# The Evolution of Social Organisms:

## Modelling Reproduction Strategy

**by**

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# Abstract

The focus of this thesis is on the study of reproduction strategy in the context of evolutionary and social-evolutionary theory. Much of the hierarchical structure that is evident in the natural world is due to major evolutionary transitions where individual subunits that once reproduced individually now reproduce only as part of a larger unit. Modelling and understanding the processes behind the evolution of this hierarchy can have applications in both biology and computer science. I argue that to explain the major transitions it is necessary to understand why an individual would reduce its reproductive success to invest instead in a higher reproductive process (i.e., reproduce collectively with other individuals).

To address this problem, a method for studying reproduction strategy was developed and is presented in this thesis. The method takes an abstract physiological approach to reproduction. It considers an individual as a quantity of resources and set of genes which define its reproduction strategy. I then investigate the advantages of different reproduction strategies and identify which strategies may dominate others. The strategies considered in my investigations include: an individual reproducing on its own; an individual gambling its total resources against those of multiple other individuals; or an individual sharing its reproductive effort with a partner or several other partners.

Starting with individual reproduction, I simply study why an individual might reduce its reproductive rate when, on the face of it, it seems that maximum fecundity should be the best option. The model is also motivated in light of current literature on life history and microbial ecology in particular. The results show how it can be advantageous for an individual to hedge its bets and delay reproduction; waiting instead until it has accumulated more resources and is less vulnerable to harsh periods. The results make predictions that are experimentally verifiable.

Given the model of individual reproduction, I reapply the growth equations to question whether there is any advantage to sharing reproductive effort through collective reproduction. This model also shows that it can pay to hedge one's bets and invest in the less vulnerable, but slower, collective reproductive strategy. The results show that there is a mathematical relationship between the number of parents and the up-front cost of reproduction spent on creating a new offspring – depending on the extra cost per parent, two parents may be the best strategy or perhaps many parents.

Looking in more detail at the transition from unicellular organisms to multicellular organisms, I model the macrocyst stage in the slime mould *Dictyostelium*. I consider how the macrocyst stage may be an early example of collective reproduction in protozoa. Here individuals aggregate to be ingested by a central cell which produces homogeneous

offspring. I assume that each individual is gambling on being the central cell and the model presented reveals under what conditions this is likely to be a good strategy when compared to individual reproduction. Again, the results show that there is an advantage to hedging one's bets and investing in the macrocyst rather than going it alone.

Finally I consider the origin of sexual reproduction in more detail. The traditional approach argues that the slower growth rate of sexually reproducing organisms means that there is a paradox concerning the origins and maintenance of sexual reproduction, especially when one considers males which do not contribute to their offspring. Extending the previous model of collective reproduction, I consider how many resources selfish individuals may contribute to their offspring. The results show that there is a lower bound to the resources individuals may contribute and that when there is a high amplitude of resource fluctuation, the sexual strategy can dominate an asexual strategy.

As well as the main body of work on the topic of individual reproduction, some further background work is also presented. The models use both mathematical and computer simulation models. These two approaches are compared and contrasted with reference to their value in generating good scientific explanations of the sorts of phenomena found in the types of systems I am studying.

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# Declarations

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**J. Bryden and J. Noble**, "Computational modelling, explicit mathematical treatments, and scientific explanation", *Artificial Life X*. (2006) 520–526.

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# Chapter 1

# Introduction

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A profound and mysterious theme in nature is the prevalence of hierarchical systems. In fact, the biota of our planet can be viewed as one large hierarchical system, with proteins made up of chains of amino acids, cells containing proteins, eukaryotic cells composed of organelles, multicellular organisms formed from eukaryotic cells, societies of multicellular organisms, and ecologies consisting of species groups.

Study of our natural history (Maynard Smith and Szathmáry 1995) indicates that (unsurprisingly) the lower levels of the hierarchy evolved before the higher levels. This recurring pattern of lower level units forming themselves into higher level units has been dubbed a *major evolutionary transition* (Szathmáry and Maynard Smith 1995). The major evolutionary transitions are summarised in Table 1.1.

There are still many mysteries around the major evolutionary transitions however

| | | |
|---|---|---|
| Replicating molecules | $\rightarrow$ | Populations of molecules in compartments |
| Independent replicators | $\rightarrow$ | Chromosomes |
| RNA as gene and enzyme | $\rightarrow$ | DNA + protein (genetic code) |
| Prokaryotes | $\rightarrow$ | Eukaryotes |
| Asexual clones | $\rightarrow$ | Sexual populations |
| Protists | $\rightarrow$ | Animals, plants, fungi (cell differentiation) |
| Solitary individuals | $\rightarrow$ | Colonies (non-reproductive castes) |
| Primate societies | $\rightarrow$ | Human societies (language) |

Table 1.1: The major transitions in evolution, in order of occurrence. Taken from the review by Maynard Smith and Szathmáry (1995).

(Maynard Smith and Szathmáry 1995). Taken separately, none of the transitions can be said to have a satisfactory individual explanation. Further to this, we lack a more general theory as to why the transitions occurred over and over again at each of the new levels. My main topic of interest, in this thesis, is therefore to build models (and modelling approaches) that work toward a unified theory of the transitions, while at the same time address each one individually.

The repeating pattern of the major evolutionary transitions indicates that there may be some common feature in individual sub-units from every level that leads them to form themselves into larger units. Perhaps there is some kind of social evolutionary force that binds these units together? I will be investigating this question further in this thesis.

The best place to start is to look for the common features of the transitions. It has been observed that all of the transitions follow one simple common principle: sub-units that could previously reproduce on their own can now only reproduce as part of the super-unit (Buss 1987, Szathmáry and Maynard Smith 1995). There are also common features shared by the sub-units: they all make copies of themselves which may or may not be perfect copies and they all compete with other similar individuals over resources. Based on these core features, the central question of this thesis is thus:

**The central question** *What factors are important for an individual that can reproduce on its own to evolve into a sub-individual that reproduces as part of a collective?*

This question is investigated further by first reviewing background literature in the next section (Section 1.1). In the following section (Section 1.2) I present the direction in which I will approach the problem of the major evolutionary transitions. With this in mind, I review current literature on social evolution in Section 1.3, involving a discussion on the origins of altruism and its potential role in the major evolutionary transitions. After concluding that altruism may not be the answer, I look instead at theories of *fitness* (an organism's ability to survive in an environment and produce viable offspring) in more detail in Section 1.5. The conclusion that the long term fitness of an individual (or an individual's lineage) may override its short term fitness leads me to the suggestion that this may be important in the major evolutionary transitions and this is the main topic of this thesis. The aims and results of the thesis are summarised in Section 1.6 and its contributions are discussed in Section 1.7.

## 1.1   Background

Given the central question of this thesis I look, in this section, to its relevance to computer science, evolutionary computation and ALife research. The dream of many ALife researchers is to employ principles inspired by evolutionary biology to implement computer systems that will use evolution to solve problems without supervision. Therefore, ALife research is interested in how evolution works over a long period of time – the sorts of timescales over which new functionality (e.g., vision or intelligence) evolves and over which several major evolutionary transitions have taken place.

To answer this question, we need to have a clear picture as to how evolution works. What are the simplest elements needed to generate evolution in a system? Evolutionary theory has been rooted in a gradualist perspective since Darwin's original theory on natural selection (Darwin 1859). Small heritable changes in offspring, combined with survival of the fittest, mean that individuals will make accretive advances in their functionality. Since the modern synthesis (Fisher 1930, Haldane 1932, Wright 1931), recent theories [e.g., (Dawkins 1982)] of these increases in individual functionality are based on natural selection pressure on reproducing individuals that are subject to mutations of their genetic code. Those mutations that increase an organism's fitness will proliferate in the population. This creates a ratchet effect [known as Muller's ratchet (Muller 1932)] where beneficial mutations are incrementally added to an organism's gene pool: there is no reversal because any organism with a detrimental mutation will not survive.

This approach has been applied with varying success in computer science. Genetic algorithms are now widely used (Mitchell 1998) for their search and optimisation functionality. A typical genetic algorithm finds optimal solutions by running multiple generations. Each generation has several solutions which are all different. The best solutions are picked using a *fitness function* (which scores solutions according to their competence in solving a problem) and are used for the next generation. However, fitness functions are very difficult to design to solve any one specified problem. Further, a fitness function that adapts to unspecified problems is severely challenging. To produce unsupervised new functionality, the fitness function must constantly change.

An alternative perspective to using genetic algorithms is the simulation of ecosystems to understand how evolution can drive new functionality within them. Such a simulation would of course have value within evolutionary biology as well. In fact a thought experiment of Gould (1989) questioned whether there is such an inevitability to evolution. Furthermore, if there is such an inevitability, what are the factors that it is contingent upon? Many proponents of Artificial life (Bonabeau and Theraulaz 1994, Ray 1994,

Bedau 1998) have argued that simulating evolution on computers can lead to important insights to this problem.

Attempts to simulate the evolutionary time-line in computer models have not gone well so far. A common opinion in biological circles (Dawkins 1989) is that core evolutionary theory is all we need to explain the evolution of life and its astounding complexities. This idea of evolution as a mechanism implies that it may be possible to generate an unsupervised system which will develop new functionality – perhaps in a computer. However, attempts to produce ALife computer models (Ray 1996, Adami 2006), which implement all the main features of evolutionary theory, have not produced much complexity of interest. It seems clear therefore that there is something missing, perhaps important factors that evolution is contingent upon have been omitted from the models that have attempted to recreate some of the complexity of life, or perhaps there is something missing from the evolutionary theory they are based on.

Perhaps the best thing to focus on is the increase in individual complexity (Szathmáry and Maynard Smith 1995). By making an individual more complex, but still competitive in an environment, it will develop its repertoire of functionality. However this is also problematic because there is no accepted measure for individual complexity. The problem with developing a measure for complexity is that there are at least three ways of quantifying it: genetic complexity (Adami, Ofria and Collier 2000), morphological complexity (Carroll 2001) and behavioural complexity (Bonner 1988). We currently lack an accepted measure for individual complexity (Adami 2002, Szathmáry and Maynard Smith 1995) that successfully captures all these different attributes.

Some have argued that we should take a neutral perspective on the adaptive advantages of individual complexity. Gould argues that since there is a lower bound to individual complexity, random drift would mean that it should naturally increase (Gould and Eldredge 1993). While this is an attractive point of view, the major evolutionary transitions show obvious increases in individual complexity – so, any theory explaining them would run against Gould's thesis and potentially shed some light on the problem of open-ended evolution.

Given the need for explanations of the mysteries of the major evolutionary transitions, and the desire for theory which demonstrates increases in individual complexity and perhaps open-ended evolution, I have chosen the major evolutionary transitions as the topic in this thesis. In the next section I outline the problems of the major evolutionary transitions in more detail.

## 1.2   The major evolutionary transitions

For the major transitions to occur, this requires that an individual must invest in a higher reproductive process. I define a higher reproductive process as follows:

**Higher reproductive process** *A process of generating new offspring with two or more individuals having some genetic stake in, and contributing resources to, the new offspring.*

From this perspective, I seek to explain why an individual might evolve to be part of a higher reproductive process and investigate, based on current data on the evolutionary transitions, the nature of these higher reproductive processes.

From the perspective of the evolution of complexity, it is this investment in a higher reproductive process that is important. Rather than explaining the individual transitions separately, a general theory that explains why an individual would invest in a higher reproductive process could explain the increase in complexity that is required to take part in a higher process, and explain the increase in complexity of the individual.

Of course, just explaining an individual's contribution to a higher reproductive process does not fully explain the evolutionary transitions – these require (Maynard Smith and Szathmáry 1995) that the sub-individuals may not freely reproduce[1] on their own, they may only reproduce as part of the higher reproductive process. What can be said is that a stable higher reproductive process is likely to become a permanent one. However, in some cases (e.g., the evolution of eukaryotes or the evolution of multicellular organisms) they also require the evolution of a new super-individual, made up from sub-individuals. Indeed, each transition has different peculiarities (only two are considered in more detail in this thesis). These are interesting topics, but they are outside the scope of this thesis.

In this thesis I therefore assume that, for a transition to occur, an individual that normally reproduces on its own must reduce its ability to make copies of itself and contribute resources instead to a group of individuals performing a higher reproductive process. The group must also be resistant to selfish individuals that, instead of contributing to the group, make proliferative copies. In light of this, I review current literature in social evolution in the next section before considering the plausibility of attempts to apply social evolutionary theory to the task of explaining the major transitions and especially to the question of why an individual might contribute to a higher reproductive process.

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[1]In multicellular organisms, many cells reproduce for the benefit of the super organism, however if they reproduce freely then they are cancerous

|  |  | Positive | Negative |
|---|---|---|---|
| | | \| *Outcome for recipient* | |
| *Outcome for* | **Positive** | Mutualism | Selfishness |
| *actor* | **Negative** | Altruism | Spite |

Table 1.2: The four different outcomes of social behaviour for an actor and a recipient. The way an outcome is measured is through the change in the *fitness* of an individual. Fitness is here defined as the change in the reproductive success of an individual over its lifetime.

