Evolution in structured populations

by

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Abstract

I study the evolution of altruistic behaviour in structured populations with overlapping generations. Two kinds of populations are considered: a simple patch structure and a spatially-explicit stepping-stone structure in both one and two dimensions. I use an inclusive fitness analysis which requires a calculation of the relatedness between interactants and knowledge of the dispersal rate. Dispersal is interpreted as an altruistic behaviour and for this reason I calculate and use the evolutionarily stable dispersal rate in the analysis of altruism.

Structured populations facilitate the evolution of altruism in conjunction with overlapping generations. In contrast, when generations are non-overlapping, altruism is favoured if and only if the direct benefit to the altruist is greater than the cost of altruistic act. In all cases, increasing the overlap between generations favours altruism. In a structured population, increasing survival probability affects the relatedness between nearby individuals, encouraging more altruistic behaviour.

Altruistic acts can either affect fecundity or survival probability. Altruism which affects fecundity is more favourable in a structured population than in an unstructured population, while acts which affect survival are less favoured than in an unstructured population. The difference between fecundity and survival effects can be understood by thinking of a survival benefit as a special class of extra offspring that have the appropriate probability of winning a site, but that do not disperse, so they must remain on their home site. This increases the local competition among offspring and reduces the advantage of altruism.

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Chapter 1

Introduction

A mathematical description of biological evolution must account for the following features of the theory of evolution. First, the genes in combination with environmental conditions control some of the physical, chemical, and behavioural traits of organisms. Second, individuals with different genes often have a different probability of surviving until they reproduce or may have different numbers of offspring. Third, the genes are transmitted from parents to offspring, although occasionally mutations can affect the transmission of genes to the next generation. The number of copies of the gene arising in the next generation from a single copy is referred to as the fitness of the gene. Individuals contribute, on average, offspring in proportion to their fitness (which depends on the fitness of their genes) and thus the abundance of genes in subsequent generations will change according to their effect on an organism's fitness.

Much of the work in mathematical evolutionary biology has examined the consequences of these general principles. The fitness of an individual often depends not only on its genes, but also on the genes of the organisms it interacts with. This is a form of frequency-dependent fitness and is the basis of the study of the evolution of social behaviours. Specific features of a biological system, such as mating patterns, the genetic system (haplo-diplontic, diplontic), and life-history traits can all have major influence on the mathematics used to analyse the evolution of specific genes. The abiotic context, such as temporal and spatial changes in biological and environmental variables will also have an impact on the evolution of specific traits. In this research, I'm particularly interested in the role of spatial patterns in the distribution of genes in an otherwise homogeneous environment and what influence that can have on the fitness of organisms and the evolution of certain traits, specifically altruistic behaviours.

1.1 The evolution of altruistic behaviours

An individual (the actor) is said to be behaving altruistically if it exhibits a costly behaviour which benefits others (the recipients). Sometimes a distinction is made between weak altruism in which the actor receives enough of the benefit to more than offset the cost of the act and strong altruism in which even if the actor receives a direct benefit, the net direct effect of the behaviour on the actor is costly (Wilson, 1979). I use the term altruism to describe strong altruism.

The evolution of strong altruism initially appears inexplicable: an action which reduces the ability of the actor to make copies of its genes, specifically the altruism gene, should not evolve. Nevertheless, altruistic interactions are ubiquitous, affecting all living things. Examples of altruistic behaviour include parental care of offspring, sterile insect workers who devote their lives to support the reproduction of a few members of their colony (Wilson, 1971), and animals which take turns acting as sentinel, forgoing foraging and mating opportunities while standing on guard (Bednekoff, 1997; Zahavi and Zahavi, 1997). Altruism is important on much smaller scales as well. Chromosomes require cooperation from their constituent genes, and eukaryotic cells contain symbiotic organelles (mitochondria and chloroplasts) with their own genetic information. The constituent parts reproduce as a whole, trading the disadvantage of slower reproduction for the benefits of being part of a larger structure (Szathmáry and Maynard Smith, 1995).

A standard model for the evolution of altruism permits two behaviours: act normally and receive a fitness of 1, or act altruistically at a cost of c and benefit your neighbours by increasing their total fitness by b. Hamilton's rule (Hamilton, 1964) tells us that the behaviour will be favoured if the cost of the altruistic act to the individual, c, is less than the benefits, b, conferred on others weighted by the average relatedness, R, of the recipients to the actor, (i.e. the probability the recipient shares with the actor an identical by descent copy of the gene for the altruistic act):

$$bR > c. \tag{1.1}$$

This simple description doesn't specify any structure in the population. In the interest of developing simple models, researchers have often avoided realistic spatial distribution of individuals (for an overview of non-spatial theories of cooperation see Dugatkin, 1997). In recent years many researchers have turned to computer simulations of evolutionary models with spatial structure. Simulation data are notoriously hard to draw conclusions from not only because of a lack of analysis of key parameters but also because simulations often involve many apparently innocuous assumptions and these vary from program to program, making the comparison of published results difficult.

Next, I describe the importance of spatial structure in evolutionary models, some convenient mathematical descriptions of population structure, and outline how I investigate the effect of population structure on the evolution of altruism.

1.2 Population spatial structure

In Nature, organisms with similar traits are often found together and the clustering of altruists within the population may facilitate the evolution of altruism (Hamilton, 1964; van Baalen and Rand, 1998). A low density of altruists spread uniformly throughout a population is unlikely to confer benefits on other altruists. Clustering means that even when altruists are rare, the probability an altruist interacts with another altruist can be high. Thus altruists can benefit from other altruists and decrease the probability that non-altruists take advantage of their behaviour. However, altruists surrounded by altruists may be competing among themselves for resources, and this means clustering can also work against altruists.

Experiments with two strains of *Escherichia coli* provide a demonstration of the importance of spatial dynamics to the evolution of social (in this case, allelopathic) behaviour (Iwasa *et al.*, 1998, and references therein). Two strains of *E. coli* were grown; one that produces colicin (a toxin) and one that is sensitive to colicin. In the absence of the colicin, the colicin-sensitive strain has a slightly larger growth rate. When the bacteria are grown in a well-mixed chemostat so that every bacterium experiences the same concentration of toxin, the outcome depends on the relative frequency of the two strains. If one strain dominates strongly in number, it will defeat the other species and in all cases one strain out-competes the other and eventually eliminates the slowly growing strain. In contrast, when these bacteria compete on an agar substrate which allows spatial structure in the colicin concentration to develop, qualitatively different results are found. In particular, if the cost of producing colicin is small enough, a few spiteful colicin-producing bacteria can invade a large population of colicin-sensitive bacteria, which was impossible in the chemostat (Chao and Levin, 1983; Wiener, 1996).

Non-spatial theoretical models and experiments in well-mixed chemostats agree. Both the colicin-production and colicin-sensitive strategies are evolutionarily stable (Durrett and Levin, 1997). If there is a large enough concentration of colicin-producers, the damage done to the colicin-sensitive bacteria will be greater than the cost of the production of the colicin and the colicin-sensitive strain will be driven to extinction. If the initial relative concentration of colicin-sensitive bacteria is sufficiently high, the colicin-producers have a small effect on the colicin-sensitive bacteria, and their slower growth rate drives them to extinction.

Iwasa *et al.* (1998) have analysed a spatial version of the allelopathy problem using the

pair-approximation method which describes spatial structure in a local, limited fashion. Their results agree qualitatively with the results from experiments in which the bacteria were grown on an agar substrate. Unlike the chemostat problem, conditions exist in which a few rare colicin-producers, concentrated in one region, can now invade a population of colicin-sensitive bacteria, if the cost of producing the toxin is sufficiently low. Similar results have been found with spatially structured simulations (Durrett and Levin, 1997).

Different models of structured populations in the literature give conflicting results, some identical to the unstructured population, and some suggesting structure favours the evolution of altruism (Wilson *et al.*, 1992; Taylor, 1992*a*,*b*; Nakamaru *et al.*, 1997, 1998; Iwasa *et al.*, 1998; Durrett and Levin, 1997). I work with three different kinds of populations: unstructured, a simple patch structure, and a spatially explicit stepping-stone structure (Figure 1.1).

Patch-structured populations

Patch-structured populations provide a simple description of spatial structure. A population is subdivided into local groups with a very simple concept of distance between patches: all patches are nearest-neighbours. An individual which leaves one patch arrives at any of the other patches with equal probability. The inclusive fitness calculations are straightforward and their simplicity means analytic results can often be obtained.

Stepping-stone populations

Unlike patch-structured populations, stepping-stone populations place patches (or sites) at regularly spaced lattice points. Altruistic interactions and local dispersal are only between nearest-neighbours giving the population a geographically rich structure. The name of the model evokes the correct image: for an influence of one patch to be felt by another, the effect must step from neighbour to neighbour until it reaches the remote site. In a one-dimensional population, relatedness between patches is not hard to calculate (Crow and Kimura, 1970; Taylor, 1994). In two or three dimensions relatedness calculations are more difficult, but relatednesses can be obtained analytically (Weiss and Kimura, 1965; Malécot, 1975; Irwin and Taylor, 2000*b*).

Many variations on these two structures are possible, including different kinds of metapopulation models (Hanski and Gilpin, 1997) and lattice populations with different mobilities (diffusion rates) for different strategies (Ferriere and Michod, 1996). Patch models are often used because they are easy to analyze, while lattice models are often studied with computer simulations because of their complexity, although I am able to find exact analytic results for both kinds of population structure.

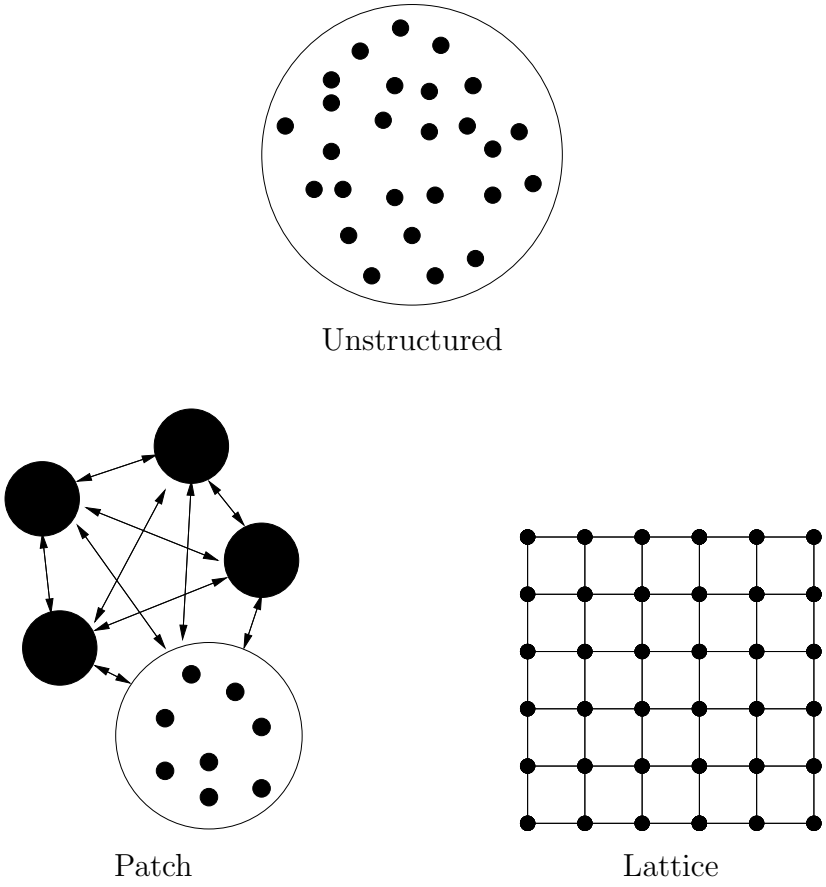

Figure 1.1: Three kinds of population structures. In an unstructured population each individual has an equal probability of interacting with any other individual, while in a structured population each individual distinguishes between neighbours and more distant individuals. I work with infinite populations similar to the finite populations shown.

1.2.1 Dispersal

A critical part of any model of a structured population is the description of the dispersal strategies of offspring. Dispersal enables successful individuals to 'export' their strategy to other patches and also reduces competition among relatives. On the negative side, altruist dispersal reduces the probability an altruist receives a benefit from another altruist. Some models have local dispersal creating correlation between altruists which favours altruism but incorporate a global mixing stage during the life cycle (Wilson *et al.*, 1992). This global mixing also favours altruism by reducing competition among relatives. I do not allow global mixing at any stage.

Offspring must make a choice: stay close to where they were born, or disperse and take their chances in a new environment. Dispersal carries many potential risks, including a cost of dispersing and the possibility of arriving in an inhospitable environment. I assume that the environment is of constant quality; there are no good and no bad sites to disperse to. I will also assume that dispersal is passive rather than active: dispersers select among the available target habitats randomly rather than making a selection based on quality or the strategy used by the occupants. In addition to the neighbourhoods and dispersal patterns, the type of population regulation must be specified to completely describe the population structure.

1.2.2 Population regulation

The competition between neighbours is influenced by the stage at which selection acts. In a small, growing population there may be very limited selection, but the density of individuals will rapidly increase. In an unstructured population, individuals which produce more offspring should have greater fitness if there is fair competition among all offspring. In a subdivided population, the situation is a bit more complicated.

In my structured population models, the next generation is formed in the following way. Each individual produces a large number of offspring and fecundity may be affected by altruistic interactions. In patch-structured populations, offspring disperse to a single 'dispersal pool' and the pool is spread uniformly over the patches. In stepping-stone populations, dispersal is restricted to nearest-neighbours. Note that the number of emigrants from a patch will in general be different from the number of immigrants to the same patch – because the average fecundity varies among patches, different patches contribute different numbers of offspring to the dispersal pool. Selection then reduces the number of offspring (native and immigrant) on a patch to the number of breeding spots available on the patch.

Other variations are possible. For example, Charlesworth (1979) and Rogers (1990) create patches in the next generation composed of a constant proportion of natives and the

remainder drawn from the migrant pool. This differs from my choice in which a constant proportion of offspring remain on the patch and the probability an adult is native to a patch varies with the frequency of altruists in the neighbourhood. Allowing altruists to contribute more to the dispersal pool creates more favourable conditions for the evolution of altruism (Kelly, 1992). Another variation allows patches with more altruists to support more adults. This is called an elastic population structure because the number of adults on a patch can be stretched by the effects of altruism. Mitteldorf and Wilson (2000) suggest that this change will facilitate the evolution of altruism. This aspect is not examined in this thesis, but I plan to investigate it in the future (see section 5.2.1).

1.2.3 The role of overlapping generations

In many theoretical studies, generations are assumed to be discrete and non-overlapping. This simplifies model analysis significantly, and may be a good description of many biological situations (e.g. annual plants, semelparous animals). Models studied with computer simulations can easily incorporate overlapping generations, but since these models often differ in many respects, it is difficult to identify the effect of overlapping generations.

In a simulation study, Olivieri *et al.* (1995) found that overlapping generations has an effect on the evolution of dispersal in a metapopulation model. An empirical study on annual and perennial species of Compositae shows a difference in seed dispersal structures and mean seed dispersal distances: annual plants tend to have shorter dispersal ranges than perennial species (Venable and Levin, 1983).

Discrete generations can be non-overlapping or overlapping. In a non-overlapping generation model, all individuals reproduce at the same time, the adults die and the offspring interact only with other individuals in the offspring generation. If generations overlap, then each individual has a probability of dying each generation, and if the population is at its carrying capacity, the only offspring which survive to breed are those born into the gaps left by a death.

In the models I study, the population structure has no effect on altruistic traits when generations are non-overlapping (Taylor, 1992*a*,*b*). Given the theoretical and empirical results mentioned above, introducing overlapping generations is a natural choice. One of my main results is that the differences commonly explained as an effect of population structure are in fact due to the combination of discrete overlapping generations and spatial structure.

In the next section, I describe the fundamental definitions and results necessary for my modeling approach. I begin by defining the idea of evolutionary stability and then describe genetic and inclusive fitness models.

1.3 One-locus genetic and inclusive fitness models of evolution

The study of the evolution of social behaviour has produced a rich set of modeling approaches. Complicated genetic models can frequently be replaced with analytically simpler and conceptually more powerful inclusive fitness models. In a genetic model, the fitness of an allele is the number of copies in the next generation produced by a single copy in the current generation. The relative fitness determines the frequencies of alleles in the next generation. An individual's inclusive fitness is an extension of this usual idea of fitness. Instead of just counting the number of descendents produced, the inclusive fitness also includes descendents produced by relatives weighted by their relatedness to the actor. For example, in diploid populations, offspring are formed by the combination of one gamete from each parent, and the relatedness between parent and offspring is $\frac{1}{2}$ since the offspring obtains half of its genes from each parent, on average. (This assumes there is no inbreeding — the parents are not related.) Similarly, identical twins have relatedness 1, sibs $\frac{1}{2}$, and half-sibs $\frac{1}{4}$. The gene for an act which benefits two of my sibs each an amount just greater than the cost to me should be favoured since there will be a net benefit to the gene. Below, I provide some definitions of evolutionary stability and describe genetic and inclusive fitness models.

Evolutionary stability

It's important to be able to identify stable populations, that is, configurations which will persist under the effects of selection and mutation. The usual approach is to determine if there are any monomorphic populations in which all individuals use the same strategy x^* which persist even when rare mutants are introduced into the population.

The fitness of an x mutant in a population with average strategy x_0 is $W(x, x_0)$. A strategy x^* is evolutionarily stable (is an ESS) if for all x,

$$W(x, (1-\epsilon)x^* + \epsilon x) < W(x^*, (1-\epsilon)x^* + \epsilon x) \tag{1.2}$$

for sufficiently small $\epsilon > 0$ and $x \neq x^*$. The condition requires that an x strategist has a lower fitness than the ESS x^* in the population, taking into account the fact that the average strategy is altered by the presence of the x strategists. If the fitness function W is linear in the population mean strategy, (1.2) is equivalent to the original definition of a global ESS x^*,

$$\text{either } W(x, x^*) < W(x^*, x^*) \tag{1.3}$$

$$\text{or } W(x, x^*) = W(x^*, x^*) \text{ and } W(x, x) < W(x^*, x) \tag{1.4}$$

for all x (Maynard Smith, 1982; Taylor, 1996). Condition (1.3) requires that all strategies $x \neq x^*$ have lower fitness than x^* in a population with average strategy x^*. The second half of the definition (1.4) says that if x is as good as x^* in an x^* population, then x^* must have greater fitness than x in an x population.

