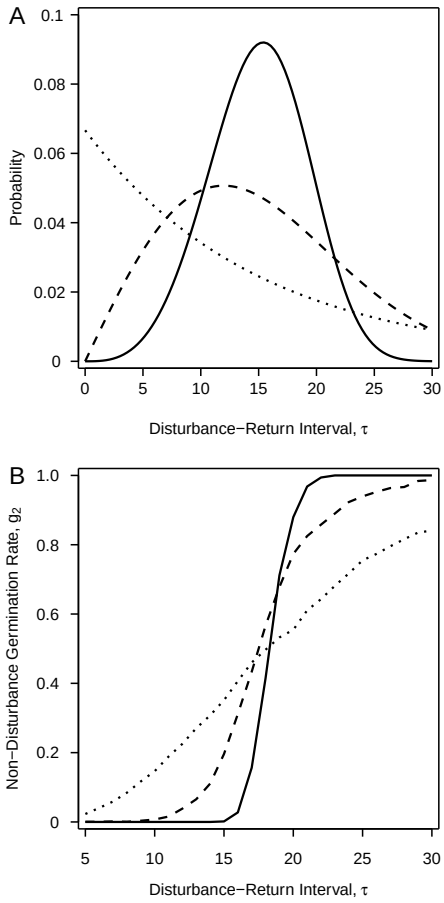

Figure 2.5: The effect of variability in disturbance-return interval on the evolution of conditional germination. A) The shape parameter affects the Weibull distribution used for the fire-return interval (τ). The mean in each case is 15 years between disturbances. The dotted line shows the probability of selecting disturbance-return intervals when the Weibull shape parameter (β) is 1 and is equivalent to a exponential distribution with expected value 15. The dashed and solid lines are for $\beta = 2$ and 4 respectively and represent increasing regularity due to an increasing hazard with time since the last disturbance. B) The solid, dashed and dotted lines show the corresponding average germination rate in non-disturbance years (g_2) that yielded the highest long-term growth rate in our simulations.

2.5 Discussion

In this paper, we explored three mechanisms by which a developmental delay (e.g., seed dormancy) can be favoured in certain environments but not in others. This work builds upon the model of annual plants developed by Cohen (1967) but allows for demographic structure. While Cohen predicted that optimal germination strategies would match the yields from any one environment, demographic structure complicates the picture because yield must be calculated over multiple time steps and hence over multiple

environments. We identified three mechanisms by which a developmental delay triggered by the state of the environment (conditional germination) can evolve.

Mechanism 1: If seedling survival is sufficiently low in 'bad' environments, it is optimal to limit germination to 'good' patches. In desert plants, seedling survival is much higher in years with high rainfall, and germination rates are correspondingly higher when early season rainfall is high (Evans et al. 2007). Similarly, 'classical disturbances' (White and Pickett 1985), such as fires, create discrete patches in which resources are higher due to decreased biological use, an ash-bed effect (Serrasolses and Vallejo 1999, Pausas et al. 2003) and/or increased decomposition. For non-annuals, only the extreme case in which seedling survival is impossible in 'bad' environments has been formally considered (in the context of post-fire germination responses, Lamont et al. 1991). Empirically, the establishment ability of seeds germinating in post-fire environments is not always elevated, and establishment in other years is often not negligible (e.g., O'Dowd and Gill 1984, Cowling and Lamont 1987, Brewer 1999, Quintana-Ascencio and Menges 2000, Liu et al. 2005). As Bond and Wilgen (1996, p142) point out, it was not previously obvious whether reported differences in seedling survival are large enough to select against germination in 'bad' years.

We used a simple model lacking trade-offs and temporal variation to find a threshold level of seedling survival in 'bad' patches below which conditional germination is expected to be advantageous. The conditions for conditional germination to evolve via this mechanism are broader when 'good' environments are common, seed survival is high, adult survival is low and seed production is low. These results can thus guide empirical work to determine whether demographic parameters would or would not favour conditional germination in a particular species.

In sections 1 and 2, we used the simplifying assumption that a random proportion of the (many) patches experience each environment in each time step, with no reference to the previous environments experienced. Thus, after germination and seedling survival occurs in a particular environment, there is no link between the environment experienced at the time of germination and the subsequent environments experienced by adults. We predict that, in a spatially explicit model where the environment experienced across the life span depends on the environment at the time of germination, low adult survival and fecundity (not just low seedling survival) in 'bad' environments could also favour conditional germination, assuming seeds can experience different environments by delaying germination.

We also incorporated intraspecific competition affecting seedling survival

and found that our results were quantitatively altered but qualitatively unaffected. Similarly, the density independent annual plant model by Cohen (1966) was extended to include density dependence by Bulmer (1984), Ellner (1985*a*) and Ellner (1985*b*). In these annual plant models with temporal environmental variation, density dependence can exacerbate the effect of environmental variation on germination fraction (or create temporal variation via deterministic dynamics, Ellner 1987). In addition, we note that annual plant models show that spatial structure can introduce sibling competition, which can reduce the optimal germination fraction (Ellner 1987). Gremer and Venable (2014) find that annual plant models with density dependence included predict germination fractions more accurately than density independent models. We caution that our model of competition was highly idealized in order to make analytical headway. While density-dependent competition was experienced equally everywhere in our model, competition should be lessened in patches that have recently experienced low adult survival. A more appropriate but complex model would be spatially explicit with differences in seedling survival affected by competitive interactions only within the same patch.

Mechanism 2: Trade-offs can make it difficult to germinate equally well in all environments, making conditional germination more likely to evolve. We considered a direct physical or developmental trade-off between germination rates, such that a plant would have to decrease germination rate in environment 1 to increase the germination rate in environment 2. This trade-off is over-and-above the fact that seeds that germinate in one environment are unavailable to germinate in the future, which can also be seen as a form of trade-off that underlies all models of delayed development.

Trade-offs are likely to arise whenever the features that protect seeds from the environment also alter their ability to germinate. For example, thickened seed coats or retention in cones may prevent germination in most environments but allow seeds to survive fires and thus allow increased germination in a post-fire environment. Indeed, many species with temperature-induced germination produce a mixture of seeds that are specialized for either post-fire or for inter-fire germination (Keeley 1995). This suggests that individual seeds cannot do both well, which will generate a trade-off if the total number of seeds is limited. Previously, Tonnabel et al. (2012) considered a trade-off between seed production and maintenance (b and s_S here). They assume seedling survival in 'bad' environments is negligible so that selection should maximize germination in 'good' (post-fire) environments only, which occurs at an intermediate (mixed) strategy with their trade-off.

To demonstrate the effects of trade-offs on conditional germination we considered a direct trade-off between germination rates, which has not been explored before. We show that, with convex trade-off shapes (dashed line in figure 2.2), specialized germination strategies are favoured, even for parameters that did not favour conditional germination in the model without trade-offs. By contrast, concave trade-offs (solid line in figure 2.2) can favour a mixed strategy with some germination in both environments, which maximizes reproductive opportunities across all patches. Thus, intermediate germination rates can be favoured because of trade-offs, in addition to bet hedging caused by temporal environmental variation (see next section).

Mechanism 3: Limiting germination to disturbance events minimizes the risk of experiencing another disturbance before reproducing. The timing of insect diapause is thought to depend on the risk of seasonal disturbances (e.g., frost or drought) occurring before reproductive maturation is reached (Cohen 1970, Taylor 1980, Hairston and Munns 1984, Taylor and Spalding 1989, Bradford and Roff 1993, Spencer and Colegrave 2001). We explored similar risks in a model where germination strategy depends on environmental state rather than time. We showed that conditional germination is more likely to evolve when plants are prone to population-scale disturbances, promoting life-histories that are more synchronized with these disturbances. As in our first model, conditional germination is more likely to evolve by this mechanism when seeds survive disturbances well but adults do not.

Our analytical results indicate that conditional germination should only evolve if severe (detrimental to adult survival) disturbances can occur before a significant number of offspring are produced (figure 2.3). In particular, organisms that take multiple years to reach reproductive maturity should have an increased risk of dying during disturbances before reproducing. A previous model with pre-reproductive age classes by Enright et al. (1998) suggested that conditional germination strategies have higher long-term growth rates even without differences in seedling survival but the mechanism favouring conditional germination was not discussed or made explicit. We produced a model based on that of Enright et al. (1998) but reduced the number of years until reproductive maturity to show that this eliminates the benefits of conditional germination (figure 2.4). To our knowledge, avoiding death before reaching reproductive maturity has not previously been theoretically investigated as an important driver for the evolution of conditional germination strategies, most likely because it requires a relatively complex demographic model with environment-dependent germination.

Interestingly, a synchronization advantage continues to favour conditional germination strategies even when the period between disturbances

is variable. In this case, incomplete rather than complete disturbance-dependent germination strategies often have the highest long-term growth rate because they bet hedge (Philippi and Seger 1989) between experiencing long and short intervals. This is an example of 'germ banking', as defined by Evans and Dennehy (2005), where unpredictable environmental variation favours an intermediate strategy. Figure 2.5 shows that, even when the disturbance probability is exactly the same in each year ($\beta = 1$, exponentially distributed disturbance intervals) and there is no difference in seedling survival, conditional germination is expected to evolve when plants take multiple years to reach reproductive maturity. Demonstrating this case explicitly is significant because many types of disturbance are likely to be non-periodic ($\beta = 1$). For fires, a Weibull shape parameter of around 2 (see figure 2.5A) has been estimated in some ecosystems (Polakow and Dunne 1999, Moritz et al. 2008). Fire hazard is thought to increase with years since a fire due to vegetation build up (Baeza et al. 2002, De Luís et al. 2004), causing a negative autocorrelation in fire intervals (Dodson et al. 2005) and making fires more uniformly spread over time (as in our periodic model). On the other hand, a positive temporal autocorrelation between disturbances (clumping, e.g., due to climate phenomena) would reduce the efficacy of the synchronization mechanism because disturbance risk is increased in the years following a disturbance.

In this paper, we determine the conditions under which these three mechanisms allow the evolution of environment-dependent germination. We first explored the most commonly envisioned mechanism (mechanism 1, low seedling survival in 'bad' environments) and then show that trade-offs and synchronization effects (mechanisms 2 and 3) can favour environment-dependent germination even when there is no difference in seedling survival. These models provide a framework for exploring which mechanisms might be responsible for conditional germination in empirical systems. For example, we have shown that the fact that it takes several years for the Australian shrub *Banksia hookeriana* to mature greatly facilitates the evolution of environment-dependent germination in this system (figure 2.4). Thus, by obtaining the required demographic parameters and using the models to determine what conditions favour conditional germination, future empirical work promises to inform us why some species wait for particular environments to continue development.

Chapter 3

Evolution of Haplont, Diplont or Haploid-Diploid Life Cycles When Haploid and Diploid Fitnesses Are Not Equal

3.1 Summary

Many organisms spend a significant portion of their life cycle as haploids and as diploids (a haploid-diploid life cycle). However, the evolutionary processes that could maintain this sort of life cycle are unclear. Most previous models of ploidy evolution have assumed that the fitness effects of new mutations are equal in haploids and homozygous diploids, however, this equivalency is not supported by empirical data. With different mutational effects, the overall (intrinsic) fitness of a haploid would not be equal to that of a diploid after a series of substitution events. Intrinsic fitness differences between haploids and diploids can also arise directly, e.g., because diploids tend to have larger cell sizes than haploids. Here, we include intrinsic fitness differences into genetic models for the evolution of time spent in the haploid versus diploid phases, in which ploidy affects whether new mutations are masked. Life cycle evolution can be predominantly determined by intrinsic fitness differences between phases, masking effects, or a combination of both. We find parameter ranges where these two selective forces act and show that the balance between them can favour convergence on a haploid-diploid life cycle, which is not observed in the absence of intrinsic fitness differences. Specifically, haploid-diploid life cycles can evolve when diploids have higher intrinsic fitness but the net effect of new mutations favours haploidy.

3.2 Introduction

Sexual reproduction in eukaryotes requires an alternation of haploid and diploid phases in the life cycle. Across taxa, there is a great deal of variation in the amount of growth (and time spent) in each of the haploid and diploid phases (see Valero et al. 1992, Klinger 1993, Richerd et al. 1993, Bell 1994; 1997, Mable and Otto 1998, Coelho et al. 2007). Some organisms, including almost all animals, are diplontic (somatic development occurs only in the diploid phase) and others, including dictyostelid slime moulds, and some green algae (e.g., *Chara*), are haplontic (somatic development occurs only in the haploid phase). However, a large and phylogenetically diverse group of eukaryotes, including most land plants, basidiomycete fungi, most brown algae, red algae and some green algae, undergo some mitotic growth in both the haploid and diploid phases, which is referred to as a haploid-diploid life cycle here (sometimes called diplohaplontic or haplodiplontic) to avoid confusion with arrhenotoky ('haplodiploid' sex determination). While several theoretical studies have explored the conditions that should favour expansion of the haploid or diploid phases, there are still relatively few studies that show how a haploid-diploid life cycle could be maintained by selection.

A prominent theory for the evolution of either haplont or diplont life cycles involves the direct consequences of ploidy level on the expression of deleterious mutations. The fitness effects of a deleterious mutation can be partially hidden by the homologous gene copy in diploids, which is favourable if a heterozygote has a higher fitness than the average fitness of the two component haploids. Thus modifier models, in which the extent of haploid and diploid phases is determined by a second locus, have found that diplonty is favoured when deleterious mutations are partially recessive and haplonty is favoured when deleterious mutations are partially dominant (Perrot et al. 1991, Otto and Goldstein 1992, Jenkins and Kirkpatrick 1994; 1995). As a consequence of mutations being partially concealed, an expanded diploid phase allows mutations to reach a higher frequency and thus increases mutation load (Crow and Kimura 1965, Kondrashov and Crow 1991). Modifiers that expand the diploid phase therefore become associated with lower quality genetic backgrounds. These associations are broken apart by recombination and so diplonty is favoured over a wider parameter range when recombination rates are higher (Otto and Goldstein 1992).

The evolution of life cycles in sexual organisms appears to be similarly influenced by beneficial mutations. Using a numerical simulation approach, Otto (1994) and Orr and Otto (1994) show that diplonty is favoured during

sweeps of beneficial mutations that are partially dominant. Increasing the length of the diploid phase of the life cycle increases the amount of selection experienced by heterozygotes and, with partial dominance, heterozygotes have higher fitness than the average fitness of the two component haploids. Conversely, haplonty is favoured when beneficial mutations are partially recessive. Again, lower recombination rates between the life cycle modifier and beneficial mutations broaden the parameter range over which haplonty is favoured because of associations between the modifiers expanding the haploid phase and higher quality genetic backgrounds that evolve when beneficial mutations are not masked.

These models typically assume that the overall fitness of haploids or diploids is the same. However, even with identical genomes, haploid and diploid cells typically differ in size and often in shape (e.g., Mable 2001), and growth and survival often differs between haploid and diploid phases. The phase with higher fitness and the magnitude of fitness differences varies widely and is heavily dependent on environmental context (Mable and Otto 1998, Thornber 2006). In *Saccharomyces* yeast, differences between haploid and diploid growth rates measured by Zörgö et al. (2013) range from being negligible to substantial (one phase can have growth rates up to 1.75 times higher) in different environments. Similar differences in growth rate and survival are observed between haploid and diploid phases of the red algae *Gracilaria verrucosa* and *Chondracanthus squarrulosus* in some laboratory conditions (Destombe et al. 1993, Pacheco-Ruíz et al. 2011). In addition, the fitness effect of new mutations may be unequal when present in haploids or in homozygous diploids, as reported by Gerstein (2012) and Zörgö et al. (2013). Therefore, following a series of substitution events, the overall (intrinsic) fitness of a haploid and a diploid should not be equal, as explored here.

The models discussed above assume that selection is independent of the densities of haploid and diploid individuals. These models also predict that either haplonty or diplonty evolves but not biphasic, haploid-diploid life cycles. Hughes and Otto (1999) and Rescan et al. (2016) consider density-dependent selection in which haploids and diploids occupy different ecological niches and show that haploid-diploid life cycles can evolve in order to exploit both the haploid and diploid ecological niches. In this study, we complement these studies by considering only density independent selection in order to focus on intrinsic fitness differences between haploids and diploids.

The effect of intrinsic fitness differences on the evolution of the life cycle may seem obvious - selection should favour expansion of whichever phase (haploid or diploid) has higher fitness, as found by Jenkins and Kirkpatrick (1994; 1995). However, Jenkins and Kirkpatrick (1994; 1995) only consid-

ered the case where the differences in intrinsic fitness is either much larger or much smaller than the genome-wide deleterious mutation rate. Here, we consider the case where the two forces are of similar strength and quantify the parameters (e.g., mutation rate) for which this is true. In addition, we consider the effect of beneficial mutations on life cycle evolution when there are intrinsic fitness differences between haploids and diploids. We show that haploid-diploid life cycle can evolve even in the absence of density dependent selection due to a balance between intrinsic fitness differences between phases and the genetic effects of masking/revealing mutations. We also consider branching conditions and find that, in haploid-diploid populations, sexually interbreeding mixtures of haploid and diploid specialists can be favoured (see also Rescan et al. 2016).

3.3 Model

We consider life cycle evolution using a modifier model in which the proportion of time spent in the haploid and diploid phases depends on the genotype at a modifier locus. Selection on the modifier results from viability selection on a set of L other loci. We first present a two-locus model, in which there is one viability locus and one modifier locus. We then extrapolate our results to the evolution of a modifier locus linked to many loci under selection; selection on a modifier caused by many loci is well approximated by the sum of the selective effect of each pairwise interaction considered separately (e.g., Jenkins and Kirkpatrick 1995, Otto and Bourguet 1999, Hough et al. 2013), assuming that the viability loci are loosely linked, autosomal and nonepistatic and the modifier has a small effect. We then test this approach by comparing our results to an explicit multi-locus simulation. Finally, we show that beneficial mutations can generate selection on the life cycle similar to that caused by deleterious mutations.

3.3.1 Analytical Model

In the modifier model presented here (figure 3.1b), zygotes are formed during synchronous random mating. The diploid genotype (ij) at the modifier locus (MM, Mm, or mm) determines the timing of meiosis and hence the proportion of time each individual spends as a diploid ($1 - t_{ij}$) and as a haploid (t_{ij}). Here, S_h and S_d represent selection acting across the genome due to intrinsic fitness differences between haploids and diploids. As our initial focus will be on the selection experienced at each of L selected loci, we also define $\sigma_h = S_h/L$ and $\sigma_d = S_d/L$ as the intrinsic fitnesses per

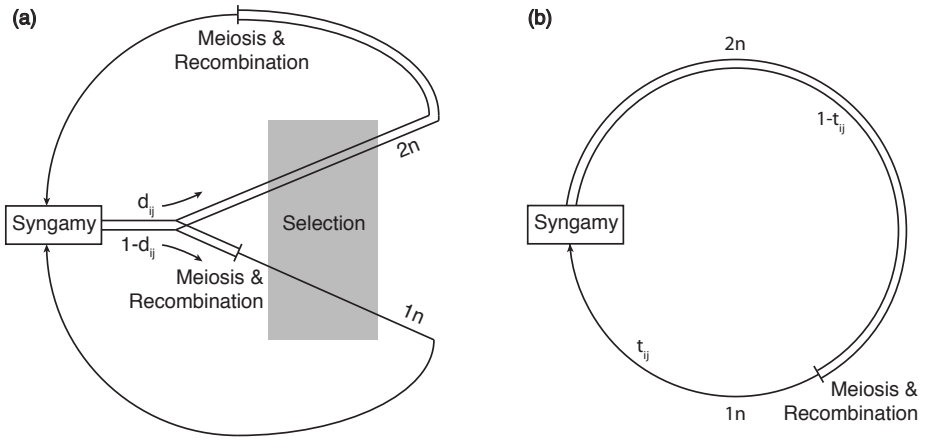

Figure 3.1: Model (a) discrete selection and (b) continuous selection haploid-diploid life cycles. Single lines represent haploid phases and doubled lines indicate diploid phases. In (a), modified from Perrot et al. (1991) and Otto and Goldstein (1992), zygotes with the modifier genotype ij undergo selection as diploids with probability d_{ij} or undergo meiosis and recombination before experiencing selection as haploids with probability $(1-d_{ij})$. In (b), after Jenkins and Kirkpatrick (1994; 1995) and Otto (1994), all zygotes with genotype ij experience viability selection as a diploid for a proportion $(1-t_{ij})$ of their life cycle before undergoing meiosis and recombination and then experiencing viability selection as a haploid for the remainder of the life cycle.

viability locus. When $\sigma_h > \sigma_d$, haploids have higher fitness than diploids and the fitness of diploids is higher when $\sigma_d > \sigma_h$. At each viability locus, we consider a wild type and mutant allele (alleles A and a). The mutant allele at each viability locus, a, can have a different effect on fitness when present in a haploid (s_h) or in a homozygous diploid (s_d). The fitness of heterozygous diploids depends on the dominance of these mutations, given by h. When considering deleterious mutations, s_h and s_d are both negative, and when considering beneficial mutations, s_h and s_d are both positive. The fitnesses of the various genotypes are given in table C.1. Recombination between the modifier and viability locus (at rate r) and mutation (from A to a, at rate μ per viability locus) occur at meiosis followed by haploid selection and then gamete production. The frequencies of genotypes MA, Ma, mA and ma are censused in the gametes (given by x_1, x_2, x_3 and x_4 respectively).

Previous models have made various different life cycle assumptions, summarized in table 3.2. In 'discrete selection' models, selection occurs once per generation and modifiers affect whether selection occurs during the haploid or diploid phase, figure 3.1a. On the other hand, 'continuous selection'

Table 3.1: Fitnesses of different genotypes.

Genotype	Fitness
A	$w_A(t_{ij}) = \exp[t_{ij}\sigma_h]$
a	$w_a(t_{ij}) = \exp[t_{ij}(\sigma_h + s_h)]$
AA	$w_{AA}(t_{ij}) = \exp[(1 - t_{ij})(\sigma_d)]$
Aa	$w_{Aa}(t_{ij}) = \exp[(1 - t_{ij})(\sigma_d + hs_d)]$
aa	$w_{aa}(t_{ij}) = \exp[(1 - t_{ij})(\sigma_d + s_d)]$

models assume that selection occurs continuously throughout the life cycle, figure 3.1b. In addition, some models have assumed that mutations occur upon gamete production, and others assume that mutations occur at meiosis. Thus, there are four possible life cycles, recursion equations for these different life cycles are provided in the appendix B. Generally, our results are unaffected by using these alternative models, these analyses can be found in the supplementary *Mathematica* file (Wolfram Research Inc. 2010). However, there are two cases in which life cycle assumptions qualitatively impact results.

Table 3.2: Life cycle assumptions used in various modifier models.

	Mutations at Gamete Production	Mutations at Meiosis
Discrete Selection (Figure 3.1a)	Perrot et al. (1991) Otto and Goldstein (1992) Otto and Marks (1996) Rescan et al. (2016)	Hall (2000)
Continuous Selection (Figure 3.1b)	Otto (1994)[a]	Orr and Otto (1994) Otto (1994)[a] Jenkins and Kirkpatrick (1994; 1995)

[a] Otto (1994) allows mutations to occur at both gamete production and meiosis.

Firstly, Hall (2000) showed that 'polymorphic' haploid-diploid life cycles can evolve if mutations occur at meiosis and selection is discrete. This life cycle allows diploids to escape selection on new mutations for one generation, generating an advantage to diploids, which allows convergence to occur when deleterious mutations favour haploids. As shown below, meiotic mutation does not favour haploid-diploid life cycles in the continuous selection model

(figure 3.1b) because new mutations experience selection the instant they appear in diploids.

Secondly, alternative mating schemes have previously only been considered by Otto and Marks (1996), who assume discrete selection and mutations at gamete production (and no differences in intrinsic fitness between haploids and diploids). They found that haploidy is favoured over a larger parameter range when selfing, asexual reproduction or assortative mating is common. In the appendix, we include selfing into all four life cycle models and show that this conclusion only applies when the fitness of haploids and homozygous diploids are assumed to be equal (e.g., no intrinsic fitness differences), otherwise additional effects of selfing are observed because selfing generates homozygotes. Furthermore, even when there are no intrinsic fitness differences, we show that selfing can increase or decrease the parameter range in which haploids are favoured when mutations occur at gamete production. This effect is presumably due to benefits that selfed diploids can accrue following a period of haploid selection on new mutations and illustrates again that the impact of increased selfing on these models is not equivalent to reduced recombination.

3.3.2 Multilocus Simulations

We used individual-based simulations (C++ program available in the Dryad Digital Repository) to test predictions from our analytical model when deleterious mutations segregate at L loci. Each individual carries either one or two copies of a chromosome (depending on its ploidy level) represented by a modifier locus (located at the midpoint of the chromosome) and a sequence of L bits (0 or 1) corresponding to the different loci.