## 1.3   Review of Social Evolution

The majority of biological work in social evolution theory applies to the evolution of eusocial insects (Hamilton 1964, Wilson 1975, Bourke and Franks 1995, Maynard Smith and Szathmáry 1995, Queller 2000, Wilson and Hölldobler 2005, Foster, Wenseleers and Ratnieks 2006) and microorganisms (Hudson, Aukema, Rispe and Roze 2002, West, Griffin, Gardner and Diggle 2006). Many social evolution models consider organisms that show reproductive specialisation: only a few privileged individuals can reproduce and most are sterile workers which cannot. Social evolution theory makes predictions as to why such systems are stable to mutations from non-reproducing individuals into reproducing individuals.

In general, social evolution studies the actions that individuals perform which have consequences on others. It is most simple to consider the case where there is an actor and a recipient. There are, then, four different outcomes for the behaviour of the actor and its consequences on the recipient. These are shown in Table 1.2.

The standard way of analysing such a table is to compare like for like. Individuals that perform an action are compared with individuals that are identical in every other way except that they do not perform that action. In these cases one can read from the opposite corner of the table – e.g, not performing a selfish action will have an altruistic outcome, or not performing a spiteful action will have a mutualistic outcome.

A naive analysis of Table 1.2 leads to some simple conclusions. Any actor which repetitively performs actions that have a negative outcome will lower its own fitness. One that performs altruistic actions, in particular, will lower its own fitness at the expense of increasing the fitness of a competitor. Therefore, it seems that any heritable trait which leads to an altruistic action will be less represented in the next generation and will eventually become extinct.

A famous example of the consequences of selfish behaviour is the tragedy of the commons (Hardin 1968, Foster 2004). Here an open pasture (a commons) is considered where herdsmen may graze as many cows as they like. For each herdsman, the best

strategy is to graze as many cows as they possibly can – a herdsman that grazes his cows more profits more and can therefore buy more cows. Unfortunately this strategy, when adopted by many herdsmen, leads to over-grazing and the pasture is ruined. There is a need for unselfish, or altruistic, behaviour from all the herdsmen for the community to survive.

Another example is eusocial insects. Considering *hymenoptera*, worker bees are performing an altruistic action. These individuals do not lay eggs and therefore have zero fitness.[2] However, *prima facie*, they can promote their own genes by laying eggs. The offspring would then be egg-laying themselves and this could the go on to destroy the collective reproduction mechanism and possible damage the whole colony, much in the same way as the herdsmen destroy the commons, or cancer cells invade a multicellular organism. Of course, since bees are quite mobile, a bee from one colony may then invade other colonies – leading, potentially, to the destruction of eusocial behaviour in the whole species. There are, however, theories as to why this doesn't happen: these are outlined in the next section.

### 1.3.1 Altruism

Given that a selfish organism will be more likely to reproduce and give birth to other selfish organisms, it seems unlikely that any altruistic traits can survive. However, there are various theories that show how altruism can indeed survive in a population (Nowak 2006). These theories do depend on different concepts of altruism [see a recent review by Kerr, Godfrey-Smith and Feldman (2004)] but the basic principle given in Table 1.2 still holds. The theories most relevant to the questions looked at in this thesis are kin selection and green beard theory and these are outlined below. I also consider two-player games which have also proved useful in understanding how altruism can survive in a population.

#### 1.3.1.1 Kin selection and inclusive fitness

One theory looks at relatedness between individuals (Hamilton 1964). It has been noted that individuals in a colony are likely to be related to one another. Looking at human families as an example, a mother will typically share 1/2 of her genes with her offspring (the other 1/2 will come from the father who is unlikely to be related to the mother). When we consider genes that differ from the population average, all of her offspring will also have 1/2 of their genes in common with each other. Hamilton called this ratio *relatedness*

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[2]Actually the correct terminology here is *direct fitness* – this is defined as an individual's reproductive success against its local neighbours, see Section 1.3.1.1

and denoted it by the variable $r$. Simply put, the basic theory is that when an individual contributes to a related other, it is effectively promoting the survival of its own genes. Hamilton shows that, when an altruistic act has a fitness benefit $B$ to a recipient and a fitness cost $C$ to the actor, then the act will be advantageous when $rB > C$. Maynard Smith (1966) coined the term *kin selection* to describe this effect. Groups of kin should cooperate, or work together, to maximise each others' fitness.

The way Hamilton approached relatedness was to consider how related individuals are due to ancestral and dependent relations. A more recent approach (Frank 1998) instead considers gene frequencies in a population. This is based on the Price equation (Price 1970, Frank 1998) which models how a character trait might change in a population:

$$\bar{w}\Delta\bar{z} = \text{Cov}(w,z) + \text{E}(w\Delta z) \,, \tag{1.1}$$

where $w$ is fitness and $z$ is a quantitative character trait ($\bar{w}$ and $\bar{z}$ are population means). The covariance term ($\text{Cov}(w,z)$) in this equation shows how a character trait will change its frequency in a population due to its correlation with fitness of those that express the trait. The expectation term ($\text{E}(w\Delta z)$) covers all the other factors that influence fitness.

The Price equation can be used to encapsulate the notion of a trait that increases the fitness of an individual according to the frequency of its expression in others around it and Hamilton's formula can be derived (Frank 1998). The difference here is that this focuses on a trait and surrounding individuals that express the trait need not be related by descent. When the effects of other individuals that have a trait are included in the measurement of its fitness, this called *inclusive fitness*. This can be contrasted with *direct fitness*, which is the way a trait will change in a population without any effects due to its frequency in the population.

### 1.3.1.2 Green beard

The ability to recognise altruistic traits in others may well be an explanation for altruistic behaviour. Dawkins (1982) gives the example of the green beard. This gene has a trait which makes a quantifiable difference (such as having a green beard) to the way an organism is perceived by, or interacts with, other organisms. The same gene also has a second cooperative/altruistic trait that means it will be altruistic to those that display the characteristics of the gene. It is simple to see how such a gene would proliferate through a population – all those with the gene would be at an advantage. The green beard strategy is susceptible to cheating behaviour [such as Müllerian mimicry (Müller 1879)] however.

|  |  | Opponent's action | |
|---|---|---|---|
|  |  | **Cooperate** | **Defect** |
| Player's | **Cooperate** | 2 | 0 |
| action | **Defect** | 3 | 1 |

Table 1.3: The prisoner's dilemma payoff matrix – a player's payoff depends on what its opponent does. The joint pay-off for cooperator-cooperator is 4, cooperator-defector is 3 and defector-defector is 2. Cooperate-cooperate is clearly the best joint strategy but defecting is the best individual strategy whatever the other player is doing.

### 1.3.1.3    Two-player games

Other abstract theories looking at altruism consider two-player games. The standard form of a two-player game involves individuals choosing one of two options. According to which option is chosen, individuals receive some reward from a pay-off matrix. The two arrangements of the matrix that involve some form of altruism are the prisoner's dilemma game and the hawk-dove (or snowdrift) game. These games are interesting and well studied because they yield to simple analysis (Maynard Smith 1982).

The prisoner's dilemma game looks at two prisoners which have been caught on some minor offence. They have both committed a more serious offence and are both in a position to give evidence against the other prisoner and receive a pardon from the minor offence. However, if they both do this, they will both be convicted of the major offence. The game is written in the form of a payoff (where a payoff corresponds to the value of the outcome of a decision) matrix in Table 1.3. The important point to note is that the joint pay-off for two cooperators is better than the joint pay-off for other strategies. However, an individual's best strategy is to defect when the other is cooperating.

When the prisoner's dilemma game is played repetitively, without memory of past actions, between two opponents (or indeed between random opponents in a group), all players quickly descend to playing defect each turn. Reading down each column on the matrix, the best strategy for a player is always to defect – if the opponent cooperates, then it's best to defect; if the opponent defects, then it's also best to defect. However, it has been shown that a 'Tit-for-tat' strategy can work quite well between two opponents when each has a memory of the actions of the previous round (Axelrod 1984). Here, a player starts by cooperating and then records the opponents move each turn and plays the same move in the next round. This strategy cooperates with other cooperating strategies and defects with other defecting strategies, and performs well when it encounters itself. The idea that a viscous group (a group which sticks together) could promote internal cooperation by playing the game only with each other was also presented (Axelrod and Hamilton 1981, Axelrod 1984). The viscous group theory has been disputed however

|  |  | Opponent's action | |
| --- | --- | --- | --- |
|  |  | **Cooperate** | **Defect** |
| Player's | **Cooperate** | 3 | 1 |
| action | **Defect** | 4 | 0 |

Table 1.4: The snowdrift (otherwise known as hawk-dove) payoff matrix. The joint payoff for cooperator-cooperator is 6, cooperator-defector is 5 and defector-defector is 0. Cooperate-cooperate is clearly the best joint strategy but it only pays to defect when the other player is cooperating.

(Marshall and Rowe 2003a, Marshall and Rowe 2003b).

The other game of interest to altruism is the hawk-dove game (Maynard Smith 1982). This is also known as the snowdrift game as it makes a good story. In this story, two people driving home in their cars encounter a snowdrift. If neither person gets out of their car and clears the drift then they will both be stuck. Though one person can clear the drift on their own, it is better if both help each other. The game is written in the form of a payoff matrix in Table 1.4.

The difference between this game and the prisoner's dilemma game is that if both players defect, then it pays for one to cooperate. Conversely, if both players cooperate then it pays for one to defect. So, when a population of competing individuals playing this game repetitively is considered, this will result in a mixed population of defectors and cooperators. If there are too many cooperators, then defectors will do well and if there are too many defectors, then cooperators will do well. The stable ratio of cooperators to defectors can be predicted (Maynard Smith 1982).

### 1.3.1.4   Summary of Altruism

I have presented two types of altruism in this section. The first type is inclusive fitness where an individual will reduce its fitness so that its genes may proliferate through other related individuals.

The other type is the prisoner's dilemma game where we don't assume individuals are related. When played only once, it would never be a good strategy for an individual to cooperate, however when played many times (the iterated prisoner's dilemma) social rules may emerge (such as Tit-for-tat) which mean that cooperation can be a good strategy. This said, evidence of the iterated prisoner's dilemma in biological systems is scarce (Brembs 1996).

In summary, the models I have presented on altruism in this section have a common theme. While an individual does in fact do something that is detrimental to their own reproductive chances, there is always a pay-off – through genetic relatedness or recipro-

cation. This means that any so-called 'altruistic' action is, in fact, a selfish one. In the next section I present and critique applications of this theory to the problem of the major evolutionary transitions.

## 1.4 Explaining the major transitions

Given the preceding outline of the theory of social evolution, I look in this section at its potential application to the question of why an individual might invest in a higher reproductive process. Essentially there are two styles of approach in the literature; these differ on whether the cooperative behaviour is affects the reproductive strategy of an individual or not. First, I consider that individuals may, through non-reproductive cooperative interactions, form themselves into a unit which will then benefit from a centralised reproductive strategy. The second approach is that the reproductive mechanism is essentially cooperative in the first place – through sibling relatedness.

### 1.4.1 Non-reproductive cooperative interactions

Much work on the transition to multicellularity (Wolpert 1990, Michod 1999, Pfeiffer and Bonhoeffer 2003) assumes that the main benefits of multicellularity are from the division of labour between cells. Examples of this are cooperative ATP production (Pfeiffer and Bonhoeffer 2003) or cooperation between reproductive cells and motile cells (Michod 1999). These models require that individuals cooperate when labour is divided. Michod (1999) gives an explanation of such cooperation through the evolution of policing – there being a mechanism whereby defecting cells are harmed. The other model (Pfeiffer and Bonhoeffer 2003) suggests that clustering itself may be good way of both maximising the benefits of cooperation and reducing the potential of non-cooperators to invade.

Alternatively, there is an argument that, as local relatedness goes up (through reproducing within higher units), so interactions get more cooperative, and these fitter individuals will dominate (Witting 2002b, Witting 2002a, Witting 2003). Thus, the beneficial interactions from this greater cooperation can facilitate transitions from asexual reproduction to sexual reproduction and eusocial reproduction (Witting 2002b).

One criticism of this approach comes from analysis of cooperative interactions between kin. Models, and some experimental data, predicts that the benefits of interactions between kin can be negated from the increased competition between kin caused by an increased local growth rate (Queller 1994, West, Pen and Griffin 2002). Secondly, if we accept that cooperation interactions can be so beneficial that a cooperative group may be

formed, this still leaves open the question as to how, or why, some individuals started to reproduce on behalf of others.