The usual way to find ESS candidates is to use the local equilibrium condition

$$\left.\frac{\partial W}{\partial x}\right|_{x=x^*} = 0 \tag{1.5}$$

and test them for stability with

$$\left.\frac{\partial^2 W}{\partial x^2}\right|_{x=x^*} < 0. \tag{1.6}$$

Another condition, convergence stability (CS), expresses the idea that in a monomorphic population away from the ESS, say at $\hat{x}$, a mutant which is closer to the ESS should have greater fitness. The expectation is that a population using $\hat{x}$ will be able to evolve towards x^* through a series of mutations. Formally, the condition requires that for $\hat{x} < x^*$,

$$\begin{aligned} W(x,\hat{x}) > W(\hat{x},\hat{x}) \quad & \text{if } \hat{x} < x < x^* \\ W(x,\hat{x}) < W(\hat{x},\hat{x}) \quad & \text{if } x < \hat{x} < x^* \end{aligned} \tag{1.7}$$

and similar conditions hold if $\hat{x} > x^*$. These conditions are usually checked with the local criterion

$$\frac{d}{dx_0}\left[\left.\frac{\partial W}{\partial x}\right|_{x=x_0}\right]_{x_0=x^*} = \left[\frac{\partial^2 W}{\partial x^2} + \frac{\partial^2 W}{\partial x \partial x_0}\right]_{x=x_0=x^*} < 0 \tag{1.8}$$

(Eshel and Motro, 1981; Christiansen, 1991; Taylor, 1996). This condition is checked in inclusive fitness analyses and is informally called the ESS condition.

Genetic models

Consider a population with fitness determined by a gene at one locus. A genetic model is an exact description of the effect of selection at the locus. Let the random variable g be the genotype of an individual and w is its fitness which may depend on the behaviour of each individual. The change in population average genotype in one generation is the difference in the expectation over the population of g in the next generation and the current generation. The expectation of g in the next generation can be calculated by weighting g in this generation by its contribution to the mean fitness $w/\bar{w}$. Thus, the change in average genotype can be written as

$$\begin{aligned} \Delta\bar{g} &= \frac{\overline{wg}}{\bar{w}} - \bar{g} \\ &= \frac{1}{\bar{w}}\operatorname{cov}(w,g). \end{aligned} \tag{1.9}$$

This is known as Price's covariance formula (Price, 1970). It is a very general expression: instead of the genotype of an individual, g can be the phenotypic value of a trait, or the average genotypic value of a group of individuals. Price's formula describes the change in population average genotype due to selection and thus can be used to study the evolution of a gene. Sometimes an extra term is included on the right hand side of (1.9) to describe changes in the average value of g not resulting from selection, such as meiotic drive and genetic drift (Queller, 1992; Frank, 1998).

Inclusive fitness models

The inclusive fitness method considers the effect of a small change in the behaviour of an individual (the actor) in a monomorphic population. This change will affect the actor's fitness and may also affect the fitness of others in the population (the recipients). The inclusive fitness effect is the sum of all these fitness changes weighted by the relatedness of each recipient to the actor.

I write x_i for the behaviour (phenotype) of the i^{th} individual, Δw_i for the change in fitness of the i^{th} individual due to the actor's change in behaviour, and r_i for the relatedness of the actor to individual i. The inclusive fitness effect is

$$\Delta w_{IF} = \sum_i \Delta w_i r_i. \tag{1.10}$$

The idea is that if $\Delta w_{IF} > 0$ the mutant behaviour will be selected.

The relatedness between a random actor and a random interacting partner in any one relatedness class (e.g. patchmate, sib) is defined as the covariance between actor genotype g and partner phenotype X divided by the covariance between actor genotype and phenotype x,

$$r = \frac{\text{cov}(g, X)}{\text{cov}(g, x)} \tag{1.11}$$

(Michod and Hamilton, 1980; Frank, 1998). If phenotype is linearly related to genotype, then relatedness can be calculated simply in terms of the genotype of the actor and partners (G),

$$r = \frac{\text{cov}(g, G)}{\text{cov}(g, g)}. \tag{1.12}$$

(Michod and Hamilton, 1980). If there are several different kinds of interactant (as is the case in a stepping-stone population), several different relatednesses will be needed.

Inclusive fitness models find the same equilibrium strategies as genetic models and the two approaches provide the same condition for convergence stability (1.8) assuming weak selection (mutations have small effects) and semi-dominance (Grafen, 1985; Taylor, 1996).

The problem with strong selection (mutations which have large effects) is that the mutant gene can alter its own distribution. In the case of a deviant dispersal or altruism gene this could affect the relatedness between neighbours. In inclusive fitness models, relatedness is calculated assuming neutral alleles. Incorporating selection into the relatedness calculation is much more difficult. The assumption that genetic effects are additive is important in diploids, but is automatically satisfied with weak selection under which a differential approximation can be used to linearize the fitness function.

1.4 Thesis outline

In this thesis I examine the evolution of social behaviour in structured populations with discrete overlapping generations using inclusive fitness methods. Altruistic individuals incur a cost and confer a benefit on neighbours. Other kinds of interactions with different effects on actor and recipients are easily incorporated into the framework. I will consider only models which affect either fecundity or survival, but allelopathic behaviour which costs the actor (reducing fecundity) and also hurts neighbours (reducing survival probability) could also be described. A brief discussion of the equivalence of these two effects is addressed in section 4.1.3.

The remainder of this thesis is organized as follows. In Chapter 2, the relatedness between pairs of individuals is calculated for both patch and stepping-stone structures with discrete overlapping generations. Chapter 3 computes the ES dispersal rate in the populations. Chapter 4 describes the analysis of altruistic behaviour and computes cost-benefit thresholds based on dispersal at the ES rate. Two kinds of altruistic interactions are considered: the simple cost-benefit example described above and the iterated prisoner's dilemma. Chapter 5 provides a brief summary of my results and a discussion of future research.

My research builds on a large literature and much of the original research described in this thesis is either accepted for publication or in review. The work on patch-structured populations extends the results in the literature by introducing overlapping generations and is reported in Taylor and Irwin (2000). This research was inspired by the overlapping generations work of Pen (2000) and much of the analysis was done by Taylor; I contributed to the analysis of dispersal (section 3.1) and the iterated prisoner's dilemma example (section 4.3). The stepping-stone population sections add a new treatment of overlapping generations including the calculation of some new relatedness coefficients. The relatedness calculations in Chapter 2 rely primarily on the method of Weiss and Kimura (1965) and also Kimura and Weiss (1964) and Malécot (1975). Weiss and Kimura were primarily interested in relatedness between distant relatives; I've added calculations of the relatedness of nearby individuals necessary for the inclusive fitness calculations in Chapters 3 and 4. Gandon and Rousset

(1999) consider the evolution of dispersal in patch and stepping-stone populations using the methods of Malécot (1975) but their main focus is on finite populations and they don't consider overlapping generations. Numerous authors have considered the evolution of altruism in stepping-stone populations, but none have an analytic treatment comparable to the inclusive fitness approach I provide here and in Irwin and Taylor (2000*a*) and Irwin and Taylor (2000*b*).

Chapter 2

Relatedness

My goal in this chapter is to compute relatedness coefficients needed in the analysis of dispersal and altruism in patch and stepping-stone structured populations with overlapping generations. The definition given in the previous chapter defines relatedness as a ratio of covariances (1.12). This is equivalent to a definition in terms of pedigree, that is, a ratio of coefficients of consangunity,

$$r_{xy} = \frac{g_{xy}}{g_{xx}}, \tag{2.1}$$

where x and y are the genotypes of a random actor and recipient from any single class of interactants, respectively. In the case of diploid individuals, an additional assumption of semi-dominance is necessary (Michod and Hamilton, 1980; Grafen, 1984; Taylor, 1996).

The coefficient of consangunity g_{xy} is the probability that an allele picked from each of x and y are identical by descent. The probability two individuals have identical by descent copies of the gene can be found with a one-generation recursion for the probability in the next generation in terms of the probability in the current generation and the life cycle dynamics (e.g. dispersal and survival probabilities). In the haploid populations considered in this thesis the calculation is quite straightforward. Note in particular that $g_{xx} = 1$ since a single allele must be identical to itself!

The calculation of relatedness in a patch-structured population is presented first, followed by the more complicated analysis for lattice-structured populations.

2.1 Patch structure

Consider an infinite asexual haploid population, distributed on patches with N individuals per patch. Each individual has a large number of (clonal) offspring who either disperse to another patch (with probability d) or remain on their native patch (with probability $1 - d$). I assume that dispersers incur a cost k, that is, only a fraction $1 - k$ of dispersing offspring

successfully find a new patch. The next generation is formed as follows. Each of the N original adults survives with probability s and in this case retains possession of its breeding spot. The offspring, native and immigrant, then compete on an equal basis for the vacated spots, with death to the offspring who do not win a spot, and the cycle begins again. The dispersal and cost parameters are both subsumed into the parameter

$$h = \frac{1-d}{1-kd} \tag{2.2}$$

which is the probability that a competing juvenile is native to the patch.

Let r denote the relatedness of a breeding individual to a random breeder on its patch including itself and $\hat{r}$ denote the relatedness of a breeding individual to another breeder on its patch. Then the relatedness r between two breeders on the same patch selected at random with replacement is

$$r = \frac{1}{N} + \left(\frac{N-1}{N}\right)\hat{r}. \tag{2.3}$$

The relatedness between any two individuals on different patches is 0. Now consider two different individuals breeding on the same patch (who therefore have relatedness $\hat{r}$) and ask for the relatedness of their "ancestors" exactly one generation ago. With probability s^2 they were fellow breeders who survived, and in that case they also had relatedness $\hat{r}$. With probability $2s(1-s)$ one of them is a survivor and the other is a new offspring and in that case the new offspring is native with probability h and is a clone of a fellow breeder of the survivor, giving a relatedness of r. Finally, with probability $(1-s)^2$ neither are survivors, and in this case they are both native with probability h^2, and then have relatedness r. This gives the recursion equation (a prime denotes a relatedness in the next generation):

$$\hat{r}' = s^2\hat{r} + 2s(1-s)hr + (1-s)^2h^2r. \tag{2.4}$$

Equations (2.3) and (2.4) can be solved at equilibrium ($\hat{r}' = \hat{r}$) to give

$$r = \frac{1+s}{N(1+s) - (N-1)(2s+h-sh)h}. \tag{2.5}$$

This patch-structured model with overlapping generations was introduced by Pen (2000) in a study of the fecundity-survival tradeoff at equilibrium.

2.2 Stepping-stone structure

Now consider an infinite asexual haploid population arranged in a regular lattice with 1 individual per site. Each individual has a large number of (clonal) offspring who either

disperse to a nearby site (with probability d) or remain on their native site (with probability $1-d$). I assume that dispersal incurs a cost k, that is, only a fraction $1-k$ of dispersing offspring successfully find a new site. The next generation is formed as follows. Each individual survives with probability s and in this case retains possession of its breeding spot. The offspring, native and immigrant, then compete on an equal basis for the vacated spots, with death to the offspring who do not win a spot, and the cycle begins again.

Initially I consider non-overlapping generations and dispersal with no cost. The effects of generation overlap and dispersal cost will be incorporated with a simple change of variables. This notational simplification was not possible in the patch-structured population because the patches had more than one individual per patch.

Relatedness between individuals on site i and site j, r'_{ij} in the next generation (denoted by a prime) is a weighted sum of relatednesses r_{kl} in the present generation with the weights determined by the dispersal pattern, dispersal cost, and survival of adults. For a general lattice structure with one individual per site the recursion is

$$r'_{ij} = \begin{cases} \sum_{kl} r_{kl} p_{ik} p_{jl} & \text{if } i \neq j \\ 1 & \text{if } i = j \end{cases} \tag{2.6}$$

where p_{ij} is the probability an adult on site i in the next generation is genetically identical to an adult on site j in the current generation. There are a variety of ways to describe the p_{ij} depending on the details of the specific model. The simplest description uses offspring dispersal probabilities; more detailed versions include dispersal costs and adult survival probability.

In the absence of any long-range dispersal or mutation the equilibrium relatednesses are all 1 on the one- and two-dimensional lattice, so I introduce as a technical device an additional dispersal rate μ which brings in unrelated individuals from 'infinity'. This can also be thought of as a mutation rate (Crow and Kimura, 1970, p. 267). I typically consider approximations to first order in $\sqrt{\mu}$ and in the analysis of dispersal and altruism I will be able to ignore terms $O(\mu)$. Without mutations or dispersal to infinity, the equilibrium relatednesses are all 1 because in the limit of an infinite number of generations, the whole population will be descended from a single ancestor. Mathematically the relatednesses are 1 because of the boundary condition that relatedness to self is 1 ($r_{ii} = 1$).

With non-overlapping generations ($s = 0$) and no-cost dispersal ($k = 0$) the p_{ij} are simply the probability a juvenile competing on site i was born on site j,

$$p_{ij} = \begin{cases} 1-d-\mu, & i = j \\ \dfrac{d}{N}, & i,j \text{ are nearest neighbours; } N \text{ is the number of nearest neighbors} \\ \mu, & i \text{ or } j = \infty \\ 0, & \text{otherwise.} \end{cases} \tag{2.7}$$

In a stepping-stone population, offspring either remain on their patch, or disperse to a nearest-neighbour. I assume there is no preferred direction for offspring to drift so that each neighbouring site has the same expected number of immigrants. The p_{ij} notation can be simplified to p_0, the probability an offspring is native, $p_1 = (1-p_0-p_\infty)/N$, the probability an offspring disperses to each of the N nearest-neighbours, and p_∞, the probability of dispersal to infinity.

Dispersal cost reduces the expected number of dispersing offspring which arrive at a new patch from d to $(1-k)d$. This also reduces the total number of competing offspring on a site. The probability an offspring is native is

$$\frac{1-d-\mu}{1-k(d+\mu)} \tag{2.8}$$

and with a survival probability of s the probability is

$$s + \frac{(1-s)(1-d-\mu)}{1-k(d+\mu)} = 1 - \frac{(d+\mu)(1-s)(1-k)}{1-k(d+\mu)}. \tag{2.9}$$

Thus the effects of dispersal cost and survival can be incorporated into (2.7) with the following substitutions,

$$\begin{aligned} d &\rightarrow \hat{d} = d(1-s)\left(\frac{1-k}{1-k(d+\mu)}\right) \\ \mu &\rightarrow \hat{\mu} = \mu(1-s)\left(\frac{1-k}{1-k(d+\mu)}\right) \end{aligned} \tag{2.10}$$

so $p_0 = 1-d-\mu$ becomes $1-\hat{d}-\hat{\mu}$ which is the same as (2.9). The distinction between relatedness between random patch-mates chosen with and without replacement which arose in section 2.1 is not necessary here because there is only one individual per site. With more than one individual per site the effect of survival can't be so easily incorporated into the p_i.

2.2.1 One dimension

Let r_j be the average relatedness of an individual to one j sites to the right in the one-dimensional lattice. I work with a homogeneous lattice and therefore relatedness depends only on the separation between individuals, not their absolute position. The subscript on r now denotes separation distance instead of identifying two sites. The recursion for the relatedness in the next generation r'_j in terms of relatedness in the current generation is

$$r'_j = \begin{cases} A(r_{j-2}+r_{j+2}) + B(r_{j-1}+r_{j+1}) + Cr_j + Dr_\infty, & j \neq 0 \\ 1, & j = 0 \\ 0, & j = \infty \end{cases} \tag{2.11}$$

where

$$\begin{aligned} A &= p_1 p_{-1} \\ B &= p_0 p_1 + p_0 p_{-1} \\ C &= p_0^2 + p_1^2 + p_{-1}^2 \\ D &= 2p_\infty(1-p_\infty) + p_\infty^2 \end{aligned}$$

and p_i is the probability a patch is won by an individual from a site i steps to the right. This equation displays the relatedness in the next generation as a weighted average of relatedness in the current generation. For example, the weight A is obtained by noticing that the distance between parents and their offspring can change by 2 in a generation only if offspring from each site disperse 1 step in opposite directions. The weight D is the probability individuals j sites apart are unrelated which can happen in two ways: if one comes from infinity and the other doesn't (with total probability $2p_\infty(1-p_\infty)$) or both come from infinity (p_∞^2). With no external pressures on dispersal, I expect symmetric dispersal and set $p_{-1} = p_1$.

Substituting $\hat{d}$ and $\hat{\mu}$ for d and μ in the p_i for non-overlapping generations and zero-cost dispersal obtains the general p_i if the modified dispersal rates are defined by (2.10).

A standard method to analyse (2.11) transforms the recursion into a linear system of four first-order difference equations (Kimura and Weiss, 1964; Taylor, 1994, see A.1 for details). Solving the recursion for the relatedness between neighbours and next-nearest neighbours yields

$$\begin{aligned} r_0 &= 1 \\ r_1 &= 1 - \Phi \\ r_2 &= 1 - 4\left(\frac{\sqrt{1-\hat{d}} - 1 + \hat{d}}{\hat{d}}\right)\Phi \\ r_3 &= 1 - \frac{16(1-\hat{d})\left(1 - \frac{\hat{d}}{2} - \sqrt{1-\hat{d}}\right) + \hat{d}^2}{\hat{d}^2}\Phi, \end{aligned} \tag{2.12}$$

where

$$\Phi = \sqrt{\frac{2\hat{\mu}}{\hat{d}(1-\hat{d})}} + O(\mu). \tag{2.13}$$

The reduction to a system of first-order difference equations can't be adapted to compute relatedness in two or more dimensions. Another approach, introduced by Weiss and Kimura (1965) can be used with a regular lattice of any dimension (see also Malécot (1975) for a summary of similar techniques for finite and infinite lattice-structured populations). I use this method to find the relatedness between individuals on nearby sites in a two-dimensional stepping-stone population. This method also provides another route to (2.12) and (2.13).

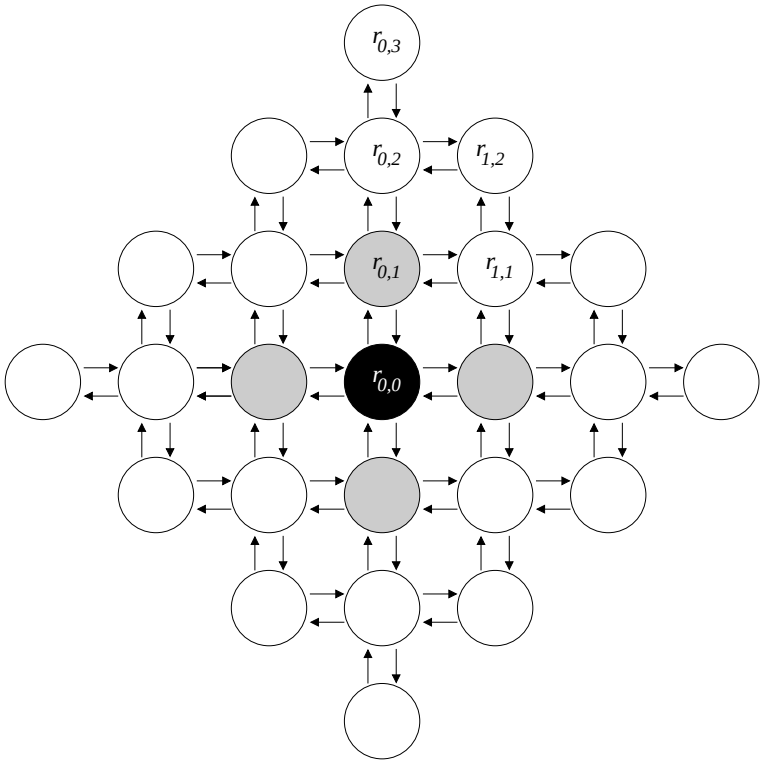

Figure 2.1: Local structure of the two dimensional stepping-stone population. The nearest neighbours of the centre (black) site are shown in grey. Sites are labeled with $r_{i,j}$, their relatedness to the centre site.