Mutations occur at a rate U per generation: the number of new mutations per chromosome is sampled from a Poisson distribution with parameter U and distributed randomly across the genome; alleles at mutant loci are switched from 0 to 1 or from 1 to 0. Mutation and back mutation thus occur at the same rate, but back mutations should generally have negligible effects under the parameter values that we use, as deleterious alleles remain at low frequencies. We assume that all deleterious alleles have the same effects on fitness (s_d, s_h, and h are constant) and that these effects multiply across loci: the fitness of a haploid carrying n deleterious alleles is given by $w_h = \exp[S_h + s_h n]$, while the fitness of a diploid carrying n_{he} deleterious alleles in the heterozygous state, and n_{ho} in the homozygous state is given by $w_d = \exp[S_d + n_{he} h s_d + n_{ho} s_d]$.

At the start of each generation, all N individuals are diploid. To produce

the 2N gametes that will form the diploids of the next generation, a diploid individual is sampled randomly among all diploids of the previous generation, and undergoes meiosis to produce a haploid; the number of cross-overs is sampled from a Poisson distribution with parameter R, while the position of each cross-over is sampled from a uniform distribution. If a random number sampled from a uniform distribution between 0 and 1 is lower than ${w_d}^{1-t}{w_h}^t$ (where w_d and w_h are the fitnesses of the diploid parent and haploid offspring), divided by its maximal possible value, then the haploid is retained; otherwise another diploid parent is sampled, until the condition is fulfilled.

At the beginning of the simulation, the modifier locus is fixed for an allele coding for an initial length of the haploid phase t_{init} (all simulations were performed for t_{init} values of 0.1, 0.5 and 0.9) and all selected loci are fixed for allele 0. Then, deleterious mutations are introduced at rate U per chromosome (the length of the haploid phase being still fixed to t_{init}) until the population reaches mutation-selection equilibrium (after generally 2,000 generations). After that, mutations at the modifier locus are introduced at a rate m_M per generation. When a mutation occurs, the length of the haploid phase coded by the mutant allele is sampled from a uniform distribution between $t_{old} - 0.1$ and $t_{old} + 0.1$, where t_{old} is the value of the parent allele; if the new value is negative or higher than 1, it is set to 0 or 1, respectively. We assume additivity among modifier alleles such that a zygote with alleles t_1 and t_2 will have a haploid phase of length $t = (t_1 + t_2)/2$. Simulations initially lasted 100,000 generations, which was sufficient in most cases for the average rate of diploidy to reach steady state, $\bar{t}$. We categorized the life cycle that evolved at the end of the simulation as haplont ($\bar{t} > 0.95$, white circles in figures 2 and 3b), diplont ($\bar{t} < 0.05$, black circles), or haploid-diploid ($0.05 < \bar{t} < 0.95$, green circles). In some cases, there was a repelling state such that the population evolved to haplonty or diplonty depending on t_{init} (red circles).

3.4 Results

3.4.1 Deleterious Mutations

We first find the frequency of deleterious mutations at mutation-selection balance ($\hat{q}_a$) when the modifier locus is fixed for a particular resident allele (MM fixed, so that the length of the haploid phase is t_{MM}). Assuming that the per locus mutation rate (μ) is small, terms of the order of the square of

the per locus mutation rate can be ignored, yielding

$$\hat{q}_a = \frac{\mu \exp[t_{MM} s_h]}{1 - \exp[t_{MM} s_h + (1 - t_{MM}) h s_d]}, \tag{3.1}$$

assuming there is some haploid or diploidy heterozygous expression so the denominator isn't near zero. When deleterious mutations are partially masked by the homologous gene copy in diploids ($hs_d/s_h < 1$), the frequency of deleterious mutations ($\hat{q}_a$) is higher when the diploid phase is longer (lower t_{MM}).

Life cycle evolution is considered by introducing an allele (m) at the modifier locus that controls the timing of meiosis and evaluating whether its frequency increases when rare. Mutants are able to invade when the leading eigenvalue of the system described by equations B.1c and B.1d, λ_l, is greater than one. Jenkins and Kirkpatrick (1994) derive a version of λ_l when $s_d = s_h$, however, they only discuss per locus intrinsic fitness differences that are of a much greater magnitude than the mutation load ($|\sigma_d - \sigma_h| \gg \mu$). To investigate the interaction between these selective forces we first present an approximation of λ_l in which the per locus fitness difference between haploids and diploids ($|\sigma_d - \sigma_h|$) is of similar magnitude to the per locus mutation rate, $O(\epsilon^2)$, the selective disadvantage of mutants (s_d and s_h) is of a larger order of magnitude, $O(\epsilon)$, and linkage is loose (r of $O(1)$) yielding

$$\lambda_l \approx 1 + (t_{Mm} - t_{MM}) \left(\sigma_h - \sigma_d + 2(-s_h)\hat{q}_a \left(\frac{h s_d}{s_h} - \frac{1}{2} \right) \right) + O(\epsilon^3). \tag{3.2}$$

Because mutation rates are small, deleterious mutations are found at low frequencies, therefore life cycle evolution depends only on the fitness of heterozygous mutants and not homozygous mutants (i.e., s_d is always found with the dominance coefficient, h). Consequently, life cycle evolution depends only on the 'effective dominance', $h_e = hs_d/s_h$, rather than dominance *per se*.

Life cycle modifiers affect the amount of selection heterozygous zygotes will subsequently experience as heterozygous diploids versus as the component haploid genotypes. Heterozygous diploids have higher fitness than the average of the two component haploids when deleterious mutations are effectively partially recessive ($0 < hs_d/s_h < 1/2$), favouring diploidy. Conversely, effectively partially dominant deleterious alleles ($hs_d/s_h > 1/2$) favour haploidy. The strength of this selection on the life cycle (caused by masking alleles) depends on the equilibrium frequency of deleterious alleles, which is greater when the diploid phase is longer (assuming $0 < hs_d/s_h < 1$).

Using this approximation, haploid-diploid life cycles are evolutionarily singular strategies when $\sigma_h - \sigma_d = 2(s_h)\hat{q}_a(h_e - 1/2)$. Without intrinsic fitness differences, there is no intermediate value of t_{MM} that solves this condition, hence either haplont or diplont life cycles are favoured. Thus, whereas Hall (2000) shows that biphasic haploid-diploid life cycles can evolve if selection occurs once per generation (figure 3.1a) and mutations occur at meiosis (as considered here), haploid-diploid life cycles in the continuous selection model (figure 3.1b) do not evolve in the absence of intrinsic fitness differences.

When diploids have higher intrinsic fitness ($\sigma_d > \sigma_h$), there are intermediate (biphasic haploid-diploid) singular strategies in the region where deleterious alleles favour haploidy. In this case, the strength of selection in favour of haploidy is strong when the diploid phase is longer (because deleterious mutations reach higher frequencies) and can outweigh the intrinsic fitness differences. When the diploid phase is short, intrinsic fitness differences dominate, favouring a longer diploid phase. This combination ensures that evolution converges towards a haploid-diploid life cycle (figure 3.2a).

When haploids have higher intrinsic fitness ($\sigma_h > \sigma_d$), either haplonty or diplonty is always favoured. Even if an intermediate singular strategies exists because deleterious alleles favour diploidy, this is a repelling point, such that either haplonty or diplonty evolves. For these parameters, selection in favour of diplonty is stronger when the diploid phase is longer, favouring even longer diploid phases (because the benefits of masking deleterious mutations is greater). Conversely, intrinsic fitness differences dominate when the diploid phase is short, favouring longer haploid phases. Thus haplonty and diplonty can both be stable strategies (figure 3.2c).

After convergence on a haploid-diploid strategy, we can then ask whether this singular strategy is evolutionarily stable. Using the same weak selection approximations as above, evolutionary stability is given by:

$$\frac{\delta^2 \lambda_l}{\delta {t_{Mm}}^2}\Big|_{t_{Mm}=t^*} = \frac{2(-s_h)(\sigma_d - \sigma_h)(hs_d/s_h - 1)(1-r)w_a[t^*]w_{Aa}[t^*]}{w_A[t^*]w_{AA}[t^*] - (1-r)w_a[t^*]w_{Aa}[t^*]}, \quad (3.3)$$

where t^* indicates the singular strategy for t, the length of the haploid phase. When convergence is stable (requiring that $\sigma_d > \sigma_h$ and $hs_d/s_h < 1$, see below), the singular strategy is evolutionarily unstable (3.3 is positive). Thus we expect weak disruptive selection after this singular point is reached. Indeed, our multilocus simulations sometimes displayed branching after 100,000 generations, such that there was a proportion t^* of haploid

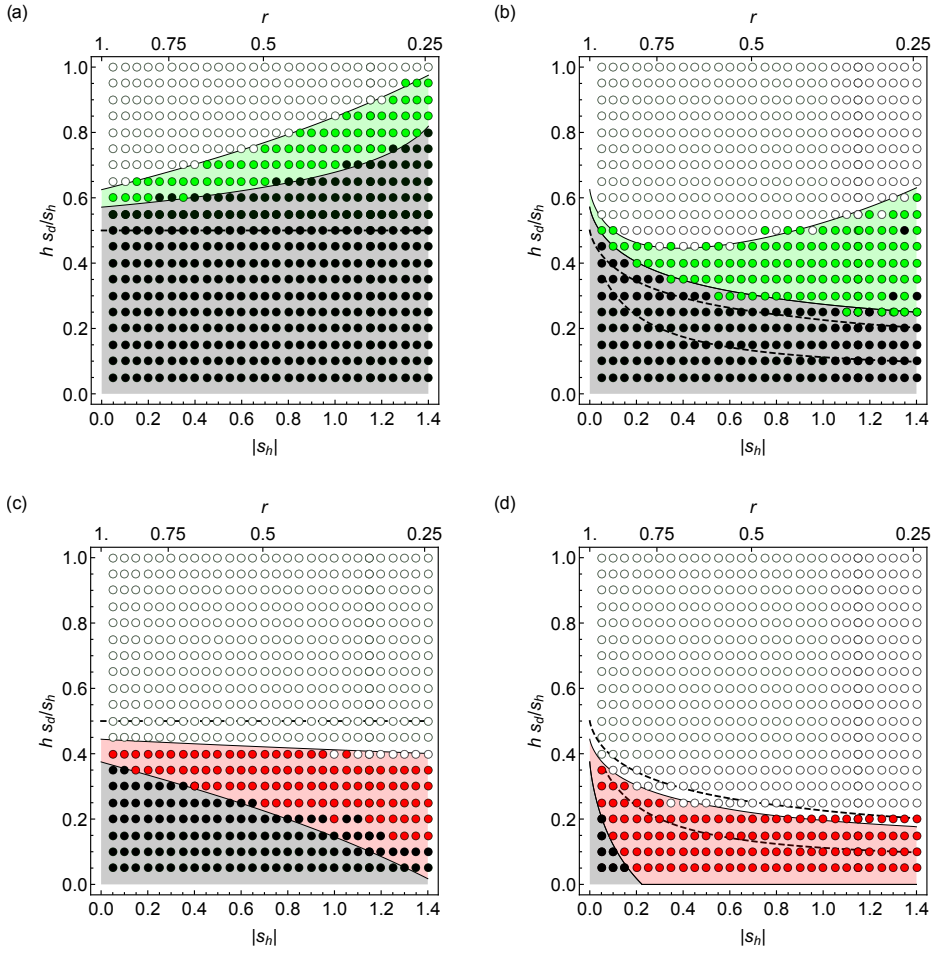

Figure 3.2: Parameter space where haplont, diplont and haploid-diploid life cycles are favoured where the strength of selection against deleterious mutations ($|s_h|$) and effective dominance hs_d/s_h is varied. The top axis gives r, the relative growth rate of mutant haploids when deleterious mutations have a fitness effect of s_h. Background colors: prediction from the two-locus stability analysis extrapolated to multiple loci. Circles: multilocus simulation results starting from three different initial haploidy rates (t_{init} = 0.01, 0.5, or 0.99), with population size 20,000. White: evolution toward haplonty. Green: convergence stable haploid-diploid life cycles. Red: either haplonty or diplonty is favoured, with a repelling state in between. Black and gray: evolution toward diplonty. (a) and (b): diploids have higher intrinsic fitness ($S_h = 0$, $S_d = 0.025$) (c) and (d): haploids have higher intrinsic fitness ($S_h = 0.025$, $S_d = 0$). Map length: $R = 100$ ((a) and (c)) and $R = 0.35$ ((b) and (d)). The dashed lines show where haplonty (above dashed lines) and diplonty (below dashed lines) are favoured when there is no difference in intrinsic fitness ($S_h = S_d = 0$). In (b) and (d), there is a repelling point between the dashed lines. Mutants change the life cycle by a small amount ($|t_{Mm} - t_{MM}| = 0.001$) and the genome-wide haploid mutation rate, $U = 0.1$.

alleles ($t_1 = 1$), and a proportion $(1 - t^*)$ of diploid alleles ($t_2 = 0$). Increasing the number of generations always lead to branching when it was not observed by this time.

The weak selection approximation above assumes that the recombination rate is large relative to selection. Without intrinsic fitness differences, Otto and Goldstein (1992) showed that haploidy is favoured over a larger range of parameter spaces when recombination rates are low because associations between haploid-promoting modifiers and the high fitness, purged genetic backgrounds they create are retained for longer. To consider tighter linkage and/or stronger selection we can use the more accurate expression of λ_l

$$\lambda_l = \exp[(t_{Mm} - t_{MM})(\sigma_h - \sigma_d)]\left(1 + \frac{\mu K_1}{K_2 K_3}\right), \tag{3.4}$$

where

$$\begin{aligned}
K_1 = 1 &- (1-r)\exp[-(t_{Mm} - t_{MM})hs_d] \\
&- r\exp[(t_{Mm} - t_{MM})(s_h - hs_d)] \\
&+ (1-2r)\{\exp[(1 - t_{Mm} - (t_{Mm} - t_{MM}))hs_d + t_{Mm}s_h] \\
&\qquad - \exp[(1 - t_{Mm})hs_d + t_{Mm}s_h]\} \\
K_2 = 1 &- \exp[-(1 - t_{MM})hs_d - t_{MM}s_h] \\
K_3 = 1 &- (1-r)\exp[(1 - t_{Mm})hs_d + t_{Mm}s_h],
\end{aligned}$$

in which the per locus mutation rate (μ) is assumed to be small, so that terms on the order of the square of the mutation rate can be ignored.

Equation (3.4) shows that singular strategies can exist without intrinsic fitness differences when recombination rates are low, $r < 1/2$, see figures 3.2b and 3.2d). As above, these singular strategies are always repelling points when $\sigma_d = \sigma_h$ such that differences in intrinsic fitness are required for haploid-diploid life cycles to evolve. Convergence upon a haploid-diploid life cycle still requires that diploids have higher intrinsic fitness ($\sigma_d > \sigma_h$). However, as selection becomes less weak relative to recombination rates (such that the approximation in 3.2 is not appropriate), haploid-diploid life cycles can evolve when $hs_d/s_h < 1/2$, see figure 3.2b. In addition, convergence stability requires $hs_d/s_h < 1$, such that the frequency of deleterious mutations ($\hat{q}_a$) increases with the length of the diploid phase, see figure 3.3a.

We next extend our two-locus result to consider deleterious mutations across L viability loci by assuming that these loci are loosely linked, autosomal and nonepistatic. With these assumptions (e.g., Jenkins and Kirkpatrick 1995, Otto and Bourguet 1999, Hough et al. 2013, Rescan et al.

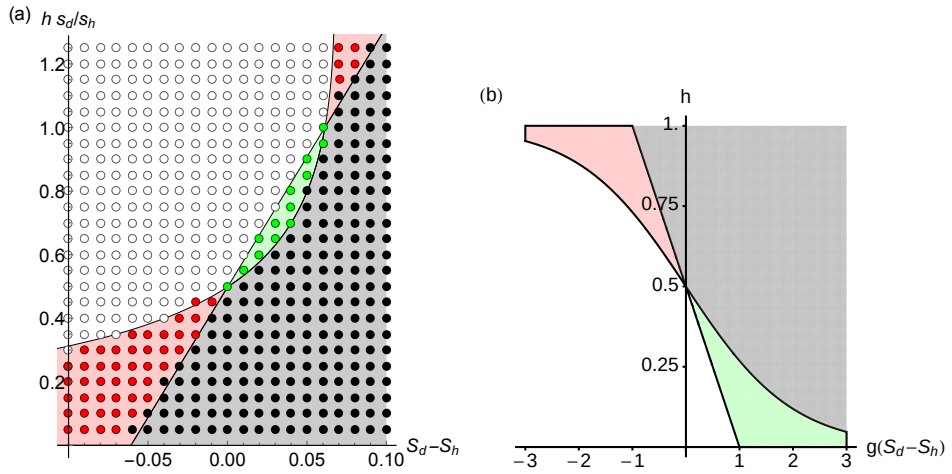

Figure 3.3: Parameter space for which (a) deleterious mutations and (b) beneficial mutations favour haplont, diplont and haploid-diploid life cycles as a function of the difference in intrinsic fitness between haploids and diploids ($S_d - S_h$). (a) Shows the effective dominance of deleterious mutations (hs_d/s_h) against intrinsic fitness differences ($S_d - S_h$), parameters and symbols as in figures 3.2a and 3.2c with $|s_h| = 0.4$. (b) Regions in which particular life cycles are favoured in the presence of beneficial mutations, evaluated using equation 3.11. g is the number of generations between fixation events. Population size, N, is 20000.

2016), invasion of a modifier of weak effect is given by

$$\lambda_{net} = 1 + \sum_{l=1}^{L} (\lambda_l - 1). \tag{3.5}$$

In figures 3.2 and 3.3a we plot where this approximation predicts haplont, diplont or haploid-diploid life cycles to evolve for comparison to the explicit multi-locus simulation (described above).

Above, as in previous work, we consider the average dominance and selection coefficients (h, s_d and s_h). We can approximate the effect of small amounts of variation (and covariation) among loci in these coefficients by performing a Taylor expansion, as described in Lynch and Walsh (1998), Appendix 1. Because we have assumed that deleterious mutations are rare, s_d is always found with h and we consider variation in s_h and the compound parameter hs_d. Assuming that deviations between coefficients and their mean value are of order ϵ and that selection is weak (as assumed in equation

3.2), yields

$$\begin{aligned}\lambda_{net} \approx & 1 + (t_{Mm} - t_{MM})\Bigg(\sigma_h - \sigma_d + 2(-s_h)L\hat{q}_a\left(\frac{hs_d}{s_h} - \frac{1}{2}\right) \\ & + \frac{(1+t_{MM})L\hat{q}_a(-s_h)}{\mu^2}\Bigg((1-t_{MM})\left(\frac{hs_d}{s_h}\operatorname{Cov}(hs_d, s_h) - \operatorname{Var}(hs_d)\right) \\ & + t_{MM}\left(\frac{hs_d}{s_h}\operatorname{Var}(s_h) - \operatorname{Cov}(hs_d, s_h)\right)\Bigg)\Bigg) + O(\epsilon^3)\end{aligned} \tag{3.6}$$

Based on this analysis, variation in s_h generally makes haploniy more stable to invasion (reduces λ_{net} for $t_{MM} = 1$, $t_{Mm} < 1$). Similarly, variation in hs_d makes diploniy more stable to invasion (where $t_{MM} = 0$, $t_{Mm} > 0$). Positive covariation between hs_d and s_h has the opposite effect. Yeast deletion data indicate that the heterozygous effects of deleterious mutations may be much less variable than their homozygous effects, due to a negative correlation between h and s (Phadnis 2005, Agrawal and Whitlock 2011, Manna et al. 2011). Even if s_d and s_h are on average the same, it may thus be that the variance of hs_d is much lower than the variance of s_h.

3.4.2 Beneficial Mutations

Whereas deleterious alleles are maintained at mutation-selection balance, beneficial mutations sweep to fixation. The time taken for a sweep to occur depends on the length of the diploid phase; selective sweeps take longer in predominantly diploid populations. During a selective sweep, heterozygotes are present in the population. Life cycle modifiers can affect whether heterozygous zygotes subsequently experience selection as heterozygous diploids or as haploids. Thus, the strength of selection exerted by beneficial mutations on modifiers depends on the time taken for fixation to occur, which depends on the life cycle of the current population. Therefore, as with deleterious alleles, the direction of selection exerted by beneficial mutations depends on dominance. Here we evaluate how these genetic considerations are expected to influence life cycle evolution and include differences in intrinsic fitness between haploids and diploids.

We obtain analytical results using a quasi-linkage equilibrium (QLE) approximation, in which selection is assumed to be weak relative to recombination so that linkage disequilibrium ($D = x_1x_4 - x_2x_3$) equilibrates quickly relative to the rate of change of allele frequencies ($p_A = x_1 + x_3$ and

$p_M = x_1 + x_2$). Assuming weak selection, $O(\epsilon)$, and low mutation rates, $O(\epsilon^2)$, the leading order term for the quasi-equilibrium value of linkage disequilibrium ($\hat{D}_Q$) is given by

$$\hat{D}_Q \approx \delta_t \frac{s_h}{r} p_M(1-p_M)p_A(1-p_A)\left(1 - p_A\frac{hs_d}{s_h} - (1-p_A)(1-h)\frac{s_d}{s_h}\right) + O(\epsilon^2), \tag{3.7}$$

where $\delta_t = (p_M(t_{Mm} - t_{MM}) + (1-p_M)(t_{mm} - t_{Mm}))$ is the effect of the modifier on the length of the haploid phase (δ_t is positive if m increases the haploid phase with $t_{mm} > t_{Mm} > t_{MM}$ and negative if $t_{mm} < t_{Mm} < t_{MM}$).

Linkage disequilibrium is a measure of associations between alleles at different loci. When $D > 0$, alleles A and M are more often found together, as are alleles a and m. When $s_h = s_d$ and $0 < h < 1$, as assumed in Otto (1994) and Orr and Otto (1994), equation (3.7) shows that m alleles that increase the length of the haploid phase ($\delta_t > 0$) are associated with the beneficial mutation, a ($\hat{D}_Q > 0$). These associations are broken apart by recombination so associations are stronger ($|\hat{D}_Q|$ larger) when the recombination rate is low. Therefore lower recombination rates should favour haplonty, as found numerically by Otto (1994) and Orr and Otto (1994).

The change in the frequency of the modifier allele, m (Δq_m) can then be expressed as a function of linkage disequilibrium ($\hat{D}_Q$) and allele frequencies, p_A and p_M. Assuming that selection is weak and mutation rates are low, the leading order term of Δq_m is given by

$$\Delta q_m \approx \delta_t p_M(1-p_M)\left(\sigma_h - \sigma_d + s_h(1-p_A)\left(1 - 2p_A\frac{hs_d}{s_h} - (1-p_A)\frac{s_d}{s_h}\right)\right) + O(\epsilon^2). \tag{3.8}$$

Unlike deleterious mutations, beneficial mutations reach high frequencies in the population, so the dynamics of the modifier depend on the fitness of both heterozygous and homozygous mutants. Equation (3.8) shows that, when fixed ($p_A = 0$), a beneficial mutation with a different effect size in haploids and diploids ($s_d \neq s_h$) affects life cycle evolution in a similar manner to intrinsic fitness differences (σ_d and σ_h). However, there is also transient selection on the life cycle that occurs during the fixation of a beneficial mutation. We isolate the transient selection on the life cycle from the effect on intrinsic fitnesses by considering the case where $s_d = s_h = s$ so that

$$\Delta q_m \approx \delta_t p_M(1-p_M)(\sigma_h - \sigma_d + 2p_A(1-p_A)(1/2-h)s) + O(\epsilon^2). \tag{3.9}$$

Equation (3.9) demonstrates that, in the absence of intrinsic fitness differences ($\sigma_d = \sigma_h$), haplonty is favoured during sweeps of partially recessive ($h < 1/2$) beneficial mutations and diplonty is favoured during sweeps of partially dominant ($h > 1/2$) beneficial mutations (as found numerically by Orr and Otto 1994).

Whether life cycle evolution is dominated by differences in intrinsic fitness or transient selection generated by beneficial mutations depends on the rate at which beneficial mutations occur and how long they segregate in the population. The fixation time of beneficial mutations is different for different life cycles (longer when diploid phases are longer). We assume that the mutant life cycle allele is rare or similar enough to that of the resident that the time taken to fix a beneficial mutation depends on the life cycle of the resident and then measure the transient selection on the modifier over the entire time course of the sweep using

$$\int p_M(1-p_M)2p_A(1-p_A)p_A(1/2-h)s\,\mathrm{d}t. \tag{3.10}$$

This integral can then be evaluated assuming that a beneficial mutation will initially be found at frequency $1/N$, where N is the population size.