Models that show differentiation between reproducing *germ-line* individuals and non-reproducing *soma* individuals require that the population is genetically homogeneous (Michod 2005, Michod, Viossat, Solari, Hurand and Nedelcu 2006, Michod 2006). The benefit of this is that labour can be divided between survival (e.g., vegetation) and reproductive effort. Every individual unit must make a trade-off between its investment into survival (*v*) and its investment into reproductive effort (*b*) – so the fitness of an individual is given by $w = vb$. Given an homogeneous population (and therefore no selection between groups) Michod (2006) argues that the fitness of the group can be defined by the mean viability of the group (*V*) times the mean fecundity of the group (*B*). The fitness of the group (*W*) is therefore greater than the fitness of the average cells when there is a negative covariance between viability (*v*) and fecundity (*b*) at the cell level, or,

$$W = VB = \bar{w} - \text{Cov}[v, b] \tag{1.2}$$

A negative covariance between fecundity and viability means that cells must specialise into fecundity (be germ-line cells) or viability (be vegetating soma cells).

So, if we believe that clonal (genetically homogeneous) groups may be formed, then it makes sense that some individuals can reproduce on behalf of others. However, if the group is not homogeneous then the group is susceptible from mutant cells which breed for themselves (and therefore don't contribute to group fecundity *B*), but cooperate in every other way. Group selectionist arguments may explain this, however they have long been contentious (Maynard Smith 1964, Williams 1966a). In fact, recent work has shown to be of some promise (Traulsen and Nowak 2006), but requires that selection within groups to be very weak – this is not plausible in the case of a mutant replicator. In the next section, I consider organisms that are not necessarily genetically homogeneous, but are related to each other in such a way that they will cooperate with a higher reproductive process.

### 1.4.2 Sibling relatedness

Considering the transition to eusociality, Hamilton's theory of relatedness (Hamilton 1964) has proved to give important insights into the reproduction strategy of eusocial insects. This transition is characterised by the evolution of a differentiation between two types of female: worker females that cannot reproduce and queens that reproduce on behalf of the worker individuals. One interesting characteristic of bees is that male bees are *haploid* (have only one set of chromosomes) whereas female bees are *diploid* (having two

sets of chromosomes). This means that females only give half of their genes to offspring (through meiosis) whereas males give all of their genes to offspring.

Given Hamilton's theory, an explanation can be given as to why worker bees do not lay their own eggs. A queen that mates once with a male bee, and produces many offspring, will generate sisters that have a relatedness to each other of 3/4 (Maynard Smith and Szathmáry 1995) – each sister having half of the queen's genes and a whole set of the male bee's genes. Any sister that then reproduces will still only be related at 1/2 to their own offspring. So, there is a benefit to protecting one's kin's offspring over one's own.

However, this simple application of Hamilton's relatedness theory to social insects appears to be flawed (Bourke and Franks 1995, Queller 2000, Wilson and Hölldobler 2005, Foster et al. 2006). While sisters in insect colonies are closely related to each other (3/4), they are also less related to males (1/4). This means that, on average, siblings have the same relatedness as mothers do to offspring (1/2). It does not seem plausible that siblings could recognise the difference between males and females at larval stages at the origin of eusociality (Maynard Smith and Szathmáry 1995). Another argument (Grafen 1986) suggests that different sex-ratios at different times may explain why this strategy might persist. However, if a sister is able to self-fertilise and produce clones as offspring (relatedness of 1) then this would invade.

Even if we are convinced by the haplodiploid theory, there is still one more problem. Termites have similar reproduction mechanisms to the other haplodiploid eusocial organisms, yet they are not haplodiploid (Maynard Smith and Szathmáry 1995). Since the haplodiploid perspective does not appear to explain all the eusocial insects, it may not be a good general theory for explaining why there is differentiation between worker organisms and the queen (or reproducing) organisms.

### 1.4.3 Summary

In this section, I have discussed various theories attempting to explain the origins of major evolutionary transitions. While kin selection shows that an individual can invest in the reproductive success of another that is related to it, Hamilton's theory predicts that the benefits must significantly outweigh the costs. When it comes to reproduction itself, the reason why a mutant individual should choose to invest resources in a higher reproductive process rather than invest resources in its own (100% related) offspring is still unclear. There must be some benefit to doing this, and that benefit is not explained by current models. To approach this question from a different perspective, I look at fitness in more detail in the next section.

## 1.5 Fitness

In the previous section, I reviewed social evolution, where an individual may transfer its direct fitness to others so that its genes may prosper. This approach does not seem to be appropriate for the major evolutionary transitions. One assumption taken by the social evolutionary literature is that fitness is defined as the number of adult offspring (Hamilton 1964) – *Hamiltonian fitness*. This does not take into account that this may be dependent on the environment. In this section I review literature concerning how we might define a measure of fitness, including those definitions that do consider the importance of the environment.

Historically, agreement on a simple, but empirically applicable, mathematical definition of fitness has always been a controversial issue. At first, fitness metrics just counted the number of offspring. However, there are conflicts between the number of offspring and the viability of the offspring (Williams 1966a, Sober 2001, Michod 2006). Thus an individual's reproductive success was seen to be the most significant factor. This can be represented by the expected number of offspring (Mill and Beatty 1979).

The significance of randomly varying environments was highlighted by the work of Cohen (Cohen 1966, Cohen 1968). This showed that the arithmetic mean of the population growth rate over variable environments was not the appropriate way to calculate the long-run growth rate. Rather the geometric mean should be used (see Section 1.5.1 for more details). The theory was applied to the question of delayed reproduction (Tuljapurkar 1989, Tuljapurkar 1990a, Tuljapurkar 1990b). This work shows that delayed reproduction is advantageous in fluctuating environments through the mechanism of generating dormant immatures (e.g., seeds) that are invulnerable to the environmental fluctuations.

In general, this approach is about looking for evolutionary attractors (Metz, Nisbet and Geritz 1992) which are the optimal strategy over a range of extrinsic environments. The approach becomes more difficult however when the environment is also defined by its residents (Metz, Mylius and Diekmann 1996). A typical approach is that the environment is defined by the density of the local population (Roughgarden 1971, Roughgarden 1979). Roughgarden (1971) shows that the direct fitness of a trait can depend on population density.

Given the significance of the environment, much modelling is moving away from the simple ideas of maximisation of Hamiltonian fitness. Under density dependence there is no population growth – thus it suffices to consider the invasion of mutant types into the population (Metz et al. 1992, Mylius and Diekmann 1995). Rather than maximising fit-

ness, relative fitness (or 'soft selection') becomes more important (Geritz, Kisdi, Meszéna and Metz 1998, Meszéna and Gyllenberg 2005) especially when considering models of speciation or evolutionary branching.

As well as density-dependence, models may also consider frequency-dependent selection (Maynard Smith and Price 1973, Maynard Smith 1982). Here a trait's fitness will vary according to its frequency in the population. From this perspective came the idea of an *Evolutionarily Stable Strategy* (ESS) – a strategy (or a community of strategies) which is stable to invasion from mutant invaders. When models that combine both frequency- and density-dependence are considered, frequency-dependent selection is at best trivial when environmental feedback (uni-dimensional feedback environments) is the same for all traits (Heino, Metz and Kaitala 1998). Multi-dimensional feedback environments, where individuals have a different response to the environment, are required.

The inclusion of density-dependent selection, and environmental fluctuation, perspectives therefore means that fitness should be calculated over the long term, not the short term. In fact, the reproductive success explanation of fitness has been criticised (Beatty and Finsen 1989, Sober 2001) due to the fact that it only considers fitness over a short term. As Beatty and Finsen point out (Beatty and Finsen 1989), a strategy that has a high number of offspring in the short term may actually have a lower number of offspring in the long term. This is illustrated by the cartoon in Figure 1.1.
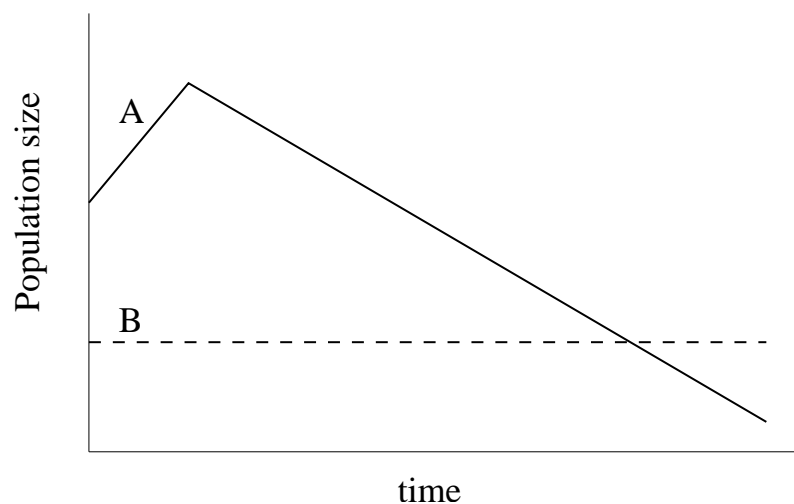


Figure 1.1: A comparison of short and long term strategies. At first, population A is more successful. However, over a long time period of many generations, population B becomes the more successful.

## 1.5.1 Geometric mean fitness

In order to introduce the importance of the geometric mean fitness, I look at bet-hedging (Seger and Brockmann 1987). To give an overview of bet-hedging theory, we may imagine a gambler with a sizeable amount of stake money. There are various horses in various races that she may back. If she bets all her money on the best horse she will have the highest *expected value* (the average profit she will make if the race was run several times). However, if the horse loses she will lose all her stake money. The best long term strategy is to gamble her money (hedge her bets) on several horses and make a small profit on the few that win.

In biological terms, bet hedging theory argues that an individual in an unpredictable environment may reduce its average fitness (i.e., the arithmetic mean of its growth rate over the different environments) if this also reduces its variance of fitness (Seger and Brockmann 1987). So, by adopting strategies that may produce fewer offspring under some circumstances, an individual can increase its offspring over a longer period of time.

The long term growth rate is calculated by considering the geometric mean growth over variable environments (Cohen 1966, Cohen 1968, Seger and Brockmann 1987). Consider two environments A and B. The population grows with rates $r_A$ and $r_B$ respectively in the two environments. For example, in environment A,

$$p_{t+1} = r_a p_t, \tag{1.3}$$

where $p$ is the size of the population. The geometric mean growth rate over time $T$ is given by

$$r_g = (r_A^{T_A} r_B^{T_B})^{1/T}, \tag{1.4}$$

where $T_A$ and $T_B$ are the amount of time spent in environments A and B respectively. The number of environments can be increased by adding extra terms to Equation (1.4). This also allows us to analyse empirical population growth data to test the theory (Boyce and Perrins 1987, Wilbur and Rudolf 2006, Venable 2007).

From a modelling perspective, the geometric mean can therefore be used to model long term growth rates of different strategies under fluctuating environments (Tuljapurkar 1990a, Tuljapurkar, Horvitz and Pascarella 2003). Typically environments extrinsically fluctuate under some distribution – the environments are not determined by the residents. When we look at modelling fitness under density dependence, the environment is determined by the population density and thus the distribution of environments is undetermined.

### 1.5.2 Summary

To summarise, I outline the various concepts of fitness covered in this section and the previous section. These are summarised in Table 1.5.

| Type of fitness | Description |
| --- | --- |
| Direct fitness | The expected reproductive success of a trait/organism due to its phenotype |
| Inclusive fitness | The expected reproductive success of a trait/organism due to its phenotype and the frequency of the trait/related organisms in the population |
| Group mean fitness | The average fitness over a group |
| Group fitness | The success of a group in making new copies of itself (reproducing itself) |
| Arithmetic mean fitness | The arithmetic mean of the reproductive success of an organism over different environments |
| Geometric mean fitness | The geometric mean of the reproductive success of an organism over different environments |

Table 1.5: Various measures of fitness found in biological literature. Direct / inclusive fitness are introduced in (Hamilton 1964, Frank 1998) (see Section 1.3.1.1), group mean / group fitness are introduced in (Michod 2005) (see Section 1.4), and arithmetic / geometric mean fitness are introduced in (Seger and Brockmann 1987) (see Section 1.5.1).

The theory presented in this section is interesting and relevant to the topic in this thesis because it shows how individuals may reduce their reproductive success for some longer term advantage. It is important to consider fitness over a long term and/or a wide range of environments – i.e., an ecological perspective.[3] By considering reproductive strategy from this ecological perspective, it may be possible to explain why an individual might invest in a higher reproductive process.

## 1.6 The thesis

The central question of this thesis is to study why an individual might contribute to a higher reproductive process (see Section 1.2). I have therefore developed models exploring this question. The perspective I have taken has largely been inspired by ALife modelling (see Section 1.1).