2.2.2 Two dimensions

Define $r_{j,k}$ to be the relatedness of a focal individual to an individual j sites away in the horizontal direction and k sites away in the vertical direction in the lattice. It's convenient to write these relatednesses in an infinite matrix $\mathbf{r} = \{r_{j,k}\}$ for all $j, k \in \mathbb{Z}$. Following the recursion above (2.11), I write a recursion for relatedness in the next generation in terms of relatedness in the present generation

$$\mathbf{r}' = \begin{cases} L^2\mathbf{r}, & (j,k) \neq (0,0) \\ 1, & (j,k) = (0,0). \end{cases} \tag{2.14}$$

where

$$\begin{aligned} L &= p_0 + p_1(S_1 + S_1^{-1} + S_2 + S_2^{-1}) \\ &= 1 - \hat{d} - \hat{\mu} + \frac{\hat{d}}{4}(S_1 + S_1^{-1} + S_2 + S_2^{-1}). \end{aligned} \tag{2.15}$$

The S_i are shift operators on the relatedness matrix which move all the entries left or down one step,

$$\begin{aligned} S_1\{r_{j,k}\} &= \{r_{j+1,k}\} \\ S_2\{r_{j,k}\} &= \{r_{j,k+1}\}. \end{aligned}$$

The operator L 'disperses' offspring with each term; the first term keeps a fraction $p_0 = 1 - \hat{d} - \hat{\mu}$ at the natal site, and the next four terms send $p_1 = \hat{d}/4$ to each of the nearest-neighbours. L^2 appears in (2.14) because I need a weighted sum of relatednesses for juveniles coming from any of the five sites for each juvenile (ignoring unrelated individuals dispersing from sites infinitely far away, see Figure 2.1). The recursion (2.14) holds for stepping-stone dispersal in a lattice of any dimension with an appropriate choice of L. For example, a recursion for one dimension can be obtained from (2.14) by defining $L = 1-\hat{d}-\hat{\mu}+\frac{\hat{d}}{2}(S+S^{-1})$ where S is the shift operator on an infinite vector of relatedness $\{r_j\}$.

The relatedness coefficients at equilibrium are now found by solving (2.14) at equilibrium. It is straightforward to verify that

$$L\{\cos(j\theta_1)\cos(k\theta_2)\} = H(\theta_1,\theta_2)\{\cos(j\theta_1)\cos(k\theta_2)\}, \tag{2.16}$$

where

$$H(\theta_1,\theta_2) = 1 - \hat{d} - \hat{\mu} + \frac{\hat{d}}{2}(\cos\theta_1 + \cos\theta_2). \tag{2.17}$$

This says that for each θ_1 and θ_2, L has an eigenvector $\{\cos(j\theta_1)\cos(k\theta_2)\}$ with eigenvalue $H(\theta_1,\theta_2)$. I introduce a function $F(\theta_1,\theta_2)$ which is a linear combination of these eigenvectors with the $r_{j,k}$ as weights,

$$F(\theta_1,\theta_2) = \sum_{j,k} r_{j,k}\cos(j\theta_1)\cos(k\theta_2). \tag{2.18}$$

The $r_{j,k}$ are the Fourier coefficients of the function F and can be recovered with the Fourier transform of F,

$$r_{j,k} = \frac{1}{(2\pi)^2}\int_0^{2\pi}\int_0^{2\pi} F(\theta_1,\theta_2)\cos(j\theta_1)\cos(k\theta_2)\; d\theta_1 d\theta_2. \tag{2.19}$$

Now, apply $(1-L^2)$ to (2.19)

$$(1-L^2)\mathbf{r} = \frac{1}{(2\pi)^2}\int_0^{2\pi}\int_0^{2\pi} F(\theta_1,\theta_2)(1-L^2)\{\cos(j\theta_1)\cos(k\theta_2)\}\; d\theta_1 d\theta_2 \tag{2.20}$$

and use the eigenvectors of L (2.16) to obtain

$$\big[(1-L^2)\mathbf{r}\big]_{j,k} = \frac{1}{(2\pi)^2}\int_0^{2\pi}\int_0^{2\pi} F(\theta_1,\theta_2)(1-H^2(\theta_1,\theta_2))\{\cos(j\theta_1)\cos(k\theta_2)\}\; d\theta_1 d\theta_2. \tag{2.21}$$

The left-hand side of (2.21) is the vector of Fourier coefficients of $F(1-H^2)$ and it follows from (2.14) and the equilibrium condition $\mathbf{r}' = \mathbf{r}$ that they are given by

$$\left[(1-L^2)\mathbf{r}\right]_{j,k} = \begin{cases} 0, & (j,k) \neq (0,0) \\ c, & (j,k) = (0,0) \end{cases} \tag{2.22}$$

where c is a constant to be determined later. Since all the Fourier coefficients of $F(1-H^2)$ are 0 except at $j = k = 0$,

$$F(\theta_1, \theta_2) = \frac{c}{1 - H^2(\theta_1, \theta_2)}. \tag{2.23}$$

Now combine (2.19) and (2.23) to obtain a formal solution to the recursion

$$r_{j,k} = \frac{c}{(2\pi)^2} \int_0^{2\pi} \int_0^{2\pi} \frac{\cos(j\theta_1)\cos(k\theta_2)}{1 - H^2(\theta_1, \theta_2)} \, d\theta_1 d\theta_2 \tag{2.24}$$

with the constant c determined by $r_{0,0} = 1$. The problem now is to evaluate these integrals.

In one dimension analogous arguments show

$$r_j = \frac{c}{4\pi} \int_0^{2\pi} \cos(j\theta) \left(\frac{1}{\hat{\mu} + \hat{d} - \hat{d}\cos\theta} + \frac{1}{2 - \hat{\mu} - \hat{d} + \hat{d}\cos\theta} \right) \, d\theta. \tag{2.25}$$

These integrals can be evaluated to obtain the results in (2.12).

In two dimensions the integrals must be simplified using various ingenious transformations. The necessary calculations appeared in Weiss and Kimura (1965) and Comins (1982) but I have adapted and extended them for this calculation.

First rewrite (2.24) using a partial fraction expansion,

$$\frac{1}{1-H^2} = \frac{1}{2}\left(\frac{1}{1-H} + \frac{1}{1+H} \right)$$

and write the two terms with a convenient notation,

$$r_{j,k} = c\left(A_{j,k}(z_1) + (-1)^{j+k} A_{j,k}(z_2) \right) \tag{2.26}$$

where

$$A_{j,k}(z) = \frac{1}{(2\pi)^2 \hat{d}} \int_0^{2\pi} \int_0^{2\pi} \frac{\cos(j\theta_1)\cos(k\theta_2)}{z - \cos\theta_1 - \cos\theta_2} \, d\theta_1 d\theta_2 \tag{2.27}$$

and

$$\begin{aligned} z_1 &= 2 + \frac{2\hat{\mu}}{\hat{d}} \\ z_2 &= 2\left(\frac{2 - \hat{\mu}}{\hat{d}} - 1 \right). \end{aligned} \tag{2.28}$$

The factor $(-1)^{j+k}$ comes from the substitution $\theta = \hat{\theta} + \pi$ which introduces negative signs in the denominator and a factor of $\cos(j\pi)\cos(k\pi) = (-1)^{j+k}$.

There are two cases which arise in the evaluation of $A_{j,k}(z)$. If $j = k$ then the integrals can be simplified using special functions eventually obtaining forms involving elliptic integrals. If $j \neq k$, use trigonometric identities to express the integrals in terms of $A_{j,j}(z)$.

On the diagonal $j = k$, transform the denominator using $\frac{1}{z} = \int_0^\infty e^{-zt}\, dt$ so that $A_{j,j}$ can be expressed using Bessel functions with imaginary argument and Legendre functions of the second kind,

$$A_{j,j}(z) = \frac{1}{\hat{d}} \int_0^\infty e^{-zt} I_j^2(t)\, dt = \frac{(-1)^n}{\hat{d}\pi i} Q_{j-1/2}\left(1 - \frac{z^2}{2}\right) \tag{2.29}$$

(Watson, 1958). This can be rewritten using the complete elliptic integrals of the first, $K(\cdot)$, and second, $E(\cdot)$, kinds

$$A_{0,0}(z) = \frac{2}{\pi z} K\left(\frac{2}{z}\right) \tag{2.30}$$

$$A_{1,1}(z) = \frac{1}{\pi}\left(z - \frac{2}{z}\right) K\left(\frac{2}{z}\right) - \frac{z}{\pi} E\left(\frac{2}{z}\right), \tag{2.31}$$

(Abramowitz and Stegun, 1964) and $A_{j,j}(z)$ for $j > 1$ can be evaluated recursively

$$Q_{n+1/2}(-z) = \frac{2z}{2n+1}\left[\left(n - \frac{1}{2}\right) Q_{n-3/2}(-z) - 2n Q_{n-1/2}(-z)\right]. \tag{2.32}$$

Off-diagonal elements are evaluated with algebraic manipulations and trigonometric identities. For example, the integrand in $A_{0,1}$ can be rewritten

$$\frac{\cos\theta_2}{z - \cos\theta_1 - \cos\theta_2} = -1 + \frac{z - \cos\theta_1}{z - \cos\theta_1 - \cos\theta_2}$$

so $A_{0,1}(z) = -\frac{1}{2} + \frac{z}{2} A_{0,0}(z)$. Similarly, $A_{0,2}$ can be rewritten using the identity $\cos 2\theta = 2\cos^2\theta - 1$ and the manipulation used for $A_{0,1}(z)$ giving

$$A_{0,2}(z) = 2z A_{0,1}(z) - 2A_{1,1}(z) - A_{0,0}(z). \tag{2.33}$$

If the mutant affects the fecundity of the nearest neighbours, two more relatedness coefficients are needed:

$$A_{1,2}(z) = z A_{1,1}(z) - A_{0,1}(z) \tag{2.34}$$

$$A_{0,3}(z) = 2z(A_{0,0}(z) + A_{0,2}(z)) - 2A_{1,2}(z) - 5A_{0,1}(z) - 2 \tag{2.35}$$

The arguments of the elliptic integrals are abbreviated as

$$\begin{aligned} a_1 = \frac{2}{z_1} &= \frac{\hat{d}}{\hat{d}+\hat{\mu}} \\ a_2 = \frac{2}{z_2} &= \frac{\hat{d}}{2-\hat{d}-\hat{\mu}}. \end{aligned} \tag{2.36}$$

Exact formulae for the first two relatedness coefficients needed in the dispersal analysis are

$$r_{0,1} = \frac{K(a_1) - K(a_2)}{a_1 K(a_1) + a_2 K(a_2)} \tag{2.37}$$

$$r_{1,1} = \frac{(z_1 - a_1)K(a_1) - z_1 E(a_1) + (z_2 - a_2)K(a_2) - z_2 E(a_2)}{a_1 K(a_1) + a_2 K(a_2)} \tag{2.38}$$

where a_i and z_i are in (2.28, 2.36). Other relatedness coefficients have more complicated formulae but can be readily computed as indicated.

2.3 Discussion of assumptions

I now have the relatedness coefficients necessary to proceed with the inclusive fitness analysis of dispersal and altruism in patch and stepping-stone structured populations with discrete overlapping generations. Before proceeding with those problems, I summarize some of the key assumptions of my approach.

First, I assume an infinite population. Finite population effects (e.g. genetic drift) are thus eliminated from the whole population, but finite-size effects are felt in local interaction groups. In fact, this is an important part of my structured population models — assuming an infinite population outside the local group not only simplifies the analysis, but makes the results much clearer by finding the right level of detail to be included in the model.

Second, I assume a monomorphic population with rare deviant mutant behaviours which have small effects on fitness. The 'small effects' assumption is essential to the analysis but is not overly restrictive since non-lethal, potentially beneficial, mutations are usually thought to have small effects. Although fitness functions are commonly non-linear, the small effects restriction linearizes the fitness function around the dominant strategy so that additive effects is a good approximation.

Third, relatedness coefficients are calculated with neutral alleles. Calculating relatedness with the deviant dispersal gene or altruistic effects turned on is much more difficult. Working with selection will change the distribution of alleles and relatednesses, but the 'neutral alleles' assumption is not a serious problem since I'm interested in the initial effect of a rare mutant invading a monomorphic population.

Fourth, generations are commonly taken to be discrete and non-overlapping. Thus each generation, adults reproduce, die, the offspring compete, and those which survive form the next generation of adults. Although this describes annual plants and some semelparous species (e.g. salmon) well, it does not seem appropriate for perennials and iteroparous animals in which individuals of different age classes interact. My simple approach is to allow some adults to survive into the next generation. I don't use a complicated age structure: all adults are reproductively active and have the same mortality. Another possible choice would be to introduce continuous generations, giving each individual an instantaneous mortality rate and eliminating simultaneous deaths. I discuss this briefly in a discussion of extensions to this research (section 5.2.2).

Finally, my analysis ignores potentially complicated spatial dynamics and considers only a static analysis of equilibria. Simulations of lattice populations show a variety of confusing and potentially conflicting results. There may be approximately periodic dynamic equilibria which permit the coexistence of several strategies or there may be a final winner determined by the initial frequency of the strategies (Nowak and May, 1992; Durrett and Levin, 1994, 1997). Additionally, for a given finite initial configuration of the spatial Prisoner's dilemma, whether one strategy comes to dominate the population is formally undecidable (Grim, 1997). Simulations on finite lattices may distort our view of might happen in an infinite population, or may provide meaningful insight into real finite populations. Gandhi *et al.* (1998) showed that the time for competitive exclusion to eliminate a strategy can be very long, and can be difficult to capture even in large simulations. Bistability, in which an initially dominant strain wins, may be theoretically impossible in an infinite population – with two strategies present on a lattice, one strategy should always eliminate the other (Iwasa *et al.*, 1998). I consider monomorphic populations with a mutant introduced infrequently and assume that the mutant will either be eliminated or come to dominate the population. The question of whether spatially heterogeneous polymorphisms persist in a spatially structured population is not studied here.

Chapter 3

Dispersal

Dispersal of offspring, especially in plants, is an important element of the life-history of a species determining local competiton, the rate at which species spread over the landscape, and gene-flow between sub-populations. The amount of dispersal is affected by traits such as seed-size (in plants) and dispersal distances and may vary with habitat type (isolated on islands, early-successional stages). A basic question is why offspring disperse at all, and what amount of dispersal is optimal.

Using a game-theoretic approach, Hamilton and May (1977) constructed the first model which showed that it was beneficial for a mother to disperse some of her offspring, even if they incur a high mortality cost. The benefit to dispersal is reduced competition for resources at home. They worked with a patch-structured model with one breeder per patch ($N = 1$) and no generation overlap ($s = 0$). This model was extended by Comins *et al.* (1980), Frank (1986) and Taylor (1988) to allow $N > 1$, and to encompass sexual reproduction and more general genetic systems. Many of the diverse approaches are reviewed in a study of the trade-off between seed-size and dispersal in plants (Levin and Muller-Landau, 2000). I extend these analyses further to allow overlapping generations, $s > 0$, and in addition to patch-structured models I consider stepping-stone populations with one individual per patch.

The dispersal problem is an interesting one, but I have an additional reason for my interest in dispersal. In the next chapter I study the evolution of altruism in spatially-structured populations. The altruism thresholds found depend on the probability an offspring is native to its patch and this depends on dispersal rate. Finally, it is worth pointing out that since dispersal of offspring reduces the level of competition for neighbours (relatives), dispersal can be regarded as altruistic, and the results of this chapter forshadow the analysis of altruism in the next.

The dispersal results in this chapter were first reported for patch-structure by Taylor and Irwin (2000) and for stepping-stone structure by Irwin and Taylor (2000*b*).

3.1 Patch structure

Consider the patch-structured population described in section 2.1. I measure fitness in terms of offspring (after dispersal) ready to compete for the spaces available in the next generation. Dispersal is under maternal control, although in a haploid model offspring have the same strategy as their parent. A mother who keeps an extra offspring at home loses $1-k$ offspring on a distant patch and gains 1 offspring at home. This extra offspring at home displaces another who will be a native with probability h and in this case will have average relatedness r to the mother.

The overall inclusive fitness effect is

$$\Delta w_{IF} = -(1-k) + 1 - hr = k - hr. \tag{3.1}$$

Increased dispersal will be favoured if this is negative which gives the condition

$$hr > k. \tag{3.2}$$

This suggests that dispersal is favoured if h is sufficiently large (which means that the dispersal rate d is sufficiently small) but it's not quite that simple, as r itself depends on h. However, since $1/r$ is a quadratic polynomial in h (2.5) this condition can be solved explicitly for h, and hence for d, and it then says that dispersal will be favoured if d is small enough, which gives a stable intermediate dispersal rate d^* (Frank, 1986; Taylor, 1988).

So far the argument is identical to the standard case of $s = 0$. The effect of breeder survival comes into play through the dependence of r on s (2.5). I find the equation for the evolutionarily stable (ES) dispersal rate and show analytically that this rate increases with s. From (3.2), the ES value of the parameter h is a solution to the equation

$$h^* r = k, \tag{3.3}$$

where $r = r(h^*, s)$ is the value of the relatedness coefficient at the ESS. Differentiate (3.3) with respect to s:

$$\frac{dh^*}{ds} r + h^* \left(\frac{\partial r}{\partial s} + \frac{\partial r}{\partial h} \frac{dh^*}{ds} \right) = 0 \tag{3.4}$$

where r and its partial derivatives are evaluated at h^*. Rearranging:

$$\frac{dh^*}{ds} \left(r + h^* \frac{\partial r}{\partial h} \right) = -h^* \frac{\partial r}{\partial s}. \tag{3.5}$$

I now show that both $\frac{\partial r}{\partial h}$ and $\frac{\partial r}{\partial s}$ are positive, and it follows that $\frac{dh^*}{ds}$ is negative. Calculating

derivatives of $1/r$ to simplify the calulations,

$$\frac{d}{dh}\left(\frac{1}{r}\right) = -\frac{N-1}{1+s}\left(2s+2h(1-s)\right) \tag{3.6}$$

$$\begin{aligned}\frac{d}{ds}\left(\frac{1}{r}\right) &= -(N-1)h\frac{d}{ds}\left(\frac{2s+(1-s)h}{1+s}\right) \\ &= -(N-1)h\left(\frac{(2-h)(1+s)-2s+(1-s)h}{(1+s)^2}\right) = -\frac{2(N-1)h(1-h)}{(1+s)^2}.\end{aligned} \tag{3.7}$$

Since h is a decreasing function of dispersal rate d (2.2) it follows that the ES dispersal rate d^* increases with s.