Assuming that the rate of adaptation is limited by the rate of environmental change so that a beneficial mutation fixes every g generations and considering selection on the life cycle from all L loci, the average invasion fitness of a rare life cycle modifier per generation is

$$\begin{aligned}\Delta\bar{q}_m \approx & \delta_t p_M(1-p_M)\Bigg((S_h - S_d) \\ & - \frac{1}{g}\ln\left[\frac{1}{N} + \frac{(N-1)(h(1-t_{MM})+t_{MM})}{N(1-h(1-t_{MM}))}\right]/(1-t_{MM})\Bigg),\end{aligned} \tag{3.11}$$

where the last term accounts for the fact that the beneficial mutations occur only once every g generations.

As with deleterious mutations, there can be haploid-diploid life cycles ($0 < t_{MM} < 1$) that are evolutionarily singular strategies. Assuming that the population size is large, mutants that increase the length of the haploid phase ($\delta_t > 0$) can only invade a resident population that has a short haploid phase ($t_{MM} = 0$) if beneficial mutations are partially recessive ($0 < h < 1/2$). Similarly, mutants that decrease the length of the haploid phase ($\delta_t < 0$) can only invade a resident population that has a long haploid phase ($t_{MM} \approx 1$) if beneficial mutations are partially recessive ($0 < h < 1/2$).

Therefore, a haploid-diploid life cycle can only be convergence stable when $0 < h < 1/2$ (green in figure 3.3b). Figure 3.3b also shows the region in which both haplonty and diplonty cannot be invaded by small life cycle modifiers, in which case the singular strategy represents a repelling point (red).

When the rate of adaptation is not limited by the rate of environmental change, but by the rate of fixation of beneficial mutations, the time between fixation events depends on the occurrence of beneficial mutations ($1/g$) and their fixation probability (P_{fix}), which is given by $2s(t_{MM} + (1 - t_{MM})h)$. Fixation probability decreases when the diploid phase is longer because beneficial mutations are partially hidden by the extra chromosomal copy in diploids. Under mutation-limited adaptation g can be replaced in equation (3.11) by g/P_{fix}. In this case, haploid-diploid life cycles are never maintained by selection. Thus, beneficial mutations can only favour haploid-diploid life cycles if the rate of adaptation is not mutation-limited.

3.5 Discussion

Empirical evidence suggests that the fitness effects of new mutations are not generally the same in haploids and diploids (Gerstein 2012, Zörgö et al. 2013). We show that, when the average fitness effect of new deleterious mutations is unequal in haploids and diploids, whether deleterious mutations favour haploidy or diploidy depends on their effective dominance (hs_d/s_h). Most mutation accumulation studies in *Saccharomyces* yeast estimate either the average heterozygous (hs_d) or haploid (s_h) effect of mutations on fitness (Wloch et al. 2001, Zeyl and DeVisser 2001, Joseph 2004, Hall et al. 2008), from which effective dominance could be estimated. However, because the expectation of a ratio is not generally equal to the ratio of expectations, estimates of effective dominance would be more accurate if calculated from the same strains. In such a study, Korona (1999) took relevant haploid and diploid fitness measures but did not estimate effective dominance. In addition, Szafraniec et al. (2003) found deleterious mutations affected haploid fitness more strongly than diploid fitness but they caution that the haploid spores were required to germinate, which may have biased their fitness measurements in favour of diploids. Thus, further empirical estimates of the effective dominance of deleterious mutations would better inform our understanding of how life cycles are impacted by deleterious mutations.

Haploid and diploid phases can also differ in their intrinsic fitnesses (Thornber 2006, Zörgö et al. 2013). While life cycle evolution depends on

the effective dominance when there are no intrinsic fitness differences, large differences in intrinsic fitnesses favour expansion of the phase with higher fitness (Jenkins and Kirkpatrick 1994). In this study, we show how life cycles are expected to evolve when life cycles experience selection due to both dominance and intrinsic fitness differences. To leading order, these selective forces both contribute when intrinsic fitness differences are similar in magnitude to the haploid genome-wide mutation rate. For example, figure 3.3A shows how life cycles are expected to evolve when the deleterious mutation rate per haploid genome (U) is 0.1, approximately equal to estimates of the deleterious mutation rate in *Amsinckia* and *Arabidopsis* plants (Schoen 2005, Halligan and Keightley 2009). Figure 3.3A suggests that these forces are of similar strength when the intrinsic fitness difference between haploids and diploids ($S_d - S_h$) is between 2% and 5%. Estimates of the deleterious mutation rate per haploid genome vary across studies and organisms (Halligan and Keightley 2009). For deleterious mutation rates that are a factor f larger, the scale of the x-axis on this figure can be multiplied by f to determine when selection on the life cycle due to deleterious mutations should be approximately the same strength as selection due to differences in intrinsic fitness. We note that mutation rate estimates in yeast and *Chlamydomonas* (Morgan et al. 2014) are lower but are typically calculated per mitotic cell division. However, the relevant mutation rate for models of life cycle evolution is per sexual cycle (i.e., per meiosis), which has been estimated to involve tens of thousands of mitotic generations in natural *S. cerevisiae* populations (Magwene et al. 2011).

In laboratory environments, substantial differences in fitness between haploid and diploids phases of *Saccharomyces* yeast and algae have been observed in some environments (Mable and Otto 1998, Destombe et al. 1993, Pacheco-Ruíz et al. 2011, Zörgö et al. 2013). However, measuring the fitness of yeast in natural environments is challenging. Some demographic studies of natural red algae populations of *Mazzaella flaccida* and *Chondrus crispus* have shown that diploids have moderately increased survivorship relative to haploids ($S_d - S_h \approx 0.1$, Bhattacharya 1985, Thornber and Gaines 2004). Other studies have found no difference in survivorship, perhaps because there is limited power to detect smaller differences in mortality in the field (e.g., Engel et al. 2001, Thornber and Gaines 2004). Overall, estimates of the magnitude of intrinsic fitness differences are still uncertain, partly because existing algal studies do not compare survivorship of isogenic haploids and diploids, which would be required to remove the effect of masked mutations in heterozygotes.

For haploid-diploid life cycles to evolve by selection, individuals with

longer diploid phases must be favoured in predominantly haploid populations and individuals with longer haploid phases must be favoured in predominantly diploid populations. Previous models predicting the evolution of biphasic haploid-diploid life cycles have posited indirect benefits from decreasing senescence by reducing phase-specific generation time (Jenkins 1993), reducing the frequency of sexual reproduction (Richerd et al. 1993), or exploiting more ecological niches (Bell 1997, Hughes and Otto 1999, Rescan et al. 2016). However, haploid-diploid life cycles are not a unique way of accessing these benefits. For example, diplont or haplont species can reduce generation times or the frequency of sexual reproduction without evolving haploid-diploid life cycles. Similarly, differentiated life cycle stages (Steenstrup alternations), phenotypic plasticity or genetic polymorphism can allow diplontic or haplontic species to exploit multiple ecological niches without tying growth form to the sexual cycle. Here, we use a population genetic model to show that haploid-diploid life cycles can evolve as a direct consequence of ploidy if the intrinsic fitness of haploids and diploids is not equal.

In species where intrinsic fitness differences and genome-wide mutation rates are of a similar magnitude to one another, haploid-diploid life cycles can only evolve according to the model presented here if diploids have higher intrinsic fitness than haploids and deleterious/beneficial mutations favour haploidy. In this case, the frequency of deleterious mutations (or time taken for beneficial mutations to fix), and thus the strength selection in favour of haploidy, is largest in predominantly diploid populations and weakest in predominantly haploid populations generating the type of frequency-dependent selection needed for haploid-diploid life cycles to evolve. In theory, a diploid intrinsic fitness advantage may be particularly likely due to several previously proposed hypotheses. Firstly, Orr (1995) showed that diplonty can protect organisms from partially recessive somatic mutations (e.g., masking potentially cancerous mutations that arise during development). Although Orr (1995) did not explicitly explore whether haploid-diploid life cycles could evolve, considering somatic mutations that are partially recessive in his model generates a diploid advantage of the type considered here (see *Mathematica* file). Secondly, Haig and Wilczek (2006) proposed that, when diploid growth is partly provisioned by the female haploid (e.g., if diploids grow on haploids), paternally expressed genes will favour greater female allocation to his diploid offspring, improving the fitness of that phase.

Given that deleterious mutations are typically partially recessive (Simmons and Crow 1977, Agrawal and Whitlock 2011, Manna et al. 2011), the region in which a haploid-diploid life cycle evolves is unlikely to be commonly encountered, except in two circumstances. First, if mutations are more dele-

terious in homozygous diploids than in haploids ($s_d > s_h$), haploid-diploid life cycles can be favoured when deleterious mutations are partially recessive (figure 3.2a). Second, when recombination rates are low, the region in which haploid-diploid life cycles are favoured moves into the zone where deleterious mutations are partially recessive (figure 3.2b).

A previous investigation by Otto and Marks (1996) found that haploidy was also favoured by recessive deleterious mutations when selfing, asexual reproduction or assortative mating is common (similar to low recombination). These results were interpreted via the fact that these mating schemes partly cause the effective recombination rate to be reduced, e.g., recombination has no impact in a selfed, homozygous individual. However, this analysis assumed that homozygotes and haploids have equal fitness, thus increased homozygosity had no direct impact on fitness. Here, we show that, when haploids and diploids have unequal fitness and/or when new mutations occur during the life cycle (e.g., at meiosis), the net effect of selfing can favour haploidy or diploidy (Appendix B). We also note that the frequency of deleterious mutations, and thus their relative impact on life cycle evolution, is also decreased with increased selfing because they are exposed to selection in the homozygous state (Appendix B). Thus, if the fitness of haploids and homozygous diploids differs, we caution against generally predicting that haplont and haploid-diploid life cycles should be more common in species where selfing, asexual reproduction and assortative mating are frequent. For example, this may explain why a survey by Mable and Otto (1998) found no correlation between haploidy and the estimated degree of sexuality in protists or green algae.

When the balance between intrinsic fitness differences and the effect of mutations favours convergence on haploid-diploid strategies, disruptive selection then arises such that polymorphisms can evolve with alternative alleles coding for longer haploid and longer diploid phases (i.e., a polymorphic strategy of specialists). In our simulations, a single modifier locus is able to confer fully haplont or diplont life cycles, polymorphism at this locus therefore means that these specialists life cycles can be relatively common (along with the life cycle of the heterozygote at the modifier locus). If genetic control of the life-cycle instead involves many modifier loci, each of which was limited to a having a small effect on the length of the haploid phase, a higher proportion of intermediate phenotypes would be observed in a population experiencing disruptive selection due to mating and recombination. This is especially true when modifier loci are loosely linked because associations between alleles at different loci (linkage disequilibria) are small when recombination is large relative to selection (e.g, Otto and Day

2007, equation 9.45). Disruptive selection was also observed in a density-dependent model where haploids and diploids occupy different niches with or without deleterious mutations (Rescan et al. 2016). Temporal variability of niche sizes can, however, stabilize obligatory alternation between phases (Rescan et al. 2016). Thus, for haploid-diploid life cycles to be favoured over a polymorphic population of specialist haploids and diploids appears to require constraints on the genetic architecture underlying life cycle variation or external variability.

It is intuitively and empirically reasonable that haploids and diploids should both differ in intrinsic fitness and in the extent to which new mutations are masked/revealed to selection. Here, we find the conditions under which these selective forces are approximately balanced and show that this suggests a new hypothesis for the evolution of haploid-diploid life cycles. A significant strength of this hypothesis is that haploid-diploid life cycles evolve in species undergoing an alternation of haploids and diploid phases without positing any extrinsic benefits.

Chapter 4

The Role of Pollen and Sperm Competition in Sex Chromosome Evolution

4.1 Summary

To date, research on the evolution of sex chromosomes has focused on sexually antagonistic selection, which has been shown to be a potent driver of the strata and reduced recombination that characterize many sex chromosomes In this study, we expand our view of the forces driving sex chromosome evolution by considering also selection among haploids, which is likely to occur predominantly among male gametes in angiosperms and animals, i.e., during pollen or sperm competition. We find that suppressed recombination is favoured on the sex chromosomes, even without selective differences between male and female diploids. Reduced recombination is favoured because it creates a stronger association between haploid beneficial alleles and the male determining region (Y or Z), which experiences haploid selection most often. Similarly, reduced recombination creates linkage between alleles selected against in the haploid stage and the female determining region (X or W). In XY systems, these associations also result in biased sex ratios at birth. Overall, we predict that whether and how fast recombination suppression evolves on the sex chromosomes can depend on the degree of haploid competition, not just on selective differences between the diploid sexes. Based on our models, sex chromosomes should become enriched for genes that experience haploid selection, as is expected for genes that experience sexually antagonistic selection. Thus, we generate a number of promising predictions that can be evaluated in emerging sex chromosome systems.

4.2 Introduction

In organisms with diploid genetic sex determination, recombination is typically suppressed between the X and Y chromosomes or Z and W chromosomes. Suppressed recombination appears to begin near the sex-determining region (SDR) and then expand to include larger segments of each sex chromosome (Bergero et al. 2007, Nam and Ellegren 2008, Lemaitre et al. 2009, Wang et al. 2012, Charlesworth 2013). In the absence of recombination, the sex-limited chromosome (Y or W) accumulates deleterious mutations (including gene losses) within the non-recombining region and 'genetic degeneration' occurs (Rice 1996, Charlesworth and Charlesworth 2000, Bachtrog 2006, Marais et al. 2008). Thus, the selective forces driving reduced recombination on sex chromosomes are fundamental to our understanding of sex chromosome evolution.

Typically, selective differences between males and females have been evoked to explain the suppression of recombination around established sex-determining regions (Fisher 1931, Bull 1983, Rice 1987). Charlesworth and Charlesworth (1980) showed that loci where males and females differ in equilibrium allele frequency due to selection (for example, sexually antagonistic selection) should evolve complete linkage with the sex-determining locus via translocations or fusions. More recently, Lenormand (2003) demonstrated that sex differences in allele frequencies at equilibrium are not required in order to favour reduced recombination with the sex-determining region. In fact, recombination suppression can evolve around the sex-determining region even if selection favours the same allele in both sexes as long as that allele is favoured more strongly in one sex than the other. In essence, these studies have demonstrated that suppressors of recombination can be favoured because they strengthen the association between the sex in which an allele is favoured and the chromosome that is present in that sex more often, e.g., between male beneficial alleles and the Y or Z and between female beneficial alleles and the X or W (Otto et al. 2011).

While differences in selection between the diploid sexes has attracted the most theoretical and empirical attention, the haploid gametes/gametophytes produced by males and females also experience different selective environments from one another, with particularly intense competition typically occurring among pollen and sperm (Mulcahy et al. 1996, Bernasconi 2004, Joseph and Kirkpatrick 2004). To the extent that pollen and sperm success reflects differences in their haploid genotypes, selection among these gametes/gametophytes is qualitatively distinct from selection among diploid males. That is, diploids cannot be assigned fitness values that also account

for the fitness of their haploid gametes (Immler et al. 2012). In plants, selection among haploid male gametophytes is thought to be pervasive (Skogsmyr and Lankinen 2002, Moore and Pannell 2011, Marshall and Evans 2016); in *Arabidopsis*, 60-70% of all genes are expressed during the haploid phase (Borg et al. 2009), and pollen expressed genes exhibit stronger signatures of purifying selection and positive selection (Arunkumar et al. 2013, Gossmann et al. 2014). For agricultural breeding, pollen has been exposed to a variety of selection pressures *in vivo* and *in vitro*, including temperature (Hedhly et al. 2004, Clarke et al. 2004), herbicides (Frascaroli and Songstad 2001), metals (Searcy and Mulcahy 1985), water stress (Ravikumar et al. 2003), and pathogens (Ravikumar et al. 2012), resulting in an increased frequency of resistant genotypes among the diploid sporophytic offspring. In animals, expression during the haploid sperm stage is traditionally thought to be suppressed (Hecht 1998), although recent evidence suggests that the extent and selective importance of postmeiotic gene expression may be underestimated (Zheng et al. 2001, Joseph and Kirkpatrick 2004, Vibranovski et al. 2010, Immler et al. 2014).

In this study, we include selection during the haploid phase in models for the evolution of recombination around the sex-determining region (XY or ZW). Specifically, we include a period of selection among the gametes/gametophytes produced by one sex, e.g., competition among pollen or sperm but not among ovules or eggs. Thus, we investigate whether sex differences in the selective environment experienced by haploid gametes/gametophytes can drive the evolution of suppressed recombination on sex chromosomes, as with sex differences in diploid selection. One complication is that haploid selection can cause zygotic sex ratios to become biased. For example, a high fitness allele that becomes more associated with the Y than the X will cause Y-bearing pollen/sperm to outcompete X-bearing pollen/sperm. Thus, increased fertilization success of Y-bearing pollen/sperm will lead to an excess of male zygotes, even if X-bearing and Y-bearing pollen/sperm are produced in equal proportions by males. Tighter linkage with the sex determining region allows greater differences in fitness between X- and Y-bearing pollen/sperm to evolve and thus greater sex ratio biases, figure 4.1. Some models that include haploid selection have found that mothers experience selection to equalize zygotic sex ratios (Hough et al. 2013, Otto et al. 2015). Here, we find that a period of selection among haploid pollen/sperm favours suppressed recombination on the sex chromosomes despite causing biased zygotic sex ratios.

4.3 Model Background

Recombination evolution on sex chromosomes is usually modelled by considering a locus under selection, the sex-determining region, and another locus that modifies the recombination rate between them, where modifiers include inversions, fusions, hotspot changes, and changes to genes involved in double strand breaks and recombination repair. Thus, a general model includes three loci and the recombination rates between them, which is typically too complex to interpret without further simplifying assumptions (Otto and Day 2007). Lenormand (2003) assumed that the recombination rates between these loci are large relative to selection, such that the linkage disequilibrium between loci equilibrates on a faster timescale than changes in allele frequencies (a 'quasi-linkage equilibrium' approximation). This analysis is most appropriate for selected loci that are far from the sex-determining region on sex chromosomes and when modifiers of recombination are weak and loosely linked (e.g., autosomal modifiers of recombination machinery). Secondly, Charlesworth and Charlesworth (1980) assumed that the selected locus is initially autosomal and then considered fusions with (or translocations to) the sex-determining region, where their analysis assumed these rearrangements became closely linked to the selected locus. Their model also corresponds to modifications on sex chromosomes (e.g., inversions) that change the recombination rate with the sex-determining region from a very high to a very low level. Finally, Otto (2014) considered modifiers of recombination between the sex-determining region and selected loci when the linkage between them is initially very tight.

Here, we study recombination evolution in a manner akin to Charlesworth and Charlesworth (1980) and Otto (2014) except that we include a period of selection among haploid male gametes/gametophytes. The model of Lenormand (2003) is very general and allows a period of haploid selection (assuming weak linkage); he recognizes but does not discuss the potential of such sex-specific haploid selection to favour suppressed recombination on sex chromosomes. Here, our goal is to complete the set of recombination evolution analyses that include a period of haploid pollen/sperm competition and explicitly describe why loci that experience haploid selection can drive the evolution of reduced recombination near sex-determining regions. Models where haploid selected loci and the sex-determining region can become tightly linked are particularly significant because strong associations between haploid selected alleles and the sex-determining region (that can build up when linkage is tight) will cause zygotic sex ratios to become biased, figure 4.1.

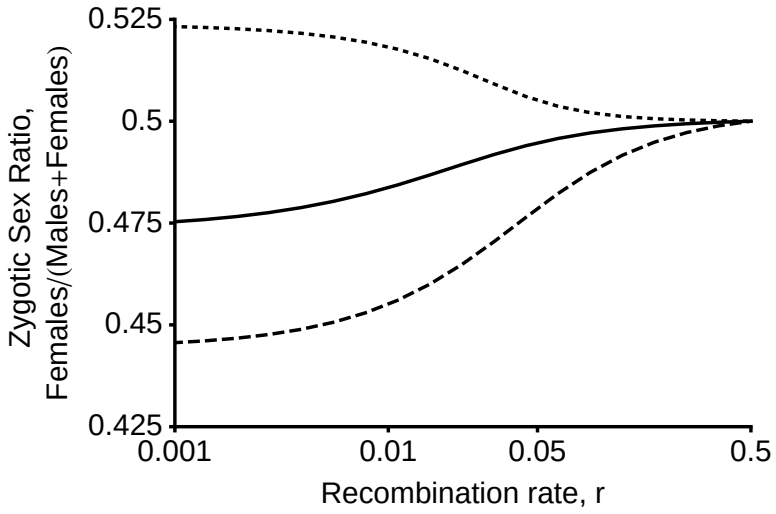

Figure 4.1: Here, we assume that the population is fixed for a particular modifier of recombination such that all individuals have the same recombination rate, r. We then allow the **A** locus to reach an equilibrium frequency and calculate the zygotic sex ratio. Alleles with high fitness during pollen/sperm competition typically become associated with the Y, causing sex ratios to become male-biased (solid and dashed lines). However, female biased sex ratios can arise if the haploid-beneficial allele is also strongly female-beneficial, causing it to become associated with the X (dotted line). The parameters used in this plot are: solid line ($w^m_{ij} = w^f_{ij} = w_{ij}$, $w_{aa} = 1$, $w_{Aa} = 0.97$, $w_{AA} = 0.91$, $w_a = 0.9$, $w_A = 1$) dashed line ($w^m_{ij} = w^f_{ij} = w_{ij}$, $w_{aa} = 1$, $w_{Aa} = 1.12$, $w_{AA} = 1.24$, $w_a = 0.8$, $w_A = 1$), dotted line ($w^m_{aa} = 1$, $w^m_{Aa} = 0.94$, $w^m_{AA} = 0.8$, $w^f_{aa} = 1$, $w^f_{Aa} = 1.14$, $w^f_{AA} = 1.2$, $w_a = 0.9$, $w_A = 1$).

4.4 Model

We consider a modifier model in which the recombination rate between a locus under selection (selected locus, **A**, with alleles A and a) and the sex-determining region (SDR) depends on the genotype at the modifier locus (**M**, with alleles M and m). In our model, male haploid gametes/gametophytes experience selection according to their genotype at the **A** locus (see table C.1) before random mating with female gametes/gametophytes. The resulting zygotes develop as males or females depending on their genotype at the sex-determining region. Diploid genetic sex determination systems are either male heterogametic (females XX and males XY) or female heterogametic (females ZW and males ZZ). There are therefore two asymmetries in the model, the sex in which haploid selection occurs and the sex that is heterogametic. For simplicity, we primarily describe XY sex determination with male gametophytic selection (pollen/sperm compe-

tition), although we also present results for ZW sex determination and male gametophytic selection.

After a period of selection among diploid males and females (table C.1), meiosis with recombination occurs to produce haploid gamete/gametophytes. Because females are homozygous at the SDR (with XY sex determination), the only recombination event of consequence in females is between the **A** and **M** locus, which occurs at rate R_f. In males, recombination similarly occurs between the selected locus **A** and the modifier locus **M** at rate R_m. Recombination can also occur between the SDR and the **A** locus in males, this recombination rate is controlled by the modifier locus and is given by r_{ij}, where ij is the genotype at the **M** locus (MM, Mm, or mm), allowing this recombination rate to evolve. Double recombination events in males occur at rate χ_{ij}, such that any ordering of the loci or type of modifier (genic, inversion, fusion) can be modelled with appropriate choices of χ_{ij}, r_{ij}, and R_m. We track the frequencies of MA, Ma, mA and ma genotypes among female eggs/ovules, male X-bearing sperm/pollen, and male Y-bearing sperm/pollen separately to allow sex-specific allele frequencies and disequilibria. The recursion equations describing the change in genotype frequencies after a single generation of this life cycle are provided in the Appendix C.

In our first analysis, we assume that selection is weak relative to the initial recombination rate (r_{MM}), such that allele frequency differences between males and females are small. We then evaluate the spread of modifiers of recombination (m) that cause recombination rates to become very small (assuming r_{Mm}, χ_{Mm}, and R_m are all small). These modifiers could be translocations or fusions from autosomes to sex chromosomes or, if the selected locus (**A**) begins on the sex chromosome, inversions or expansions of the non-recombining region. We assume that chromosomes are still able to disjoin regularly from their homologs during meiosis.

In our second analysis, following Otto (2014), we assume that the **A** locus begins at equilibrium and in tight linkage with the SDR (r_{MM} and χ are on the order of a small term, ϵ). We then consider whether any modifiers can invade that increase this recombination rate slightly (where the change in recombination rate, $r_{Mm} - r_{MM}$, is on the order of ϵ). The recombination rate between the modifier locus and these sex chromosome loci (R_f and R_m) is not constrained. This analysis focuses on the final stages of sex chromosome evolution, asking when complete recombination is favoured or not.

4.5 Results

Considering a population originally fixed for the M allele at the modifier locus, the frequency of the A allele among X-bearing eggs/ovules, X-bearing sperm/pollen, and Y-bearing sperm/pollen is given by p_{Xf}, p_{Xm}, and p_{Ym} respectively. The spread of rare mutants that change the recombination rate can be evaluated using the leading eigenvalue, λ, of the system described by equations (A1c), (A1d), (A2c), (A2d), (A3c), and (A3d).