I consider individuals as resource containers which acquire resources (which may represent energy but may also represent territorial ownership, money and many other things)

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[3]See work by (Coulson, Benton, Lundberg, Dall and Kendall 2006) for more discussion on the importance of ecology in evolution

and expend them on maintenance (including growth) and reproduction. The strategies that an individual might employ to encounter and store resources and reproduce depend on its genes. Since I am interested in reproductive strategy, I compare strategies of individuals that are basically the same in every way except the way in which they reproduce. The modes of reproduction I have studied are inspired by the major evolutionary transitions.

My review of fitness in Section 1.5 shows that, through bet hedging, individuals may produce fewer viable offspring in some seasons so that they may optimise their reproductive success over many different seasons. Another idea, shown by Figure 1.1 has that an individual's fitness over a long term is more important than its fitness over the short term. The ALife approach is well suited to analysing a very long term perspective of reproduction strategy. Rather than considering a model of a few seasons, a computer model of a population can analyse the effects over many generations. Large fluctuations in population numbers can be modelled. This gives the potential to study reproduction strategy over a timescale where it is possible that transitions could occur.

As detailed in Section 1.2, the major evolutionary transitions require that an individual must reduce its direct fitness to contribute to a higher reproductive process. I have argued that, while inclusive fitness theory may well play a major role in the transitions, it has not yet provided a simple explanation as to why an individual may do this. If an individual may reduce its reproductive success when there is some pay-off in the long term, then maybe this is a selfish (or at least mutualistic) reason to invest resources for some genetic stake in a higher reproductive process?

The central questions considered in this work are therefore as follows. From a methodological perspective, see Section 1.5, I look for analytic tools that will allow for comparison of strategies over density dependent environments – i.e., environments where the availability of resources fluctuates between conditions for growth and decline of the population. Density dependence is likely to be important in the major evolutionary transitions because, under growth conditions, Hamiltonian fitness models apply.

The first model covers questions that are applicable to the resource allocation strategies of clonal reproducers. I consider the models of delayed reproduction developed by Tuljapurkar (1990a). I question whether the results may be reproduced in terms of resource allocation strategy under resource variation (instead of through dormant immatures). This means that, with analytic tools for comparing strategies under density dependence, I can identify optimal strategies under density dependent conditions.

The second model considers the strategy of an individual allocating resources to reproduction as part of a larger group, rather than allocating resources to clonal reproduction. Again, this may be modelled under density dependent conditions. Effectively, this

model questions whether it is plausible to invest in a higher reproductive process. It also questions whether unpredictable resource fluctuations therefore a significant factor.

Normally an individual will reproduce when it is healthy and has accumulated a fair number of resources. In the third model, I consider if there are other points in an individual's life history where it may reduce it's chances of survival to increase its chances of reproduction? When an organism has a low number of resources, its survival is increasingly unlikely. Are there biological examples of social mechanisms showing how an individual may completely sacrifice its short-term fitness and invest instead with a larger group of which it has a small long-term stake? What are the consequences of this on the transition from unicellular organisms to multicellular ones?

Finally, when many individuals are involved in a shared reproductive effort, there is a need for individuals to cooperate. If one contributes less resources than the others it may get some unfair advantage. It is possible that, through invasion of uncooperative individuals, the benefits of collective reproduction could be negated. Considering the simplest case (where only two parents contribute to offspring), what are the consequences of this on the transition from individually reproducing organisms to sexually reproducing organisms?

### 1.6.1 Thesis overview

After this chapter, I continue the thesis with a discussion of the methodology that I will use throughout the work. Based on the paper by Bryden and Noble (2006), Chapter 2 considers the two modelling approaches in the work presented here: computer simulation and mathematical modelling. Much of the mathematical modelling I have done was first done by computer simulation. Computer simulation has allowed me the freedom to explore the often complex dynamics of evolutionary systems. I have also derived mathematical theory which presents an alternative analytic approach.

The modelling chapters of this thesis employ an artificial life perspective [in line with (Fontana, Wagner and Buss 1994, Judson 1994, Epstein and Axtell 1996, Axelrod 1997, Kitano, Hamahashi, Kitawaza, Takao and Imai 1997, Stewart 1997, Bedau 1999, Bedau, McCaskill, Packard, Rasmussen, Adami, Green, Ikegami, Kaneko and Ray 2000, Kitano 2002, Wheeler, Bullock, Di Paolo, Noble, Bedau, Husbands, Kirby and Seth 2002, Di Paolo, Noble and Bullock 2000, Silverman and Bullock 2004)] to the study of reproduction strategy. The results of these studies are then used to address the problems introduced in Section 1.6. I have endeavoured to produce explanations that address the important issues, but are minimally simple. All the models therefore use a very similar

modelling approach which is intended to capture the most important attributes of a system in which evolution may occur. Individuals are placed in a closed environment of limited size; they receive resources and may grow and/or reproduce. The input of resources to the system may be stable or fluctuate. When an individual's resource levels reach a lower threshold, the individual will die – thus the models can implement large population fluctuations (as required by the questions in Section 1.6. Given this simple setup, I investigate the optimal reproduction strategies under various scenarios.

The first modelling chapter (Chapter 3) looks exclusively at individual reproduction. Here individuals receive resources from the environment and, when they reach a threshold, split into two new equal organisms. The model is motivated in light of the literature on delayed reproduction, resource allocation between growth and reproduction and unicellular reproduction the model is introduced and motivated in this light. The model studies how an individual may change an upper resource threshold which determines how many resources it will accumulate before reproduction. By increasing this threshold this means that the individual is, rather than increasing its direct fitness by reproducing immediately, hedging its bets. The work replicates results from the previous models of delayed reproduction demonstrating that unpredictable resource fluctuations are significant. This shows that the mechanism of delayed reproduction presented is a valid one. By saving resources for later, individuals (or their offspring) are less vulnerable to unpredictable resource fluctuations that cause population increases and decreases (like those associated with density dependence).

Two analytic techniques are employed in the chapter. These are used to consider trade-offs, under different amplitudes of resource fluctuation, between the threshold at which an individual may reproduce and two parameters: the upfront cost of reproduction and the cost of growth/maintenance. The first analytic technique uses Theorem 2 (in Appendix A) to show what happens when an individual has a different reproductive success (I actually consider the lineage growth rate, but this translates into reproductive success) over two different resource regimes. The theorem is a more generalised version of Corollary 3 which shows that the ratio of an individual's (or a lineage with the same reproduction strategy) growth rate over the two regimes defines which individuals are the fittest (see Figure 1.2 for an example). This theorem, applied through this model, assumes that there is no trend in the total population and thus allows for analysis of varying growth over different environments under the assumption of density dependence.

The second type of analysis considers what happens when the population is close to an equilibrium level and resource levels are dependent on population density. I show how an individual which has a lower equilibrium resource level will invade a population with a
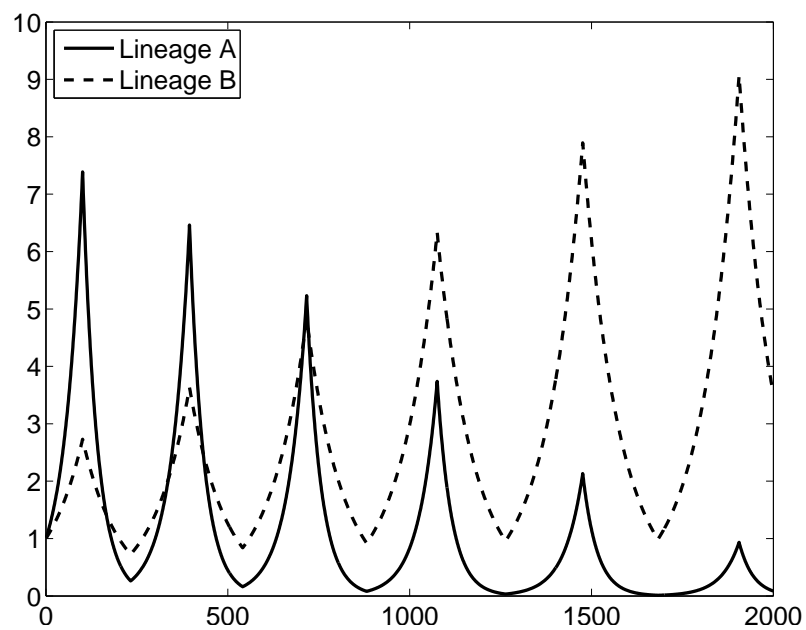
Figure 1.2: Two lineages compete with each other. Lineage A has both faster growth and decline rates compared with lineage B. However, lineage B has a greater ratio of growth to death rates so, as predicted by Corollary 3 (in Appendix A), will dominate when there is no overall trend in total population growth. Lineage A: grows at 0.02 births per individual per timestep, declines at 0.025 deaths per individual per timestep. Lineage B: grows at 0.01, dies at 0.01.

higher equilibrium resource level (see Figure 3.7 for an explanatory cartoon). The mathematical models predict the equilibrium resource levels for similar parameter regimes as those that were used for comparing growth ratios.

Chapter 4 describes an abstract model which looks at whether there is a selfish advantage to reproduce as part of a collective. Theorem 2 (from Appendix A) is used to show parameters where the growth ratio (i.e., the long term fitness) for a collectively reproducing individual is better than that of a individually reproducing individual. Collectively reproducing individuals invest less in their offspring so are less vulnerable to periods of low resources. Different numbers of parents are considered but all parents are considered to be unselfish in this model (i.e., resources are shared evenly between parents and offspring, after reproduction).

In light of the results from Chapters 3 and 4, a model of collective reproduction in slime mould is considered in Chapter 5. This model studies the macrocyst stage of slime mould which is triggered by harsh environmental conditions (Raper 1984). Starving individuals may hedge their bets by gambling all their remaining resources, their lives and their genes on being the genotype of the macrocyst and then surviving the harsh conditions.

Given that parents are unselfish in Chapter 4, Chapter 6 considers a scenario where parents are selfish and may contribute less resources to offspring. For simplicity only two parents are considered, so the model is motivated in light of the literature on sexual reproduction. This chapter uses game theory to model what is the expected lower bound to the number of resources a selfish parent may contribute before they start to damage their own reproduction chances. This is then compared with individual reproduction, using Corollary 3, to show parameter regimes where sexual reproduction will dominate.

## 1.7 Contributions

In this section I outline the contributions of the thesis in context of the literature reviewed in this chapter. The main importance of this work is in the perspective on bet hedging, and its application to the transitions to higher units. I first consider the scientific contributions of the work presented here in light of the literature introduced in the introduction before considering the conceptual contributions.

### 1.7.1   Scientific contributions

The main topic of the thesis is the transition from units that reproduce on their own to units that reproduce as part of a larger unit. The approach I have chosen to take here is to consider abstract models where an individual will reduce its reproductive success and instead contribute to a higher reproductive process. Chapter 4 shows how, when parents share resources unselfishly, individuals may contribute to a higher reproductive process. In fact it shows that, depending on the impact on the upfront cost of reproduction, the more parents that reproduce, the better.

The main methodological contribution of the work is through the development of Theorem 2 (in Appendix A). This theorem (illustrated in Figure 1.2) assumes that there is no overall trend in population numbers. By taking this assumption, the theorem is a useful tool for modelling density dependent systems. When growth is linearly dependent on the availability of resources, the type of environmental fluctuations can be abstracted away from the model – which makes for potentially very simple but powerful models. When growth is not linearly dependent, Corollary 3 may still be used to demonstrate under which strategies may dominate under what fluctuation regimes.

The transition from individual to sexual reproduction is studied in Chapter 6, where selfish parents (which may vary their own resource contributions to offspring) are introduced. This has important consequences for our understanding of the origins of sexual reproduction. The traditional explanations of sexual reproduction come from the advantages of genetic mixing [reviews are given by Otto and Lenormand (2002) and Agrawal (2006), Watson (2006) gives more information on the benefits of genetic mixing for compositional evolution], the approach outlined in this thesis indicates that genetic mixing may only be of secondary benefit. The primary benefit of sexual reproduction being the ability of two parents to hedge their bets against future unpredictable fluctuations in resource availability.

A more complex major evolutionary transition is that from unicellular organisms to multicellular organisms. This is studied in more detail in Chapter 5. The model shows that parameters exist where an individual is prepare to gamble its life, resources and genes against a reproductive pot. This reproductive pot then produces a colony of homogeneous organisms. Interestingly, Michod's work on germ-soma differentiation (Michod 2005, Michod et al. 2006, Michod 2006) requires a genetically homogeneous population as a precursor so the combination of the results of Chapter 5 with this model maps a complete transition from individuals that reproduce on their own to individuals that differentiate into germ line cells and soma cells.

Considering the life history field the modelling approach in Chapter 3 outlines a novel

perspective on delayed reproduction. It shows how delaying reproduction by accumulating more resources can be a mechanism for delaying reproduction (instead of producing dormant immatures). Trade-offs are demonstrated between this mechanism for delaying reproduction and changes in the up-front cost of reproduction and the cost of maintenance. This, more general, result is relevant to microbial ecology and may inspire experiments that may be carried out in the laboratory.