In fact, I can compute the ES dispersal rate d^* explicitly,

$$d^* = \frac{2k(N+s)+1+s-\sqrt{(1+s)^2+4k(N-1)(s^2(1-k)+s+Nk)}}{2k(N(1+k)-Ns(1-k)+2s)}. \tag{3.8}$$

This reduces to $d^*=1$ with no cost of dispersal ($k=0$) and to

$$d^* = \frac{1}{1+k} \tag{3.9}$$

for a patch with only a single ($N=1$) adult. Figure 3.1 shows the ES dispersal rate for patches with $N=5$ adults as a function of s for two different dispersal costs.

3.2 Stepping-stone structure

I now find the ES dispersal rate in a one- and two-dimensional stepping-stone population with discrete overlapping generations. A mutant adopts a deviant dispersal probability $d'=d+\delta$. The inclusive fitness effect of the mutant is the sum over the whole population of the fitness change due to the mutant for each individual weighted by its relatedness to the mutant:

$$\Delta w_{IF} = \sum_i \Delta w_i r_i. \tag{3.10}$$

The fitness of a breeder on site i is the expected number of its breeding descendents on all sites in the next generation. This is the sum of the probability of survival s and the expected number of offspring which obtain a breeding spot. If an adult on site i sends n_{ij} offspring to compete on site j, there will be $n_j=\sum_k n_{kj}$ juvenile competitors on site j. The probability an offspring from site i wins site j is the product of the probability the current occupant dies and n_{ij}/n_j. Thus, the fitness of the adult on site i is

$$w_i = s+(1-s)\sum_j \frac{n_{ij}}{n_j}. \tag{3.11}$$

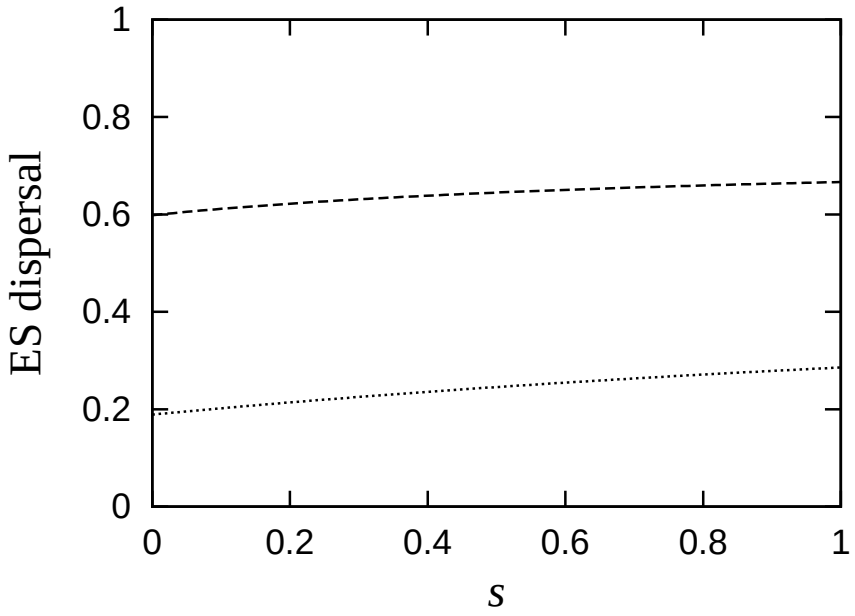

Figure 3.1: ES dispersal rate d^* in patch as a function of survival s with $N = 5$. The upper curve corresponds to low-cost dispersal ($k = 1/10$) and the lower curve $k = 1/2$. For no-cost dispersal ($k = 0$), $d^* = 1$.

The change in fitness due to mutant behaviour on site i is

$$\Delta w_i = w'_i - w_i = (1 - s) \sum_j \left(\frac{n'_{ij}}{n'_j} - \frac{n_{ij}}{n_j} \right). \tag{3.12}$$

3.2.1 One dimension

In a one-dimensional stepping-stone population, the non-zero n_{ij} are

$$n_{ii} = 1 - d - \mu \tag{3.13}$$

$$n_{i,i\pm1} = \frac{d}{2}(1 - k). \tag{3.14}$$

The mutant only affects dispersal away from the origin, so the only n'_{ij} which differ from n_{ij} in the previous generation are

$$n'_{00} = 1 - d' - \mu \tag{3.15}$$

$$n'_{0,\pm1} = \frac{d'}{2}(1 - k). \tag{3.16}$$

The mutant affects the fitness of individuals one and two steps away because it changes the number of competitors on its own site and sites one step away. The fitness changes are

obtained by substituting these n_{ij} into (3.12),

$$\begin{aligned}
\Delta w_0 &= (2-3d-kd)\frac{(1-k)(1-s)\delta}{2(1-kd)^2} \\
\Delta w_1 &= -(1-2d)\frac{(1-k)(1-s)\delta}{2(1-kd)^2} \\
\Delta w_2 &= \frac{-d(1-k)^2(1-s)\delta}{4(1-kd)^2}
\end{aligned} \tag{3.17}$$

(see section A.2 for more detail.) Selection is weak so I include only terms linear in δ and ignore terms $O(\mu)$ since I retained terms $O(\sqrt{\mu})$ in the relatednesses. The mutant gene is favoured if

$$\Delta w_{IF} = \Delta w_0 + 2\Delta w_1 r_1 + 2\Delta w_2 r_2 > 0 \tag{3.18}$$

using the relatedness from (2.12). The ES dispersal rate is

$$d^* = \frac{1}{2k} \cdot \frac{2(1-\sqrt{1-k(1-k)(1-s)}) - k(1-s)}{1-\sqrt{1-k(1-k)(1-s)} - k(1-s)}. \tag{3.19}$$

If dispersal has no cost (considering the limit $k \to 0$) then

$$d^* = \frac{3+s}{4}. \tag{3.20}$$

3.2.2 Two dimensions

In two dimensions, the method of calculation is the same, but more sites are affected by the mutant. A change in its dispersal rate affects the number of competitors on its site and on the nearest-neighbour sites. The fitness of individuals which disperse offspring to any of these 5 sites must be included in the inclusive fitness. In total, individuals on thirteen sites are affected, representing four distinct relatedness groups as shown in Figure 2.1. The change in fitness of individuals on these four groups of sites to first order in δ is

$$\begin{aligned}
\Delta w_{0,0} &= (4-5d-3dk)\frac{(1-k)(1-s)\delta}{4(1-kd)^2} \\
\Delta w_{0,1} &= -(1-2d)\frac{(1-k)(1-s)\delta}{4(1-kd)^2} \\
\Delta w_{1,1} &= -d\frac{(1-k)^2(1-s)\delta}{8(1-kd)^2} \\
\Delta w_{0,2} &= -d\frac{(1-k)^2(1-s)\delta}{16(1-kd)^2}.
\end{aligned} \tag{3.21}$$

The inclusive fitness effect is obtained by adding these together, weighted by their relatedness and the number of each different site type,

$$\Delta w_{IF} = \Delta w_{0,0} + 4\Delta w_{0,1} r_{0,1} + 4\Delta w_{1,1} r_{1,1} + 4\Delta w_{0,2} r_{0,2}. \tag{3.22}$$

The ES dispersal rate d^* is a solution of

$$2(2d^* sk - s - 1)K\left(\frac{(1-s)(1-k)d^*}{d^*(1-s+k+sk)-2}\right) + \pi(2 - d^*(1-s+k+sk)) = 0. \tag{3.23}$$

Figure 3.2 shows the ES dispersal rate d^* as a function of survival rate s for one- and two-dimensional stepping-stone populations. The ES dispersal rate for a $N = 1$ patch model (3.9) is also shown for comparison. The top panel is for zero-cost dispersal and the bottom panel shows the effect of a small cost of dispersal ($k = 1/10$), reducing the ES dispersal rate slightly.

3.3 Discussion

At the centre of the analysis of dispersal is a tension between local competition and the cost of dispersal. Offspring which remain on their natal patch are likely to compete with sibs, while those which pay the cost of dispersal are more likely to compete with more distant relatives.

The main result is that increasing the survival rate of reproductively active adults promotes greater dispersal rates. Olivieri *et al.* (1995) found an analogous result with a computer simulation of a metapopulation model. A similar pattern is seen in natural populations: annual plants tend to have shorter dispersal ranges than perennial species (Venable and Levin, 1983). This is a result of the effect of survival probability on the relatedness between neighbours. There is a technical difference between relatednesses in stepping-stone and patch-structured populations. In a patch-structured population, the relatedness between patch-mates increases with survival while an a stepping-stone population, the relatedness between neighbours decreases as survival increases. Increased dispersal in the patch structure is favoured because increasing survival increases the competition among relatives on the home patch. Another way to think about this is that survival tends to increase the between-patch genetic variance because the "mixing" effects of dispersal are reduced, even with the same offspring dispersal rate.

Two features create the differences between one- and two-dimensional stepping-stone models. The first and most obvious is that there are more interacting neighbours in two dimensions. Similar to increasing the patch size, this is expected to decrease relatedness between neighbours and favour increased dispersal and indeed my results show that the

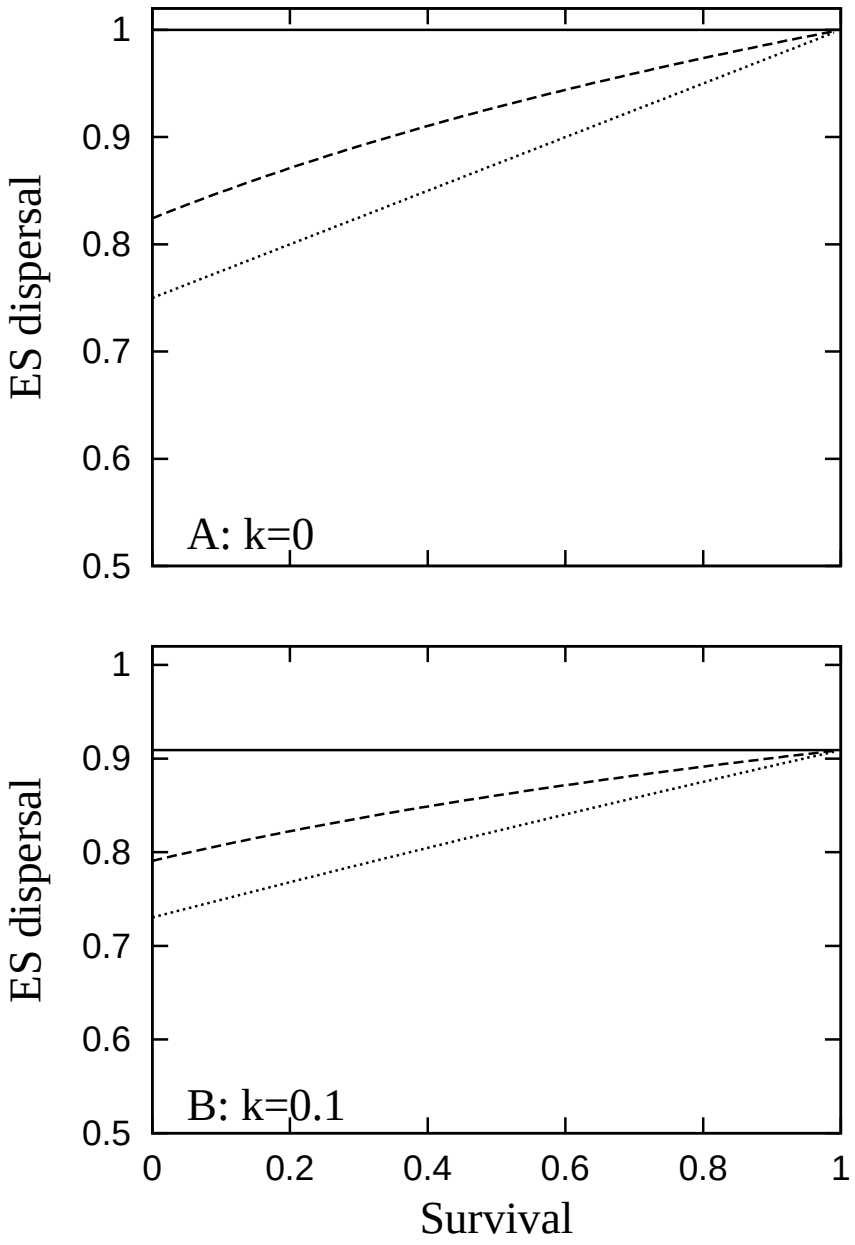

Figure 3.2: Evolutionarily stable dispersal rates. Starting from the top the lines correspond to the patch model ($N = 1$, solid), the 2-dimensional stepping-stone with four neighbours (dashed) and the 1-dimensional stepping-stone (dotted). Zero-cost dispersal (A) and $k = 1/10$ (B) are shown.

ES dispersal rate is greater in a two-dimensional stepping-stone population than in one dimension (Fig. 3.2). The second difference is that in two-dimensions there are pairs of sites separated by the same number of dispersal steps with different relatednesses. For example, among pairs of sites two steps apart there are two different relatednesses: one type is separated by two steps in a line (with relatedness $r_{0,2}$) and the other is separated by one step horizontally and one step vertically (with relatedness $r_{1,1}$, see Fig. 2.1). It's not clear what effect this will have on the relatedness or ES dispersal rate, but it makes the solution to the relatedness recursions (2.11, 2.14) more complicated in two dimensions than in one. This difference may appear to be rather artificial from a biological viewpoint, but even if there are not crisp examples of one- and two-dimensional lattices in real systems, this model identifies an important difference between 'narrow' (one-dimensional) and two-dimensional habitats.

Now consider the similarities between the stepping-stone populations and the patch-structured population with one individual per patch ($N = 1$). In a patch-structured population individuals can disperse from one patch to any other, but in a stepping-stone population dispersers can only arrive from neighbouring sites (ignoring the dispersal rate from infinity, $\mu \to 0$). In this sense, a patch population is the limit of a stepping-stone population as the number of neighbours goes to infinity and the relatedness between neighbours goes to zero. The ES dispersal rate in stepping-stone populations increases as the number of neighbours increases and the ES dispersal rate for the $N = 1$ patch population is greater than in either stepping-stone population (Fig. 3.2). An extra difference is that the ES dispersal rate is independent of survival probability s for the $N = 1$ patch population. This is understandable because the effect of s on relatedness vanishes when the relatedness between different individuals is 0.

In one dimension, I obtain an analytic expression for ES dispersal, including a remarkably simple result when there is no cost of dispersal (3.20). The two-dimensional problem is inherently more complicated, but working with standard special functions it is possible to compute exact numerical dispersal rates without requiring simulations. The two-dimensional stepping-stone population has obvious application to communities which live on a surface, but the one-dimensional lattice may be superior to the two-dimensional lattice for examining a population in an edge habitat (e.g. alpine or coastal).

Chapter 4

Altruism

An altruistic act confers a benefit on the recipients and costs the altruistic actor. This is an abstract way to describe many different possible biological interactions. This includes individuals in a colony taking turns on 'defensive watch' or genes cooperating with chromosomes by not replicating independently. As such, the evolution of altruistic behaviour is an important part of the evolution of complex biological entities (cells, discrete organisms, symbioses, and social groups).

The main result is that increasing the overlap between generations favours altruism. This is a consequence of the effect of increased survival on the relatedness between interactants and population structure. Specifically, I find the cost-benefit threshold as a function of survival for altruism to be favoured in the population structures introduced in Chapter 1. A second result is that overlapping generations, which are created by introducing survival between generations, means there is a difference between altruistic acts which affect fecundity and acts which affect survival. In structured populations, altruism which affects fecundity is generally more favourable and altruism which affects survival is less favoured compared with unstructured populations. Finally, in non-overlapping generations the net cost of the act to the actor and not the benefit on other recipients determines the fate of the altruism gene. This simple result vanishes with overlapping generations. These results are reported in Taylor and Irwin (2000) and Irwin and Taylor (2000a).

4.1 Patch Structure

I begin by considering the problem of the evolution of altruism in a patch-structured population with overlapping generations.

I assume that just prior to breeding, the N individuals interact at random on the breeding patch and there is the possibility for individual behaviour to be altruistic. Each altruistic

act incurs a cost c to the altruist and provides a total benefit b to the individuals on the patch. Thus, on average, each individual (including the altruist) gets benefit b/N from each altruistic act on the patch. These costs and benefits can represent changes in either fecundity or survival, and this leads to two different versions of the model. I assume that costs and benefits are additive and that selection is weak, that is, b and c are small compared to the baseline fitness. Our objective is to find conditions on b and c for which the altruistic behaviour is selectively favoured.

4.1.1 Fecundity effects

The inclusive fitness approach (Hamilton, 1964) counts all next generation individuals who are produced from or whose fitness is affected by an altruistic act, each one weighted by its relatedness to the actor. Suppose that an altruistic act creates b offspring (of the recipient) with average relatedness r to the altruist, and destroys c offspring (of the altruist) with relatedness 1. Now in case any of these $b-c$ extra offspring succeed in breeding, they will be found on a distant patch with probability $1-h$ and will in that case have no effect of the fitness of a relative. However with probability h they will be found on the natal patch and they will then displace $b-c$ individuals competing for vacant spots in the next generation, and these will be native to that patch with probability h and in this case will have average relatedness r to the actor. This gives an overall weighting of h^2r to each of these displaced individuals. The overall inclusive fitness effect is:

$$\begin{aligned} \Delta w_{IF} &= (1-s)(-c+br-h^2r(b-c)) \\ &= (1-s)\left(b(r-h^2r)-c(1-h^2r)\right). \end{aligned} \tag{4.1}$$

The altruistic trait will spread if $\Delta w_{IF} > 0$ which, using the relatedness (2.5), can be written (after some algebra)

$$\frac{b}{c} > N-(N-1)\frac{2hs}{(1+h)(1+s)}. \tag{4.2}$$

If the last term in the inequality is zero the condition reads

$$\frac{b}{c} > N \tag{4.3}$$

which is just the condition that the direct benefit b/N to the altruist exceed the cost c. Now there are three ways that this can happen if $N=1$ or $h=0$ or $s=0$. The first two are expected. In case $N=1$, the altruist has no one to interact with except itself and its inclusive fitness is $b-c$. And in case $h=0$ all offspring disperse, interactions are essentially at random in the whole population, and the only relative that the altruist interacts with is itself.