Complete suppressors of recombination ($r_{Mm} = 0$) that are closely linked to the **A** locus ($R_f = R_m = \chi = 0$) experience the strongest selective force. These modifiers can bring either the A or the a allele into tight linkage with either the X or Y chromosome. Thus, the invasion of these mutants can be evaluated separately and is given by λ_{ij}, where ij is the haplotype at the newly linked SDR and **A** loci.

The spread of modifiers that create tight linkage between the Y and A allele is given by

$$\lambda_{YA} = \bar{w}^m_{YA}/\bar{w}^m \tag{4.1}$$

where $\bar{w}^m_{YA}$ is the marginal fitness of YA haplotypes and $\bar{w}^m$ is the mean fitness of males, see table C.2. Such modifiers will spread if $\lambda_{YA} > 1$, which is true when $\bar{w}^m_{YA} > \bar{w}^m$.

Invasion of modifiers that create a strong linkage between the X and a allele is determined by the largest solution to the characteristic polynomial (C.4). For such modifiers, the leading eigenvalue λ_{Xa} is greater than one if

$$\bar{w}^{mat,f}_{Xa}/\bar{w}^f + (\bar{w}^{mat,m}_{Xa}/\bar{w}^m)(\bar{w}^{pat,f}_{Xa}/\bar{w}^f) > 2 \tag{4.2}$$

where $\bar{w}^f$ is the mean fitness of females and $\bar{w}^{i,j}_{Xa}$ indicates the marginal fitness of Xa haplotypes when inherited from the mother ($i = mat$) or father ($i = pat$) and found in offspring of sex j. This condition demonstrates that the newly formed sex chromosome is able to invade if its marginal fitness is higher than average (once appropriately weighted across carriers of maternal and paternal copies).

Here, we consider the case where the **A** locus is initially at an intermediate frequency maintained by selection. Polymorphisms can be maintained by a combination of sexually antagonistic selection, ploidally antagonistic selection, and/or overdominance (Immler et al. 2012). We write λ_{YA} in terms of the difference in fitness between haploid genotypes ($\delta_H = w_A - w_a$) and the difference in equilibrium allele frequency between Y-bearing pollen/sperm and ovules/eggs ($\delta = \hat{p}_{Ym} - \hat{p}_{Xf}$) where the caret indicates an equilibrium

frequency. We can then write equation (4.1), for the invasion of modifiers that bring the A allele into tight linkage with the Y chromosome, as

$$\lambda_{YA} = 1 + \frac{r_{MM} w^m_{Aa}}{\hat{p}_{Ym} \bar{w}^m} \left(\delta + V_m \delta_H / \bar{w}_H\right) \tag{4.3}$$

where $V_m = \hat{p}_{Ym}(1 - \hat{p}_{Ym})$ is the variance among Y-bearing pollen/sperm and $\bar{w}_H = (\hat{p}_{Ym} w_A + (1 - \hat{p}_{Ym}) w_a)$ is the mean fitness of haploid male gametes/gametophytes. If there is no selection among haploid genotypes ($w_A = w_a$), equation (4.3) is equivalent to equation (A3) in Charlesworth and Charlesworth (1980), in which case these tightly linked YA haplotypes invade if the A allele is more common in males than females ($\hat{p}_m - \hat{p}_{Xf} > 0$), as expected if the A allele is beneficial in males with sexually antagonistic selection. We also find an additional term, demonstrating that tight linkge is also favoured when the A allele is beneficial during haploid selection ($w_A > w_a$), even in the absence of frequency differences between males and females ($\hat{p}_{Ym} = \hat{p}_{Xf}$), i.e., even when there is no difference in selection between diploid males and females.

Here, in order to solve (C.4) for λ_{Xa}, we will assume that linkage is initially loose between the SDR and **A** locus ($r_{MM} = 1/2$), such that segregation in males is random and $\hat{p}_{Xm} = \hat{p}_{Ym} = \hat{p}_m$. In Appendix C we present equivalent results for cases where we do not assume that recombination is initially free ($r_{MM} < 1/2$). We will further assume that selection is weak, such that the difference in frequency between A alleles in males and females ($\delta = \hat{p}_m - \hat{p}_{Xf}$) and the difference in fitness between haploid genotypes ($\delta_H = w_A - w_a$) are small (δ and δ_H of order ϵ^2). Ignoring terms of order ϵ^3 and higher

$$\lambda_{Xa} = 1 + \frac{1}{3} \frac{w^m_{Aa}}{2(1 - \hat{p}_m)\bar{w}^m} \left(\delta + V_m \delta_H\right). \tag{4.4}$$

Thus, the same conditions that favour linkage between the Y and the A allele, favour linkage between the X and the a allele. Specifically, when the a allele is more common in females ($\delta > 0$, e.g., a is a female beneficial allele) and when the A allele is favoured during haploid competition ($\delta_H > 0$). In the special case where there is no difference in selection between male and female diploids ($w^m_{ij} = w^f_{ij} = w_{ij}$), we can find an exact expression for λ_{XA} by solving for $\hat{p}_m$ and $\hat{p}_{Xf}$ without assuming that selection is weak, which confirms the expectation from (4.4) that linkage between the X chromosome and alleles deleterious in haploid pollen/sperm is favoured by selection, see Appendix C.

It may not be intuitively obvious why an association with the allele that is less fit during haploid selection should be favoured. This result comes from the fact that the a allele is initially maintained at an equilibrium frequency by selection. At equilibrium, selection against a in haploid male gametes/gametophytes must be balanced by selection in favour of A in female and/or male diploids. The X chromosome is found in males less often than an autosomal or loosely linked locus and therefore experiences haploid selection less frequently. Thus linkage between the a locus and the X is favoured because it allows the a allele to experience haploid selection less often. Similarly, equation (4.3) indicates that linkage between the Y, which experiences haploid selection most often, and a haploid beneficial allele is favoured.

As with previous analyses (Charlesworth and Charlesworth 1980, Charlesworth and Wall 1999, Lenormand 2003), we find that the strength of selection in favour of recombination modifiers is strongest on Y chromosomes because these are always found in only one sex whereas the X will sometimes be found in males and sometimes in females. In particular, (4.3) and (4.4) differ by a factor of 1/3 once we account for the difference between the probability of linkage arising with the A allele, p_m, or the a allele, $(1-p_m)$. However, mutations causing linkage with the Y (e.g., fusions) should also arise at a lower rate because there are three times as many X chromosomes as Y chromosomes in the population, such that the overall establishment rate of recombination modifiers is the same on the X and Y (Pennell et al. 2015).

The tight linkage case considered above is the best case scenario for generating selection in favour of recombination suppressors. For a few parameters, Charlesworth and Charlesworth (1980) find numerically that recombination suppressors spread, but at lower rates, if R_m and R_f are larger. Here, we find analytical results by assuming the recombination rates between the **A** locus, the **M** locus, and the SDR are small (χ, R_m and R_f or order ϵ^3). Neglecting terms of order ϵ^4 and higher, the growth rate of such mutants ($\lambda_{\tilde{ij}}$) is.

$$\lambda_{\tilde{YA}} \approx \lambda_{YA} - \frac{(1-\hat{p}_m)w^m_{Aa}R_m}{\bar{w}^m} - \chi_{Mm} \tag{4.5a}$$

$$\lambda_{\tilde{Xa}} \approx \lambda_{Xa} - \frac{\hat{p}_m}{3}\left(\frac{2w^f_{Aa}R_f}{\bar{w}^f} + \frac{w^m_{Aa}R_m}{\bar{w}^m}\right) - \frac{\chi_{Mm}}{3} \tag{4.5b}$$

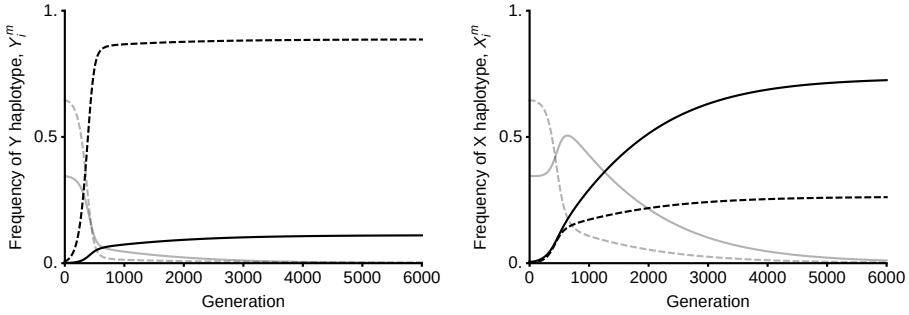

Figure 4.2: Here, we iterate the recursion equations C.1, C.2, C.3 to track the change of genotype frequencies among X-bearing female haploids (X_i^f), X-bearing male haploids (X_i^m), and Y-bearing male haploids (Y_i^m), respectively. Across this plot, X-bearing haploids in males and females have very similar haplotype frequencies so we plot X_i^m only. We assume that the population initially has loose linkage between the **A** locus and the SDR ($r_{MM} = 0.5$, where M is initially fixed) and allow allele frequencies to reach a polymorphic equilibrium. We then introduce a modifier allele m that reduces the recombination rate between **A** locus and the SDR ($r_{Mm} = r_{mm} = 0.01$); in generation 0, m is at frequency 0.01 and in linkage equilibrium with M. We assume that the **M** locus lies between the **A** locus and the SDR such that $\chi_{ij} = (r_{ij} - R_m)/(1 - 2R_m)$, where $R_m = R_f = 0.005$. Fitness parameters are as in the solid line in figure 4.1. That is, there are no differences in selection between diploid sexes and selection is ploidally antagonistic with A favoured by haploid selection, thus $\hat{p}_{Xf} = \hat{p}_{Xm} = \hat{p}_{Ym}$ initially, see Appendix C Lines show the frequencies of the A allele (dashed), the a allele (dotted) the recombination suppression mutant, m (solid). Due to continuing recombination between the **A** locus, **M** locus, and the SDR, a particular haplotype does not fix on the Y chromosome, as is the case when $r_{ij} = 0$ (see Appendix C). However, after recombination has evolved to a lower level, the haploid beneficial allele (A, dashed lines) becomes more common on the Y and less common on the X.

where λ_{ij} corresponds to the tight linkage results (4.3) and (4.4). The additional terms in (4.5) illustrate that the spread of linked haplotypes is slowed when the alternative **A** allele recombines onto the modifier and SDR background (recombination rate R_m or R_f) or when the modifier recombines onto the opposite sex chromosome (which occurs at rate χ_{Mm} in males). In figure 4.2, we track the spread of a recombination modifier where $R_m, R_f, \chi, r_{Mm} \neq 0$, such that both **M** alleles and both **A** alleles can recombine onto both sex chromosomes. As predicted from equation (4.5), a recombination suppressor increases in frequency and the X and Y chromosomes become associated with the a and A alleles, respectively.

We derive equivalent results for ZW sex chromosome systems (where males are ZZ and females are ZW) with a period of haploid selection among male gametes/gametophytes. We again consider invasion of a modifier that creates tight linkage between the **A** locus and the **M** locus (r_{Mm}, χ, R_m and R_f of order ϵ^3) in a population in which linkage is initially loose between

the SDR and **A** locus ($r_{MM} = 1/2$). Here, we present λ_{Wa} and λ_{ZA} under the same assumptions as (4.5), where the difference in frequency of the A allele between males and females and the difference in fitness between haploid genotypes are small ($\delta = \hat{p}_{Zm} - \hat{p}_{Wf}$ where δ and δ_H are of order ϵ^2), yielding

$$\lambda_{\tilde{Wa}} \approx 1 + \frac{r_{MM} w^f_{Aa}}{(1-\hat{p}_f)\bar{w}^f}(\delta + V_f \delta_H) - \frac{\hat{p}_f w^f_{Aa} R_f}{\bar{w}^f} - \chi_{Mm} \tag{4.6a}$$

$$\begin{aligned} \lambda_{\tilde{ZA}} \approx & 1 + \frac{1}{3}\frac{w^f_{Aa}}{2\hat{p}_f \bar{w}^f}(\delta + V_f \delta_H) \\ & - \frac{(1-\hat{p}_f)}{3}\left(\frac{2w^m_{Aa} R_m}{\bar{w}^m} + \frac{w^f_{Aa} R_f}{\bar{w}^f}\right) - \frac{\chi_{Mm}}{3} \end{aligned} \tag{4.6b}$$

where we discard terms of $O(\epsilon^4)$. λ_{ZA} and λ_{Wa} show that, when the A allele is more common in males ($\delta > 0$), linkage between the male Z chromosome and the A allele and linkage between the female specific W chromosome and the a allele are both favoured. In addition, linkage is favoured between the Z and the allele favoured during haploid selection (A if $\delta_H > 0$) and between the female specific W chromosome and the allele with low haploid fitness (a if $\delta_H > 0$). Thus, recombination suppression allows an association between the chromosome that is present in males most often (Z) and alleles favoured during pollen/sperm competition.

Finally, we evaluate the evolution of recombination during the final stages of sex chromosome evolution by considering the evolution of small amounts of recombination around the sex-determining region (SDR). Considering diploid selection alone, Otto (2014) demonstrated that a small amount of recombination around the SDR can be maintained by selection. This result is counterintuitive because, as discussed above, linkage allows associations to build up between the sex-determining region and selected loci. Because these associations arise due to selection, they are generally favourable and one would expect that breaking them apart by recombination would be detrimental. However, particular forms of selection, combined with the asymmetrical inheritance patterns of sex chromosomes can favour loosely linked modifiers that increase recombination around the SDR.

With tight linkage between the SDR and a selected locus (**A**), the Y chromosome always becomes fixed for one allele or the other, see Appendix

C. Without loss of generality, we will assume that selection on the Y favours the A allele, which becomes fixed on the Y. X chromosomes will therefore be paired with a YA haplotype in diploid males; this alters selection experienced by X chromosomes found in diploid males versus those found in diploid females. For example, the **A** locus will never be homozygous for the a allele in males but could be in females. When there is a polymorphism maintained (such that recombination can have an effect), the X can either be fixed for the a allele or be polymorphic. In either case, the effect of increasing the recombination rate with the sex-determining region is to produce more Ya and XA haplotypes among pollen/sperm. Ya haplotypes always have low fitness given that the Y was originally fixed for the A allele. However, the XA haplotypes produced by recombination can be favoured because they are found in male gametes/gametophytes. X-bearing male gametes/gametophytes first experience pollen/sperm competition and then produce females in the next generation. Thus, the XA haplotypes produced by recombination do not experience the same selective environment as average X chromosomes. Certain selection regimes favour XA haplotypes in pollen/sperm (even if the X is fixed for the a allele), which can favour modifiers that increase recombination around the sex-determining region.

With diploid selection only, increased recombination around the SDR can only evolve if selection in females favours the A allele (which is fixed on the Y) because XA pollen/sperm produced by recombination will next be found in a female (Otto 2014). For this to occur, selection in males must be overdominant (a necessary but not sufficient condition). With overdominance in males, the a allele has the highest fitness on the X chromosome in males because it is always paired with an A allele on the Y. Thus, the a allele can be maintained (or even fixed) on X chromosomes and yet the A allele can be favoured during selection in females. However, with pollen/sperm competition, it is possible for a small increases in recombination to be favoured under a wider variety of selective regimes in diploids, including overdominance, underdominance, sexually antagonistic selection and ploidally antagonistic selection. In Appendix C, we show that the evolution of increased recombination requires either that the a allele is favoured by selection on the X in males ($w^m_{Aa} > w^m_{AA}$ or that the A allele is favoured during selection among haploid male gametes/gametophytes ($w_A > w_a$). If the A allele is selected against on male X chromosomes, it is possible for it to be favoured on female X chromosomes and yet still maintain the a allele. In addition, XA haplotypes produced by recombination will be found in pollen/sperm and thus experience haploid selection before becoming diploid females. Therefore, if haploid selection favours the A allele, XA haplotypes in pollen/sperm can

have high fitness.

Given that XA pollen/sperm have an advantage through haploid competition and/or high fitness in female diploids, the fitness advantage of XA pollen/sperm must outweigh by the cost of producing low-fitness Ya pollen/sperm. Thus, increased recombination around the SDR only evolves in particular regions of parameter space (figure C.1). In addition, the evolution of increased recombination requires that the modifier is sufficiently loosely linked to the SDR (R_f and R_m are sufficiently large), e.g., autosomal modifiers. This allows the modifier to gain a short term advantage before recombining onto a different background. If R_f and R_m are small, the modifier remains linked to the haplotypes it creates (XA and Ya), such modifiers never spread because increased recombination breaks down the association between alleles that are favoured on average in a sex and that sex.

4.6 Discussion

Even in predominantly diploid organisms such as animals and angiosperms, there is considerable potential for selection among haploid male gametes (sperm/pollen). Here, we demonstrate that linkage between the diploid sex-determining region (XY or ZW) and a locus that experiences haploid selection is typically favoured by selection. Thus, along with selective differences between diploid sexes, selection among haploids could be a potent driver of recombination suppression on sex chromosomes.

In ZW sex determination systems, the sex ratio among diploids is unaffected by selection among male haploids. However, in XY sex determination systems, the number of males and females in each generation depends on the frequency of X and Y gametes after haploid selection. Despite this, we find that selection on recombination modifiers is not primarily driven by balancing the sex ratio of diploids. In fact, the evolution of recombination suppression should lead to Y-bearing gametes/gametophytes that have high fitness during haploid selection. Thus, we predict that sex ratios at birth can become male biased in the early stages of sex chromosome evolution.

Biased flowering sex ratios, especially male-biased sex ratios, are common among dioecious plants (Field et al. 2013). However, in *Rumex*, more intense pollen competition appears to result in more female biased sex-ratios among the progeny (Conn and Blum 1981, Stehlik and Barrett 2006, Field et al. 2012). This phenomenon may reflect the accumulation of deleterious mutations on the Y-chromosome following recombination suppression (Lloyd 1974, Stehlik and Barrett 2005), as suggested by the prevalence of female

sex ratio bias in species with heteromorphic rather than homomorphic sex chromosomes (Field et al. 2013). Thus, the net effect of experimentally manipulating the intensity of haploid selection may depend on the stage of sex chromosome degeneration, as well as the alleles associated with the Y. The increasing availability of sex-linked markers should allow sexes to be identified before reproductive maturity in plants, thus allowing changes in the sex ratio to be directly evaluated across haploid and diploid phases in species with differing degrees of Y chromosome degeneration.

The emergence of both haploid expression profiles (Joseph and Kirkpatrick 2004, Borg et al. 2009) and a larger number of sex chromosome systems (Ming et al. 2011, Charlesworth 2013; 2015, Bachtrog et al. 2014, Vicoso and Bachtrog 2015) provides an excellent opportunity to evaluate whether sex chromosomes are enriched for genes selected during the haploid phase, as predicted by our models. If possible, a stronger signal of association with sex-determining regions should occur among loci explicitly shown to exhibit variation in haploid competitive ability (Travers and Mazer 2001) or loci where mutants affect fitness in both haploid and diploid phases (Muralla et al. 2011). Finally, we predict that the strength of haploid competition partly determines whether and how fast recombination suppression evolves. Evaluating a related hypothesis, Lenormand and Dutheil (2005) correlate heterochiasmy (differences in autosomal recombination between sexes) with the degree of sex specific haploid selection, using outcrossing rate as a proxy for male haploid selection. We would predict a similar pattern for recombination suppression around sex-determining regions. Estimates of pollen limitation could also be used as proxy for the intensity of haploid competition (Vamosi et al. 2006, Friedman and Barrett 2009).

As in a previous analysis by Otto (2014), we find that a small amount of recombination can be selectively maintained around the sex-determining region. Otto (2014) considered only diploid selection and found that overdominance in males was required for recombination to be selectively maintained. Here, we include a period of selection among haploids and find that increased recombination can be favoured with various forms of selection among diploids, including sexually antagonistic selection and ploidally antagonistic selection (figure C.1), as long as the allele fixed on the Y is favoured in haploids and/or females. However, increased recombination is never favoured when modifiers of recombination act locally, such that they are also closely linked to the sex-determining region. Therefore, while these dynamics may influence the maintenance of small amounts of recombination around sex-determining regions when polymorphisms with the right form of selection arise (e.g., within the coloured regions in figure C.1), suppressed

recombination will be favoured in most circumstances.

Meiotic drive is not exactly equivalent to a period of haploid selection. In particular, meiotic drive can only occur in heterozygotes and often involves an interaction with a separate susceptible/resistant locus (Bull 1983). However, meiotic drive is also usually sex specific, either acting during spermatogenesis in males or polar body formation in females. In this respect, we expect loci experiencing meiotic drive to behave similarly to those experiencing haploid selection. In particular, we predict that selection should favour linkage between alleles favoured by drive and the sex chromosome for the sex in which drive occurs (e.g., with the Y or Z when drive occurs during spermatogenesis). Despite theoretical interest in related topics (Feldman and Otto 1989, Haig 2010, Brandvain and Coop 2012, Patten 2014, Rydzewski et al. 2016), such as the evolution of recombination between an X chromosome that experiences drive and another selected locus (Feldman and Otto 1989, Rydzewski et al. 2016), this process has yet to be explicitly modelled and is worthy of future exploration.

Overall, as well as providing several predictions, our model offers a new perspective on drivers of sex chromosome evolution. Traditionally, sex differences in selection are thought to provide the raw material driving recombination suppression on sex chromosomes. However, even where diploid sexes exhibit very few morphological or ecological differences, the selective environment of their haploid gametes may be very divergent. We have shown that this condition - differences in fitness among pollen or sperm - should also favour suppressed recombination. Consequently, our view of sex chromosome evolution is expanded to incorporate the degree of sex specific selection in haploids along with that in diploids.

Chapter 5

Conclusions

In this Chapter, I briefly review some of the results presented in this thesis and highlight instances where the results obtained were not apparent at the outset. Finally, following Chapter 4, I further discuss the relationship between haploid selection (e.g., pollen/sperm competition) and sex ratios.

5.1 Developmental Delays

A traditional explanation for the evolution of developmental delays is that some environments are unsuitable for growth and thus continuing development 'does not pay' (Cohen 1967, Levins 1968, Schaal and Leverich 1981). We model the evolution of seed dormancy (a developmental delay) in Chapter 2, in which we consider non-annual plants. Perhaps unsurprisingly, the optimal germination rate for non-annuals depends on the environments that might subsequently be experienced, not just the environment in the year that germination occurs (equation 2.6). However, it might not be immediately obvious that this allows dormancy to evolve in environments that are not 'bad' *per se*.

Firstly, we show that dormancy can evolve in environments where seedling survival is low (mechanism 1), even if this environment is not intrinsically 'bad'. Consider the case where the population would grow if it experienced a particular environment (environment 2) all of the time. If the population also experiences another environment (environment 1) with higher seedling survival, it can be favourable to evolve dormancy in environment 2 so that seeds can germinate in environment 1 in future years. Initially, I anticipated that dormancy would only evolve in environments with negative growth rates because we generally expect immediate development to maximize growth rate in all favourable environments (Bulmer 1985, Philippi and Seger 1989, Rees 1996). However, this heuristic is primarily based on models of short-lived organisms; by explicitly modelling demographically structured populations we were able to modify our intuition, finding that decreased seedling survival can be sufficient to favour dormancy. We also note that dormancy can evolve in favourable environ-

ments if there are physical or developmental trade-offs that make it difficult to have germination rates that are equally high in all environments.

Secondly, when large disturbance events occur, we found that dormancy can evolve in apparently favourable environments in order to avoid 'immaturity risk': the risk of dying in a disturbance before reaching reproductive maturity. We might expect this risk to be significant; previous models predict that insects should enter diapause once there is a significant risk of failing to reproduce before a catastrophe occurs (e.g., a winter frost, Cohen 1970, Taylor 1980, Hairston and Munns 1984, Taylor and Spalding 1989, Bradford and Roff 1993, Spencer and Colegrave 2001). On the other hand, before constructing these models, it was not clear that immaturity risk could be significant when the probability of a disturbance is the same in every year. In this case, it seems intuitively reasonable that all years have the same immaturity risk for newly germinating seeds. However, even with a constant risk of disturbance, strategies where germination occurs immediately after the previous disturbance will maximize the age at the time of the next disturbance, relative to strategies in which germination occurs indiscriminately.

Overall, I think one of the key features of this study is that we demonstrate how the mechanisms we present can be isolated. For example, removing differences in seedling survival eliminates mechanism 1 and reducing the number of years required to reach maturity can eliminate mechanism 3. This approach can be applied to carefully collected demographic data, e.g., where stimulated seeds or young seedlings are transplanted into different environments to measure the survival rates if the germination rate was the same. Thus, the contribution of these mechanisms to the evolution of dormancy in a particular environment could be estimated. However, most currently available demographic data does not include transplants into environments in which germination does not occur (e.g., Enright et al. 1998, used as an example in Chapter 2).