### 1.7.2 Conceptual contributions

Given the applicability of the results to understanding the major evolutionary transitions, one of the most important contributions to evolutionary biology and ALife of this thesis is therefore conceptual. I present models that show how an individual can hedge its reproductive bets by reproducing as part of a collective, rather than reproducing on its own. This therefore implies an insight into the major evolutionary transitions: that inclusive fitness is not needed to explain why an individual might reduce its reproductive success for the benefit of its group (altruistic explanations of the major evolutionary transitions have been discussed in detail in Section 1.3.1). This is not to say that inclusive fitness is not important in the transitions, however the approach of this thesis opens a new line of enquiry.

The thesis introduces two new analytic techniques which are applied in Chapters 3, 4 and 6: Theorem 1 (see Appendix A) is used in all three chapters and the analysis of equilibrium resource levels is used in Chapter 3. These analytic techniques are likely to be of value in further extending the work of the thesis and in problems outside the context of the thesis.

The reusability of the mathematical analysis is an important aspect of this thesis. Much of the research was done by experimenting with computer simulation models. The research done actually started with the model in Chapter 5, which has complex dynamics. The mathematical analysis done, short of reproducing the computer simulation model with mathematical equations, helps to explain the behaviour of the computer simulation model however. This methodology has been spelled out in more detail, contrasting the freedom of computer simulation models with the explanatory power of mathematical models in Chapter 2. The outlining of the methodology used will hopefully help others to follow or refine my approach.

One of the initial motivations of the model is to understand how complexity may increase in an individual. All the models provide interesting insights into this question. Simply put, I argue that the investment of resources into anything other than fast repro-

duction will facilitate the increase in individual complexity – through optimised energy storage (see Chapter 3 for more details) or through investment into social reproduction (see Chapter 4 for more details).

# Chapter 2

# Philosophical considerations on the method

## 2.1   Introduction

In this chapter I consider some of the philosophical and methodological issues relevant to the two styles of modelling I have used in this thesis: computational and mathematical modelling. Much of the research was initially done using computational modelling. As insights were found from the computational models, mathematical models were generated. I present an outline of the two modelling approaches and identify key differences between them.

It seems possible that computer simulation modelling could become the new modelling paradigm in biology. As transparent, tractable, computer simulation models are developed, their relaxed assumptions will, in comparison with traditional explicit mathematical treatments, make for considerably more realistic models that are closer to the data. The 'Virtual Biology Laboratory' has been proposed (Kitano et al. 1997) where a cycle is proposed for the development of computer simulation models and biological experiments in tandem: the results from each procedure inspiring the direction of the other. Animals, such as *C. elegans*, have been well studied using computational models, e.g., work on locomotion control (Bryden and Cohen 2004, Bryden and Cohen 2008). Indeed the formation of a complete model of the organism has been identified as a po-

tential grand challenge for computing research (Harel 2002). However, a full exploration of the relationship between mathematical and computational models in biology has not yet been achieved. Questions remain: for instance, whether both forms of modelling can peacefully coexist, whether mathematical models should aspire to the complexity of computational models, and conversely whether a computational model can ever be as precise as a mathematical treatment.

The scope of this chapter is mainly concerned with the scientific modelling (both computational and mathematical) of biological systems, however I hope that the findings can be applied more generally. Biological systems are made up of many different subsystems. Put simplistically, genes and proteins are made of molecules, genes produce proteins which interact with other proteins, genes and other molecules to form cells; cells can interact to form multicellular organisms; organisms interact to form ecological communities and societies. ALife models often reside in the interface between one level and the next and can become extremely complex, especially as entities from any level can interact with entities from other levels.

The discipline of computer simulation modelling allows modellers previously unheard-of freedom to build and understand systems of many interacting parts. This new expressive freedom appears to have the potential to become the new modelling paradigm in science, perhaps overriding traditional techniques which use explicit mathematical treatments. However, this freedom does not come without a cost: as more and more detail is added computer simulation models can quickly become unwieldy and too complicated to understand.

How then can computer models contribute to the task of producing scientifically acceptable explanations? The use of a complex yet poorly understood model may be acceptable as some sort of loose analogy. However, Di Paolo et al. (2000) have argued that without a proper understanding of the internal workings of a computer simulation model, it can be impossible to say whether such a model makes a valuable contribution to the scientific problem it is addressing. They describe such problematic models as 'opaque thought experiments', arguing the need for explanations of the phenomena modelled. They suggest that modellers should use an 'experimental phase' in which manipulations are made to the computer model, the results of these manipulations hopefully generating insights into the workings of the system. Once the internal mechanisms are understood, the transparent model can then not only give new insights into the system being modelled but can also become a powerful predictive tool.

I question whether a computer simulation model can, in and of itself, constitute a scientific explanation. For example, one might produce a model in which individual or-

ganisms are explicitly represented and a particular population-level phenomenon appears to emerge. But this does not constitute an explanation of how entities from one level of a biological hierarchy produce interesting phenomena at another level. Di Paolo et al. (2000) argue that some explanation is required above a basic description of the model and the system it represents. In this chapter I look further into what an adequate explanation of a model's mechanisms should entail. I will compare the account that I construct with the more basic position, sometimes seen in the artificial life literature, that a bare-bones description of a biological system with a computer model that qualitatively produces similar behaviour – with little or no extra analysis or explanation – can constitute a scientific explanation of some phenomena.

Given the above concerns regarding computer simulation modelling I must also consider the traditional methodology of explicit mathematical treatment. By explicit mathematical treatment I mean a model which is complete and contains no implicit steps. It must be noted that computer simulation models are fundamentally mathematical constructions. However, given that many of the mathematical steps take place computationally, these are *implicit* rather than the *explicit* steps used by formal mathematical models. An explicit mathematical treatment takes logical axioms and specifies a number of clear explicit steps that deductively generate some result. In this chapter I compare this traditional treatment with the new computational approach.

First, I set the context; I look at a framework for scientific modelling. Then, by looking at two examples of a similar system, I identify some properties that characterise an explicit mathematical treatment which a computer simulation is unlikely to share. However, having established that explicit mathematical treatment is the ultimate goal of any modelling enterprise, I look at how computer simulation models do indeed still have value. I look at how complex and unwieldy computer simulations may be simplified to more easily generate explicit mathematical treatments – proposing that this can be done by decomposition into simpler systems. Finally I set out, in an order of merit, the various different modelling approaches discussed.

## 2.2 A framework for scientific modelling

To understand how modelling is important and relevant within scientific investigation, I present a framework for scientific investigation with the scientific modelling cycle highlighted. Figure 2.1 presents a diagram of the framework.

The primary focus of scientific investigation is the building of a good *conceptual model* of the *real world*. Explanations of the real world reside in the conceptual mod-
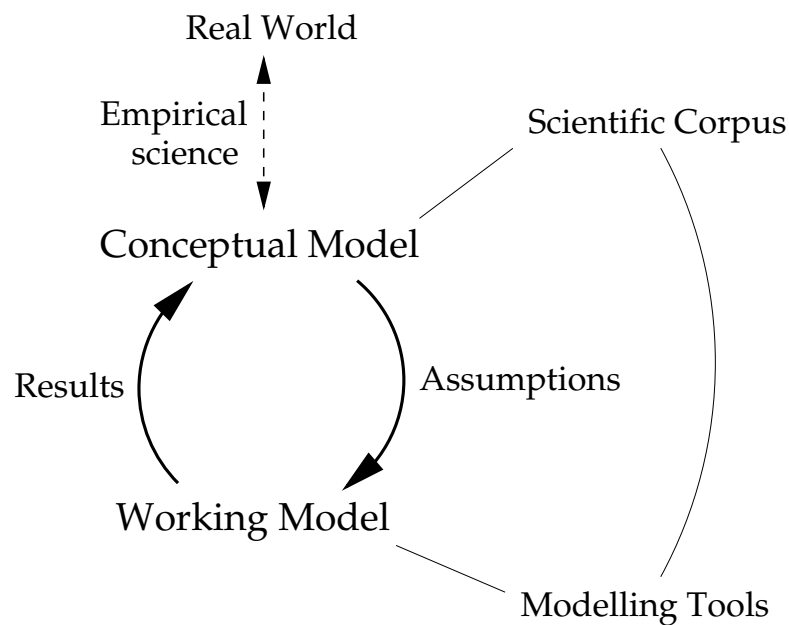
Figure 2.1: The cycle of enquiry in scientific modelling within the context of scientific investigation.

elling area of the framework; these are recorded in the *scientific corpus*. The basic scientific process involves the submission of concepts to the twin tests of *empirical science* and scientific modelling. The main focus of the framework, however, is on scientific modelling and the interface between a conceptual model and a *working model*.

Both computer simulation models and explicit mathematical treatments reside in the working model area of the framework. I take a working model to be a deterministic and completely specified model of a system. (Whereas a conceptual model may remain vague in places, a working model must be completely fleshed out.) Logical processes are applied to the axioms and the results of this process are recorded. Logical processes can include mathematical equations, logical deductions and computations. Working models produce *results* which are used to refine and update the conceptual model.

Before I specifically look at the sorts of results that can be generated by explicit mathematical treatments or computer simulation models, I discuss the types of *assumptions* that can be used to generate a working model. An assumption is essentially an abstraction from a more complex system. There will be many abstractions from the real world in the conceptual model (tested by empirical science) and it will normally be necessary to make further abstractions for ease of modelling. One of the main benefits of computer simulation modelling (Di Paolo et al. 2000) is that assumptions can be very easily added to or removed from models to see if they are significant or important. Explicit mathemat-

ical treatments tend to be more fixed in their assumptions. The types of abstractions used by either explicit conceptual models or computer simulation models can be divided into two groups, *reductionist* and *analogous* abstractions. I take inspiration for this distinction from Bedau's discussion of 'unrealistic' models (Bedau 1999).

In order to highlight the important differences between the use of computational and mathematical techniques in building a working model, I must first consider the outcomes of a successful working model for the broader scientific project. The more valuable results generated by a working model will form some kind of explanation of why some phenomenon is present in the conceptual model. Other, less valuable, results include those that generate predictions. With an explanation generated by a model to hand, an empirical scientist can easily and quickly generate good empirical experiments to test whether an explanation is valid or not. A working model may indicate that some factors are more important than others for a particular phenomenon. This may point empirical science toward a more fruitful direction. The value of a result can depend not only on the type of working model used to generate the result, but also the assumptions used to generate the working model in the first place.

## 2.3   Competence and performance in scientific modelling

The previous section has set out the tasks necessary before embarking on a modelling enterprise: Once a conceptual model has been chosen that builds a picture of what is known about some real-world phenomenon, assumptions are then chosen to simplify this conceptual picture into logical units and axioms that can be built into a model. Up to this point everything is quite similar between the two logical modelling styles. Perhaps it is natural to assume that since both modelling techniques are analytical, the style of the results will also be quite similar?

To answer this question I must consider a thought experiment based on a specific example which can easily be understood and modelled by either a computer simulation model or an explicit mathematical treatment. The Lotka-Volterra system is a mathematical treatment of a predator-prey system. Two equations model the dynamics of the system:

$$\frac{dx}{dt} = Ax - Bxy \tag{2.1}$$

$$\frac{dy}{dt} = -Cy + Dxy \tag{2.2}$$

where x is the frequency of prey, y is the frequency of predators and $A, B, C, D$ are con-

stants. This system of equations has been shown [see, e.g., (Murray 2002)] to generate oscillations between predator and prey frequencies. The mathematical treatment can be considered alongside an individual based computer simulation model of the same phenomenon.

An example of a computer simulation model of this system would use an individual based model. Software objects would represent individual predators and prey using a variable that represents energy. Prey individuals receive energy from the environment and predators receive energy by encountering prey (predator-prey encounters may be probabilistic, or predators and prey may be placed on a spatial grid and encounters modelled through distances). Both predator and prey individuals reproduce (make copies of themselves in the system) and die based on their internal energy levels.

Without wanting to go into too much detail, I assume, for the purposes of argument, that the computer simulation of the predator-prey system has very similar dynamics to the mathematical system. That is, both systems will make the same predictions about any particular predator-prey system to which they might be applied. The computational model can now be compared with the mathematical treatment in context of the initial question concerning the nature of the scientific explanation that may be derived from each modelling enterprise.

To answer that question I draw on a distinction introduced by Chomsky between *competence* and *performance* (Chomsky 1986). Chomsky's approach considers whether the linguistic corpus can be used as a source of empirical evidence for linguistic enquiry. He distinguishes between competence (our internal unconscious capacity for language) and performance (actual instances of language production). Regarding linguistic inquiry, he argues that we should take this distinction into account when considering models of linguistic competence above models of linguistic performance.

I use Chomsky's distinction to shed light on the differing styles of scientific explanation that are likely to follow from the use of computational versus mathematical treatments of a particular problem. From this point of view, the computer simulation model must merely be considered as a performance of a scientific explanation, whereas the explicit mathematical treatment can be considered as having competence (an innate capacity) as a scientific explanation.