The third condition, $s = 0$, is unexpected and is interesting. This is the case of non-overlapping generations, and (4.2) says that in this case the benefit conferred on relatives is exactly balanced by the competitive effects of the extra offspring created. This is the result discovered by Wilson *et al.* (1992) in a simulation study of a lattice-structured population and verified analytically in a patch- and lattice-structured population by Taylor (1992*a*,*b*).

The significant finding of (4.2) is that if none of these conditions hold, that is, if $N > 1$, $h > 0$ (dispersal is incomplete) and $s > 0$ (some probability of breeder survival), then altruism can be favoured with a cost c that exceeds the direct benefit b/N to the altruist.

4.1.2 Survival effects

Here I suppose that an altruistic act confers an extra probability b (in total) of survival to random breeders on the patch (with relatedness r) and the altruist loses survival probability c. With net probability $b - c$, individuals will survive because of the act, and the offspring who are displaced from a vacant spot in these instances will be native with probability h and in this case will have average relatedness r to the actor. Thus each of these displaced individuals gets a weighting of hr. The overall inclusive fitness effect is:

$$\begin{aligned} \Delta w_{IF} &= s\left(-c + br - hr(b-c)\right) && (4.4)\\ &= s\left(b(r-hr) - c(1-hr)\right). && (4.5) \end{aligned}$$

The altruistic trait will spread if $\Delta w_{IF} > 0$ which, using the relatednesses (2.5), can be written

$$\frac{b}{c} > N + (N-1)\frac{h(1-s)}{(1+s)}. \qquad (4.6)$$

As above, when $N = 1$ or $h = 0$, the condition reduces to (4.3) and for altruism to be favoured we need the direct fitness effect on the altruist to be positive. This will also be true in the biologically unrealistic case that $s = 1$ (infinite lifetimes). But in all other cases, the last term in the inequality exceeds zero and a gift of survival is actually less beneficial to the actor than it would be in the complete mixing case ($h = 0$).

4.1.3 The fecundity/survival tradeoff for cost

According to the analysis above, extra fecundity displaces offspring at rate $h^2 r$ whereas extra survival displaces offspring at the larger rate hr. For altruism to have the best chance of succeeding, one might think that the benefit should be given in fecundity but the cost should be borne in survival, giving us the inclusive fitness:

$$\Delta w_{IF} = b(r - h^2 r) - c(1 - hr). \qquad (4.7)$$

However this argument fails. As long as the altruist has an evolutionarily stable balance between survival and fecundity, it makes no difference whether the cost is incurred in units of fecundity or survival. This follows from a recent analysis of Pen (2000) which I now summarize.

As shown above, a change of ΔS units of survival counts $\Delta S(1-hr)$ towards inclusive fitness and a change of ΔF units of fecundity counts $\Delta F(1-h^2r)$ towards inclusive fitness. At evolutionary equilibrium for survival-fecundity trade-off within the altruist, these should be equal, so that

$$\Delta S(1-hr) = \Delta F(1-h^2r). \tag{4.8}$$

Now in (4.7), the cost c is playing the role of ΔS let's call it c_S. If we want to convert it to a fecundity cost, c_F then (4.8) means I must replace $c_S(1-hr)$ by $c_F(1-h^2r)$ and that gives (4.1). So (4.7) is really equivalent to (4.1); they just provide two different ways of measuring cost.

There is one cryptic aspect of this argument, and that is a question of units. For the purpose of the above argument it is enough to note that for (4.7) to make sense, fecundity and survival must be measured in comparable units, and leave it at that. But for the sake of completeness, I will elaborate. The obvious choice of a common unit is probability of next generation occupancy. In this case, ΔS would be measured in straight units of survival (e.g. $\Delta S = 0.01$ would mean an extra 1% survival), but since only $1-s$ of the spots are available for offspring in each generation, ΔF will have to be relative numbers of offspring multiplied by $1-s$. For example, if survival is 75% then only 25% of the spots are available, and to get an increase 1% in occupancy ($\Delta F = 0.01$), I would have to increase my offspring number by 4%. Thus if I used f to denote fecundity measured in numbers of offspring (4.7) would be written

$$\Delta S(1-hr) = \frac{\Delta F}{f}(1-s)(1-h^2r). \tag{4.9}$$

and this is the notation of Pen (2000).

4.1.4 The effect of s on the threshold b/c

In both equations (4.2) and (4.6), the threshold value of the benefit/cost ratio decreases with increasing s. The conclusion is that for either a fecundity or a survival benefit, increased overlap between generations promotes an increased level of altruism.

But there is a subtlety here. When I say that the threshold in both equations decreases with s, I am supposing that h is being held constant. But h depends on the dispersal rate d, and the ES value of d is expected to depend on s. So as s increases, the ES value of h (which I call h^*) will also change, and it is not so clear whether the thresholds of equations (4.2) and (4.6) will still decrease.

In chapter 3, I used the ESS condition on h^* (3.3) to show that h^* decreases with increasing s and that the ES value of d^* increases with increasing s (3.8), assuming a constant cost k of dispersal (this is expected as dispersal is an altruistic behaviour). With this result I can easily analyze the threshold value of b/c for the case of survival benefit (4.6). Since h^* and $\dfrac{1-s}{1+s}$ both decrease with increasing s, the right hand side of (4.6) decreases with s and so therefore does the threshold value of b/c. The same result holds for the case of fecundity benefits (4.2) although this is not immediately clear as h^* and s change in opposite directions. The s derivative of b/c (from (4.2)) is

$$\frac{d}{ds}\left(\frac{b}{c}\right) = -2(N-1)\left(\frac{h^*(1+h^*)+s(1+s)\frac{dh^*}{ds}}{(1+h^*)^2(1+s)^2}\right). \tag{4.10}$$

This is negative because although the derivative of h^* with respect to s is negative,

$$-\frac{dh^*}{ds} = \frac{h^*}{1+s}\left(\frac{2h^*(1-h^*)}{\left(\frac{N}{N-1}\right)(1+s)+h^{*2}(1-s)}\right) < \frac{h^*(1+h^*)}{s(1+s)}, \tag{4.11}$$

it is small in magnitude.

The decreasing relationship between the threshold b/c and s is illustrated in Figure 4.1 for the parameter values $N = 5$, $k = 1/2$. The conclusion is that for both fecundity and survival benefits in a patch-structured population with overlapping-generations, altruism becomes more favoured as s increases.

4.2 Stepping-stone structure

I now consider populations in a one-dimensional lattice with two nearest-neighbour sites and a two-dimensional lattice with four nearest-neighbours. The lattice is infinite with one asexually reproducing haploid individual per site. Prior to reproduction, nearest-neighbours interact with the possibility of altruistic behaviour. Each individual produces a large number of offspring which disperse to one of the neighbouring sites with total probability d. Dispersers incur a cost and only a proportion $1-k$ arrive at a new site. The effect of d and k are combined into a single variable p_{j-i} the probability an offspring competing on site j was born on site i,

$$p_{j-i} = \begin{cases} \frac{1-d}{1-kd}, & i = j \\ \frac{d(1-k)}{N(1-kd)}, & i,j \text{ are nearest-neighbours; } N \text{ is the number of nearest-neighbors} \\ 0, & \text{otherwise.} \end{cases} \tag{4.12}$$

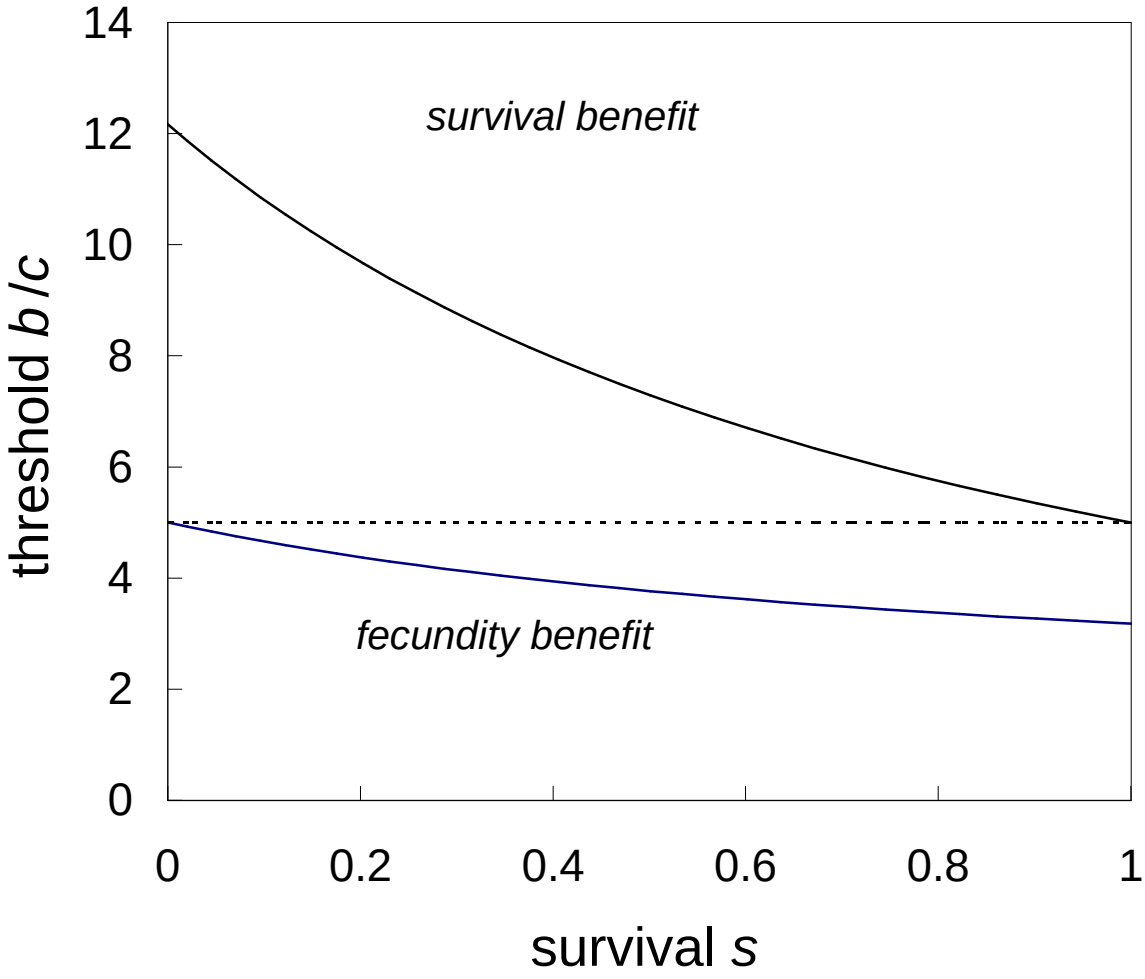

Figure 4.1: The threshold benefit-cost ratio b/c (above which altruism will be favoured) plotted against the probability s that a breeder will survive to reproduce in the next generation. The graphs plotted are for fecundity benefit (4.2) and for survival benefit (4.6). The parameter values are $N = 5$ breeders on a patch and cost $k = 1/2$ of dispersal, giving a probability h that a breeder is native of around 0.85. Observe that the random mixing model predicts a threshold b/c of $N = 5$, and this is the value obtained for $s = 0$ (fecundity benefit) and $s = 1$ (survival benefit). Observe also that both graphs decrease – greater survival promotes altruistic behaviour.

Table 4.1: Notation used in the main altruism model. In a homogeneous lattice, variables with two indices depend only on the separation between sites and are sometimes written to reflect this, e.g. $r_{j-i} = r_{ij}$. In two dimensions this is a vector difference and I index with these differences, e.g. $r_{0,\pm1} = r_{\pm1,0}$ is the relatedness of nearest-neighbours.

d	dispersal rate,
δ_i	effect of altruist at the origin on the fecundity or survival of an individual on site i,
k	dispersal cost; a fraction $1-k$ arrive at the new site,
N	number of neighbours, $N = 2$ in 1 dimension and $N = 4$ in 2,
n_{ij}	average number of offspring competing for site j who come from site i,
$n_j = \sum_i n_{ij}$	average number of offspring competing for site j,
$p_{ij} = n_{ij}/n_j$	probability an offspring competing for site j came from site i,
r_{ij}	equilibrium relatedness between an adult on site i and an adult at site j,
s	the probability an adult survives to breed in the next generation,
w_i, Δw_i	fitness (and change in fitness due to the mutant) of an adult on site i.

Thus p_0 is the probability an offspring is native. After reproduction, each adult survives and breeds again in the next generation with probability s. On a site in which the resident adult dies, the offspring, both native and immigrant, compete on an equal basis for the vacant spot. Offspring which do not win a site die and the cycle begins again. My notation is summarized in Table 4.1.

An altruistic act costs the actor and benefits its neighbours, affecting either fecundity or survival. I denote by δ_i the direct effect of an altruist at any site j on the breeding individual at site $j+i$. For example in the fecundity model, δ_i is the multiplicative change in the number of offspring. In a stepping-stone population δ_0 is the effect on the actor, δ_1 the effect on each nearest-neighbour, and all other δ_i are zero. These effects δ_i are not changes in fitness but they alter the fitness of individuals by an amount which depends on dispersal and survival rate. For both fecundity and survival effects, an individual's fitness is its expected number of offspring which breed in the next generation plus its probability of surviving to breed again.

An inclusive fitness calculation adds up the fitness effects of a mutant weighted by the relatedness between the affected individual and the actor. I denote the relatedness between adults at sites i and j by r_{ij}. The lattice is homogeneous so I often abbreviate this as r_{j-i} noting only the displacement between sites. Relatednesses for several pairs of nearby sites are computed using the lattice structure and stepping-stone dispersal pattern with no selection

(see chapter 2). I assume that the costs and benefits are small (weak selection), so the inclusive fitness results are exact to first order in δ_i and will give reasonable approximations for small δ_i (Taylor, 1996). I assume dispersal at the ES dispersal rate which depends on survival rate (see chapter 3).

In the following sections I find conditions on the δ_i which favour altruistic acts, considering first those which affect fecundity, then survival. Within each section I write a general expression for the inclusive fitness effect and find conditions in terms of the altruistic effects (δ_i) which select altruism in one- and two-dimensional stepping-stone populations. Finally, I adapt the general results to two examples of altruistic interactions: a simple cost-benefit altruism and the iterated prisoner's dilemma.

4.2.1 Fecundity effects

I now analyse the effect of an altruistic mutant at site 0. The altruistic behaviour alters the fecundity of the mutant and individuals on neighbouring sites. Since there is some dispersal from sites ± 1 to sites ± 2, there will be an effect on the number of competing juveniles at sites ± 2. Since some offspring born on sites ± 3 will disperse to sites ± 2, these offspring will experience a different amount of competition (see Fig. 2.1). Thus the inclusive fitness effect includes fitness changes of individuals up to three steps away.

The fitness of the adult on site i is the sum of 1 times the probability the adult survives and the probability its offspring out-compete other offspring on each site, $\sum_j p_{ij}$, times the probability the resident adult dies,

$$w_i = s + (1-s)\sum_j p_{ij}. \tag{4.13}$$

The number of individuals from site i which compete on site j once the mutant has been introduced n'_{ij} is the original number n_{ij} increased by a small relative change,

$$n'_{ij} = n_{ij}(1+\delta_i). \tag{4.14}$$

The average number of individuals competing for site j is $n_j = \sum_i n_{ij}$ and with the mutant this is

$$n'_j = \sum_i n'_{ij} = n_j\left(1 + \sum_i \delta_i p_{ij}\right). \tag{4.15}$$

The new probability of an individual from site i obtaining a vacant breeding spot on site j is p'_{ij}, written to first order in δ

$$p'_{ij} = \frac{n'_{ij}}{n'_j} = p_{ij}\left(1 + \delta_i - \sum_k \delta_k p_{kj}\right). \tag{4.16}$$

The fitness of the adult on site i with a mutant at site 0 is

$$w_i' = s + (1-s)\sum_j p_{ij}', \tag{4.17}$$

and together with (4.13) and (4.16) I obtain the inclusive fitness effect of the mutant

$$\Delta w_{IF} \;=\; \sum_i \Delta w_i r_i = (1-s)\sum_{ij} r_i p_{ij}\left(\delta_i - \sum_k \delta_k p_{kj}\right). \tag{4.18}$$

Symmetry allows the simplifications $p_{ij} = p_{j-i}$ and $p_{-j} = p_j$; the probability of dispersal to a site depends only on the separation between the two sites and not the absolute position of the sites. The inclusive fitness effect Δw_{IF} can be written as

$$\Delta w_{IF} = (1-s)\left(\sum_i \delta_i r_i - \sum_{ijk} \delta_k p_{j-i} p_{j-k} r_i\right). \tag{4.19}$$

If $s = 0$, the relatedness recursion permits a simplification of (4.19), obtaining

$$\Delta w_{IF} = \delta_0\left(r_0 - \sum_{ik} r_i p_k p_{k-i}\right). \tag{4.20}$$

(Taylor, 1992*b*). With non-overlapping generations the inclusive fitness effect is independent of the effect of the mutant on other breeders – none of the other $\delta_i, |i| \geq 1$ appear in (4.20). This is a general result for any dispersal pattern or range of altruistic interactions on a lattice of arbitrary dimension.

One dimension

In a one-dimensional stepping-stone population the inclusive fitness effect (4.19) simplifies to

$$\Delta w_{IF} \;=\; r_0\Delta w_0 + 2r_1\Delta w_1 + 2r_2\Delta w_2 + 2r_3\Delta w_3 \tag{4.21}$$

where the net fitness effects on breeders at a distance i, Δw_i, are

$$\begin{aligned}
\Delta w_0 &= (1-s)\left(\delta_0(1-p_0^2-2p_1^2) - \delta_1(4p_0p_1)\right)\\
\Delta w_1 &= (1-s)\left(-2\delta_0 p_0 p_1 + \delta_1(1-p_0^2-3p_1^2)\right)\\
\Delta w_2 &= -(1-s)\left(\delta_0 p_1^2 + 2\delta_1 p_0 p_1\right)\\
\Delta w_3 &= -(1-s)\delta_1 p_1^2.
\end{aligned}$$

If $s = 1$ these fitness effects are all 0 as expected. The altruistic behaviour is favoured if

$$2\delta_1 \left[\frac{r_1 - r_3 - p_0(4r_0 - 7r_1 + 4r_2 - r_3)}{2(r_0 - r_2 + p_0(3r_0 - 4r_1 + r_2))} \right] + \delta_0 > 0. \tag{4.22}$$

The quantity in square brackets is an altruism threshold: the cost-benefit ratio $-\delta_0/(N\delta_1)$ must be smaller than this, assuming $\delta_1 > 0$, for altruism to be favoured. Survival probability does not appear explicitly in this threshold, but is felt through the relatedness r_i.