Incorporating temporal variation in the environment into a demographically structured population model is challenging and generally requires extra approximations or assumptions (Tuljapurkar 1990*b*). For example, to obtain analytical results in Chapter 2, we assume that temporal variation in the environment is cyclical. Similarly, approximations are often required to consider how the evolution of life history traits (e.g., dormancy) are affected by density dependence in non-demographically structured populations (e.g., annual plants) if there is temporal environmental variation (Bulmer 1984, Ellner 1985*a*;*b*, **?**). A challenge for future research will be to incorporate density dependence into a model with a temporally varying environment and demographic structure. This problem is intuitively important, e.g., the

overshadowing of younger plants by older plants is likely to be a key factor in the evolution of dormancy but is not explicitly included in current models.

5.2 Haploid-Diploid Life Cycles

Ploidy significantly affects the way in which alleles are exposed to selection. Intuition commonly suggests that diploidy is favoured in order to mask the effect of deleterious alleles. For example, H.J. Muller is said to have announced that he wasn't concerned about the mutagenic effects of pepper by stating "that's why we're diploid" (Kirkpatrick 1994). On the other hand, because deleterious alleles are liable to be removed by selection in haploids, the frequency of deleterious mutations is typically lower in haploid populations (Crow and Kimura 1965). Thus, it is not immediately clear how to weigh the immediate masking of deleterious mutations with the change in allele frequency in subsequent generations. Evolutionary invasion analyses (in the form of 'modifier models') have clarified this problem by specifically evaluating the success of mutations that alter whether selection occurs predominantly in the haploid or diploid phase (Perrot et al. 1991, Otto and Goldstein 1992, Jenkins 1993, Jenkins and Kirkpatrick 1995, Hall 2000).

Unlike previous models, in Chapter 3, we allowed haploids and homozygous diploids to differ in intrinsic fitness and considered the interaction between intrinsic fitnesses and the masking effects of ploidy. At the outset of this project, I considered the possibility that the balance between these two forces could favour life cycles that have both haploid and diploid phases. As predicted, our results show that intermediate haploid-diploid life cycles can evolve if diploids have higher intrinsic fitness and deleterious mutations favour haploidy. However, unexpectedly, we found that the reverse situation - where haploids have high intrinsic fitness and deleterious mutations favour diploidy - does not favour convergence upon haploid-diploid life cycles. This is because the frequency of deleterious alleles is highest in predominantly diploid populations. Therefore, selection due to deleterious alleles is strongest when diploidy is common, which prevents convergence upon a haploid-diploid strategy if deleterious alleles favour diploidy. Thus, an intrinsic diploid advantage is a strong condition for haploid-diploid life cycles to have evolved via the mechanism we present. This condition can be examined by measuring fitness components of isogenic haploids and diploids in a natural environment.

Taken together, these models have greatly clarified how haploidy and diploidy influence life cycle evolution. The main contribution of Chapter

3 is to remove the assumption that haploids and homozygous diploids are equivalent. From this, we found a novel hypothesis for the evolution of haploid-diploid life cycles.

5.3 Sex Chromosome Evolution

One feature that characterizes many sex chromosome systems is that, along most of the length, recombination with the opposite sex chromosome has been lost (Skaletsky et al. 2003, Bergero et al. 2007, Nam and Ellegren 2008, Lemaitre et al. 2009, Wang et al. 2012, Wright et al. 2014). The primary explanation for this phenomenon involves sexually antagonistic selection between diploid sexes (Fisher 1931, Bull 1983, Rice 1987, Charlesworth 2013; 2015). For example, reduced recombination allows a a stronger association between male beneficial alleles and the Y and between female beneficial alleles and the X. However, the haploids produced by males and females also experience very different selective environments; particularly intense selection occurs among pollen and sperm (Mulcahy et al. 1996, Bernasconi 2004, Joseph and Kirkpatrick 2004). Lenormand (2003) indicates (among other results) that sex-specific haploid selection could favour weak recombination suppressors. However, this possibility is not commonly cited as an important driver of sex chromosome evolution. In Chapter 4, I wanted to examine whether selection during the haploid phase could allow strong suppressors of recombination (e.g., inversions or fusions) to spread. An important aspect of our study is that, while we tend to think of physical size as a proxy for the importance of haploid and diploid phases, we might want to alter our perspective to think about how much selection occurs during the haploid stage.

Before conducting this study, we expected sex ratio selection to be an important driver of recombination evolution. The sex of offspring in an XY sex determination system is determined by the chromosome carried by the successful pollen/sperm (after haploid selection). Thus, strong associations between haploid beneficial alleles and the Y (which can build up when recombination is strongly suppressed) will cause sex ratios to become male biased. In general, there is strong selection selection in favour of balancing sex ratios (Fisher 1930, Hamilton 1967). Therefore, we expected that the biasing of sex ratios might prevent recombination suppression. However, our results suggest that sex ratio selection has little impact on the evolution of recombination suppression. This can be understood via the fact that sex ratios are affected by pollen or sperm competition, which is a male-like period

in which selection tends to maximize siring success rather than balance sex ratios (Otto et al. 2015).

We found that selection among haploid genotypes in pollen or sperm can drive the evolution of suppressed recombination between sex chromosomes. Our result presents a number of promising avenues for empirical investigation. In particular, we predict that sex chromosomes will become enriched for genes that experience haploid selection. This prediction can be examined by finding the genomic locations of genes that potentially experience haploid selection (Joseph and Kirkpatrick 2004, Borg et al. 2009) and/or those shown to be essential in both phases (Muralla et al. 2011). More generally, we would expect the rate of sex chromosome evolution to reflect the degree of pollen or sperm competition, for which we might be able to use a proxy like pollination syndrome or outcrossing rate (e.g., Lenormand and Dutheil 2005).

5.4 Extraordinary Sex Ratios: Revisited

In Chapter 4, we show that sex chromosomes should evolve to become linked to alleles that are selected in the haploid phase, resulting in biased sex ratios. Generally, any sex-linked gene that harbours genetic variation in haploid fitness should cause sex ratios to become biased. Sex ratio bias caused by pollen competition has previously been discussed in the context of Y-linked deleterious mutations, which are thought to build up after recombination suppression evolves (Lloyd 1974, Stehlik and Barrett 2005). Sex ratios can also become biased due to meiotic drive; in a classic paper, Hamilton (1967) showed that X- or Y-linked alleles that experience meiotic drive will bias sex ratios. He assumed that driving alleles are under directional selection and spread to fixation but such alleles can also be maintained at intermediate frequencies by selection (Feldman and Otto 1989, Holman et al. 2015). When sex ratios are biased, other loci are expected to evolve to restore equal sex ratios. Indeed, alleles that negate the effect of sex-linked meiotic drivers and restore equal sex ratios have been identified (Stalker 1961, Smith 1975). A similar process occurs with cytoplasmic male sterility alleles (that cause biased sex ratios) and nuclear 'restorer' genotypes (Frank 1989).

When sex ratio bias occurs due to haploid selection, a natural class of sex ratio 'restorers' exist because haploid selection often occurs in a context that is determined by the diploid parents. For example, the intensity of pollen competition can be manipulated by altering style length (Travers and Shea 2001, Lankinen and Skogsmyr 2001, Ruane 2009), delaying stigma receptiv-

ity (Galen et al. 1986, Lankinen and Madjidian 2011) and/or delaying pollen tube growth in the pistil (Herrero 2003). Where the X and Y have fixed fitness differences, Hough et al. (2013) demonstrated that mothers should generally evolve to balance sex ratios by reducing the intensity of haploid competition. However, reducing competition among haploids also reduces the potential for harmful deleterious mutations to be purged. When deleterious mutations are included, the optimal intensity of haploid selection can reflect a balance between maximizing offspring fitness and equalizing sex ratios.

As part of a collaborative project (Otto et al. 2015), I considered the evolution of the haploid 'selective arena' in cases where the X chromosome harbours a polymorphism that affects haploid fitness. Mothers again primarily evolve to restore equal sex ratios. However, modifying haploid selection also affects the X-linked genotypes that are inherited by offspring. Specifically, increasing the intensity of haploid selection increases the proportion of daughters (all progeny of X-bearing sperm/pollen are female) that inherit the allele with high haploid fitness. If this allele has high fitness in daughters, mothers can be selected to increase the intensity of haploid selection; otherwise, decreased selection among haploids is favoured. Thus, because altering haploid selection intensity affects the alleles that are inherited by daughters, mothers can favour slightly biased sex ratios. In addition, I found that stronger sex ratio biases can be favoured by paternal manipulations of the haploid 'selective arena' because fathers are strongly selected to maximize their own siring success (above selection to equalize the sex ratio).

Several aspects of the relationship between haploid selection (e.g., pollen or sperm competition) and sex ratios remain to be explored. For example, new sex-determining systems (particularly transitions between male and female heterogamety) can be favoured in order to restore equal sex ratios in populations that have a sex ratio bias (Bull 1983, Kozielska et al. 2010, Úbeda et al. 2015). Based on the results of Chapter 4, we would expect that sex ratio biases would occur via associations between sex-determining loci and loci that experience haploid selection. However, these associations should also select against transitions between sex-determining systems, as has been found with sexually antagonistic selection (van Doorn and Kirkpatrick 2007; 2010). It is not clear how the spread of new sex determination systems would be influenced by the combination of sex ratio biases and favourable associations between haploid selected loci and sex-determining regions. Finally, Hamilton (1967) pointed out that biased sex ratios can affect population size because the number of offspring in each generation is typically determined by the number of females. Population density can, in

turn, affect the intensity of pollen/sperm competition in future generations because fewer males are available to donate pollen/sperm in a particular area. Thus, a feedback could occur between population densities and haploid selection, which has not yet been investigated.

A satisfactory theory for a difficult problem, such as the evolution of complex life cycles, often requires a cluster of specific models exploring different facets (Levins 1966). In this thesis, I have developed models designed to investigate several aspects of life cycle evolution. These studies demonstrate how mathematical models can advance our understanding by yielding new and unexpected insights.

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Appendix A

Evolution of Developmental Delays Analysis

A.1 Differences in Seedling Survival

Here we provide an outline of our proofs; we also provide a *Mathematica* (Wolfram Research Inc. 2010) file, which can be used to re-derive our results and contains additional details.

The transition matrix for the case of two environments can be written

$$\mathbf{T_D} = \begin{pmatrix} ps_{S1}(1-g_1) + (1-p)s_{S2}(1-g_2) & pb_1 + (1-p)b_2 \\ ps_{S1}s_{Y1}g_1 + (1-p)s_{S2}s_{Y2}g_2 & ps_{A1} + (1-p)s_{A2} \end{pmatrix}, \quad \text{(A.1)}$$

which is a specific form of the more general matrix:

$$\mathbf{T_E} = \begin{pmatrix} a[g_1, g_2] & b \\ c[g_1, g_2] & d \end{pmatrix}. \quad \text{(A.2)}$$

Writing (A.1) in this more general form simplifies the presentation below and allows more general insights to be obtained. The long-term growth rate ($\lambda[g_1, g_2]$) is given by the larger root of the characteristic polynomial for this matrix,

$$\lambda[g_1, g_2]^2 - a[g_1, g_2]\,\lambda[g_1, g_2] - d\,\lambda[g_1, g_2] + d\,a[g_1, g_2] - b\,c[g_1, g_2] = 0. \quad \text{(A.3)}$$

Dynamics of Germination Rate in 'bad' Patches, g_2

The effect of a small mutation on the growth rate is obtained by differentiating this polynomial with respect to g_1 or g_2. For the g_2 case, re-arranging gives

$$\frac{\partial \lambda[g_1, g_2]}{\partial g_2} = \frac{\lambda[g_1, g_2]\frac{\partial a[g_1, g_2]}{\partial g_2} - d\frac{\partial a[g_1, g_2]}{\partial g_2} + b\frac{\partial c[g_1, g_2]}{\partial g_2}}{2\lambda[g_1, g_2] - d - a[g_1, g_2]}. \quad \text{(A.4)}$$

We can then make a simplification in cases where seed survival ($a[g_1, g_2]$) and seed germination ($c[g_1, g_2]$) rates are linear functions of g_2 and g_1, as in equation (A.1), so that $\frac{\partial a[g_1,g_2]}{\partial g_2}$ is proportional to $\frac{\partial c[g_1,g_2]}{\partial g_2}$. Specifically we assume that $\frac{\partial c[g_1,g_2]}{\partial g_2} = \beta \frac{\partial a[g_1,g_2]}{\partial g_2}$, where β is the proportionality constant ($\beta = -s_{Y2}$ in equation A.1). With this simplification, equation (A.4) becomes

$$\frac{\partial \lambda[g_1, g_2]}{\partial g_2} = \frac{\frac{\partial a[g_1,g_2]}{\partial g_2}(\lambda[g_1, g_2] - d + b\,\beta)}{2\lambda[g_1, g_2] - d - a[g_1, g_2]}. \tag{A.5}$$

A potential ESS germination rate (g_2) occurs if $\frac{\partial \lambda[g_1,g_2]}{\partial g_2} = 0$. This condition requires that either

$$\frac{\partial a[g_1, g_2]}{\partial g_2} = 0 \tag{A.6}$$

or

$$\lambda[g_1, g_2] = d - b\,\beta. \tag{A.7}$$

For the parameters in (A.1), $\frac{\partial a[g_1,g_2]}{\partial g_2} = -(1-p)s_{S2}$, which is negative. Hence solution (A.6) does not provide a relevant ESS. However, solution (A.7) does and can be re-written in terms of the original parameters as presented in equation (2.7).

Special Case $s_{Y1} = s_{Y2}$

Here we show that a conditional germination does not evolve when $s_{Y1} = s_{Y2}$ (dashed line in figure 1B). Assuming that $s_{Y1} = s_{Y2} = s_Y$ and re-arranging the transition point in equation (2.7) gives

$$b_1 = \frac{(1-p)(s_{S2} - s_{A2} - b_2 s_Y) + p(s_{S1} - s_{A1})}{p s_Y}. \tag{A.8}$$

Substituting this point into transition matrix $\mathbf{T_D}$ in equation (A.1) gives the population dynamics at this point. We then calculated the eigenvalues (λ_1 and λ_2) for this new transition matrix in order to assess whether the long-term growth rate could be at or above replacement ($\lambda \geq 1$). These eigenvalues are:

$$\lambda_1 = (1-p)s_{S2} + p s_{S1} \tag{A.9a}$$

$$\lambda_2 = (1-p)(s_{A2} - g_2 s_{S2}) + p(s_{A1} - g_1 s_{S1}), \tag{A.9b}$$

both of which must be less than 1. Note that the parameters in (A.9) are rates or proportions that must be between 0 and 1. Thus the long-term growth rate when this transition point occurs is below replacement rate ($\lambda < 1$) when $s_{Y1} = s_{Y2}$.

Furthermore, it can be shown that increasing the germination rate in 'bad' patches (g_2) has a positive effect on growth rate ($\frac{\partial \lambda[g_1,g_2]}{\partial g_2} > 0$) when $s_{Y2} = s_{Y1}$, assuming that the population is able to grow ($\lambda > 1$). This proof uses equation (A.5), which can be written as:

$$\frac{\partial \lambda[g_1, g_2]}{\partial g_2} = (\lambda[g_1, g_2] - d + b\,\beta)\frac{\frac{\partial a[g_1,g_2]}{\partial g_2}}{2\lambda[g_1, g_2] - d - a[g_1, g_2]}. \tag{A.10}$$

The fraction in equation (A.10) is always positive, assuming that $\lambda[g_1, g_2] > 1$. We also show that the remaining part ($\lambda[g_1, g_2] - d + b\,\beta$) is always positive when $s_{Y1} = s_{Y2}$ and $\lambda[g_1, g_2] > 1$. The details of this proof can be found in the supplementary *Mathematica* file. Hence, we show that, when seedling survival in 'bad' environments (s_{Y2}) is near its maximum value (assuming $s_{Y1} \geq s_{Y2}$), increasing germination rate in 'bad' environments (g_2) should increase the long-term growth rate. While the transition to evolution favouring conditional germination cannot occur within self-sustaining populations ($\lambda > 1$) with $s_{Y1} = s_{Y2}$, the transition (7) can occur with lower survival rates (e.g., as shown in figure 1B). In addition, we can show that there is, at most one positive transition point satisfying (7) as the juvenile survival rate in normal years (s_{Y2}) is varied and that increasing g_2 will decrease the long-term growth rate for values of s_{Y2} below this point (see supplementary *Mathematica* file).

Dynamics of Germination Rate in 'good' Patches, g_1

The same analysis for mutations to germination rate in 'good' patches (g_1) shows that g_1 is always expected to increase when the population growth rate is at or above replacement ($\lambda \geq 1$) and $s_{Y1} \geq s_{Y2}$. An invasion analysis, as conducted above, yields the solution

$$\lambda[g_1, g_2] = d - b\,\gamma, \tag{A.11}$$

which is analogous to equation (A.7), except that $\gamma = -s_{Y1}$ (whereas $\beta = -s_{Y2}$). We substituted the parameters from (A.1) back into this equation

and re-arranged to get:

$$b_1 = \frac{\big((1-p)(s_{S2}s_{Y1} + g_2 s_{S2}s_{Y2} - b_2 {s_{Y1}}^2 - g_2 s_{S2}s_{Y1} - s_{A2}s_{Y1}) + ps_{S1}s_{Y1} - ps_{A1}s_{Y1}\big)}{p{s_{Y1}}^2} \tag{A.12}$$

We then substituted this point into the transition matrix in equation (2.4) and obtained the following eigenvalues:

$$\lambda_1 = (1-p)\Big(s_{S2}\Big(1 - g_2\left(1 - \frac{s_{Y2}}{s_{Y1}}\right)\Big)\Big) + ps_{S1} \tag{A.13a}$$

$$\lambda_2 = (1-p)\left(s_{A2} - \frac{g_2 s_{S2}s_{Y2}}{s_{Y1}}\right) + p(s_{A1} - g_1 s_{S1}), \tag{A.13b}$$

neither of which can be greater than or equal to 1 (assuming $s_{Y1} \geq s_{Y2}$). That is, there is never a transition point at which $\frac{\partial\lambda[g_1,g_2]}{\partial g_1} = 0$ assuming that $\lambda[g_1, g_2] > 1$ and $s_{Y1} \geq s_{Y2}$. Therefore, unlike germination rate in 'bad' patches (g_2), which can undergo a transition for sufficiently small s_{Y2}, there is no transition point for germination rate in 'good' patches, g_1.

We can also show that the sign of $\frac{\partial\lambda[g_1,g_2]}{\partial g_1}$ is positive assuming that $s_{Y1} \leq s_{Y2}$ and $\lambda[g_1, g_2] > 1$ (see supplementary *Mathematica* file). Hence, increasing the germination rate in 'good' patches, g_1, is always expected to increase the long-term growth rate given that the population is able to grow.

Effect of other parameters on the size of the region in which conditional germination evolves

We re-write transition point (7) in terms of b_1 (called b_{1crit} for critical b_1 value) here:

$$b_{1crit} = \frac{ps_{S1}g_1(s_{Y1} - s_{Y2}) + s_{Y2}((1-p)s_{S2} - (1-p)s_{A2} - (1-p)b_2 s_{Y2} + p(s_{S1} - s_{A1}))}{p{s_{Y2}}^2}. \tag{A.14}$$

As shown in the supplementary *Mathematica* file, conditional germination is favoured for values of b_1 below this point but not above. Thus any parameter that decreases b_{1crit} will decrease the parameter space over which conditional germination evolves. Taking the derivative of b_{1crit} with respect to s_{A1}, s_{A2}

and b_2 gives:

$$\frac{\partial b_{1crit}}{\partial s_{A1}} = -\left(\frac{1}{s_{Y2}}\right), \tag{A.15a}$$

$$\frac{\partial b_{1crit}}{\partial s_{A2}} = -\left(\frac{(1-p)}{p s_{Y2}}\right), \tag{A.15b}$$

$$\frac{\partial b_{1crit}}{\partial b_2} = -\left(\frac{(1-p)}{p}\right), \tag{A.15c}$$

which are all negative, indicating that increasing adult survival or the number of seeds produced in 'bad' patches will decrease b_{fcrit} and therefore restrict the conditions under which conditional germination evolves. In contrast, the effect of changes in s_{S1}, s_{S2}, s_{Y1} and g_1 on b_{1crit} are given by:

$$\frac{\partial b_{1crit}}{\partial s_{S1}} = \frac{g_1 s_{Y1} + (1-g_1) s_{Y2}}{{s_{Y2}}^2}, \tag{A.16a}$$

$$\frac{\partial b_{1crit}}{\partial s_{S2}} = \frac{(1-p)}{p s_{Y2}}, \tag{A.16b}$$

$$\frac{\partial b_{1crit}}{\partial s_{Y1}} = \frac{g_1 s_{S1}}{{s_{Y2}}^2}, \tag{A.16c}$$

$$\frac{\partial b_{1crit}}{\partial g_1} = \frac{s_{S1}(s_{Y1} - s_{Y2})}{{s_{Y2}}^2}, \tag{A.16d}$$

which are all positive (assuming that $s_{Y1} > s_{Y2}$). Hence, increasing seed survival, seedling survival in 'good' patches, or the germination rate in 'good' patches all broaden the conditions under which conditional germination evolves. Increasing p also generally increases b_{1crit} but this proof also requires the assumption that the population can grow in 'bad' patches. The effect of a change in p on b_{1crit} is

$$\frac{\partial b_{1crit}}{\partial p} = \frac{s_{A2} + b_2 s_{Y2} - s_{S2}}{p^2 s_{Y2}}. \tag{A.17}$$

For the population to be able to grow in the absence of 'good' patches requires that the leading eigenvalue with $p = 0$ be greater than one, which in turn implies that

$$b_2 > \frac{1 - s_{A2} + (1-g_2) s_{A2} s_{S2} - (1-g_2) s_{S2}}{g_2 s_{S2} s_{Y2}}. \tag{A.18}$$

Rearranging (A.18) in the form of equation (A.17) gives:

$$\frac{s_{A2} + b_2 s_{Y2} - s_{S2}}{p^2 s_{Y2}} > \frac{(1 - s_{S2})((1 - s_{A2}) + g_2 s_{S2})}{p^2 g_2 s_{S2} s_{Y2}}. \tag{A.19}$$

The right hand side of equation (A.19) is always positive, thus $\frac{\partial b_{1crit}}{\partial p}$ must also be positive, indicating that increasing the proportion of 'good' patches, p, broadens the conditions under which conditional germination is expected to evolve.

A.2 Trade-Offs

equation (2.8) is a form of the more general transition matrix

$$\mathbf{T_F} = \begin{pmatrix} a[g_2] & b \\ c[g_2] & d \end{pmatrix}. \tag{A.20}$$

The effect of a small mutation altering the germination rate on the long-term growth rate ($\frac{d\lambda[g_2]}{\mathrm{d}g_2}$) is now given by

$$\frac{\mathrm{d}\lambda[g_2]}{\mathrm{d}g_2} = \frac{\lambda[g_2]\frac{\mathrm{d}a[g_2]}{\mathrm{d}g_2} + b\frac{\mathrm{d}c[g_2]}{\mathrm{d}g_2} - d\frac{\mathrm{d}a[g_2]}{\mathrm{d}g_2}}{2\lambda[g_2] - d + a[g_2]}. \tag{A.21}$$

In this case, $\frac{\mathrm{d}a[g_2]}{\mathrm{d}g_2}$ is not always proportional to $\frac{\mathrm{d}c[g_2]}{\mathrm{d}g_2}$. To clarify this, we write $\frac{\mathrm{d}a[g_2]}{\mathrm{d}g_2}$ and $\frac{\mathrm{d}c[g_2]}{\mathrm{d}g_2}$ in terms of their original parameters (from equation 7):

$$\frac{\mathrm{d}a[g_2]}{\mathrm{d}g_2} = -\big((1-p)s_{S2} + p s_{S1} g_1'[g_2]\big) \tag{A.22a}$$

$$\frac{\mathrm{d}c[g_2]}{\mathrm{d}g_2} = (1-p)s_{S2}s_{Y2} + p(s_{S1}s_{Y1}g_1'[g_2]). \tag{A.22b}$$

The simplification that $\frac{\mathrm{d}c[g_2]}{\mathrm{d}g_2}$ is proportional to $\frac{\mathrm{d}a[g_2]}{\mathrm{d}g_2}$ requires that seedling survival rates are constant ($s_{Y1} = s_{Y2}$). If we make this simplification then solutions (A.6) and (A.7) again describe the potential ESS (where $\frac{\mathrm{d}\lambda[g_2]}{\mathrm{d}g_2} = 0$). The proof used above to show that there is no relevant ESS solution from equation (A.7) when $s_{Y1} = s_{Y2}$ continues to apply with trade-offs. Equation (A.6) now does yield a potential ESS, which occurs at equation (2.9).