Simulation runs have the same sorts of problems as those Chomsky identifies for linguistic performances. They are subject to faults (in code as well as in run-time conditions) and each simulation model is merely a single data point and may not reveal the complete potential of a system. In a similar way, it is possible to hide flaws in the performance from the audience. Simulations can be set up so that the data points presented make the best

possible case for whatever it is the modeller is trying to argue.

Alternatively, explicit mathematical treatments, assuming they are done correctly, are analytically complete: flaws in the system are immediately obvious. In addition, mathematical treatments are not limited to some narrow range of parameters but provide universal coverage of all variables included in the model. These two properties were identified by Chomsky as arguments in favour of looking at linguistic competence over linguistic performance.

Furthermore, explicit mathematical treatments have more powerful identity conditions than do computational models. By this I mean that one mathematical treatment can automatically be established as the same as, or different to, another treatment, just by comparing the logic. Computer simulation runs, on the other hand, may produce similar results for the same problem, but have very different underlying explanations. The opposite can also occur, in that two computer simulations may be driven by the same underlying process without this being obvious to an observer.

Mathematical treatments are more reusable than computer simulation models. Some give good clean results which can instantly be applied to systems, others benefit from the ease with which they can be written down in full and passed on. Such models can then be used as logical axioms for other models with their competence passed on. In contrast, although computer models can certainly be transferred from one author to another their results are rarely used, in practice, as axioms for other models.

To put this in context with regards to the example of the Lotka-Volterra system, the mathematical treatment of this system [see Murray (2002) for details] has competence as an explanation of oscillations between predators and prey. In comparison, only graphs (or other data visualisations) showing oscillations from performances of simulation runs may be presented from the computer simulation model, only certain parameters may be tested and simulations can only be run over a finite length of time.

One might argue at this point that we can distinguish the code for a computer simulation model from an individual execution of the code. The argument continues that a simulation run is merely a performance of the code, the code itself has competence. This argument can be rejected by stating that if the code has competence then it is basically a mathematical treatment in itself (or at least part of a mathematical treatment). Returning to the Lotka-Volterra example, the predator-prey oscillations are a macroscopic phenomenon. There are only two cases possible here. Either, without an execution of the code these macroscopic phenomena are not deducible from reading the source code[1], or, if the macroscopic function is deducible from the code, then this deductive process

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[1]Such non-deducible phenomena were described as being emergent by Bedau (Bedau 1997)

would necessarily form an explicit mathematical treatment. If this deductive process is impossible, any explanation generated must be teased out by analysis of simulation runs and is thus a performance.

At this point, we are left with a conundrum. If computer simulation models are viewed as mere instances (performances) rather than as systematic explanations (having competence), how can they be of use to science? The answer is that there are many areas, identified especially in the ALife field, which do not yet yield to mathematical modelling but in which simulation models can already be produced. Such simulation models not only have scientific power as proofs of concept and for generation of insights for performing empirical science, but they can also have some explanatory power (Di Paolo et al. 2000).

When considering a complex simulation in which there is no explanation of the effects produced, some explanation can be deduced by performing experiments on the simulated system in the same way that one would do for an empirical investigation. In this mode of enquiry a *control* simulation is generated in which some important phenomenon does not happen. This is normally done through some manipulation of the system. The control simulation is compared with the untampered system and the results are used as evidence that the changes made by the manipulations are part of the explanation of the phenomenon.

The above procedure is very similar to the normal mode of empirical science. A conceptual model can be built of the working model system and this conceptual model acts as an explanation. I will now look further into how this form of explanation relates to an explicit mathematical treatment.

### 2.3.1   Analytic explanation versus synthetic explanation

To attempt to understand the difference between an explanation generated through the use of a working model in explicit mathematical form and an explanation generated by experimental manipulations of a computer simulation model, I consider a distinction used by the logical positivists – that of *analytic* and *synthetic* truths.

According to Frege's reworking (Frege 1980) of Kant's original distinction, an analytic truth is one that can be deduced through logical laws alone. A synthetic truth is one which needs some other means, generally empirical investigation, to establish its truth or falsity.

I use this distinction to identify modes of truth for explanations generated by a working model. As pointed out previously, I assume all working models are using the same assumptions, i.e., they start from the same set of logical axioms. I distinguish between an analytic explanation – one which follows logically from the initial assumptions – and a

synthetic explanation – one which must be determined by some other means.

Naturally an explicit mathematical treatment is in itself an analytic explanation. However, empirical experiments done on a computer simulation can only form synthetic explanations. These synthetic explanations require validation in the same way empirical science must be validated. The evidence backing up these validations relies on measurements taken from performances and is thus open to disconfirmation, reproduction and revalidation.

There is an ongoing debate about the analytic/synthetic distinction, some arguing that it is not a black and white distinction but more a question of degree (Quine 1953). While Quine's arguments are concerned with statements about the real world rather than statements about a closed set of logical axioms, I agree that our distinctions of explanations should not be black and white. A working model can, like a biological system, be large and complex. Some parts of such a system will yield to explicit mathematical treatment, whereas with other parts I may have to rely on empirical-style experiments of the kind discussed by Di Paolo et al. The final explanation generated through such a process will consist of a mixture of analytic and synthetic statements.

In the next section I present an account of how systems can be decomposed into smaller parts to identify explicit mathematical treatments. Successful mathematical treatments will render the resulting explanations more analytic in the way I have just described.

## 2.4   Decomposition of systems

A system can become hard to analyse when it is made up from many inter-dependent subsystems. In fact, the identification of subsystems is a good first step when tackling such a complicated system. However, this is rarely simple. When subsystems are inter-dependent it is not possible to manipulate one subsystem independently without affecting another: both subsystems, at the same time, affect the overall system. The situation becomes increasingly difficult when the subsystem's components are not mutually exclusive from each other.

Simon (Simon 1996) describes a 'nearly decomposable system' as being one in which components are independent in the short term, but dependent in the long term. This is a useful way to divide a system up and this has been expanded further (Watson 2005, Polani, Dauscher and Uthmann 2005) considering modular dynamical systems. Watson introduces a concept called *modular interdependence* to describe a system with modules that are decomposable but not separable. A hierarchy can be formed from subsystems and it is easy to see how complex behaviour can be generated. This hierarchical perspective

is a valuable decomposition of a complex system. If it is possible to divide up a set of microscopic entities into subsets this will allow us first to tackle the mechanisms of the subsets, before understanding how they interact with each other.

In the next section I consider a more general perspective for decomposing systems. Rather than breaking up the set of microscopic entities into subsets, I consider a more arbitrary way of decomposing a system into subsystems that contain a simplified version of the dynamics of the supersystem.

### 2.4.1   Mechanistic subsystem

I propose information theoretic definitions of a mechanistic subsystem and interdependence in mechanistic subsystems. This style of definition has been used by McGregor and Fernando (2005) to formalise *hyperdescriptions*. I then go on to discuss how these definitions relate to our intuitive notions of these concepts before looking at examples in the next section.

Define a system $S$ as being a set of mathematical entities, their interactions and their parameters. Take a descriptor function $d(S) = M$ that will map the system $S$ to a set of descriptors $M$. Define the *entropy* of a random variable $X$ as

$$H(X) = -\sum_{x \in X} p(x) \log p(x) \,,$$

the *conditional entropy* between two random variables $X$ and $Y$ as

$$H(Y|X) = -\sum_{x \in X} p(x) \sum_{y \in Y} p(y|x) \log p(y|x)$$

and the *mutual information* as

$$I(X;Y) = H(Y) - H(Y|X) \,.$$

Take a system $S_1$, such that $d(S_1) = M_1$. Then, $S_1$ is a mechanistic subsystem of $S$ if

$$S_1 \quad \subset \quad S \tag{2.3}$$

$$H(M_1|M) \quad = \quad 0 \tag{2.4}$$

$$I(M_1;M) \quad > \quad 0 \tag{2.5}$$

$$I(M_1;M) \quad < \quad H(M) \,. \tag{2.6}$$

The mechanistic subsystem $S_1$ is a constrained version of its supersystem $S$. The

constraints can take place in the parameter space, the number of entities, the nature of the entities, or their interactions. I list the Equations [(2.3) to (2.6)] and describe their meaning: (2.3) $S_1$ is a subset of $S$; (2.4) all information in $M_1$ is predicted by $M$; (2.5) $M_1$ and $M$ share some information; (2.6) there is information in $M$ that is not predicted by the information shared by $M_1$ and $M$.

The information theoretic definition presented includes many of the important concepts of a mechanistic subsystem. However, a useful mechanistic subsystem should have two further properties. Firstly, it should be transparent, i.e., it is possible to understand why and how it produces its macroscopic effects. Secondly, its macroscopic effects should be of interest when compared to the macroscopic effects of the main system. I need to avoid specifying macroscopic subsystems that are either equally complex to the main system with only some negligible reduction, or are so simplistic that they are of no analytic value.

Following on from this definition of a mechanistic subsystem, I draw on Polani et al.'s definition (Polani et al. 2005) of a system that is decomposable but not separable to identify how two mechanistic subsystems can be interdependent. Take a system $S$ and two mechanistic subsystems $S_1$ and $S_2$, the subsystems are interdependent if

$$0 < I(M_1; M_2) < \min[H(M_1), H(M_2)] \ .$$

The two subsystems are neither independent nor completely dependent.

With this approach identified, It can be seen how it is possible to break up a complex system of many interacting parts into simpler mechanistic subsystems.

## 2.4.2 Examples of Mechanistic Subsystems

I consider, as an example, the spatial embedding of reproducing agents. Space has been shown to be an important factor in the maintenance of cooperation in a population (Boerlijst and Hogeweg 1991, Di Paolo 2000). The common feature of these models is that two regimes are considered. The models are considered and analysed in a non-spatial environment before being placed in a spatial environment. The non-spatial treatment is a mechanistic subsystem of the spatial treatment. In this treatment agents are thought to be in a perfectly mixed spatial environment, a special case of the spatial component. A comparison of the interactions of agents in the spatial and non-spatial environments demonstrates how cooperation is increased.

A mechanistic subsystem (the non-spatial model) functioned primarily as a control in the above experiments. Further to this, the dynamics of mathematical treatments of the

non-spatial model (Di Paolo 2000) are considered with the final discrete spatial individual based model. Spatial localities can be considered as non-spatial models. In this way, the mechanistic subsystem provides insights on the final results.

A different model [see Bryden (2005a) and also in Chapter 5] considers collective reproduction in amoebae. This non-spatial model demonstrates that cells that reproduce individually must reproduce more slowly to maintain high energy reserves for periods of low resources. By reproducing collectively during periods of low resources, individuals can avoid the need to reproduce slowly and can dominate periods of high resources by reproducing more quickly. The model is complex and it is not easily apparent why this is occurring. To explain why individuals might reproduce more slowly, I have done a mathematical treatment (presented in this thesis in Chapter 3) which analyses a mechanistic subsystem of the main model only considering individuals that reproduce individually. This, combined with the insights from the other work in Chapter 4 show that an individual is actually hedging its bets by reproducing collectively.

A further model (Bryden 2005b) considers the effects of space on the individual reproduction mechanistic subsystem: agents in the model live and reproduce on a spatial grid. This model can be broken up into two mechanistic subsystems, firstly a non-spatial subsystem with individuals reproducing and secondly a spatial subsystem without individuals reproducing. The results indicate that the spatial effects increased the frequency of both periods of high resources and periods of low resources. The mathematical model has shown that this would increase the tendency for individuals to conserve resources and reproduce more slowly. This is an example of a system that combines two interdependent mechanistic subsystems (a spatial and a reproductive system) that interact with each other to produce a macroscopic phenomenon.

Away from the field of agent based modelling, I consider models based on neural biological systems. Neural systems have extremely complex dynamics, which are resistant to mathematical analysis. However, the use of linear stability analysis has proved useful in identifying mechanistic subsystems which can be used as building blocks within larger systems. For example, a system of coupled oscillators, based on the FitzHugh-Nagumo model, has been analysed as a mechanistic subsystem (Buckley, Bullock and Cohen 2004). This analysis demonstrates how, when the oscillators are linked to a simple gas net, the system can produce temporally distinct oscillations. Much other work continues into the identification of simple oscillatory models, such as that done in Central Pattern Generators (CPGs) (Marder and Bucher 2001). CPGs can work as mechanistic subsystems within models of animal locomotion systems.

In this section I have demonstrated how a complex system that does not yield to ex-

plicit mathematical treatment may be simplified into mechanistic subsystems which are more likely to yield to explicit mathematical treatment. We can observe from the examples chosen that the working models arrived at through such a process consist of both synthetic and analytic explanations.