Two dimensions

In two dimensions, the inclusive fitness effect involves terms from the six kinds of sites identified in Figure 2.1,

$$\begin{aligned} \Delta w_{IF} &= r_{0,0}\Delta w_{0,0} + 4r_{0,1}\Delta w_{0,1} + 4r_{1,1}\Delta w_{1,1} + 4r_{0,2}\Delta w_{0,2} \\ &\quad + 8r_{1,2}\Delta w_{1,2} + 4r_{0,3}\Delta w_{0,3} \end{aligned} \tag{4.23}$$

where the net fitness effects on breeders at a distance i, Δw_i, are

$$\begin{aligned} \Delta w_{0,0} &= (1-s)\left(\delta_0(1 - p_0^2 - 4p_1^2) - \delta_1(8p_0p_1)\right) \\ \Delta w_{0,1} &= (1-s)\left(\delta_0(-2p_0p_1) + \delta_1(1 - p_0^2 - 9p_1^2)\right) \\ \Delta w_{1,1} &= -2(1-s)\left(\delta_0(p_1^2) + \delta_1(2p_0p_1)\right) \\ \Delta w_{0,2} &= -(1-s)\left(\delta_0(p_1^2) + \delta_1(2p_0p_1)\right) \\ \Delta w_{1,2} &= -3(1-s)\delta_1 p_1^2 \\ \Delta w_{0,3} &= -(1-s)\delta_1 p_1^2. \end{aligned}$$

The altruistic behaviour is favoured if

$$4\delta_1 \left[\frac{7r_{0,1} - 6r_{1,2} - r_{0,3} - p_0(8r_{0,0} - 25r_{0,1} + 16r_{1,1} + 8r_{0,2} - 6r_{1,2} - r_{0,3})}{4(3r_{0,0} - 2r_{1,1} - r_{0,2} + p_0(5r_{0,0} - 8r_{0,1} + 2r_{1,1} + r_{0,2}))} \right] + \delta_0 > 0. \tag{4.24}$$

4.2.2 Survival effects

Now I want the altruistic behaviour to have an effect on survival probability, not on fecundity. Assuming a non-zero survival probability, I write the survival of an adult on site i as $s_i = s$ and introduce an effect of the mutant, altering the survival probability by a small relative change δ_i,

$$s_i' = s_i(1 + \delta_i). \tag{4.25}$$

I leave n_{ij} and p_{ij} as before, and note that p_{ij} can be interpreted as the probability an individual from site i wins on site j conditioned on the death of the individual at site j. The fitness of an individual on site i is

$$w_i = s_i + \sum_j p_{ij}(1 - s_j) \tag{4.26}$$

and after a mutant is introduced at site 0, the fitness is

$$\begin{aligned} w_i' &= \sum_j p_{ij}'(1 - s_j') + s_i' = \sum_j p_{ij}(1 - s_j(1 + \delta_j)) + s_i(1 + \delta_i) \\ &= w_i - \sum_j \delta_j p_{ij} s_j + s_i \delta_i. \end{aligned} \tag{4.27}$$

The inclusive fitness effect of the mutant is

$$\Delta w_{IF} = \sum_i r_i \Delta w_i = s\left(\sum_i r_i \delta_i - \sum_{ij} r_i \delta_j p_{j-i}\right). \tag{4.28}$$

One dimension

The inclusive fitness effect (4.28) in one dimension is

$$\Delta w_{IF} = \Delta w_0 + 2r_1 \Delta w_1 + 2r_2 \Delta w_2 \tag{4.29}$$

where the changes in fitness of the mutant and four nearest-neighbours are

$$\begin{aligned} \Delta w_0 &= s(\delta_0(1 - p_0) - 2\delta_1 p_1) \\ \Delta w_1 &= s(-\delta_0 p_1 + \delta_1(1 - p_0)) \\ \Delta w_2 &= -s\delta_1 p_1. \end{aligned}$$

The altruistic act is favoured if

$$2\delta_1 \left[\frac{r_0 - 2r_1 + r_2}{2(r_1 - r_0)}\right] + \delta_0 > 0. \tag{4.30}$$

Two dimensions

The fitnesses of individuals on fewer sites are affected with survival effects in two dimensions compared to fecundity effects. Individuals two steps from the mutant are affected because of the change in survival of individuals one step away. The inclusive fitness effect is

$$\Delta w_{IF} = \Delta w_{0,0} + 4r_{0,1}\Delta w_{0,1} + 4r_{1,1}\Delta w_{1,1} + 4r_{0,2}\Delta w_{0,2} \tag{4.31}$$

where the fitness changes (4.27) are

$$\begin{aligned}\Delta w_{0,0} &= s(\delta_0 - 4\delta_1 p_1 - \delta_0 p_0)\\ \Delta w_{0,1} &= s(\delta_1 - \delta_0 p_1 - \delta_1 p_0)\\ \Delta w_{1,1} &= -2s\delta_1 p_1\\ \Delta w_{0,2} &= -s\delta_1 p_1.\end{aligned}$$

The altruistic act is favoured if

$$4\delta_1\left[\frac{r_{0,0} - 4r_{0,1} + 2r_{1,1} + r_{0,2}}{4(r_{0,1} - r_{0,0})}\right] + \delta_0 > 0. \tag{4.32}$$

Computational results

Figure 4.2 shows the altruism threshold $-\delta_0/(N\delta_1)$ as a function of survival s for one- ($N = 2$) and two-dimensional ($N = 4$) stepping-stone populations with both fecundity and survival effects. The horizontal line at 0 is the threshold for a randomly interacting population; altruism is selected if the cost to the actor is in fact a direct benefit ($\delta_0 > 0$). With fecundity effects, the threshold increases with s from 0 allowing altruism to be favoured for increasingly large costs. With survival effects, the threshold also increases with increasing survival but from an initial negative relatedness threshold, requiring a large negative cost to overcome the benefit given to neighbours. In the limit as $s \to 1$ the threshold approaches 0. Adding a cost to dispersal (thin lines) pushes the threshold closer to that of a random-mixing population.

4.2.3 The effect of survival on the cost-benefit threshold

I now briefly consider an altruistic act which costs the altruist c and provides a total benefit b which is shared throughout the $N+1$ sites in the neighbourhood. In one dimension, $N = 2$, so on average the altruist and its neighbours receive a benefit of $b/3$ each. Substituting $\delta_0 = -c + \frac{b}{3}$, $\delta_1 = \frac{b}{3}$ and (2.12) into the fecundity threshold (4.22) and solving for $\frac{b}{3}/c$, I obtain

$$\frac{b/3}{c} > \frac{\hat{d}(d(1-k)(1-\sqrt{1-\hat{d}}) - \hat{d}(1-kd)))}{(3\hat{d} - 4(1-\sqrt{1-\hat{d}}))(d(1-k) - \hat{d}(1-kd)) - d\hat{d}(1-k)\sqrt{1-\hat{d}}} \tag{4.33}$$

and for no-cost dispersal ($k = 0$) using the ES dispersal rate (3.20) this reduces to

$$\frac{b/3}{c} > \frac{s+3}{5s+3}. \tag{4.34}$$

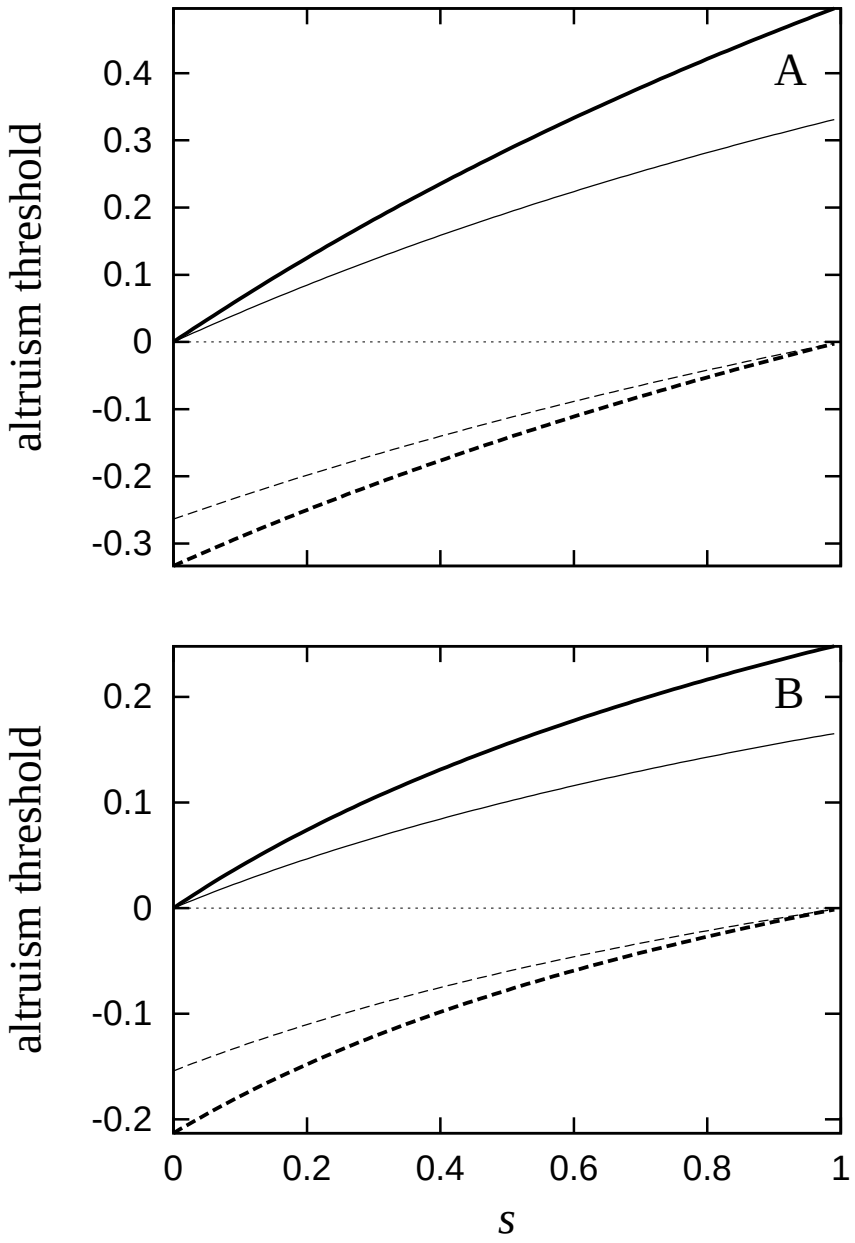

Figure 4.2: Altruism threshold $-\delta_0/(N\delta_1)$ as a function of survival s. Results for fecundity (solid) and survival (dashed) effects in one (A) and two (B) dimensions. Thick lines are for zero-cost dispersal and thin lines for a small dispersal cost ($k = 0.2$). The horizontal dotted line is the altruism threshold for random-mixing populations or $s = 0$.

For survival effects, solving $\Delta w_{IF} > 0$ from (4.29) obtains

$$\frac{b/3}{c} > \frac{\hat{d}}{3\hat{d} - 4(1 - \sqrt{1-\hat{d}})}, \tag{4.35}$$

and for $k = 0$, the threshold (4.35) has an even simpler form obtained using the ES dispersal rate (3.20),

$$\frac{b/3}{c} > \frac{s+3}{3s+1}. \tag{4.36}$$

In two dimensions, there is no explicit formula for the evolutionarily stable dispersal rate d^*; I find d^* by solving (3.23) numerically. Since there are 5 recipients, each obtains a benefit $b/5$ while the altruist incurs the full cost c. Setting $\delta_0 = -c + b/5$ and $\delta_1 = b/5$, using the relatednesses (2.24) and solving for $\frac{b}{5}/c$ yields a threshold benefit-cost ratio for altruism to be favoured. The threshold is found numerically using equations obtained with the assistance of a computer algebra system.

Figure 4.3 shows the benefit-cost threshold $b/(N+1)c$ as a function of survival s for one- and two-dimensional stepping-stone populations with both fecundity and survival effects. The qualitative features have the same interpretation as our general results reported in Figure 4.2. The horizontal line at 1 corresponds to the threshold benefit-cost ratio for a randomly interacting population where the altruistic benefit is shared among $N+1$ recipients. With fecundity effects, the threshold decreases as s increases from 1 at $s = 0$. With survival effects, the threshold also decreases with increasing survival but from an initial threshold much greater than for a random-mixing population, reaching 1 at $s = 1$.

4.3 Prisoner's dilemma models for altruistic interactions

The prisoner's dilemma (PD) is frequently used to model altruistic behaviour in biology, economics, and other fields (Trivers, 1971; Axelrod and Hamilton, 1981; Maynard Smith, 1982; Skyrms, 1996). The game encapsulates a widely observed phenomenon: cooperation is mutually beneficial but greedy or opportunistic behaviour which exploits the 'naïve' cooperation of others appears to be even better for a few defectors in a population of cooperators. The PD game reduces the basic problem down to a very simple description and the iterated prisoner's dilemma (IPD) is an extension designed to favour cooperative strategies. Recently, spatial versions of both games have been devised as another way to favour cooperative strategies (Nowak and May, 1992; Killingback *et al.*, 1999). I modify the analysis of

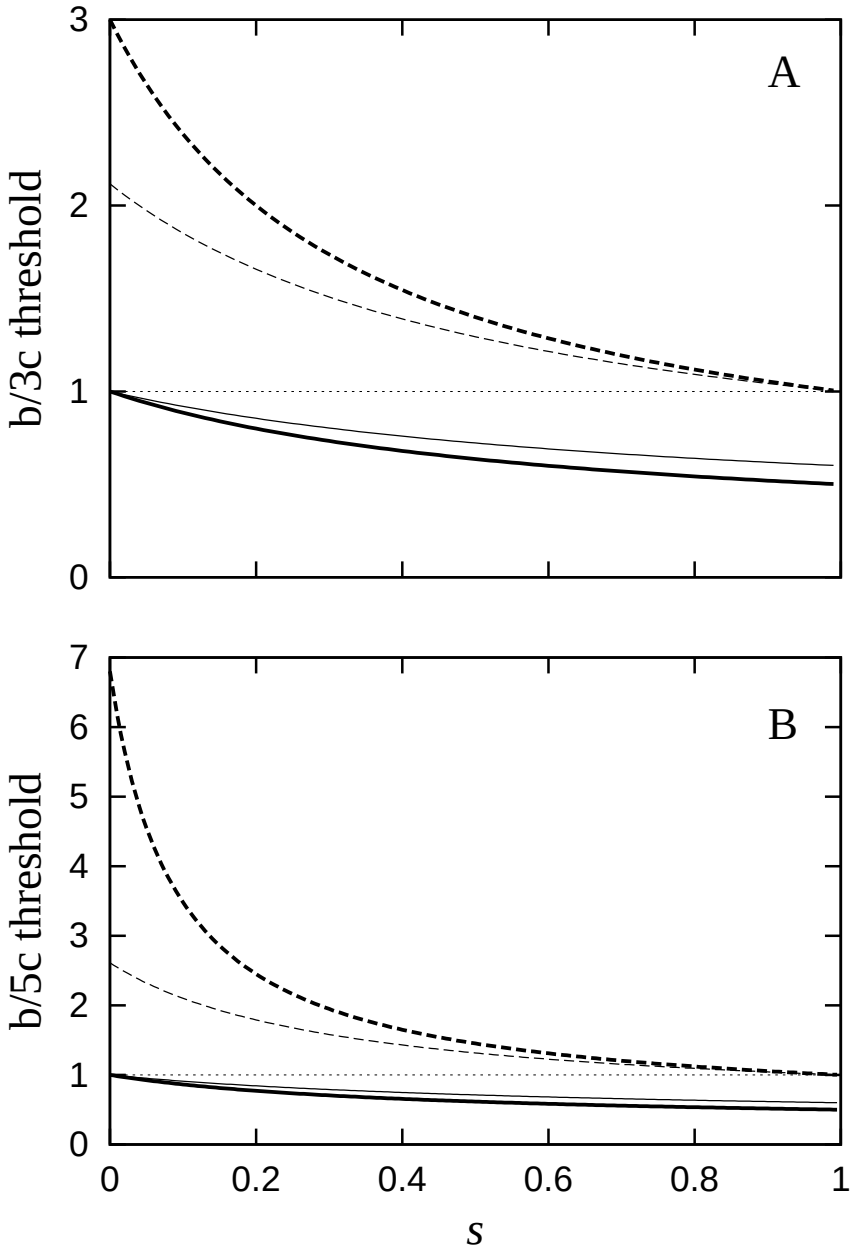

Figure 4.3: Altruism thresholds $\frac{b}{N+1}/c$ as a function of survival s. Results for fecundity (solid) and survival (dashed) effects in one (A) and two (B) dimensions. Thick lines are for zero-cost dispersal and thin lines for a small dispersal cost ($k = 0.2$). The horizontal dotted line is the threshold for random-mixing populations or $s = 0$.

		Partner	
		C	D
Actor	C	$R = 3$	$S = 0$
	D	$T = 5$	$P = 1$

Table 4.2: Payoff matrix for the prisoner's dilemma game showing standard payoffs used in numerical examples.

altruism presented above to incorporate the IPD and describe the effect of spatial structure on the evolution of altruism. I begin by describing both the PD and IPD.

The prisoner's dilemma (PD) is illustrated by a two-player game with two strategies: cooperate (C) and defect (D) which can also be thought of as behave altruistically towards your partner or behave selfishly (or non-altruistically). In this game, everyone's baseline fitness of 1 is adjusted by a small amount proportional to the payoff determined by one's own strategy and the strategy of one's partner. The game can also be extended to multi-player situations, for example, by assigning scores based on the average payoff from all possible interactions. The payoff matrix for the actor is given in Table 4.2. For example, an actor playing the C strategy receives a payoff of R against another C strategist, but S against a D player. The numbers in the table are standard values for the payoffs which will be used throughout for numerical examples. A useful mnemonic for these parameters is that the payoffs correspond to Temptation, Reward, Punishment, and Sucker. The payoffs are ordered so that $T > R > P > S$ which means that an actor can always improve her score by defecting. (Another condition usually imposed requires that $R > \frac{T+S}{2}$. The reward for cooperating is greater than the average of a temptation and sucker payoff.) Everyone knows this so everyone defects. The dilemma arises because the payoff if both players cooperate is higher than if both defect. Acting alone and making the best decision for oneself results in a sub-optimal score, not just for the pair, but on an individual basis as well. If there was a way to coordinate behaviour, everyone could benefit from the larger payoff from mutual cooperation.