Evolutionary Stability

To determine whether the singular point (2.9) represents a maximum or a minimum growth rate we take the second derivative of the characteristic polynomial (the roots of which yield the long-term growth rate) and evaluated it at the singular point:

$$\frac{\mathrm{d}^2\psi}{\mathrm{d}{g_2}^2}\Big|_{\frac{\mathrm{d}\lambda[g_2]}{\mathrm{d}g_2}=0} = 0, \tag{A.23}$$

where ψ is the characteristic polynomial:

$$\psi = \lambda[g_2]^2 - a[g_2]\,\lambda[g_2] - d\,\lambda[g_2] + d\,a[g_2] - b\,c[g_2] = 0. \tag{A.24}$$

Rearranging gives:

$$\frac{\mathrm{d}^2\lambda[g_2]}{\mathrm{d}{g_2}^2} = \frac{\lambda[g_2]\frac{\mathrm{d}^2a[g_2]}{\mathrm{d}{g_2}^2} + b\frac{\mathrm{d}^2c[g_2]}{\mathrm{d}{g_2}^2} - d\frac{\mathrm{d}^2a[g_2]}{\mathrm{d}{g_2}^2}}{2\lambda[g_2] - d - a[g_2]}. \tag{A.25}$$

In the original parameters

$$\frac{\mathrm{d}^2a[g_2]}{\mathrm{d}{g_2}^2} = -ps_{S1}g_1''[g_2] \tag{A.26a}$$

$$\frac{\mathrm{d}^2c[g_2]}{\mathrm{d}{g_2}^2} = s_{Y1}ps_{S1}g_1''[g_2], \tag{A.26b}$$

so that

$$\frac{\mathrm{d}^2c[g_2]}{\mathrm{d}{g_2}^2} = \gamma\frac{\mathrm{d}^2a[g_2]}{\mathrm{d}{g_2}^2} \tag{A.27}$$

where $\gamma = -s_{Y1}$, whether $s_{Y1} = s_{Y2}$ or not. For evolutionary stability of the singular point (9) $\frac{d^2\lambda[g_2]}{dg_2{}^2}$ must be negative. This condition can be written as:

$$\frac{\mathrm{d}^2a[g_2]}{\mathrm{d}{g_2}^2}\left(\frac{\lambda[g_2] + \gamma b - d}{2\lambda[g_2] - d - a[g_2]}\right) < 0. \tag{A.28}$$

The part in parentheses is positive if the population growth rate is at or above the replacement rate ($\lambda[g_2] \geq 1$, see equations A.11-A.13). Evolutionary stability is therefore determined by the sign of $\frac{\mathrm{d}^2a[g_2]}{\mathrm{d}{g_2}^2}$, which is negative when $g_1''[g_2] > 0$ (implying stability).

Annual Plants

Here we demonstrate how trade-offs impact MacArthur's (1972) model of germination rates in an annual system with global migration among patches. A two-environment version of equation (2.2) with a trade-off between germination rates in different environments is given by

$$S[t] = S[0]\{p((1-g_1[g_2])s_{S1}+g_1[g_2]y_1)+(1-p)((1-g_2)s_{S2}+g_2y_2)\}^t. \quad \text{(A.29)}$$

The population growth rate (λ) is therefore given by the term in braces. This term describes the number of seeds resulting from seeds in the previous year, equivalent to the upper left element of transition matrix $\mathbf{T_F}$, $a[g_2]$. Therefore, defining $a[g_2]$ from equation (A.29), we get $\frac{\mathrm{d}\lambda[g_2]}{\mathrm{d}g_2} = \frac{\mathrm{d}a[g_2]}{\mathrm{d}g_2}$, and equation (A.6) continues to yield a potential ESS, which now occurs where

$$\frac{(s_{S2}-y_2)}{(s_{S2}-y_2)-(s_{S1}-y_1)g_1'[g_2]} - p = 0. \quad \text{(A.30)}$$

Evolutionary stability can again be determined by the sign of $\frac{\mathrm{d}^2a[g_2]}{\mathrm{d}{g_2}^2}$ and will depend on the sign of $g_1''[g_2]$, as above. Therefore, trade-offs can lead to the evolution of conditional germination or intermediate germination rates in an annual plant model with purely spatial environmental variation, as in perennials.

Incorporating Density Dependence

We can account for any form of density-dependent effects on seedling survival through the transition matrix:

$$\mathbf{T_G} = \begin{pmatrix} a[g_2] & b \\ c[g_2]comp[A] & d \end{pmatrix}. \quad \text{(A.31)}$$

Here, we make the assumption that the impact of the adult population size is to reduce the survival of all seedlings, regardless of their provenance. We considered the dynamics of a rare mutant with a slightly different germination rate (${g_2}^{mut} = {g_2}^{res} + \epsilon$) and growth rate ($\lambda[{g_2}^{mut}] = \lambda[{g_2}^{res}] + \epsilon\Delta\lambda$), where we use the ^{res} superscript to denote resident values and ^{mut} for the mutant. The characteristic polynomial for the invasion of such a mutant is:

$$\begin{aligned} 0 =&(\lambda[{g_2}^{res}] + \epsilon\Delta\lambda)^2 - a[{g_2}^{res} + \epsilon](\lambda[{g_2}^{res}] + \epsilon\Delta\lambda) \\ &- d(\lambda[{g_2}^{res}] + \epsilon\Delta\lambda) + d\,a[{g_2}^{res} + \epsilon] - b\,c[{g_2}^{res} + \epsilon]comp[A^{res}] \end{aligned} \quad \text{(A.32)}$$

We then conducted a first order Taylor series expansion of ϵ around 0, assuming that the difference between mutant and resident is small, to obtain

$$\begin{aligned}0 = &\left(\lambda[{g_2}^{res}]^2 - a[{g_2}^{res}]\lambda[{g_2}^{res}] - d\,\lambda[{g_2}^{res}] + d\,a[{g_2}^{res}] - b\,c[{g_2}^{res}]comp[A^{res}]\right)\\ &+ \epsilon\Bigg(\Delta\lambda(2\lambda[{g_2}^{res}] - d - a[{g_2}^{res}]) + \frac{\mathrm{d}a[{g_2}^{res}]}{\mathrm{d}{g_2}^{res}}(d - \lambda[{g_2}^{res}])\\ &- b\,comp[A^{res}]\frac{\mathrm{d}c[{g_2}^{res}]}{\mathrm{d}{g_2}^{res}}\Bigg) + O[\epsilon^2].\end{aligned} \tag{A.33}$$

A mutant with the same trait value as the resident ($\epsilon = 0$) has the same growth rate as the resident, which is $\lambda[{g_2}^{res}] = 1$ at equilibrium with density dependence. Therefore, from equation (A.33):

$$1 - a[{g_2}^{res}] - d + d\,a[{g_2}^{res}] - b\,c[{g_2}^{res}]comp[A^{res}] = 0. \tag{A.34}$$

Re-arranging equation (A.33) and ignoring higher order terms in ϵ gives:

$$\Delta\lambda = \frac{\frac{\mathrm{d}a[{g_2}^{res}]}{\mathrm{d}{g_2}^{res}} + b\,comp[A^{res}]\frac{\mathrm{d}c[{g_2}^{res}]}{\mathrm{d}{g_2}^{res}} - d\,\frac{\mathrm{d}a[{g_2}^{res}]}{\mathrm{d}{g_2}^{res}}}{2 - d - a[{g_2}^{res}]}. \tag{A.35}$$

The similarity between this equation and (A.21) indicates that density dependence does not alter the qualitative results of the model. Indeed, equation (A.35) is the same as equation (A.21) with b now equal to $b\,comp[A^{res}]$. Thus, our results are affected by density dependence in a manner akin to having birth rates adjusted by the competitive effect of resident adults. Because we have assumed that this competitive effect is the same for types with different germination rates, the evolution of these germination rates is qualitatively unaffected. For example, if we assume that $s_{Y1} = s_{Y2}$ then, in the presence of trade-offs, solution (A.6) again represents a potential ESS given by equation (2.9).

A.3 Approximating the Cycle Matrix

The **A** and **D** matrices from equation (2.12) are

$$\begin{aligned}\mathbf{A} &= \begin{pmatrix} u_{11} & u_{12} \\ (1-u_{11}) & (1-u_{12}) \end{pmatrix} \\ \mathbf{D}^{\tau-1} &= \begin{pmatrix} {\lambda_1}^{\tau-1} & 0 \\ 0 & {\lambda_2}^{\tau-1} \end{pmatrix} \\ \mathbf{A}^{-1} &= \begin{pmatrix} \frac{1-u_{12}}{u_{11}-u_{12}} & \frac{u_{12}}{u_{12}-u_{11}} \\ \frac{1-u_{11}}{u_{12}-u_{11}} & \frac{u_{11}}{u_{11}-u_{12}}) \end{pmatrix} \end{aligned} \tag{A.36}$$

where λ_1 and λ_2 are the eigenvalues of $\mathbf{T_2}$, u_{11} and $(1-u_{11})$ are the elements of the right eigenvector associated with eigenvalue λ_1 and u_{12} and $(1-u_{12})$ are the elements of the right eigenvector associated with eigenvalue λ_2. The approximation we used was to drop the smaller eigenvalue from the normal year matrix. So that, assuming $|\lambda_2| > |\lambda_1|$:

$$\widetilde{\mathbf{D}}^{\tau-1} = \begin{pmatrix} 0 & 0 \\ 0 & {\lambda_2}^{\tau-1} \end{pmatrix} \tag{A.37}$$

and

$$\widetilde{\mathbf{T}}_2{}^{\tau-1} = \begin{pmatrix} \frac{(1-u_{11})u_{12}{\lambda_2}^{\tau-1}}{u_{12}-u_{11}} & \frac{u_{11}u_{12}{\lambda_2}^{\tau-1}}{u_{11}-u_{12}} \\ \frac{(1-u_{11})(1-u_{12}){\lambda_2}^{\tau-1}}{u_{12}-u_{11}} & \frac{u_{11}(1-u_{12}){\lambda_2}^{\tau-1}}{u_{11}-u_{12}} \end{pmatrix}, \tag{A.38}$$

in which the $\widetilde{\ }$ notation is used to indicate that these correspond to the approximation. Equation (A.38) multiplied on the right by $\mathbf{T_1}$ gives the approximate transition matrix across the entire disturbance cycle as follows,

$$\widetilde{\mathbf{T}}_{cycle} = \begin{pmatrix} \frac{s_{S1}u_{12}(1-u_{11}-g_1(1-u_{11}(1-s_y))){\lambda_2}^{\tau-1}}{u_{12}-u_{11}} & \frac{u_{12}(u_{11}s_{A1}-b_1(1-u11)){\lambda_2}^{\tau-1}}{u_{11}-u_{12}} \\ \frac{s_{S1}(1-u_{12})(1-u_{11}-g_1(1-u_{11}(1-s_y))){\lambda_2}^{\tau-1}}{u_{12}-u_{11}} & \frac{(1-u_{12})(u_{11}s_{A1}-b_1(1-u11)){\lambda_2}^{\tau-1}}{u_{11}-u_{12}} \end{pmatrix}. \tag{A.39}$$

Here again, we focus on the case where $s_{Y1} = s_{Y2} = s_Y$, which does not permit the evolution of conditional germination in our simple model that ignores temporal variation. The eigenvalues of $\widetilde{\mathbf{T}}_{cycle}$ are then are 0 and

$$\begin{aligned}\lambda_{cycle} = \frac{{\lambda_2}^{\tau-1}}{u_{12}-u_{11}}\big(&b_1(1-u_{11}) + u_{12}(s_{S1}(1-u_{11}) - b_1(1-u_{11}) \\ &- g_1 s_{S1}(1-u_{11}(1-s_Y))) - s_{A1}u_{11}(1-u_{12})\big)\end{aligned} \tag{A.40}$$

Dynamics of Disturbance-Induced Germination Rate, g_1

The change in long-term growth rate over the entire cycle, λ_{cycle}, when disturbance-induced germination rate (g_1) is slightly changed is given by

$$\frac{\partial \lambda_{cycle}}{\partial g_1} = \frac{s_{S1}u_{12}(1-u_{11}(1-s_Y)){\lambda_2}^{\tau-1}}{u_{11}-u_{12}}. \tag{A.41}$$

If this quantity is positive then increasing g_1 is expected to increase the long-term growth rate across the cycle (λ_{cycle}). To evaluate the sign of equation (A.41) we re-write $(1-u_{11}(1-s_Y))$ and $u_{11}-u_{12}$ as follows:

$$1-u_{11}(1-s_Y) = \frac{s_Y(\lambda_2 - s_{A2})}{(\lambda_2 - s_{A2}) + g_2 s_{S2}(1-s_Y)} \tag{A.42a}$$

$$u_{11}-u_{12} = \frac{g_2 s_{S2} s_Y (2\lambda_2 - (s_{A2} + (1-g_2)s_{S2})}{(\lambda_2 - s_{S2}(1-g_2(1-s_Y)))(g_2 s_{S2} s_Y + \lambda_2 - s_{A2})}. \tag{A.42b}$$

Both of which must be positive if we assume that the population is able to grow in non-disturbance years ($\lambda_2 > 1$). Thus equation (A.41) is also positive and mutants with higher g_1 values would have higher long-term growth rates.

Dynamics of Germination Rate in Non-Disturbance Years, g_2

To evaluate the effect of a small change in germination rate in non-disturbance years (g_2) on λ_{cycle} we have to take account of the fact that u_{11}, u_{12} and λ_2 are all functions of g_2 and take a derivative of λ_{cycle} with respect to g_2 to get:

$$\frac{\partial \lambda_{cycle}}{\partial g_2} = \frac{{\lambda_2}^{\tau-2}}{(u_{11}-u_{12})^2}\left(x\lambda_2 \frac{\mathrm{d}u_{11}}{\mathrm{d}g_2} - y\lambda_2 \frac{\mathrm{d}u_{12}}{\mathrm{d}g_2} - z(u_{11}-u_{12})(\tau-1)\frac{\mathrm{d}\lambda_2}{\mathrm{d}g_2}\right), \tag{A.43}$$

where

$$x = b_1(u_{12}-1)^2 + u_{12}\left(s_{A1}(u_{12}-1) + s_{S1}((1-u_{12})(1-g_1) - g_1 s_Y u_{12})\right), \tag{A.44a}$$

$$y = b_1(u_{11}-1)^2 + u_{11}\left(s_{A1}(u_{11}-1) + s_{S1}((1-u_{11})(1-g_1) - g_1 s_Y u_{11})\right), \tag{A.44b}$$

$$z = b_1(u_{11}-1)(u_{12}-1) + s_{A1}u_{11}(u_{12}-1) + s_{S1}u_{12}\left((1-u_{11})(1-g_1) - g_1 s_Y u_{11}\right), \tag{A.44c}$$

and

$$\frac{\mathrm{d}u_{11}}{\mathrm{d}g_2} = \frac{s_{S2}s_Y(\lambda_2 - s_{S2})(\lambda_2 - s_{S2}(1-g_2))}{(\lambda_2 - s_{S2}(1-g_2(1-s_Y)))^2(2\lambda_2 - s_{A2} - s_{S2}(1-g_2))}, \tag{A.45a}$$

$$\frac{\mathrm{d}u_{12}}{\mathrm{d}g_2} = \frac{-s_{S2}s_Y(\lambda_2 - s_{S2})(\lambda_2 + g_2 s_{S2} - s_{A2})}{(\lambda_2 + g_2 s_{S2}s_Y - s_{A2})^2(2\lambda_2 - s_{A2} - (1-g_2)s_{S2})}, \tag{A.45b}$$

$$\frac{\mathrm{d}\lambda_2}{\mathrm{d}g_2} = \frac{(\lambda_2 - s_{A2})(\lambda_2 - s_{S2})}{g_2(2\lambda_2 - s_{A2} - (1-g_2)s_{S2})}. \tag{A.45c}$$

We know that, as τ goes to ∞ (no disturbances), increasing g_2 would increase λ_{cycle} (assuming $\lambda_2 > 1$). However, for short cycles (low τ), $\frac{\partial \lambda_{cycle}}{\partial g_2}$ may be negative. We denote the critical value of τ at which $\frac{\partial \lambda_{cycle}}{\partial g_2}$ transitions between negative and positive as τ_c. By setting equation (A.43) equal to zero and solving we find:

$$\tau_c = \frac{x\lambda_2 \frac{\mathrm{d}u_{11}}{\mathrm{d}g_2} - y\lambda_2 \frac{\mathrm{d}u_{12}}{\mathrm{d}g_2} + z(u_{11} - u_{12})\frac{\mathrm{d}\lambda_2}{\mathrm{d}g_2}}{z(u_{11} - u_{12})\frac{\mathrm{d}\lambda_2}{\mathrm{d}g_2}}. \tag{A.46}$$

Next we simplify equation (A.43) by assuming that disturbance-induced germination rate is as high as possible ($g_1 = 1$, because increasing g_1 was found to increase λ_{cycle} above). We also set the germination rate in non-disturbance years to be high ($g_2 = 1$) to see if decreasing the germination rate from a high value will increase growth rate. Finally, we use the smallest relevant cycle length (where there is one disturbance and one non-disturbance year, $\tau = 2$) to find when $\frac{\partial \lambda_{cycle}}{\partial g_2}$ is negative for very small τ. Written in terms of τ_c, equation (A.43) then becomes:

$$\left.\frac{\partial \lambda_{cycle}}{\partial g_2}\right|_{g_1=1, g_2=1, \tau=2} = \frac{-(\tau_c - 2)c_1c_3{\lambda_2}^2(s_{A1}s_{S2}c_3 + s_{S1}c_1c_2)}{s_{S2}c_2\left((\tau_c - 2)c_1c_2c_4 + (c_2 + 2c_3)c_1\lambda_2 + c_3 s_{A2}^2\right)}, \tag{A.47}$$

which is simplified for presentation using the following positive quantities

(assuming $\lambda_2 > 1$):

$$c_1 = \lambda_2 - s_{A2}, \tag{A.48a}$$
$$c_2 = 2\lambda_2 - s_{A2}, \tag{A.48b}$$
$$c_3 = \lambda_2 - s_{S2}, \tag{A.48c}$$
$$c_4 = 2\lambda_2 - s_{S2}. \tag{A.48d}$$

Equation (A.47) is negative when $\tau_c > 2$ (assuming $\lambda_2 > 1$). Thus, as long as $\tau_c > 2$, $\frac{\partial \lambda_{cycle}}{\partial g_2}\big|_{g_1=1,g_2=1}$ will start out negative at $\tau = 2$, favouring conditional germination. As the cycle length increases, the sign of $\frac{\partial \lambda_{cycle}}{\partial g_2}$ will switch at τ_c and select against conditional germination at longer cycle lengths.

Next, we evaluate whether increasing the life-history parameters from the disturbance year (equation 11a) will increase or decrease τ_c by taking the derivative of τ_c with respect to b_1, s_{A1}, s_{S1} and g_1. More general expressions can be obtained (see supplementary *Mathematica* file, Wolfram Research Inc. 2010) but here we present the case where $g_1 = 1$ and $g_2 = 1$ to see if the region within which conditional germination strategies ($g_2 < 1$) are expected to evolve (τ_c) is increased or decreased by these parameters. For b_1 we find:

$$\left.\frac{\partial \tau_c}{\partial b_1}\right|_{g_1=1,g_2=1} = -\left(\frac{s_Y {\lambda_2}^{2\tau}(s_{S1}c_1c_2 + s_{A1}s_{S2}c_3)}{{\lambda_{cycle}}^2 c_1 {c_2}^2 c_3}\right), \tag{A.49}$$

which is negative (assuming that $\lambda_2 > 1$). Therefore, increasing the number of seeds produced in disturbance years will decrease τ_c and thus decrease the parameter space over which we expect conditional germination to be favoured. Similarly, for s_{A1} we find:

$$\left.\frac{\partial \tau_c}{\partial s_{A1}}\right|_{g_1=1,g_2=1} = -\left(\frac{{\lambda_2}^{2\tau+1}(s_{S1}c_1c_2 + s_{A1}s_{S2}c_3)((\tau_c-2)c_2c_3 + s_{A2}s_{S2} + 2c_1\lambda_2)}{s_{S2}{c_2}^2c_3{\lambda_{cycle}}^2(c_1c_2c_3(\tau_c-2) + s_{A2}^2c_3 + c_1\lambda_2(c_2+2c_3))}\right), \tag{A.50}$$

which is negative (assuming that $\tau_c > 2$ and $\lambda_2 > 1$). Thus, increasing the adult survival rates during disturbances also decreases the parameter space over which conditional germination is expected to evolve. In contrast, the

derivatives of τ_c with respect to g_1 and s_{S1} are positive:

$$\left.\frac{\partial \tau_c}{\partial g_1}\right|_{g_1=1,g_2=1} = \frac{s_{S1}{\lambda_2}^{2\tau-1}(b_1 s_{S2} s_Y(\lambda_2 + s_{S2} + c_1) + \lambda_2(2 s_{A1} s_{S2} + c_1(s_{A1} + s_{S1}))}{s_{S2}{\lambda_{cycle}}^2 {c_2}^2}, \tag{A.51}$$

$$\left.\frac{\partial \tau_c}{\partial s_{S1}}\right|_{g_1=1,g_2=1} = \frac{{\lambda_2}^{2\tau}(b_1 s_{S2} s_Y c_2 + s_{A1}\lambda_2(c_1 + s_{S2}))}{s_{S2}{\lambda_{cycle}}^2 {c_2}^2 c_3}. \tag{A.52}$$

Hence, increasing germination rate and seedling survival in disturbance years increases τ_c and therefore increases the parameter space (in terms of disturbance cycle length, τ) over which conditional germination strategies ($g_2 < 1$) have higher growth rates than maximal germination ($g_2 = 1$).

Note on the approximated cycle matrix, $\widetilde{\mathbf{T}}_{cycle}$

The above approximation is most accurate when there is a large difference between eigenvalues λ_1 and λ_2 and when the number of non-disturbance years ($\tau - 1$) is large. Care must thus be taken in interpreting the results when the cycle length is short, which is when conditional germination strategies tend to be favoured. Therefore, our approximation serves as a guide, but is not quantitatively accurate, in cases with short disturbance cycles. For example, when $g_1 = 1$, $b_1 = 0$ and $\tau = 2$, g_2 should have no effect on the long term growth rate because, in the disturbance year, all seeds germinate and no new seeds are produced, therefore there are no seeds in the seed bank in the subsequent year and g_2 cannot affect growth rate. In contrast, using our approximation, equation (A.43) can be negative at this point (see figure A.1).

Figure A.1 shows that $\frac{\partial \lambda_{cycle}}{\partial g_2}$ can have a different sign when using the approximated (equation A.39) vs full transition matrix (equation 11). For example, when $\tau = 4$, plants are able to germinate and produce seeds exactly twice between disturbances so increasing g_2 increases the long-term growth rate of the full system. In contrast, when τ is 3 or 5, increasing g_2 will reduce the seed bank and increase the number of adults that experience a disturbance before reproducing. Exploring the parameter space numerically indicates that τ_c is generally a good indicator of how short disturbance cycles must be in order for conditional germination to be favoured, but oscillations such as that observed in figure A.1 can cause some values of τ below (above) τ_c to select against (for) conditional germination.

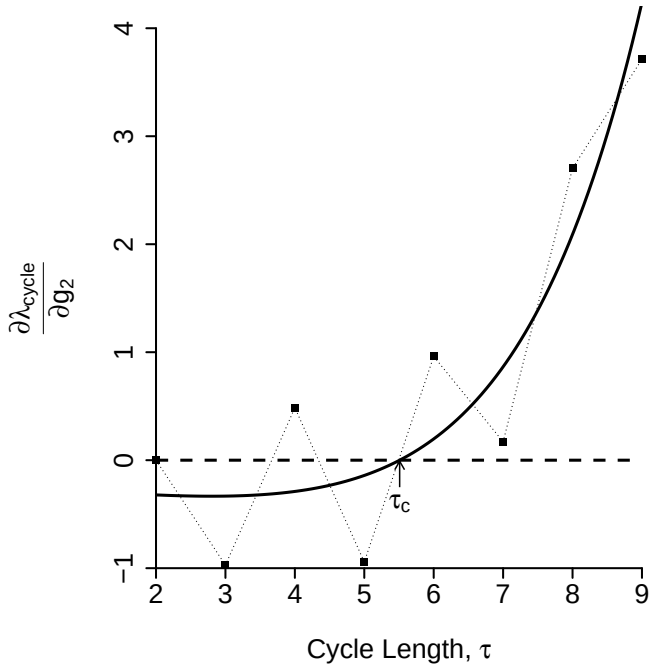

Figure A.1: An example of a discrepancy between the approximated transition matrix across a disturbance cycle ($\widetilde{\mathbf{T}}_{cycle}$) and the full matrix. The solid line shows the derivative of λ_{cycle} with respect to g_2 taken from equation (A.43) (using the approximation). The points (squares connected by a dotted line) show the same derivative where λ_{cycle} is calculated from equation (2.12) (unapproximated). Both derivatives are evaluated where $g_2 = 1$ and $g_1 = 1$. The other parameters used were $b_1 = 0$, $b_2 = 2$, $s_Y = 0.6$, $s_{S1} = s_{S2} = 0.8$, $s_{A2} = 0.7$ and $s_{A1} = 0$. τ_c is labelled with an arrow. Similar graphs may be explored numerically in the supplementary *Mathematica* file.