The process of simplification identified above is not the only way of making simpler models. By choosing different assumptions and approaching a conceptual model from a different perspective it is also possible to open up a system to explicit mathematical treatment. With computer simulation it is increasing easy to change the assumptions of a model and get a feel for how the system changes. This sort of approach is invaluable as a tool for the sort of lateral thinking needed when generating an explicit mathematical model.

## 2.5   Discussion

While this chapter has argued that an explicit mathematical treatment will provide a superior explanation of a scientific phenomenon to an equivalent computer simulation, it must be made clear that computer simulation is still a crucial part of the modeller's toolbox. The overarching goal of the scientific modeller is to build better models. The ease with which models can be produced with computers is extremely valuable. Furthermore, not only can these early efforts lead to some important scientific results, but they can also point towards new directions for mathematical models. I list below, in increasing order of merit, different styles of working models and explain how valuable each one is in generating scientific explanation. By starting with models at the beginning of the list and progressing up the list, models can become better explanations of scientific phenomenon.

- A description of an opaque computer simulation and some vague rhetorical statements that it consists of an explanation of what it is trying to model. I have argued that this approach is merely setting down a procedure for producing a performance of explanation of some phenomenon. However, this approach can still yield a proof of concept for some topic under debate, or generate insights for empirical experiments.

- The same computer simulation as before, but this time complete with well documented source code, parameters and other data that can easily be tested by other users and reused in new simulations. While this approach does not yet produce a competent explanation, it allows for more simple reproduction of the model which will help others develop it further.

- An opaque computer simulation (with well documented source code) with some manipulations and simulation runs that demonstrate how various attributes of the model explain various phenomena. I have argued that this approach can yield a competent explanation of sorts, but this is merely a synthetic explanation and is not logically grounded.

- An opaque computer simulation (with well documented source code) that has been decomposed into mechanistic subsystems. Some subsystems have been treated mathematically. Such a working model can also yield a competent explanation of sorts, this explanation is more analytic than in the previous case.

- An explicit mathematical treatment. Such a working model yields a competent fully analytic scientific explanation.

As set out above, clearly the best option is to produce an explicit mathematical treatment. However, this is rarely simple, and in many cases mathematics is not yet mature enough to approach this goal. Since we must live in the real world, science must answer questions about systems that cannot be yet modelled by mathematical approaches. Computer simulation modelling provides us with a working methodology for approaching these complex or complicated systems and taking important steps toward understanding them.

Further to this, it is important to note that computer simulation models can extend already established mathematical treatments. By extending or relaxing the assumptions made in the purely mathematical treatment, the new model will rely on the mathematical treatment as a mechanistic subsystem but may also produce new results or important insights into the mathematical model (Harris and Bullock 2002). Since computer simulation models lend themselves to more accurate, relaxed assumptions, when explanations become available they are more likely to be of value to the conceptual model under question.

What is important is that scientific models progress up the order of merit listed. A novel modelling approach that identifies a new style of working model may have value even if it merely provides a performance of some scientific explanation. Such a system can be experimented with and decomposed into mechanistic subsystems and the standard of explanation will improve. This is one of the benefits of computer simulation modelling in that it gives us tools to break down a problem so that we can get closer to an explicit mathematical treatment through an iterative process. Computer simulation models can be thought of as providing a sort of sandbox in which imagination and lateral thinking can be done.

# Chapter 3

# Modelling delayed reproduction under density dependence

## 3.1   Introduction

In this chapter I present a model of individual reproduction strategy. Within the context of the thesis, this model has two purposes: first to try to understand what evolutionary advantage there is in accumulating resources and delaying reproduction within a fluctuating environment and second to generate a baseline model of reproduction which may be extended in the later chapters (see Chapters 4 and 6). This baseline model is also relevant to other biological theories of reproduction strategy so the biological background is reviewed in more detail.

The problem of how much investment a parent might make in its offspring was identified early in the study of evolutionary biology (Fisher 1930). There is clearly a tension between the need for an individual to generate the maximum number of offspring as quickly as possible and the need for those offspring to be successful.

It has long been known that, in the absence of density dependence, populations will grow at an exponential rate (Malthus 1798). During a period of growth a fast reproducing individual will increase its frequency in a population relative to one which reproduces more slowly. In the past there has been an argument about why individuals do not always reproduce as quickly as they might. It was proposed (Wynne-Edwards 1962) that this was because fast reproduction was not beneficial to the group (or, in this case, the whole species). This *group selectionist* viewpoint came under a great deal of criticism (Maynard Smith 1964, Williams 1966a) and the belief that individuals might make adaptations for the benefit of the species is now uncommon, with the gene-centric view dominant (Dawkins 1982).

Since we may reject the group selectionist argument, any explanation as to why an individual might reproduce more slowly must include reasons for the spread amongst a population of a gene for slow reproduction. An early study looked at the clutch sizes of birds (Williams 1966b), and argued that birds which lay more eggs often produce fewer fledglings because they are unable to feed them properly. Following on from this early work, much research into reproduction strategy has been done and this is summarised in the next section.

### 3.1.1 Background on life history modelling

The analysis of problems related to reproduction strategy is wide ranging and many different perspectives have been considered. These include parent-offspring conflicts (Trivers 1974), $r/K$ selection in population ecology modelling (Reznick, Bryant and Bashey 2002), analysis of age vs. size at maturity (Stearns 2000) and the trade-off between the lifetime of a mother and the number of her offspring (Stearns 1992).

The development of good general models of life history strategy such as the "general life history problem" (Schaffer 1983) can prove elusive (Stearns 1992). This would involve the modelling of reproductive strategies favoured by natural selection which would give optimal results under various environmental conditions. In this field any prediction usually relies on many assumptions and there are often exceptions. It is therefore important to view life-history theory as a framework for studying biological systems rather than a model of how such systems might work.

Considering the $r/K$ selection approach (MacArthur and Wilson 1967, Pianka 1970, Reznick et al. 2002), this argues that environmental conditions are important for determining which reproduction strategy an organism may take. It looks at a continuum between fast-growing individuals that make a big investment in many small offspring and die young ($r$-selected) and slow-growing individuals that have a small number of larger offspring and live longer ($K$-selected). It is intended as a model of a complete ecosystem in which different species are located along the $r$-$K$ continuum. Correlates have been presented (Pianka 1970) arguing that $r$-selected species tend to experience density independent growth while $K$-selected species experience density dependence. This theory became unpopular in the early 1980s (Reznick et al. 2002) due mainly to the lack of concurring data from experimental studies to test the $r/K$ selection hypothesis (Stearns 1992).

The introduction of age-structured models proved very successful compared with the traditional $r/K$ approach (Stearns 2000). Notably, Tuljapurkar (1990a) modelled delayed reproduction by dividing the population into two age groups – adults and immatures. The

dormant immatures are not susceptible to environmental fluctuations, but adult fertility is. As the variance of adult fertility due to environmental fluctuations is increased, there is a greater proportion of immatures that delay their transition to the adult stage. The calculation of the geometric mean growth (in line with bet hedging theory, see Section 1.5.1) over varying environments is used as a metric to show how the strategy of keeping some of the population in reserve can help when fluctuations that may wipe out a significant proportion of the population are increasingly frequent. When the population is at a fixed level (i.e., there are no fluctuations), Tuljapurkar's model does not predict any delayed reproduction. It also does not consider what happens when there are fluctuations, but the population is under density dependence (i.e., where there is no long term growth of the population).

Another relevant question is concerned with the allocation of resources between growth and reproduction in animals which grow beyond reproductive events [see a review by Heino and Kaitala (1999)]. Modelling approaches measure the population growth of various strategies when fecundity is a function of size and age (Kozłowski 1991, Kozłowski 1992). These modelling approaches do not consider feedback from the environment however. Heino and Kaitala (1999) consider this a major theoretical challenge. In light of this I look at work on microbial ecology, a good example of individuals growing beyond reproductive events, in the next section

### 3.1.2 Background on microbial ecology

While age-structured modelling has proved very successful in ecological modelling of metazoa, this style of modelling is less applicable to microbes which normally reproduce by splitting into two equal halves. After mitosis there is no obvious parent. There are therefore difficulties in assessing the age of any individual microbe. Attempts to model reproduction strategy within microbial population ecology are therefore still very much based around the traditional $r/K$ approach (Andrews and Harris 1986, McArthur 2006).

In analysing the reproduction strategy of bacteria, the $r/K$ selection model was invoked by Velicer and Lenski (1999). Attempts were made to show that bacteria will adapt toward either an $r$-strategy or a $K$- strategy, depending on which of two growth regimes they have been exposed to. They exposed bacteria to fast and slow growth regimes. In the fast regime, the bacteria were constantly placed in fresh medium whenever the medium was exhausted. In the slow regime, the bacteria were fed in a chemostat with a relatively low influx of medium, but enough to maintain slow population growth. The intention was to show that those exposed to one regime were not only better adapted to that regime than

their ancestors but worse off in the alternative regime than their ancestors. This would be in line with the $r/K$ selection model. Individuals that had undergone selection were put in competition against ancestors in both regimes. The results proved to be at best inconclusive and at worst contrary to that theory with the selected individuals out-competing the ancestors in the alternative regime in 58% of the strains analysed (they always did better than ancestors in the selected regime). These results do not seem to support the $r/K$ selection model.

As an alternative to $r/K$ selection, other perspectives have been considered regarding the modelling of microbial growth. Much of this centres around application of the Monod equation (Monod 1942, Monod 1950), which is a simple equation modelling the density dependent growth of an organism (Smith and Waltman 1995). As with the Verhulst equation (used for $r/K$ selection) there is exponential growth at lower population density levels and then growth becomes bounded as population density increases. Using this equation, ecological models can be produced looking at predator prey interrelations (Smith and Waltman 1995).

Analysis of bacterial growth rates in fluctuating environments has also been considered (Vasi, Travisano and Lenski 1994). The work estimates Monod equation parameters for bacteria cultivated in an environment organised so that the population dynamics would fluctuate between a fast growth phase and a stationary phase. During the fast growth phase, individuals were simply cultured in a medium. Once the medium was exhausted, the stationary phase would begin: in this phase the individuals were left to experience a further period of time with no new medium. This process was iterated, with each new culture starting with 1% of the bacteria from the previous culture. The results found that, during the growth phase of the experiment, there was an increase in the growth rate of the bacteria subjected to this procedure. Interestingly, the ancestral bacteria used showed marginal growth during the stationary period (when the medium was exhausted). After selection, the bacteria instead showed marginal declines in numbers during the stationary period, however the change was negligible (Vasi et al. 1994). This experiment has much in common with the model of Tuljapurkar (1990a) which is concerned with the effect of a fluctuating environment on the growth rates of a population. In the next section I outline a model which considers both strands.

### 3.1.3   Scope of the models

Two relevant strands were introduced in the previous two sections: delayed reproduction in life history modelling and experimental studies of microbial ecology. In this section,

I outline the model I present in this chapter which will address both of the two strands. In essence the model studies different strategies for resource accumulation and contribution to offspring. Delayed reproduction is achieved by accumulating more resources before reproduction (rather than producing dormant offspring in Tuljapurkar's model, Section 3.1.1). This approach is therefore relevant to single celled organisms (see Section 3.1.2) because there is no age structure. The effects of density dependence (called for in Section 3.1.1) may also be studied because resource accumulation depends on population density (in harmony with the theme of density dependence introduced in Section 1.5).

From previous results (see Section 3.1.1), resource fluctuations are expected to be the cause of reproductive delay. To confirm this, the models consider cases where there are extrinsic resource fluctuations. They also consider cases where there are no extrinsic resource fluctuations and resource availability is completely density dependent to investigate what factors may influence delayed reproduction in these circumstances.

Two mathematical modelling approaches are considered here. These differ from the way an individual may encounter resources – and hence the type of individual they model. First, I consider an individual that receives resources continuously. This corresponds with a bacteria absorbing nutrients from the surrounding area – the rate of absorption depending on the nutrient concentration. The second model considers resources that are encountered intermittently – in discrete resource units. This corresponds with predatory organisms that encounter prey.

The simulation models are introduced to relax assumptions taken in the mathematical models and extend them when useful assumptions are no longer possible. (The assumptions taken are specified in the descriptions of the mathematical models in Section 3.2.)

In summary, the models are therefore designed to answer the following questions:

 (i). Is it possible to explain delayed reproduction using models of density dependence?

 (ii). Can delayed reproduction be modelled with physiological models of resource accumulation and investment in offspring?

(iii). What is the role of physiological factors, such as the up-front cost of reproduction and the cost of maintenance to delayed reproduction?

## 3.2   Mathematical modelling

In this section I look at two mathematical models of individual reproduction. In the models, reproduction strategies are genetically determined. Quite simply, an individual has

a resource threshold above which it will reproduce: i.e., determined by its genes, it will either reproduce with a lower level of resources or wait until its resources have reached a higher level. Strategies are passed on to offspring, and therefore are modelled as a complete lineage.