If the partners are permitted to interact more than once, the game is called the iterated prisoner's dilemma. The series of prisoner's dilemma games is called a tournament. The set of strategies available to an actor is much larger now since the choice of strategy in a specific round of the tournament can depend on the result of previous rounds. I consider one class of these models where after the first encounter the probability of encountering the same opponent is w, independent of the past history, and once paired, individuals play the same opponent. Players remember the outcome of the previous encounter and use this information in their strategy. The probability another round is played is constant, so the

		Partner	
		TFT	AD
Actor	TFT	$\frac{R}{1-w}$	$S+\frac{Pw}{1-w}$
	AD	$T+\frac{Pw}{1-w}$	$\frac{P}{1-w}$

Table 4.3: Payoffs for the iterated prisoner's dilemma where TFT is the cooperative strategy 'tit-for-tat' and AD is 'always defect'. The parameters T, R, P, S are the payoffs for the prisoner's dilemma and satisfy $T > R > P > S$. The probability of playing the prisoner's dilemma again with the same partner is w. I use $T = 5$, $R = 3$, $P = 1$, $S = 0$ for numerical examples. If $w = 0$ the game reduces to the prisoner's dilemma.

length of tournaments has a geometric distribution, i.e. the conditional probability a pair plays exactly i rounds is $p_i = w^{i-1}(1-w)$. The expected length of a tournament is

$$\sum_{i=1}^{\infty} ip_i = 1 + w + w^2 + \cdots = \frac{1}{1-w}. \tag{4.37}$$

Two special strategies will be taken to be the pure strategies available to players: tit-for-tat (TFT) and always defect (AD). Under TFT, a player first cooperates then repeats its opponents previous choice. AD players always defect. The payoff matrix has a simple form in this case because in any pairing, the payoffs for the second and subsequent rounds are identical (always the reward or punishment payoff) and so the expected payoff from round 2 and following is R or P times the number of games to be played after round 1 which is $w + w^2 + w^3 + \cdots = \frac{w}{1-w}$. The payoff matrix is shown in Table 4.3. The rank ordering of the payoffs (reward > punishment > sucker) is preserved for all $w \in [0, 1]$ but the temptation payoff is greater than the reward payoff if $T + Pw/(1-w) > R/(1-w)$ which is equivalent to

$$w < \frac{T-R}{T-P}. \tag{4.38}$$

This inequality is not satisfied if $w \geq 1/2$ using the payoffs from Table 4.2. This means that mutual defection is not attractive for sufficiently large re-encounter probabilities and one could expect TFT to be superior to AD. (This will be the case in an unstructured population with a large enough frequency of TFT – see the dotted line in Fig. 4.5.)

The essential feature of this extension is that the multiple interactions allow cooperative behaviour to be tested and rewarded and uncooperative behaviour can be met with

an appropriate uncooperative response. I use the IPD because it is a model of altruistic interactions between individuals and the PD and IPD have been intensively studied and are of interest in their own right. In recent years there have been a flurry of studies of the IPD in a spatially structured habitat (Nowak and May, 1992; Nowak *et al.*, 1994; Grim, 1995; Nakamaru *et al.*, 1997, 1998, for example). However, none of these have employed the inclusive fitness method and exact calculations of the cost-benefit ratio which favour altruistic behaviour. Instead workers have concentrated on simulations and *ad hoc* methods such as the pair-approximation.

I use the IPD game to determine costs and benefits for the earlier altruism model adapting the inclusive fitness argument presented earlier. Consider a monomorphic population composed of individuals who use TFT with probability p and AD with probability $1-p$. The average payoff to a p actor playing a q player is

$$W(p,q) = pq\left(\frac{R}{1-w}\right) + (1-p)q\left(T+\frac{Pw}{1-w}\right) \tag{4.39}$$

$$+ p(1-q)\left(S+\frac{Pw}{1-w}\right) + (1-p)(1-q)\left(\frac{P}{1-w}\right). \tag{4.40}$$

I translate the payoffs into the cost and benefit notation from the patch or stepping-stone altruism models presented earlier. Individuals play each of their neighbours and pay a cost equal to their mean payoff. Similarly, the benefit received by each neighbour is the average of the payoffs from each game played. The cost to an actor of using the mutant strategy $p' = p + \delta$ is

$$C = -(W(p',p) - W(p,p)) \tag{4.41}$$

and the benefit to its neighbours is

$$B = W(p,p') - W(p,p). \tag{4.42}$$

I use capital letters as a reminder that these are the net cost C to the actor and the benefit B which is divided among the neighbours. These are different from the lower-case costs and benefits introduced earlier.

Now consider the patch-structured population calculation with its costs and benefits defined by the change of variables,

$$c = C + \frac{B}{N-1} \tag{4.43}$$

$$b = \frac{NB}{N-1}.$$

The threshold benefit:cost ratio which arose in the previous analysis can be written in terms of B and C and thus W, p and δ,

$$\frac{b/N}{c} = \frac{B}{B+C(N-1)}. \tag{4.44}$$

Substituting (4.41, 4.42) into (4.44) and solving for p to first order in δ using the payoffs from Table 4.2 obtains the following threshold

$$p = \frac{1-w}{3w-1} \cdot \frac{\frac{b/N}{c}(3+N)-4}{1+\frac{b/N}{c}(N-2)} \tag{4.45}$$

where $\frac{b}{N}/c$ can be obtained from the altruism threshold for fecundity (4.2) or survival effects (4.6).

The notation for the stepping-stone population is slightly different. Substituting $C = -\delta_0$, $B = N\delta_1$ and (4.41, 4.42) to obtain an altruism relatedness threshold $-\delta_0/(N\delta_1)$ and solving for p to first order in δ using the payoffs from Table 4.2, I obtain the following threshold

$$p = \frac{1-w}{3w-1} \cdot \frac{(\frac{-\delta_0}{N\delta_1})(3+N)-4}{1+(\frac{-\delta_0}{N\delta_1})(N-2)}. \tag{4.46}$$

The threshold $-\delta_0/(N\delta_1)$ can be obtained from the appropriate section above, that is, one of equations (4.22, 4.24, 4.30, or 4.32).

Expression (4.46) is perhaps deceptively simple; the expression $-\delta_0/N\delta_1$ is a complicated function of several relatedness coefficients, dispersal rate, and survival and the exact form depends on whether fecundity or survival effects are being considered on a one- or two-dimensional lattice. If $w > 1/3$ then increased altruism (frequency of playing TFT) is selected for p greater than this threshold and decreased altruism is selected for smaller p. If $w < 1/3$ then AD is the ESS.

In randomly mixing populations (and populations with fecundity effects and non-overlapping generations), the threshold (4.46) takes a simpler form, independent of the size of the interaction neighbourhood,

$$p > \frac{1-w}{3w-1} \text{ and } w > \frac{1}{3} \tag{4.47}$$

assuming the numerical values for the payoffs from Table 4.2. For $\frac{1}{3} < w < \frac{1}{2}$, AD is the global ESS and the threshold p is 1. For $w > \frac{1}{2}$, the payoff matrix in Table 4.3 now has a greater payoff for TFT than AD when meeting a TFT partner, so AD and TFT are both local ESSs.

Threshold values of p are shown as a function of s in Figures 4.6 and 4.4 for patch and stepping-stone populations respectively and as a function of w in stepping-stone populations in Figure 4.5. Fecundity thresholds (solid lines) are below the random-mixing threshold (dotted line) and survival effects (dashed lines) are above the random-mixing threshold. Results for one (A) and two (B) dimensions are shown as well as zero-cost dispersal (thick

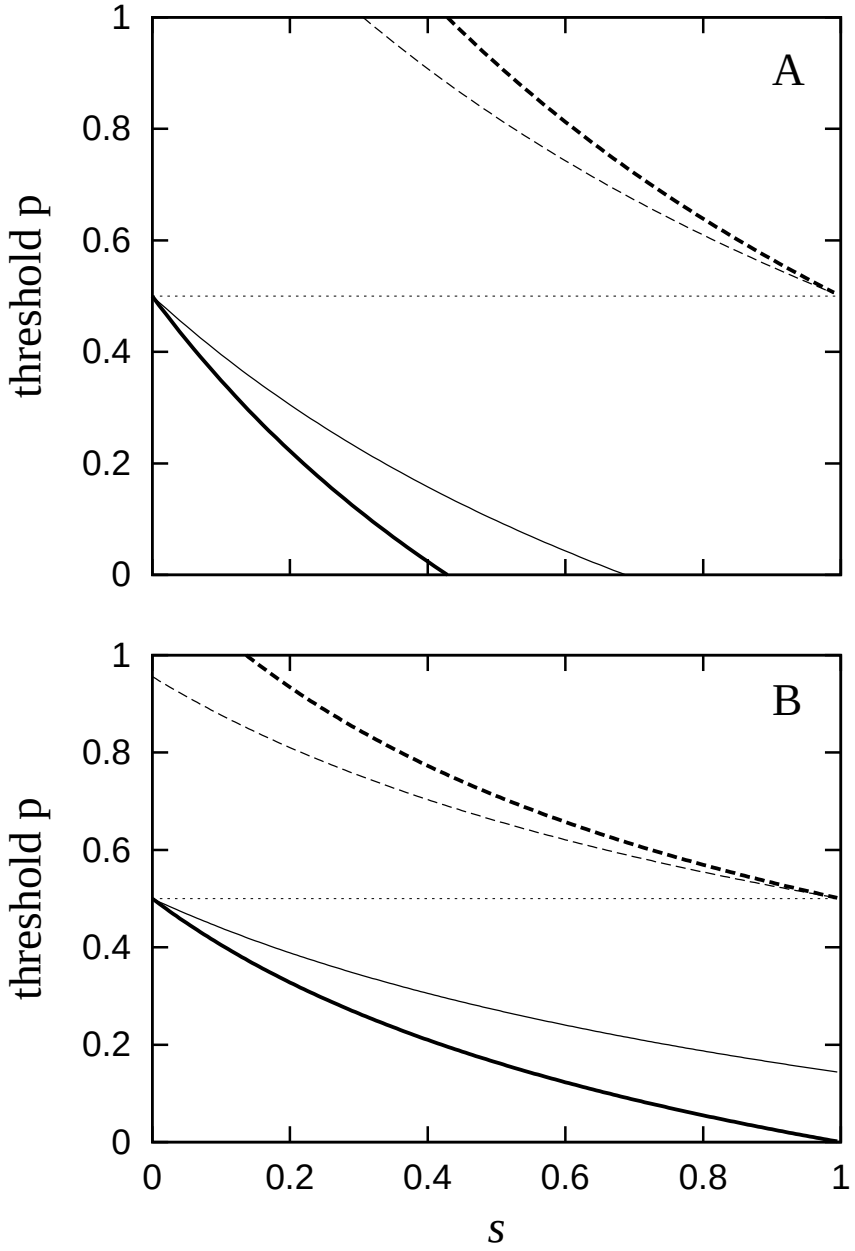

Figure 4.4: Threshold values of TFT frequency p as a function of survival s for the iterated prisoner's dilemma game with a probability of re-encounter of $w = 3/5$ in one- (A) and two-dimensional (B) stepping-stone populations. The horizontal dotted line is the threshold for random-mixing populations (4.47) or $s = 0$ and other lines are as in Figure 4.2. Increased frequency of TFT is favoured above the appropriate line. For no-cost dispersal in one dimension, TFT is the only ESS for $s > \frac{3(P-S)}{2T-3P+S} = \frac{3}{7}$ with fecundity effects and AD is the only ESS for $s < \frac{9-13w}{1+3w} = \frac{3}{7}$ with survival effects.

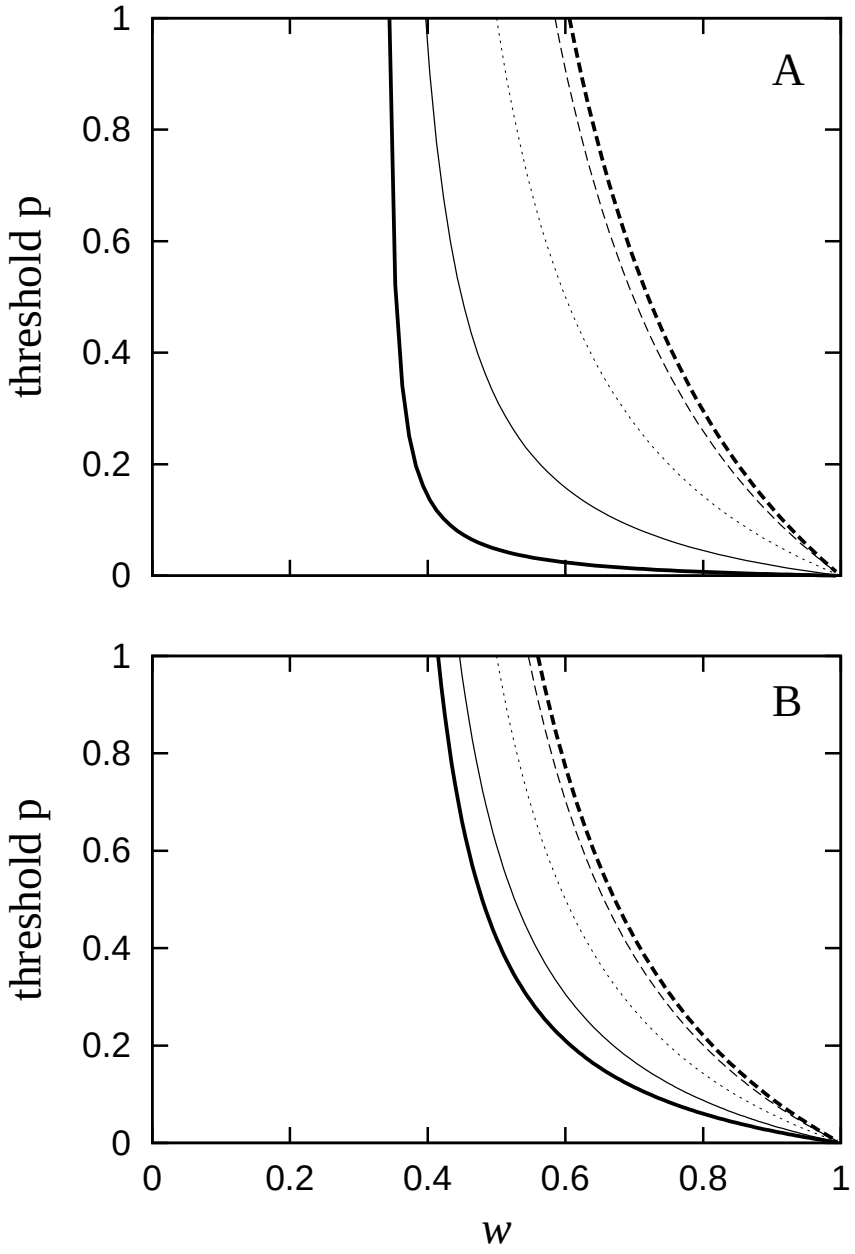

Figure 4.5: Threshold values of TFT frequency p as a function of the probability of re-encounter w for the iterated prisoner's dilemma game with survival probability of $s = 2/5$ in one- (A) and two-dimensional (B) stepping-stone populations. The dotted line is the threshold for randomly-mixing populations (4.47) or $s = 0$ and other lines are as in Figure 4.2. Increased frequency of TFT is favoured above the appropriate line.

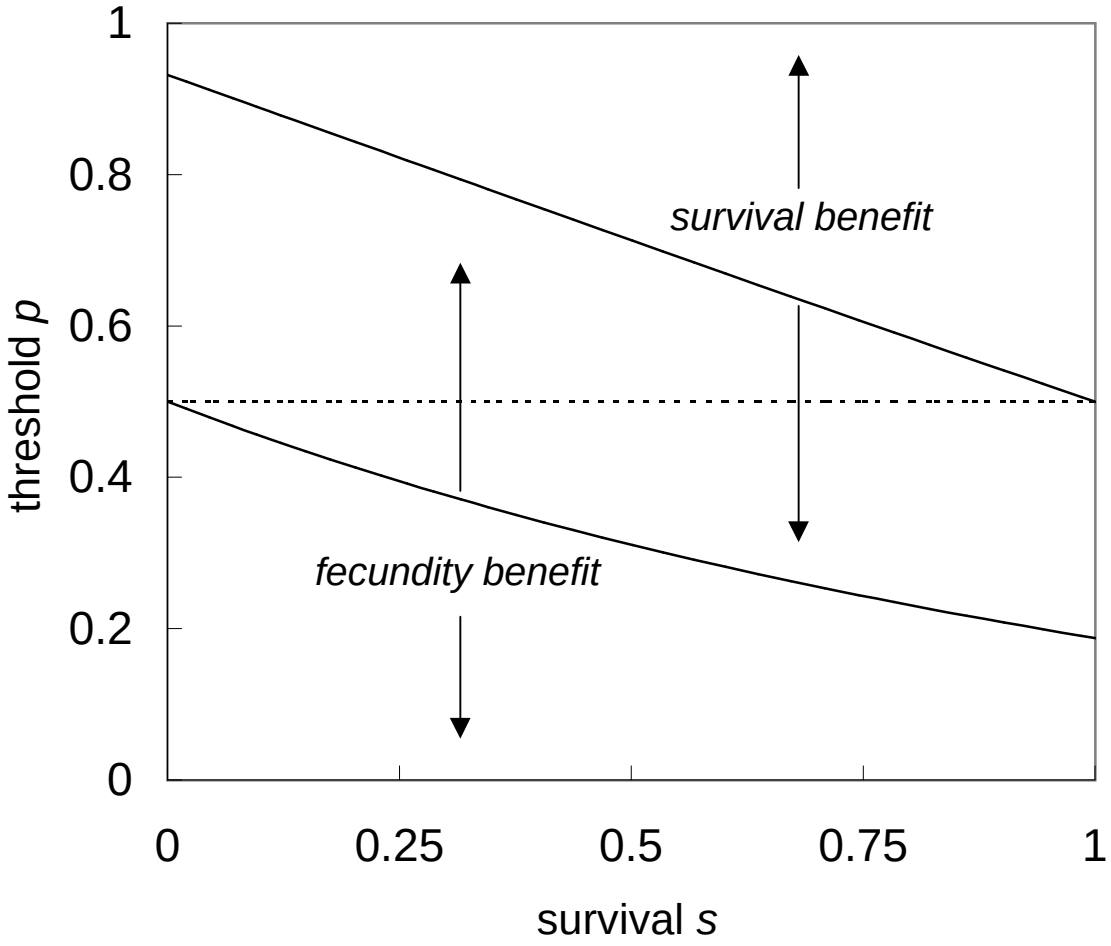

Figure 4.6: Threshold frequency of TFT p in the iterated prisoner's dilemma in a patch-structured population as a function of survival s using the payoffs in Table 4.2. Increased altruism is favoured above the lines (arrows pointing upward) and selected against below the lines (arrows pointing downward). The height of the dashed line, $p = (3w - 1)/(1 - w)$, gives the threshold p for non-overlapping generations ($s = 0$). The two solid curves are for fecundity and survival benefits. The parameter values are $N = 5$, $h = 0.8$, and $w = 3/5$.

lines) and dispersal with a small cost ($k = 0.2$, thin lines). Populations with p above the corresponding line favour increasing p, that is larger probabilities of playing TFT. Increasing dispersal cost k moves the thresholds closer to the random-mixing threshold (4.47), inhibiting altruism with fecundity effects and favouring it with survival effects. The pattern is the same as the previous altruism example: increasing s enlarges the area in parameter space where altruism is favoured. A fecundity benefit favours altruism more in a stepping-stone structured population than in an unstructured population, but survival benefits make it more difficult for altruism to evolve.