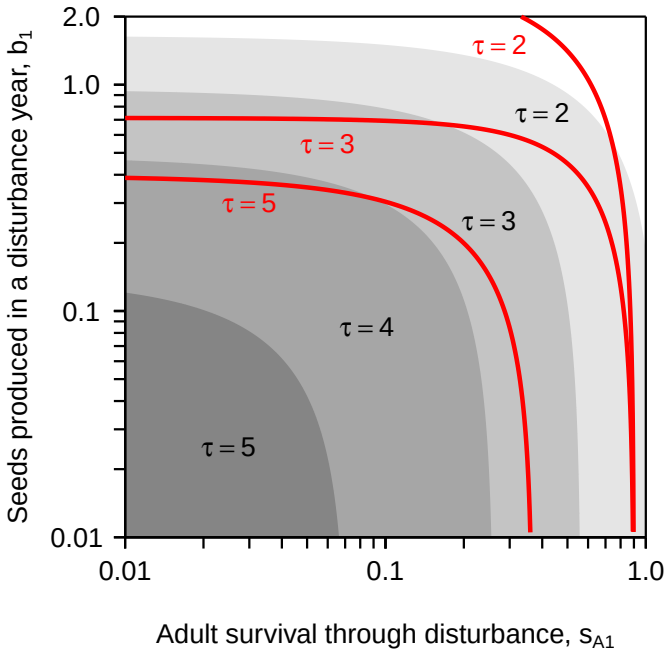

Figure A.2: A version of Figure 2.3 that is drawn using the non-approximated transition matrix $\mathbf{T}_{cycle}$. Labelled red lines enclose the parameters for which conditional germination is expected to evolve for various different cycle lengths ($\tau = 2$, , 3 and 5). This full model includes no region for which conditional germination evolves when disturbances occur every four years (no line for $\tau = 4$). Shaded areas represent the parameters for which conditional germination is expected to evolve in the approximated model (equation A.39), as shown in figure 3. Increasingly dark grey areas indicate where conditional germination is expected to evolve for cycle lengths of 2, 3, 4 and 5 (lighter regions overlap darker regions). Other parameters are $g_1 = 1$, $s_{Y2} = s_{Y1} = 0.6$, $s_{S1} = s_{S2} = 0.9$, $b_2 = 2$ and $s_{A2} = 0.7$.

Appendix B

Further Analysis Of Haploid-Diploid Life Cycle Evolution

We consider four models: two continuous selection models and two discrete selection models with mutations occurring at either meiosis or gamete production. We allow selfing to occur among gametes at rate σ, following Otto and Marks (1996). In the main text, we primarily discuss the continuous selection model with mutations at meiosis where $\sigma = 0$. We denote the genotypes MA, Ma, mA and ma by indices 1 to 4, the frequency of these

genotypes in the next generation x_1', x_2', x_3' and x_4') are given by

$$x_1' = (1-\mu)\big((1-\sigma)\big(x_1^2 w_{11,A} + x_1x_2w_{12,A} + x_1x_3w_{13,A} + x_1x_4w_{14,A} - rDw_{14,A}\big) + \sigma x_1 w_{11,A}\big)/\overline{W} \tag{B.1a}$$

$$x_2' = \big((1-\sigma)\big(x_2x_1w_{12,a} + x_2^2 w_{22,a} + x_2x_3w_{23,a} + x_2x_4w_{24,a} + rDw_{14,a}\big) + \sigma x_2 w_{22,a} + \mu\big((1-\sigma)\big(x_1^2 w_{11,A\mu} + x_1x_2w_{12,A\mu} + x_1x_3w_{13,A\mu} + x_1x_4w_{14,A\mu} - rDw_{14,A\mu}\big) + \sigma x_1 w_{11,A\mu}\big)\big)/\overline{W} \tag{B.1b}$$

$$x_3' = (1-\mu)\big((1-\sigma)\big(x_3x_1w_{13,A} + x_3x_2w_{23,A} + x_3^2 w_{33,A} + x_3x_4w_{34,A} - rDw_{14,A}\big) + \sigma x_3 w_{33,A}\big)/\overline{W} \tag{B.1c}$$

$$x_4' = \big((1-\sigma)\big(x_4x_1w_{14,a} + x_4x_2w_{24,a} + x_4x_3w_{34,a} + x_4^2 w_{44,a} + rDw_{14,a}\big) + \sigma x_4 w_{44,a} + \mu\big((1-\sigma)\big(x_3x_1w_{13,A\mu} + x_3x_2w_{23,A\mu} + x_3^2 w_{33,A\mu} + x_3x_4w_{34,A\mu} - rDw_{14,A\mu}\big) + \sigma x_3 w_{33,A\mu}\big)\big)/\overline{W} \tag{B.1d}$$

where $D = x_1x_4 - x_2x_3$ and $\overline{W}$ is the sum of the numerators. The notation $w_{ij,k}$ refers to the fitness of a zygote formed by gametes with indices i and j that produces a haploid of type k without mutation, $w_{ij,k\mu}$ is similar but where the k haploid produced by meiosis mutates. These fitnesses for the discrete and continuous selection models are given in table B.1. When mutations occur at gamete production, mutation does not affect fitness and $w_{ij,A\mu} = w_{ij,A}$. The fitness values where mutations occur at meiosis are given in table B.2.

We then calculate the frequency of the a allele ($\hat{q}_a$) when the modifier locus is fixed for a resident allele, M, which is given by

$$\hat{q}_a = \frac{\mu w_{11,A\mu}}{w_{11,A} - (1-\sigma)w_{12,a} - \sigma w_{22,a}}, \tag{B.2}$$

where we ignore terms on the order of μ^2. For the continuous selection model with mutations at meiosis and $\sigma = 0$, this is equivalent to equation

Table B.1: Fitnesses in discrete and continuous selection models

Fitness	Continuous selection	Discrete selection
$w_{11,A}$	$w_{AA}(t_{MM})w_A(t_{MM})$	$w_{AA}d_{MM} + w_A(1-d_{MM})$
$w_{12,A}$	$w_{Aa}(t_{MM})w_A(t_{MM})$	$w_{Aa}d_{MM} + w_A(1-d_{MM})$
$w_{12,a}$	$w_{Aa}(t_{MM})w_a(t_{MM})$	$w_{Aa}d_{MM} + w_a(1-d_{MM})$
$w_{13,A}$	$w_{AA}(t_{Mm})w_A(t_{Mm})$	$w_{AA}d_{Mm} + w_A(1-d_{Mm})$
$w_{14,A} = w_{23,A}$	$w_{Aa}(t_{Mm})w_A(t_{Mm})$	$w_{Aa}d_{Mm} + w_A(1-d_{Mm})$
$w_{14,a} = w_{23,a}$	$w_{Aa}(t_{Mm})w_a(t_{Mm})$	$w_{Aa}d_{Mm} + w_a(1-d_{Mm})$
$w_{22,a}$	$w_{aa}(t_{MM})w_a(t_{MM})$	$w_{aa}d_{MM} + w_a(1-d_{MM})$
$w_{24,a}$	$w_{aa}(t_{Mm})w_a(t_{Mm})$	$w_{aa}d_{Mm} + w_a(1-d_{Mm})$
$w_{33,A}$	$w_{AA}(t_{mm})w_A(t_{mm})$	$w_{AA}d_{mm} + w_A(1-d_{mm})$
$w_{34,A}$	$w_{Aa}(t_{mm})w_A(t_{mm})$	$w_{Aa}d_{mm} + w_A(1-d_{mm})$
$w_{34,a}$	$w_{Aa}(t_{mm})w_a(t_{mm})$	$w_{Aa}d_{mm} + w_a(1-d_{mm})$
$w_{44,a}$	$w_{aa}(t_{mm})w_a(t_{mm})$	$w_{aa}d_{mm} + w_a(1-d_{mm})$

Table B.2: Fitnesses of mutated types when mutations occur at meiosis

Fitness	Continuous selection	Discrete selection
$w_{11,A\mu}$	$w_{AA}(t_{MM})w_a(t_{MM})$	$w_{AA}d_{MM} + w_a(1-d_{MM})$
$w_{12,A\mu}$	$w_{Aa}(t_{MM})w_a(t_{MM})$	$w_{Aa}d_{MM} + w_a(1-d_{MM})$
$w_{13,A\mu}$	$w_{AA}(t_{Mm})w_a(t_{Mm})$	$w_{AA}d_{Mm} + w_a(1-d_{Mm})$
$w_{14,A\mu} = w_{23,A\mu}$	$w_{Aa}(t_{Mm})w_a(t_{Mm})$	$w_{Aa}d_{Mm} + w_a(1-d_{Mm})$
$w_{33,A\mu}$	$w_{AA}(t_{mm})w_a(t_{mm})$	$w_{AA}d_{mm} + w_a(1-d_{mm})$
$w_{34,A\mu}$	$w_{Aa}(t_{mm})w_a(t_{mm})$	$w_{Aa}d_{mm} + w_a(1-d_{mm})$

(3.1). As in the main text, we then evaluate the spread of a rare modifier using the leading eigenvalue (λ_l) of the system described by equations B.1c and B.1d. Full expressions of λ_l for each of the life cycles considered can be found in the supplementary *Mathematica* notebook.

In the models in which mutations occur at gamete production, and assuming that the fitnesses of A haploids and AA diploids are equal (such that $w_{11,A} = w_{13,A} = w_{33,A} = 1$), invasion occurs ($\lambda_l > 1$) if

$$\begin{aligned} 0 <& \sigma(w_{22,a} - w_{44,a})(w_{12,A} - w_{14,A}(1-r)) \\ &+ r(1-\sigma)(w_{12,A}w_{14,a} + w_{14,A}(w_{12,a} - 2w_{14,a}) \\ &+ (w_{12,A} - w_{14,A})(1 - w_{14,a}(1-\sigma) - w_{22,a}\sigma). \end{aligned} \tag{B.3}$$

Increased selfing can either increase or decrease the parameter range over

which this inequality is satisfied unless it is further assumed that the fitness of a haploids and aa diploids are equal (such that $w_{22,a} = w_{44,a}$ and the first term in B.3 is 0).

When the fitnesses of haploids and homozygous diploids are equal and mutations occur at gamete production, Otto and Marks (1996) showed that haploidy is always favoured over a larger parameter space when selfing is higher in the discrete selection model. Similarly, in the continuous selection model, where we also assume that modifiers have a small effect, $t_{Mm} - t_{MM} = \delta_{tMm}$ is of order μ, modifiers that increase the length of the haploid phase ($\delta_{tMm} > 0$) invade if

$$h(w_{AA}(t_{MM})w_A(t_{MM}) - (1-\sigma)w_{Aa}(t_{MM})w_a(t_{MM}) - \sigma w_{aa}(t_{MM})w_a(t_{MM})) > r(1-\sigma)(1-2h)w_a(t_{MM})w_{AA}(t_{MM}). \tag{B.4}$$

This condition is always met when $h > 1/2$ and is always satisfied for a greater parameter range with higher selfing rates (higher σ) if $h < 1/2$.

In the continuous selection model with mutations at meiosis, however, the impact of selfing is not so simple. Even when we assume the fitnesses of haploids and homozygous diploids is equal ($s_h = s_d$ and $\sigma_d = \sigma_h = 0$) and modifiers have a small effect ($t_{mm} - t_{MM} = \delta_{tmm}$ and $t_{Mm} - t_{MM} = h_m\delta_{tmm}$, where δ_{tmm} is of order μ and terms of $O(\mu^2)$ are discarded) and make the further assumption that recombination is free ($r = 1/2$), haploidy is favoured when

$$h > \frac{1-(1-h_m)(1-\sigma)(1+\sigma w_a(t_{MM})w_{Aa}(t_{MM})/K_1)}{2h_m}, \tag{B.5}$$

where $K_1 = w_{AA}(t_{MM})w_A(t_{MM}) - \sigma w_{aa}(t_{MM})w_a(t_{MM})$. For dominant modifiers ($h_m = 1$), this condition is satisfied if and only if $h > 1/2$, such that selfing has no effect on whether haploidy or diploidy is favoured. When $0 < h_m < 1$, increased selfing increases the right hand side of inequality (B.5). Therefore, increased selfing decreases, rather than increases, the parameter range under which haploidy is favoured. Although selfing can facilitate the evolution of haploidy when $r < 1/2$ (presumably because the impact of disequilibrium is greater), our overall finding is that when mutations occur at meiosis, selfing does not uniformly favour haploidy even when we assume that the fitness of haploids and homozygous diploids are equal.

In addition, the convergence properties of discrete and continuous selection models differ. For example, Hall (2000) found that, without selfing

or intrinsic fitness differences, haploid-diploid life cycles can evolve in the discrete selection model where mutations occur at meiosis. However, in the main text we show that haploid-diploid life cycles do not evolve in the continuous selection model where mutations occur at meiosis without intrinsic fitness differences. For the purposes of this study, one important distinction between models is whether haploid-diploid life cycles evolve for recessive deleterious mutations with selfing and loose linkage ($\sigma > 0$, $r = 1/2$). In figure B.1, we show a numerical example of life cycle evolution with selfing, loose linkage, and $s_d = s_h$. For these parameters, haploid-diploid life cycles evolve for low h in the discrete selection model but not in the continuous selection model (where mutations occur at gamete production in both cases). Thus in both the case considered by Hall (2000) (mutations at meiosis with no selfing) and in figure B.1 (mutations at gamete production with selfing), life-cycle models in which selection occurs continously (figure 3.1b) favour haploid-diploid life cycles less often than discrete life cycle models (figure 3.1a)

Finally, we clarify how selfing affects the disequilibrium between the M and A loci, which was discussed in Otto and Marks (1996). Using the same model and assumptions as Otto and Marks (1996), where $w_{AA} = w_A = 1$, $w_{Aa} = 1 - hs$, and $w_a = w_{aa} = 1 - s$ we find that the disequilibrium, $D = x_1x_4 - x_2x_3$ during invasion of a modifier is given by

$$D = \frac{(d_{Mm} - d_{mm})(1-h)\mu(1-\sigma)}{K_5(1 - d_{MM}(1-h)(1-\sigma))} \tag{B.6}$$

where $K_5 = r(1-\sigma) + s(1-d_{Mm})(1-h)(1-r) + hs(1-r)(1-\sigma) + \sigma s$ is strictly positive. Thus, disequilibrium has the same sign as $(d_{Mm} - d_{MM})$ and is positive for modifiers that increase the the diploid phase (modifiers associated with the less fit allele) and negative for modifiers that increase the haploid phase, as found by Otto and Marks (1996). However, the magnitude of this disequilibrium decreases with increasing selfing, contrary to the result stated in Otto and Marks (1996). In the supplementary *Mathematica* file we show that the magnitude of the disequilibrium increases with increasing selfing if $\hat{q}_a$ is held constant but because selfing also helps purging and reduces $\hat{q}_a$, the net effect on disequilibrium is opposite.

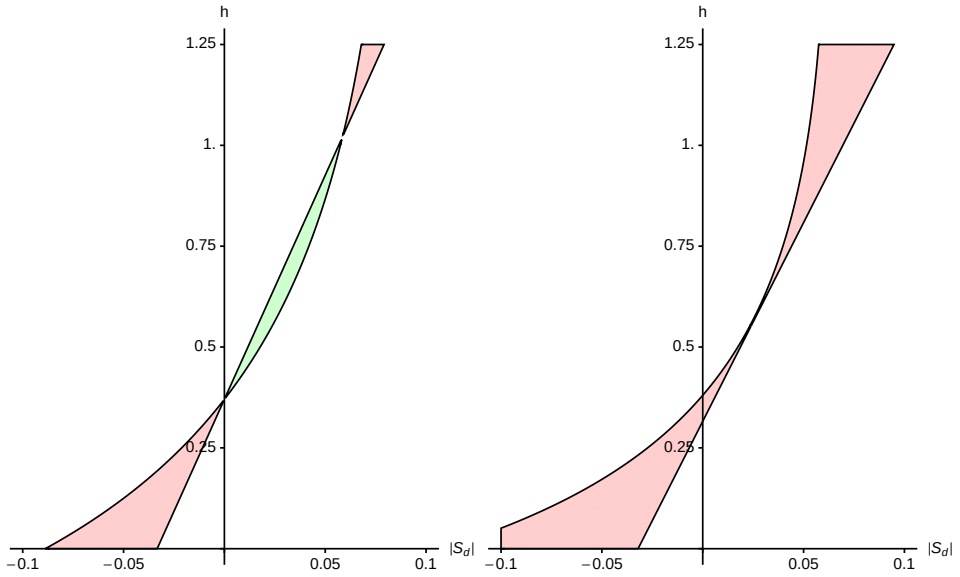

Figure B.1: Here we plot whether haplont, diplont, or haploid-diploid life cycles are favoured when there is selfing among gametes as a function of the intrinsic fitness of diploids (S_d) for (a) the discrete selection model with mutations at gamete production and (b) the continuous selection model with mutations at gamete production. To evaluate expected life cycle evolution we evaluated the stability of pure haplont ($d_{MM} = 0$, $t_{MM} = 1$) or diplont ($d_{MM} = 1$, $t_{MM} = 0$) strategies using equation (3.5) with the full expression of λ_l where terms on the order of μ^2 are discarded, which can be found in the supplementary *Mathematica* file. In both plots $\sigma = 0.4$, $r = 1/2$, $s_d = s_h = -0.3$, $U = 0.1$, $L = 1000$, $S_h = 0$, and modifiers have a small and dominant effect ($t_{mm} = t_{Mm}$, $|t_{Mm} - t_{MM}| = 1/10,000$, $d_{mm} = d_{Mm}$, $|d_{Mm} - d_{MM}| = 1/10,000$).

Appendix C

Evolution of Recombination Rate on Sex Chromosomes

C.1 Recursion Equations

In each generation we census the genotype frequencies in male and female haploids before haploid selection, e.g., sperm/pollen and eggs/ovules. Before haploid selection, the frequency of X-bearing male and female haploids are given by X_i^m and X_i^f and the frequency of Y-bearing haploids is given by Y_i^m where the index i specifies genotypes MA, Ma, mA, and ma. Selection then occurs among male haploids according to the **A** locus allele, k, carried by individuals with genotype i. Assuming that the fraction of X-bearing haploids produced by males is f, the genotype frequencies after haploid selection are $X_i^{m,s} = f w_k X_i^m / \bar{w}_H$ and $Y_i^{m,s} = (1-f) w_k Y_i^m / \bar{w}_H$, where $\bar{w}_H = \sum_{i=1}^4 f w_k X_i^m + (1-f) w_k Y_i^m$ is the mean fitness of male haploids. Random mating then occurs between gametes to produce diploid females with genotype ij at frequency $x_{ij} = X_i^f X_j^{m,s}$ and diploid males at frequency $y_{ij} = X_i^f Y_j^{m,s}$. In females, individuals with genotype ij are equivalent to those with genotype ji. For simplicity we denote the frequency of genotype ij in females to the average of these frequencies, $x_{ij} = (X_i^f X_j^{m,s} + X_j^f X_i^{m,s})/2$. Note that the sex ratio before diploid selection depends both on the production of X-bearing haploids by fathers (f) and on haploid selection (w_k). However, f does not enter into any results, indicating that the main force driving recombination evolution is not to balance the current sex ratio.

Table C.1: Fitness of different genotypes.

Genotype	A	a	AA	Aa	aa
Fitness in males	w_A	w_a	w_{AA}^m	w_{Aa}^m	w_{aa}^m
Fitness in females	1	1	w_{AA}^f	w_{Aa}^f	w_{aa}^f

Table C.2: Marginal fitnesses of Y**A** and X**a** haplotypes

$\bar{w}^{m}_{YA}$	$= (w_A(p_{Xf}w^{f}_{AA} + (1-p_{Xf})w^{f}_{Aa}))$
$\bar{w}^{mat,m}_{Xa}$	$= p_{Ym}w_A w^{m}_{Aa} + (1-p_{Ym})w_a w^{m}_{aa}$
$\bar{w}^{pat,f}_{Xa}$	$= p_{Xf}w_a w^{f}_{Aa} + (1-p_{Xf})w_a w^{f}_{aa}$
$\bar{w}^{mat,f}_{Xa}$	$= p_{Xm}w_A w^{f}_{Aa} + (1-p_{Xm})w_a w^{f}_{aa}$

Selection among diploids then occurs according to the diploid genotype at the **A** locus, k, for an individual of type ij (see Table C.1). The diploid frequencies after selection are given by $x^s_{ij} = w^f_k x_{ij}/\bar{w}^f$ in females and $y^s_{ij} = w^m_k y_{ij}/\bar{w}^m$ in males, where $\bar{w}^f = \sum_{i=1}^4 \sum_{j=1}^4 w^f_k x_{ij}$ and $\bar{w}^m = \sum_{i=1}^4 \sum_{j=1}^4 w^m_k y_{ij}$ are the mean fitnesses of females and males, respectively. Finally, these diploids undergo meiosis to produce the next generation. The haplotype frequencies in the next generation of eggs/ovules is given by:

$$X^{f'}_{MA} = \left(\sum_{j=1}^{4} x^s_{1j}\right) - R_f(x^s_{14} - x^s_{23}) \tag{C.1a}$$

$$X^{f'}_{Ma} = \left(\sum_{j=1}^{4} x^s_{2j}\right) + R_f(x^s_{14} - x^s_{23}) \tag{C.1b}$$

$$X^{f'}_{mA} = \left(\sum_{j=1}^{4} x^s_{3j}\right) + R_f(x^s_{14} - x^s_{23}) \tag{C.1c}$$

$$X^{f'}_{ma} = \left(\sum_{j=1}^{4} x^s_{4j}\right) - R_f(x^s_{14} - x^s_{23}) \tag{C.1d}$$

which only involve the recombination rate between the **A** locus and the **M** locus in females (R_f). In males, recombination between the SDR and the **A** locus or the **M** also affects the frequencies of haplotypes produced. The frequency of haplotypes among X-bearing sperm/pollen (before haploid

selection) in the next generation are given by

$$\begin{aligned} X_{MA}^{m'} = &\left(\sum_{j=1}^{4} y_{1j}^{s}\right) - r_{MM}(y_{12}^{s} - y_{21}^{s}) \\ &- (R_m + r_{Mm} - 2\chi)(y_{13}^{s} - y_{31}^{s}) - (R_m + r_{Mm} - \chi)y_{14}^{s} \\ &+ (r_{Mm} - \chi)y_{41}^{s} + \chi\ y_{23}^{s} + (r_{Mm} - \chi)y_{32}^{s} \end{aligned} \tag{C.2a}$$

$$\begin{aligned} X_{Ma}^{m'} = &\left(\sum_{j=1}^{4} y_{2j}^{s}\right) - r_{MM}(y_{21}^{s} - y_{12}^{s}) \\ &- (R_m + r_{Mm} - 2\chi)(y_{24}^{s} - y_{42}^{s}) - (R_m + r_{Mm} - \chi)y_{23}^{s} \\ &+ (r_{Mm} - \chi)y_{32}^{s} + \chi\ y_{14}^{s} + (r_{Mm} - \chi)y_{41}^{s} \end{aligned} \tag{C.2b}$$

$$\begin{aligned} X_{mA}^{m'} = &\left(\sum_{j=1}^{4} y_{3j}^{s}\right) - r_{mm}(y_{34}^{s} - y_{43}^{s}) \\ &- (R_m + r_{Mm} - 2\chi)(y_{31}^{s} - y_{13}^{s}) - (R_m + r_{Mm} - \chi)y_{32}^{s} \\ &+ (r_{Mm} - \chi)y_{23}^{s} + \chi\ y_{41}^{s} + (r_{Mm} - \chi)y_{14}^{s} \end{aligned} \tag{C.2c}$$

$$\begin{aligned} X_{ma}^{m'} = &\left(\sum_{j=1}^{4} y_{4j}^{s}\right) - r_{mm}(y_{43}^{s} - y_{34}^{s}) \\ &- (R_m + r_{Mm} - 2\chi)(y_{42}^{s} - y_{24}^{s}) - (R_m + r_{Mm} - \chi)y_{41}^{s} \\ &+ (r_{Mm} - \chi)y_{14}^{s} + \chi\ y_{32}^{s} + (r_{Mm} - \chi)y_{23}^{s} \end{aligned} \tag{C.2d}$$

and the frequencies of Y-bearing sperm/pollen haplotypes (before haploid selection) are given by

$$\begin{aligned} Y_{MA}^{m'} = &\left(\sum_{j=1}^{4} y_{1j}^{s}\right) - r_{MM}(y_{21}^{s} - y_{12}^{s}) \\ &- (R_m + r_{Mm} - 2\chi)(y_{31}^{s} - y_{13}^{s}) - (R_m + r_{Mm} - \chi)y_{41}^{s} \\ &+ (r_{Mm} - \chi)y_{14}^{s} + \chi\ y_{32}^{s} + (r_{Mm} - \chi)y_{23}^{s} \end{aligned} \tag{C.3a}$$

$$\begin{aligned} Y_{Ma}^{m'} = &\left(\sum_{j=1}^{4} y_{2j}^{s}\right) - r_{MM}(y_{12}^{s} - y_{21}^{s}) \\ &- (R_m + r_{Mm} - 2\chi)(y_{42}^{s} - y_{24}^{s}) - (R_m + r_{Mm} - \chi)y_{32}^{s} \\ &+ (r_{Mm} - \chi)y_{23}^{s} + \chi\ y_{41}^{s} + (r_{Mm} - \chi)y_{14}^{s} \end{aligned} \tag{C.3b}$$

$$\begin{aligned} Y_{mA}^{m'} = &\left(\sum_{j=1}^{4} y_{3j}^{s}\right) - r_{mm}(y_{43}^{s} - y_{34}^{s}) \\ &- (R_m + r_{Mm} - 2\chi)(y_{13}^{s} - y_{31}^{s}) - (R_m + r_{Mm} - \chi)y_{23}^{s} \\ &+ (r_{Mm} - \chi)y_{32}^{s} + \chi\ y_{14}^{s} + (r_{Mm} - \chi)y_{41}^{s} \end{aligned} \tag{C.3c}$$

$$\begin{aligned} Y_{ma}^{m'} = &\left(\sum_{j=1}^{4} y_{4j}^{s}\right) - r_{mm}(y_{34}^{s} - y_{43}^{s}) \\ &- (R_m + r_{Mm} - 2\chi)(y_{24}^{s} - y_{42}^{s}) - (R_m + r_{Mm} - \chi)y_{14}^{s} \\ &+ (r_{Mm} - \chi)y_{41}^{s} + \chi\ y_{23}^{s} + (r_{Mm} - \chi)y_{32}^{s} \end{aligned} \tag{C.3d}$$