4.4 Discussion

My primary interest in this chapter is the effect of the survival rate of breeders on the selective advantage of altruism. I have two main results which apply to both patch and stepping-stone structured populations. First, there is a distinction between benefits of fecundity and survival. Under the former, altruism is more strongly favoured and under the latter altruism is less strongly favoured than in a random-mixing population. Second, there is an effect of overlapping generations. For both types of benefit, an increased survival probability promotes altruism.

I use an inclusive fitness model to measure the effects of altruism, and in order for this to predict the course of genetic evolution, I need to assume that selection is 'weak,' that is, that altruistic behaviour has a small selective effect, which means that the costs and benefits are small (Taylor, 1996). There are several reasons for this assumption, but an important one is that the calculation of relatedness (Chapter 2) is valid only when the allele causing the altruistic behaviour is neutral. The selective advantage of altruism that I calculate is then actually the rate at which the fitness of this allele increases as the effect of the allele is increased above zero.

When breeder survival s is zero and there is no overlap between generations I find that, independent of the dispersal rate of offspring, the altruism threshold occurs where the direct effect δ_0 of the altruist on itself is positive (4.20). This is the condition that weak altruism is selected and strong altruism is at a disadvantage. This is also expected in a random-mixing population, but the point is that it is also obtained when dispersal of offspring is only partial in patch and both one- and two-dimensional stepping-stone populations. However, if breeder survival is positive, so that there is overlap between generations, a decrease in the dispersal rate does have an effect on the threshold level of altruism. But here there is a striking difference between gifts of fecundity and gifts of survival — relative to a random-mixing population, altruism is promoted under a fecundity benefit, but discouraged under a survival benefit. This is illustrated in Figures 4.2 and 4.3. The difference between these

two cases is easily understood. A survival benefit can be regarded as a special class of extra offspring that have the appropriate probability of winning a site, but that do not disperse, so they must remain on their home site. This increases the local competition among offspring and reduces the advantage of altruism.

The altruism threshold decreases as breeder survival s increases, favouring higher levels of altruistic behaviour. The analysis of this effect is complicated because the threshold depends on both s and the dispersal rate d, and the ES value of d itself increases with s (Irwin and Taylor, 2000*b*). This latter relationship, the increase of dispersal rate with s, is an example of the phenomenon I am discussing — increased altruism with higher s.

Increasing the cost of dispersal has different effects on the altruism threshold for fecundity and survival effects (Figures 4.2 and 4.3). First, with fecundity effects, increasing dispersal cost reduces the ES dispersal rate and inhibits altruism. This is because the increased cost reduces the proportion of immigrant offspring on a patch and thus increases competition among native offspring, resulting in a reduced benefit from the altruistic act. In contrast, a survival benefit increases competition on neighbouring sites. This affects the recipient's fitness only though its offspring which disperse to these sites. If dispersal cost is higher, a smaller fraction of offspring disperse, so the cost has less effect on the recipient.

Several recent studies of social behaviour in lattice-structured populations are worth comparing to my results. Nowak and May (1992) and Nowak *et al.* (1994) simulated the prisoner's dilemma game on a lattice with fecundity effects and found that cooperators do better in models with overlapping instead of non-overlapping generations. Nakamaru *et al.* (1997, 1998) studied the iterated prisoner's dilemma on the lattice with both survival and fecundity effects. They simulated populations of TFT and AD strategists in one- and two-dimensional stepping-stone populations. There are several differences between their model and mine: they assume complete dispersal ($d = 1$, an empty site can only be colonized by a neighbour, and not the offspring of the dead individual) and they use continuously overlapping generations — only one individual on the lattice dies per time-step. Despite these differences they obtained similar results: fecundity effects are generally more favourable to the evolution of altruism although unlike my results, survival effects sometimes inhibited the evolution of altruism and sometimes facilitated it. Nakamaru *et al.* (1997, 1998) attribute their results to population structure, but in my example of non-overlapping generations the results are identical in both structured and randomly mixing populations. In their model with sufficiently long games (w near 1), TFT is able to invade a population of AD individuals. In one dimension their result was especially striking: their threshold could be shown as a vertical line at $w = 3/5$ on our Figure 4.5A. With fecundity effects and no-cost of dispersal, I find that TFT is the ESS if $s > 3/7$ but that both TFT and AD are ESSs for smaller s (Fig. 4.3A). With survival effects I find no situation where a rare TFT can invade an AD

population. One of their derivations of the vertical threshold was a 'pair-edge' analysis of the velocity of the interface between clusters of TFT and AD (Ellner *et al.*, 1998; Nakamaru *et al.*, 1998). A critical assumption in this calculation is that adjacent sites on the interface can't die (and potentially be replaced with the other strategy) in the same generation. This is approximately true in my model only at high survival rates.

Chapter 5

Conclusions

5.1 Summary

In this thesis I've studied the evolution of dispersal and altruism in patch-structured and stepping-stone populations with discrete overlapping generations. I've assumed infinite, asexual haploid populations throughout. Some recent work with inclusive fitness models shows that qualitatively similar results can be obtained in finite populations (Rousset and Billiard, 2000; Taylor and Day, 2000; Taylor *et al.*, 2000).

There are two main results found in both kinds of population structure. First, increasing survival probability facilitates the evolution of altruism. Second, there is an important difference between fecundity and survival effects in altruistic interactions. With the former, altruistic behaviour has an advantage compared to unstructured populations and with the latter, altruism is less likely to be favoured.

When breeder survival s is zero and there is no overlap between generations, I find that independent of the dispersal rate of offspring, the altruism threshold occurs where the direct effect of the altruist on itself is positive. This is also expected in a random-mixing population, but the same result is obtained when dispersal of offspring is only partial in both patch and stepping-stone populations. However, if breeder survival is positive, so that there is overlap between generations, a decrease in the dispersal rate does have an effect on the threshold level of altruism. Relative to an unstructured population, altruism is promoted under a fecundity benefit, but discouraged under a survival benefit. The difference between these two cases is easily understood. A survival benefit can be regarded as a special class of extra offspring that have the appropriate probability of winning a site, but that do not disperse, so they must remain on their home site. This increases the local competition among offspring and reduces the advantage of altruism.

I've used examples of two different kinds of spatial structure. Patches are the simplest

structure which allow for limited dispersal and local genetic structure. The stepping-stone population creates a more realistic spatial structure; some individuals are close together, others are far apart and the coupling between individuals is directly proportional to the distance between them. Other mathematical representations can be used, including a continuous description of space (leading to partial differential equations) or a detailed itemization of the location and other properties of each individual (leading to individual based models usually analysed by computer simulation). A striking result of the work in this thesis is that there is no essential difference between the two kinds of spatial structure I used. Spatial structure is important to the evolution of social behaviour when combined with overlapping generations, but the kind of spatial representation does not seem to be that important.

5.2 Future work

The results of this research suggest some interesting problems which I plan to investigate.

5.2.1 Elastic populations

Throughout this thesis I have assumed that patches are inelastic. This means that local populations are at their carrying-capactity and that the number of individuals supported on a patch does not depend on the strategy adopted by its occupants. A natural extension is to allow the number of individuals on a patch to vary, e.g. instead of N individuals on a patch, there might be enough resources to support $N(1+p)$ where p is the local frequency of altruists.

In a recent paper, van Baalen and Rand (1998) use the pair approximation technique to analyse a model similar to Wilson *et al.* (1992) but their populations are not at carrying capacity. They show that the conditions for non-altruists to invade clusters of altruists in their model are more complicated than described by Taylor (1992*a*) and Wilson *et al.* (1992), and depend on features such as the background mortality rate. A careful inclusive fitness analysis of the effect of elastic populations in structured populations will clarify some of the confusion in the literature.

5.2.2 Continuously overlapping generations

The analysis of overlapping generations in this thesis extends previous analytic studies of altruism which have only considered non-overlapping generations. I chose discrete overlapping generations, but another option would be to study continuously overlapping generations by assigning an instantaneous survival probability to each individual. In my models, sev-

eral individuals die each generation but with continously overlapping generations, no two individuals would die simultaneously.

The models of Nakamaru *et al.* (1997, 1998) and others employ continuously overlapping generations. A difference between the two kinds of overlapping generations is illustrated by the moving-boundary calculation of Nakamaru *et al.* (1998). It turns out that a crucial assumption in their calculation is that it is impossible for two adjacent sites in a stepping-stone population to become vacant simutaneously. This accounts for significant deviations between their results and mine. Both situations may be biologically plausible and it is important to extend the inclusive fitness method here to describe continuously overlapping generations.

5.2.3 Hierarchical patch-deme-structured populations

Another extension would be to study a variation on the patch-structured model which perhaps more closely describes a true spatial structure. In a hierarchical patch-deme-structured population, individuals are grouped first in patches with limited dispersal between patches and then patches are grouped into demes (groups of patches) with limited dispersal (possibly at a different rate) between demes. An infinite collection of demes then comprises the whole population. Such a population structure has been studied by Kelly (1994) in the context of a resource allocation problem. An imporant feature of this model is that analytical results should be reasonably easy to obtain because Price's formula (1.9) easily generalises to a hierarchical decomposition.

Appendix A

Details of selected computations

A.1 Relatedness in a one-dimensional stepping-stone population

The recursion (2.11) can be written as a set of first-order difference equations

$$\begin{bmatrix} r_{j+2} \\ r_{j+1} \\ r_j \\ r_{j-1} \end{bmatrix} = \frac{1}{A} \begin{bmatrix} -B & (1-C) & -B & -A \\ A & 0 & 0 & 0 \\ 0 & A & 0 & 0 \\ 0 & 0 & A & 0 \end{bmatrix} \begin{bmatrix} r_{j+1} \\ r_j \\ r_{j-1} \\ r_{j-2} \end{bmatrix} \tag{A.1}$$

where A, B, and C are given in (2.11) and the boundary condition $r_0 = 1$ selects a unique bounded solution. The eigenvectors of the matrix in (A.1) have the form $\{r_j\} = \{\lambda^j\}$ where λ is the corresponding eigenvalue. Thus bounded solutions only include eigenvalues such that $|\lambda| \leq 1$.

With no mutations ($\mu = 0$) the characteristic polynomial of the matrix is

$$p(\lambda) = (\lambda - 1)^2(A\lambda^2 + (B + 2A)\lambda + A). \tag{A.2}$$

For small $\mu > 0$, $p(1) < 0$ and so the double root at 1 separates into a pair of roots, only one of which has magnitude less than 1. The other two roots are both negative, and one lies in the interval $(-1, 0)$. The two roots can be written as

$$\theta = 1 - O(\sqrt{\hat{\mu}}) \tag{A.3}$$

$$\phi = \frac{2\sqrt{1-\hat{d}} - 2 + \hat{d}}{\hat{d}} + O(\sqrt{\hat{\mu}}) \tag{A.4}$$

respectively. The solutions to the recursion are a linear combination of the eigenvectors and thus the relatednesses r_k can be written as

$$r_k = c\theta^k + d\phi^k. \tag{A.5}$$

The coefficients c and d can be found with the additional equations $r_0 = 1$ and $r_{-1} = r_1$. This finally leads to the expressions in (2.12).

The relatednesses in one dimension can also be found by reasonably straightforward (computer-assisted) integration of (2.25).

A.2 Fitness effects in dispersal model

The computations leading to (3.17) and (3.21) are quite detailed; some of the intermediate steps are provided below.

A.2.1 One dimension stepping-stone

$$\begin{aligned}\Delta\tilde{w}_0 &= \frac{n'_{00}}{n'_{00}+n'_{10}+n'_{01}} - \frac{n_{00}}{n_{00}+n_{10}+n_{01}} \\ &\quad + 2\left(\frac{n'_{01}}{n'_{01}+n'_{11}+n'_{12}} - \frac{n_{01}}{n_{01}+n_{11}+n_{12}}\right) \qquad (A.6)\\ &= \frac{1-d'-\mu}{1-d'-\mu+d(1-k)} - \frac{1-d-\mu}{1-d-\mu+d(1-k)} \\ &\quad + \frac{d'(1-k)}{\frac{1}{2}d'(1-k)+1-d-\mu+\frac{1}{2}d(1-k)} - \frac{d(1-k)}{1-d-\mu+d(1-k)} \qquad (A.7)\end{aligned}$$

$$\begin{aligned}\Delta\tilde{w}_1 &= \frac{n'_{11}}{n'_{11}+n'_{01}+n'_{12}} - \frac{n_{11}}{n_{11}+n_{01}+n_{12}} + \frac{n'_{10}}{n'_{00}+n'_{10}+n'_{01}} \\ &\quad - \frac{n_{10}}{n_{00}+n_{10}+n_{01}} + \frac{n'_{12}}{n'_{11}+n'_{12}+n'_{10}} - \frac{n_{12}}{n_{11}+n_{12}+n_{10}} \qquad (A.8)\\ &= \frac{1-d-\mu}{\frac{1}{2}d'(1-k)+1-d-\mu+\frac{1}{2}d(1-k)} - \frac{1-d-\mu}{1-d-\mu+d(1-k)} \\ &\quad + \frac{1}{2}\frac{d(1-k)}{1-d'-\mu+d(1-k)} - \frac{1}{2}\frac{d(1-k)}{1-d-\mu+d(1-k)} \qquad (A.9)\end{aligned}$$

$$\begin{aligned}\Delta\tilde{w}_2 &= \frac{n'_{12}}{n'_{11}+n'_{12}+n'_{01}} - \frac{n_{12}}{n_{11}+n_{12}+n_{01}} \\ &= \frac{1}{2}\frac{d(1-k)}{\frac{1}{2}d'(1-k)+1-d-\mu+\frac{1}{2}d(1-k)} - \frac{1}{2}\frac{d(1-k)}{1-d-\mu+d(1-k)} \qquad (A.10)\end{aligned}$$

I've omitted factors of $(1-s)$ from each Δw_i – a decoration ˜ has been added to the w to indicate this change. The substitution $d' = d + \delta$ leads to the fitness effects in (3.17).

A.2.2 Two dimension stepping-stone

$$\begin{aligned}
\tilde{w}_0 &= \frac{n_{00}}{n_0} + 4\frac{n_{01}}{n_1} && \text{(A.11)}\\
&= \frac{1-d-\mu}{1-d-\mu+d(1-k)} + \frac{d(1-k)}{1-d-\mu+d(1-k)} \\
\tilde{w}'_0 &= \frac{n'_{00}}{n'_0} + 4\frac{n'_{01}}{n'_1} && \text{(A.12)}\\
&= \frac{1-d'-\mu}{1-d'-\mu+d(1-k)} 1-d-\mu+\frac{1}{4}d'(1-k)+\frac{3}{4}d(1-k) \\
\tilde{w}_1 &= \frac{n_{11}}{n_1} + \frac{n_{10}}{n_0} && \text{(A.13)}\\
&= \frac{1-d-\mu}{1-d-\mu+d(1-k)} + \frac{1}{4}\frac{d(1-k)}{1-d-\mu+d(1-k)} \\
\tilde{w}'_1 &= \frac{n'_{11}}{n'_1} + \frac{n'_{10}}{n'_0} && \text{(A.14)}\\
&= \frac{1-d-\mu}{1-d-\mu+\frac{1}{4}d'(1-k)+\frac{3}{4}d(1-k)} + \frac{1}{4}\frac{d(1-k)}{1-d'-\mu+d(1-k)} \\
\tilde{w}_2 &= \frac{2n_{21}}{n_1} && \text{(A.15)}\\
&= \frac{1}{2}\frac{d(1-k)}{1-d-\mu+d(1-k)}
\end{aligned}$$

$$\begin{aligned}
\tilde{w}'_2 &= \frac{2n'_{21}}{n'_1} && \text{(A.16)}\\
&= \frac{1}{2}\frac{d(1-k)}{1-d-\mu+\frac{1}{4}d'(1-k)+\frac{3}{4}d(1-k)} \\
\tilde{w}_3 &= \frac{n_{31}}{n_1} && \text{(A.17)}\\
&= \frac{1}{4}\frac{d(1-k)}{1-d-\mu+d(1-k)} \\
\tilde{w}'_3 &= \frac{n'_{31}}{n'_1} && \text{(A.18)}\\
&= \frac{1}{4}\frac{d(1-k)}{1-d-\mu+\frac{1}{4}d'(1-k)+\frac{3}{4}d(1-k)}
\end{aligned}$$

Now, using $d' = d + \delta$ the four fitness effects contributing to the inclusive fitness effect

(3.21) are

$$\begin{aligned}
\Delta\tilde{w}_{0,0} &= \tilde{w}'_0 - \tilde{w}_0 \\
&= \frac{3dk^2 + 2dk - 4k + 4k\mu + 4\delta k - 5d + 4 - 4\mu - 4\delta)\delta}{(-4 + 4\mu + 4dk - \delta + \delta k)(-1 + \delta + \mu + dk)} \qquad \text{(A.19)} \\
\Delta\tilde{w}_{0,1} &= \tilde{w}'_1 - \tilde{w}_1 \qquad \text{(A.20)} \\
&\qquad\qquad 4\mu\delta k + 4k^2\mu d + 4\mu dk - 4dk - 4 - 3d\delta - 8d\mu - 4\mu^2 \\
&\qquad\qquad -4\delta\mu - 8k\mu - 4dk^2 - 8d^2k + 8d^2k^2 + 2d\delta k \\
&= -\frac{1}{4}\delta \frac{+\delta k^2 d + 4k\mu^2 + 4k + 8d + 8\mu + 4\delta - 4\delta k}{(-1 + \delta + \mu + dk)(-4 + 4\mu + 4dk - \delta + \delta k)(-1 + \mu + dk)} \\
\Delta\tilde{w}_{1,1} &= \tilde{w}'_2 - \tilde{w}_2 = -\frac{1}{2}\frac{d(1-k)^2\delta}{(-4 + 4\mu + 4dk - \delta + \delta k)(-1 + \mu + dk)} \qquad \text{(A.21)} \\
\Delta\tilde{w}_{0,2} &= \tilde{w}'_3 - \tilde{w}_3 = -\frac{1}{4}\frac{d(1-k)^2\delta}{(-4 + 4\mu + 4dk - \delta + \delta k)(-1 + \mu + dk)}. \qquad \text{(A.22)}
\end{aligned}$$

The final step ignores terms $O(\mu)$ and $O(\delta^2)$.

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1. Z. V. Finkel and A. J. Irwin. Cell size and the optimization of intracellular pigment concentration in unicellular algae. In preparation.

2. A. J. Irwin and P. D. Taylor. Evolution of altruism in stepping-stone populations with overlapping generations. *Evolution* Submitted.

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