C.2 Invasion of Recombination Modifiers

Invasion of modifiers that create a strong linkage between the X and a allele is determined by the largest solution to the characteristic polynomial

$$\lambda_{Xa}{}^2 - \lambda_{Xa}\bar{w}_{Xa}^{mat,f}/\bar{w}^f - (\bar{w}_{Xa}^{pat,f}/\bar{w}^f)(\bar{w}_{Xa}^{mat,m}/\bar{w}^m) = 0. \tag{C.4}$$

This can be solved for λ_{Xa} if we assume that the selected locus is initially loosely linked to the SDR (r_{MM}) and that there are no sex differences in selection ($w_{ij}^m = w_{ij}^f = w_{ij}$). The equilibrium frequency of the A allele when maintained at a polymorphic equilibrium by selection is then

$$\hat{p}_{Xm} = \hat{p}_{Ym} = \hat{p}_{Xf} = \frac{2w_a w_{aa} - w_{Aa}(w_A + w_a)}{2\left(w_A(w_{AA} - w_{Aa}) + w_a(w_{aa} - w_{Aa})\right)}. \tag{C.5}$$

This equilibrium is valid and stable when

$$\begin{aligned} &w_{Aa}(w_A + w_a) > 2w_A w_{AA} \text{ and} \\ &w_{Aa}(w_A + w_a) > 2w_a w_{aa}. \end{aligned} \tag{C.6}$$

Therefore, a polymorphism can be maintained either if there is heterozygote advantage in diploids ($w_{Aa} > w_{aa}$ and $w_{Aa} > w_{AA}$) or if there is antagonistic selection between haploids and diploids (e.g., $w_A > w_a$ and $w_{aa} > w_{Aa} > w_{AA}$) or a combination of both (Immler et al. 2012).

After this equilibrium is reached, the invasion of a modifier that brings the A allele into linkage with the Y is given by

$$\lambda_{YA} = 1 + \frac{(w_A - w_a)w_{Aa}(w_A + w_a)(w_{Aa}(w_A + w_a) - 2w_{AA}w_A)}{(w_A + w_a)({w_{Aa}}^2(w_A + w_a)^2 - 4w_A w_{AA} w_a w_{aa})}, \tag{C.7}$$

where $\lambda_{YA} > 1$ indicates that the modifier increases in frequency. Given that a polymorphism at the **A** locus is initially stable (conditions C.6 are met) the sign of $\lambda_{YA} - 1$ depends on the sign of $w_A - w_a$. That is, modifiers that bring the allele favoured in haploids (e.g., A when $w_A > w_a$) into tight linkage with the Y will spread.

Similarly, condition (4.2) for the invasion of modifiers that bring the a allele into tight linkage with the X chromosome is satisfied if

$$\frac{(w_A - w_a)w_{Aa}(w_A + w_a)(w_{Aa}(w_A + w_a) - 2w_{AA}w_A)}{2(w_A + w_a)(w_{Aa}(w_A + w_a) - w_A w_{AA} - w_a w_{aa})} > 0, \tag{C.8}$$

which requires $w_A > w_a$, given that conditions (C.6) are met. These results indicate that recombination modifiers invade if they bring the X into tight linkage with the allele that is less fit during haploid selection, even without the weak selection assumptions in equation (4.4) and without sex differences in selection in the diploid phase.

In the main text and above, we consider the invasion of recombination suppressors that bring the a allele into tight linkage with the X when the **A** locus is initially loosely linked to the SDR ($r_{MM} = 1/2$) such that $\hat{p}_{Xm} = \hat{p}_{Ym}$. Here, we consider cases where $r_{MM} < 1/2$ and define the difference in the frequency of the A allele between X- and Y-bearing pollen/sperm as $\delta_{XY} = \hat{p}_{Ym} - \hat{p}_{Xm}$. We assume that selection is weak relative to recombination such that δ, δ_{XY}, and δ_H are all small (of order ϵ^2). Invasion is

then given by

$$\lambda'_{Xa} = \lambda_{Xa}\left(1 - (1 - 2\ r_{MM})(3 + 2w^f_{Aa}/\bar{w}^f)\right) + \frac{w^f_{Aa}\delta_{XY}}{3\bar{w}^f} \tag{C.9}$$

Under the conditions where $\lambda_{Xa} > 1$, we would expect that the a allele is associated with the X such that $\delta_{XY} < 0$. Thus, (C.9) indicates that selection in favour of modifiers that suppress recombination is less strong when $r_{MM} < 1/2$ ($\lambda'_{Xa} < \lambda_{Xa}$), in which case intralocus conflicts are initially partially resolved by reduced recombination.

C.3 Invasion of Modifiers that Increase Recombination from an Initially Low Level

We consider a population in which linkage is tight between the **A** locus and the SDR (r_{MM} is of order ϵ, where the M allele is initially fixed). Recombination has no effect if the **A** locus is fixed for one allele, we therefore focus on the five equilibria that maintain both A and a alleles, of which four are given to leading order by:

$$\begin{aligned}
(A)\quad & \hat{p}_{Ym} = 0,\ \hat{p}_{Xf} = \frac{\alpha}{\alpha+\beta},\ \hat{p}_{Xm} = \frac{w^m_{Aa}\alpha}{w^m_{Aa}\alpha + w^m_{aa}\beta}\\
(A')\quad & \hat{p}_{Ym} = 1,\ \hat{p}_{Xf} = 1 - \frac{\alpha'}{\alpha'+\beta'},\ \hat{p}_{Xm} = 1 - \frac{w^m_{Aa}\alpha'}{w^m_{Aa}\alpha' + w^m_{aa}\beta'}\\
(B)\quad & \hat{p}_{Ym} = 0,\ \hat{p}_{Xf} = 1,\ \hat{p}_{Xm} = 1\\
(B')\quad & \hat{p}_{Ym} = 1,\ \hat{p}_{Xf} = 0,\ \hat{p}_{Xm} = 0
\end{aligned}$$

$$\begin{aligned}
\alpha =& w^f_{Aa}(w^m_{aa}w_a + w^m_{Aa}w_A) - 2w^f_{aa}w^m_{aa}w_a\\
\alpha' =& w^f_{Aa}(w^m_{AA}w_A + w^m_{Aa}w_a) - 2w^f_{AA}w^m_{AA}w_A\\
\beta =& w^f_{Aa}(w^m_{aa}w_a + w^m_{Aa}w_A) - 2w^f_{AA}w^m_{Aa}w_A\\
\beta' =& w^f_{Aa}(w^m_{AA}w_A + w^m_{Aa}w_a) - 2w^f_{aa}w^m_{Aa}w_a
\end{aligned}$$

A fifth equilibrium (C) also exists where A is present at an intermediate frequency on the Y chromosome ($0 < \hat{p}_Y < 1$). However, equilibrium (C) is never locally stable when $r_{MM} \approx 0$ and is therefore not considered further. Thus, the Y can either be fixed for the a allele (equilibria A and B) or the A allele (equilibria A' and B'). The X chromosome can then either be polymorphic (equilibria A and A') or fixed for the alternative allele (equilibria

B and B'). Since equilibria (A) and (B) are equivalent to equilibria (A') and (B') with the labelling of A and a alleles interchanged, we discuss only equilibria (A') and (B'), in which the YA haplotype is favoured (as in the previous section), without loss of generality.

We next calculate when (A') and (B') are locally stable for $r_{MM} = 0$. According to the 'small parameter theory' (Karlin and McGregor 1972*a*;*b*), these stability properties are unaffected by small amounts of recombination between the SDR and **A** locus, although equilibrium frequencies may be slightly altered. For the A allele to be stably fixed on the Y requires that $\bar{w}^m_{YA} > \bar{w}^m_{Ya}$, where the marginal fitnesses of YA and Ya haplotypes are $\bar{w}^m_{YA}$ (as above) and $\bar{w}^m_{Ya} = w^m_{Aa}p_{Xf} + w^m_{aa}(1 - p_{Xf})$, respectively. Substituting $\hat{p}_{Xf}$ from above, fixation of the A allele on the Y requires that $\gamma_i > 0$ where $\gamma_{(A')} = w_A(w^m_{Aa}\alpha' + w^m_{AA}\beta') - w_a(w^m_{aa}\alpha' + w^m_{Aa}\beta')$ for equilibrium (A') and $\gamma_{(B')} = w^m_{Aa}w_A - w^m_{aa}w_a$ for equilibrium (B'). Stability of a polymorphism on the X chromosome (equilibrium A') further requires that $\alpha' > 0$ and $\beta' > 0$. Fixation of the a allele on the X (equilibrium B') is mutually exclusive with (A') and requires that $\beta' < 0$. We will assume that these conditions are met such that population has reached a stable equilibrium at the **A** locus when considering evolution at the modifier locus.

To consider recombination rate evolution, we evaluate whether a mutant allele, m, can invade if it modifies the recombination rate between **A** and the SDR by a small amount ($|r_{mm} - r_{MM}|$ and $|r_{Mm} - r_{MM}|$ are of order ϵ). As above, we use the leading eigenvalue, λ, from a local stability analysis to evaluate the spread of a rare mutant modifier, where now λ_i determines invasion into a population at equilibrium i. Firstly, because stability of equilibrium (A') requires that $\alpha' > 0$ and $\beta' > 0$ and all fitnesses must be non-negative, we can define the following series of κ terms, which must be positive when (A') is locally stable.

$$
\begin{aligned}
\kappa_1 &= w^f_{aa}\alpha' + w^f_{Aa}\beta' \\
\kappa_2 &= w^f_{Aa}\alpha' + w^f_{AA}\beta' \\
\kappa_3 &= w^m_{Aa}\alpha' + w^m_{AA}\beta' \\
\kappa_4 &= w^f_{aa}\alpha' + w^f_{AA}\beta' \\
\kappa_5 &= w^m_{Aa}w_a + w^m_{AA}w_A \\
\kappa_6 &= w^m_{Aa}w_a w^m_{AA}w_A \\
\kappa_7 &= w^f_{aa}w^m_{Aa}w_a\alpha' + w^f_{AA}w^m_{AA}w_A\beta' \\
\kappa_8 &= w^m_{aa}\alpha'\alpha' + 2w^m_{Aa}\alpha'\beta' + w^m_{AA}\beta'\beta' \\
\kappa_9 &= w^m_{Aa}w_a\alpha' + w^m_{AA}w_A\beta' \\
\kappa_{10} &= w^f_{Aa}\kappa_9 + 2\kappa_6\kappa_4/\kappa_5
\end{aligned}
$$

These are useful in determining the magnitude of $\lambda_{(A')}$, which determines invasion of modifiers and is given by

$$\lambda_{(A')} = 1 + (r_{Mm} - r_{MM})\frac{w^m_{Aa}\alpha' K_1}{w_a R_m(w^m_{aa}\alpha' + w^m_{Aa}\beta')K_2} \tag{C.10}$$

where we neglect terms of order ϵ^2 and higher and K_2 is strictly positive,

$$
\begin{aligned}
K_2 =& R_f 2w^f_{Aa}\kappa_3\kappa_5(\alpha' + \beta')\kappa_{10} + R_f R_m w^m_{Aa}w^m_{AA}2w_a w_A K_3\kappa_3\kappa_4/\kappa_5 \\
&+ R_m w^m_{Aa}w^m_{AA}(1 - 2R_f)\big(w_a\beta'\kappa_1(2w^m_{AA}w_A\kappa_2 + \kappa_{10}) + w_A\alpha'\kappa_2(2w^m_{Aa}w_a\kappa_1 + \kappa_{10})\big)
\end{aligned}
$$

such that $\lambda_{(A')} > 1$ if and only if $(r_{Mm} - r_{MM})K_1 > 0$, where

$$
\begin{aligned}
K_1 =& -(1 - 2R_f)R_m\gamma_{(A')}\kappa_1\kappa_2\kappa_6 - R_m R_f\gamma_{(A')}\kappa_4\kappa_6\big(\kappa_7/\kappa_5 + w^f_{Aa}(\alpha' + \beta')/2\big) \\
&- R_f\gamma_{(A')}w^f_{Aa}w_a\kappa_1\kappa_3\kappa_5 \\
&+ R_f w^f_{Aa}w^m_{Aa}(\gamma_{(A')}\alpha' + R_m w_a\kappa_8)\big((w^m_{Aa} - w^m_{AA})w_a w_A\kappa_4 + (w_A - w_a)w^f_{Aa}\kappa_5(\alpha' + \beta')/2\big)
\end{aligned}
$$

Modifiers that increase recombination ($r_{Mm} - r_{MM} > 0$) therefore only spread if $K_1 > 0$. Only the last term of K_1 can be positive, and this term can only be positive if either $w^m_{Aa} > w^m_{AA}$ or $w_A > w_a$. Thus, for increased recombination to be favoured by selection ($K_1 > 0$), heterozygous males must be more fit that males homozygous for the allele fixed on the Y and/or the allele fixed on the Y must be favoured during haploid selection. Since the A allele is fixed on the Y, $w^m_{Aa} > w^m_{AA}$ implies that X chromosomes bearing

the a allele are favoured during selection in males. If a polymorphism is maintained on the X (equilibrium A'), counter-selection must favour the A allele during haploid selection and/or selection in females when $w^m_{Aa} > w^m_{AA}$. In addition, when linkage between the modifier locus and the selected locus is tight (at least in females, $R_f = 0$), K_1 is always negative and increased recombination is never favoured.

We next consider the invasion of a recombination modifier into a population at equilibrium (B'). Local stability of this equilibrium requires that $(-\beta') > 0$ and $\gamma_{(B')} > 0$. Ignoring terms of order ϵ^2 and higher,

$$\lambda_{(B')} = 1 + \frac{(r_{Mm} - r_{MM})K_4}{4(\gamma_{(B')} + R_m w^m_{aa} w_a)((-\beta') + w^f_{Aa}(R_f w^m_{Aa} w_a + R_m w^m_{AA} w_A(1 - R_f))}$$

where

$$\begin{aligned} K_4 = &- 2\gamma_{(B')}(-\beta') - (2R_f + R_m(1 - R_f))w^f_{Aa} w^m_{AA} w_A \gamma_{(B')} \\ &- R_m(-\beta')w^m_{aa} w_a \\ &+ R_f(w_A - w_a)w^f_{Aa} w^m_{Aa}(2\gamma_{(B')} + R_m w^m_{aa} w_a) \\ &+ R_f R_m(w^m_{Aa} - w^m_{AA})w^f_{Aa} w^m_{Aa} w_a w_A \end{aligned}$$

Therefore $\lambda_{(B')} > 1$ if and only if $(r_{Mm} - r_{MM})K_4 > 0$. The only terms in K_4 that can be positive again involve the factors $(w_A - w_a)$ and $(w^m_{Aa} - w^m_{AA})$, such that either $w^m_{Aa} > w^m_{AA}$ or $w_a > w_A$ are again necessary (but not sufficient) conditions for the invasion of modifiers that increase recombination.

Finally, we re-write the condition $K_4 > 0$ to obtain

$$w^f_{aa} < w^f_{Aa}\left(1 - \gamma_{(B')}R_f(2 - R_m)R_m) - \gamma_{(B')}(w^m_{Aa} - w^m_{AA})K_5 + (w_A - w_a)K_6\right)/K_7 \tag{C.11}$$

where the following terms are positive

$$\begin{aligned} K_5 =& (1 - R_f)(2\gamma_{(B')}(1 - R_m) + R_m w^m_{Aa} w_a)/w^m_{Aa} \\ K_6 =& (R_f R_m w_A {w^m_{Aa}}^2 + (w^m_{AA}(1 - R_f) + R_f w^m_{Aa})(2\gamma_{(B')}(1 - R_m) + w^m_{Aa} w_A R_m)) \\ K_7 =& 4\gamma_{(B')} + 2w^m_{aa} w_a R_m \end{aligned}$$

Thus, if haploid selection favours the A allele, then condition (C.11) can be met whether selection among diploid females favours allele A or a ($w^f_{aa} <$

w^f_{Aa} or $w^f_{aa} > w^f_{Aa}$). However, if haploid selection favours the a allele ($w_a > w_A$), the evolution of increased recombination requires that $w^m_{Aa} > w^m_{AA}$ (see above), and equation (C.11) shows that selection must favour the A allele during selection in females ($w^f_{aa} < w^f_{Aa}$). Thus, increased recombination is only favoured if the A allele is favoured during selection in females ($w^f_{aa} < w^f_{Aa}$) and/or the A allele is favoured during haploid selection ($w_A > w_a$). Only under these conditions is it possible for recombination between the XA and Ya to produce XA gametes that are favoured over the short term (in daughters and/or gametes/gametophytes, respectively).

One might not expect selection to favour XA haplotypes because an A allele on an average X background should either have the same fitness as an a allele (when a polymorphism is maintained, equilibrium A') or lower fitness (when A is fixed, equilibrium B'). However, an XA haplotype created by recombination in males is found in a male haploid (pollen or sperm), not on an average X background (which is weighted across X-bearing male sperm/pollen and female eggs/ovules). Increased recombination does not evolve if R_f and R_m are small because the modifier remains linked to the haplotypes it creates, which will eventually be found on all backgrounds. However, when R_f and R_m are sufficiently large, modifiers that increase recombination can gain a transient fitness advantage. XA pollen/sperm haplotypes can gain a transient fitness advantage during haploid selection and/or selection in females. The evolution of increased recombination is only consistent with this form of selection.

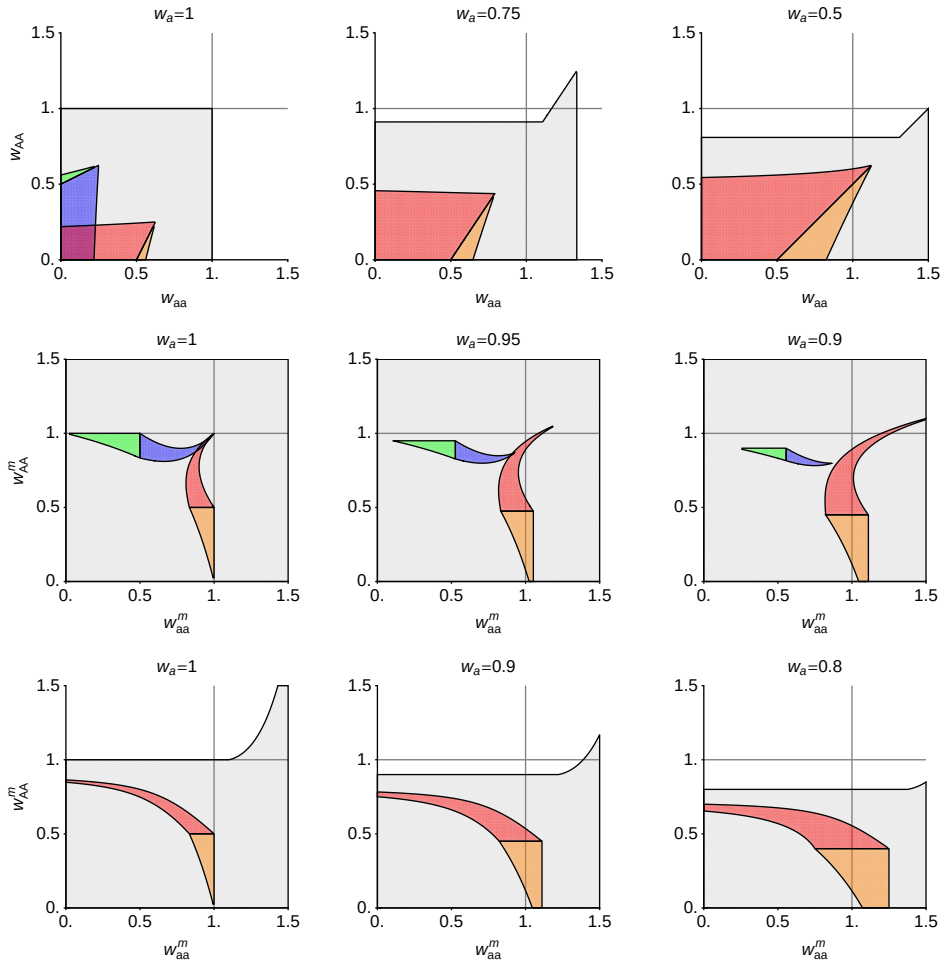
w_a=1
w_a=0.75
w_a=0.5
w_a=1
w_a=0.95
w_a=0.9
w_a=1
w_a=0.9
w_a=0.8
w_{AA}
w^m_{AA}
w_{aa}
w^m_{aa}
0.
0.5
1.
1.5

Figure C.1 *(preceding page)*: Selection can favour increased recombination between the sex-determining region (SDR) and a selected locus that is closely linked to the SDR ($r_{ij} \approx 0$), even when selection in males is not overdominant. The grey regions show where one or more of the polymorphic equilibria are stable and thus recombination modifiers can affect fitness. Coloured regions show where increased recombination is favoured in a population at equilibrium (A) in blue, (B) in green, (A') in red, and (B') in orange. Since this model is symmetrical, red/orange regions can be exchanged with blue/green regions if the labelling of A and a alleles is switched. Across columns we vary the fitness of a-bearing haploids relative to the A-bearing haploids ($w_A = 1$). Grey lines show the fitness of heterozygous diploids $w^k_{ij} = 1$. In the first row, there are no differences in selection between male and female diploids ($w^f_{ij} = w^m_{ij} = w_{ij}$), where w_{aa} and w_{AA} are varied along the x and y axes, respectively. As haploid selection becomes stronger, increased recombination can evolve with weaker overdominance in diploids and also with ploidally antagonistic selection ($w_{aa} > 1 > w_{AA}$). In the second and third rows, we consider sex differences in selection, where w^m_{aa} and w^m_{AA} are varied along the x and y axes ($w^m_{Aa} = 1$). In the second row, where selection in females is overdominant ($w^f_{AA} = 0.75$, $w^f_{Aa} = 1$, $w^f_{aa} = 0.75$), increased recombination can be favoured when selection is directional (or underdominant) in males and haploid selection is moderately strong. In the third row, selection favours the A allele in females ($w^f_{AA} = 1.05$, $w^f_{Aa} = 1$, $w^f_{aa} = 0.75$) and increased recombination can also be favoured with sexually antagonistic selection ($w^m_{AA} < 1 < w^m_{aa}$). For this plot, we assume that the modifier of recombination is unlinked ($R_f = R_m = 1/2$).