Both mathematical models derive the lineage growth rate, i.e., the number of new individuals produced by each individual in the lineage per unit of time. The two mathematical models differ based on whether the resources are continuous or intermittent (see Section 3.1.3 for more information). The first model is dubbed the deterministic model which assumes that resources are continuous and set at a static level throughout the lifetimes of individuals. The second model is dubbed the stochastic model where resources are intermittent. In this model, the mean resource uptake throughout the lifetime of an individual is set as in the deterministic model but there is variance in the resource uptake within that lifetime.

In the simplest model (the deterministic model), the growth rates are analysed under different amplitudes of resource fluctuation. Since different reproductive strategies have different growth rates under different resource conditions, I look to Theorem 2 in Appendix A to predict which strategy will dominate. This theorem considers two populations that grow at different rates in two seasons and proves which population will dominate. Since it does not require a definition of the resource uptake function, the way resources vary is as unconstrained as possible in the model. However, the one constraint introduced is that the population is in equilibrium. This means that, while population levels may fluctuate, there must be no overall positive or negative trend to population levels – the population is effectively under density dependence (see Section 3.1.3 for more information).

The stochastic model is analysed by looking at the situation where the population converges very quickly to its equilibrium level. I find that different parameters mean that some lineages have different equilibrium values (this is not shown by the deterministic model) – and therefore those that are in equilibrium at lower levels of resources might invade those that are in equilibrium at higher levels of resources.

The analysis of both the models is done by first considering individuals that are identical apart from their reproduction thresholds (which determine the speed of reproduction). The prediction made by the model, in this scenario, is that the optimal strategy is to reproduce as slowly as possible. It seems reasonable however that there may be some physiological costs involved in reproducing more slowly so I have therefore considered that an individual might encounter an increased cost of reproduction and/or an increased cost of maintenance (I have assumed that costs of growth are part of costs of maintenance)

through reproducing more slowly. I therefore analyse the trade-off between the reproduction threshold and reproduction cost and the trade-off between the reproduction threshold and maintenance cost.

### 3.2.1 Model basics

Each individual $i$ is modelled by a resource level $x(i,t)$. Every individual $i$ belongs to a lineage $j$ which determines its reproduction threshold $R_1(j)$, its cost of reproduction $R_r(j)$ and its cost of growth and maintenance $R_c(j)$.

Individual resource levels are modelled by the equation,

$$\frac{dx}{dt} = U(i,t)R_u - R_c(j) , \tag{3.1}$$

where $R_u$ is a positive constant and $R_c$ [$0 < R_c(j) < R_u$] is dependent on the lineage. *Resource uptake* (the level of resources received from the environment) is variable and modelled by $U(i,t) \in [0,1]$. This variable is treated differently in the two models presented in this chapter. In the deterministic model, $U(i,t)$ is considered to be constant over the lifetime of the individual ($U(i,t) = u$), i.e., the individual's fate is determined by the value of $u$. In the stochastic model $U(i,t)$ is modelled by white noise depending on variables $u$ and $\delta t$. Time is broken up into segments of length $\delta t$ and for each segment $\Pr(U = 1) = u$ and $\Pr(U = 0) = 1 - u$. The average over realisations ($< U >= u$) is also constant over an individual's lifetime.

If an individual's resource level decreases below the lower threshold $R_0$ (fixed for all individuals) it will die. If an individual's resource level increases above its reproduction threshold it will reproduce. All individuals pay a cost of reproduction $R_r(j)$ (dependent on their lineage) when they reproduce and share half of the remaining resources with the new offspring. In the mathematical models all offspring are assumed to be same lineage as their parents. Therefore, all individuals start their lives at

$$x(i) = \frac{R_1(j) - R_r(j)}{2} . \tag{3.2}$$

As an *ad hoc* simplification, $R_0$ is set to 0. To study different values of $R_0$, $R_c$ and $R_r$ can be scaled appropriately.

Given this starting position for individuals of lineage $j$ and a specific model for resource uptake [$U(i,t)$] the growth rate of the lineage is approximated using the equation,

$$G(j,u) = births - deaths$$

$$= \frac{F(j,u)}{W(j,u)} - \frac{1-F(j,u)}{W(j,u)}$$

$$= \frac{2F(j,u)-1}{W(j,u)} \, , \tag{3.3}$$

where $W$ is the expected generation time (the time taken for $x(i)$ to reach $R_0$ or $R_1$) of an individual and $F$ is the ratio of individuals that eventually reach the upper threshold to those that reach the lower threshold. In the deterministic model $F$ is equal to 1 or 0. However in the stochastic model $F$ will take on values between 1 and 0. For simplicity, this equation therefore assumes that, from the starting position $x$, the time taken to reach the upper threshold is equal to the time taken to reach the lower threshold. When $x$ is near to $R_1$ or $R_0$, this equation becomes less accurate[1], however the assumption can be relaxed through simulation modelling (see Section 3.3)

### 3.2.2 Deterministic model

In this section equations for $W_d$ and $F_d$ are presented for the deterministic model and a growth rate $G_d$ is derived. The derived growth equation yields to simple analysis which is also presented.

As outlined above, the individual resource level in the deterministic model is given by Equation (3.1) with $U(i,t) = u$. To generate expressions for $F_d$ and $W_d$, I first define the value of $u_0$ for which $dx/dt = 0$ $[u_0(j) = R_c(j)/R_u]$ and consider the time taken for $x$ to reach $R_1$ when $u > u_0$ $(F = 1)$ and the time take to reach $R_0(= 0)$ when $u < u_0$ $(F = 0)$. The generation time $W_d$ for an individual starting with $x$ resources is given by the number of resources needed to move to the relevant threshold divided by its rate of change of resources $(dx/dt)$:

$$W_d(x,u) = \begin{cases} \dfrac{(R_1 - x)\delta t}{uR_u - R_c(j)} & ,u > u_0 \\[3ex] -\dfrac{x\delta t}{uR_u - R_c(j)} & ,u < u_0 \, , \end{cases} \tag{3.4}$$

(as $u \to u_0, W \to \infty$).

All individuals are assumed to start just after reproduction as in Equation (3.2). This value can be put into Equation 3.4, and Equation (3.3) can now be used to predict the

---

[1] In fact, further modelling work done by Stephen Cornell shows that this approximation is good except when the bias is high, i.e., the difference between $R_c(j)$ and $uR_u$ is large.

growth rate of lineage $j$:

$$G_{\mathrm{d}}(j,u) = \begin{cases} \dfrac{2\left[uR_{\mathrm{u}} - R_{\mathrm{c}}(j)\right]}{\left[R_1(j) + R_{\mathrm{r}}(j)\right]\delta t} & , u > u_0 \\[2ex] 0 & , u = u_0 \\[2ex] -\dfrac{2\left[uR_{\mathrm{u}} - R_{\mathrm{c}}(j)\right]}{\left[R_1(j) - R_{\mathrm{r}}(j)\right]\delta t} & , u < u_0 \,. \end{cases} \qquad (3.5)$$

This equation is analysed in the next section.

### 3.2.3 Deterministic model analysis

To analyse Equation (3.5), I compare lineages with various different lineage parameters ($R_1$, $R_{\mathrm{r}}$ and $R_{\mathrm{c}}$) against each other. First, I fix $R_{\mathrm{r}}$ and $R_{\mathrm{c}}$ and look at whether there are any advantages to increasing $R_1$. Then I consider what happens when $R_{\mathrm{r}}$ increases as $R_1$ increases. Finally I consider the case where $R_{\mathrm{c}}$ increases as $R_1$ increases.

To look at increasing values of $R_1$, Equation (3.5) is plotted in Figure 3.1 for two lineages with differing values of $R_1$ and all other parameters fixed. There are two regimes on the plot, to the right of $u = u_0 = 0.0909$ both lineages grow with the growth rate ($G_{\mathrm{d}}$) increasing linearly as a function of $u$ [approximately, $G_{\mathrm{d}}(A) = 0.0169(u - u_0)$ and $G_{\mathrm{d}}(B) = 0.0096(u - u_0)$]. To the left of $u = 0.0909$ both lineages are declining, again $G_{\mathrm{d}}$ is a linear function of $u$ [approximately, $G_{\mathrm{d}}(A) = 0.0314(u - u_0)$ and $G_{\mathrm{d}}(B) = 0.0129(u - u_0)$]. For both lineages the growth rate is lower than the decline rate (the high value of $R_{\mathrm{r}}$ was chosen to accentuate this on the plot).

The results from the figure can be used to show under what conditions invasion may occur. Since the linear equations in the above paragraph take the same form as Equations (A.1) and (A.2), Theorem 2 (in Appendix A) can be used (within the scenario considered by the theorem) to predict when one population is dominant over the other. The ratio of the slope in the growth region ($u > u_0$) to the slope in the decline region ($u < u_0$) will determine which lineage will dominate: a lineage with a greater ratio will dominate a lineage with the lesser ratio. In the example shown in Figure 3.1 the ratio of the growth to decline slopes of lineage A is $0.0169/0.0314 = 0.538$ and the ratio of growth to decline slopes of lineage B is $0.0096/0.0129 = 0.744$, so lineage B is dominant.

Analysis of Equation (3.5) can therefore be done by looking at the ratio of slope in the positive regime to the negative regime, this is given by (fixing $R_{\mathrm{r}}$ and $R_{\mathrm{c}}$ for both lineages
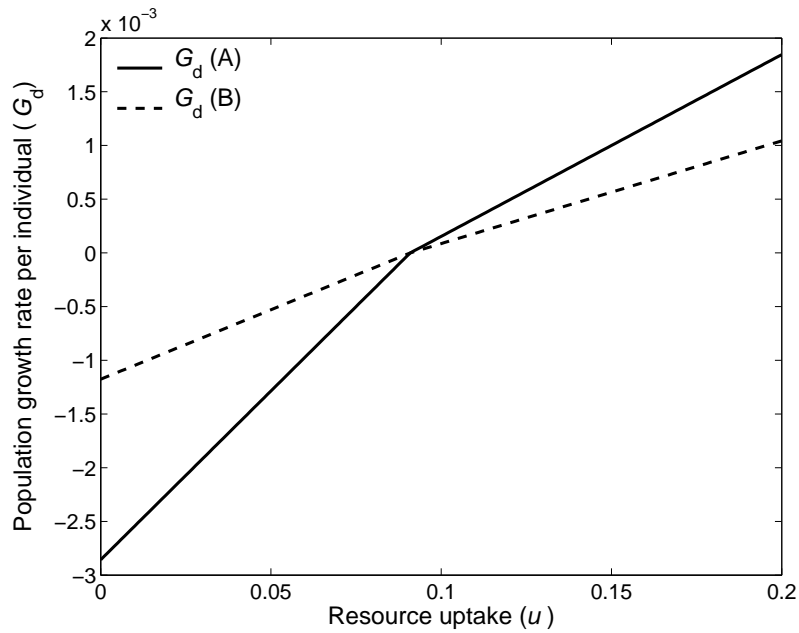
Figure 3.1: Comparison of the growth rates of two lineages A and B with different reproduction thresholds. Equation (3.5) is with parameters $R_c = 0.001$, $R_u = 0.01$, $R_r = 0.3$, $\delta t = 1$, $R_1(A) = 1.0$ and $R_1(B) = 2.0$ . For both lineages $u_0 = 0.0909$.

and taking $G' = dG/du$):

$$\Psi(j) = \frac{G'_{u>u_0}}{G'_{u<u_0}} = \frac{R_1(j) - R_r}{R_1(j) + R_r} \, , \tag{3.6}$$

which is the derivative (with respect to $u$) of the growth portion ($u > u_0$) of Equation (3.5) divided by the derivative of the declining portion ($u < u_0$). As $R_1(j)$ increases, so $\Psi$ increases. Theorem 2 shows that, given population levels fluctuating about an equilibrium value, a greater value of $\Psi$ will dominate all lower values. Therefore, when $R_r$ and $R_c$ are fixed, the optimal value of $R_1 = \infty$.

In the previous example, $R_r(j)$ is the same for all lineages. I consider the case where lineages have differing values of $R_r(j)$. Specifically, I first consider the case where $R_r(j) = \alpha R_1(j)$ ($\alpha$ is a variable). Here,

$$
\begin{aligned}
\Psi(j) &= \frac{R_1(j) - R_r(j)}{R_1(j) + R_r(j)} \\
&= \frac{R_1(j) - \alpha R_1(j)}{R_1(j) + \alpha R_1(j)} \\
&= \frac{1 - \alpha}{1 + \alpha} \, .
\end{aligned}
\tag{3.7}
$$

By the theorem, any dominant lineage will have a greater value of $\Psi$, but Equation (3.7) shows that when $\alpha$ is increasing, $\Psi$ is decreasing and *vice-versa*. Therefore, the lineage with the lowest value of $\alpha$ will dominate. In other words, the ratio between an individual's reproduction threshold ($R_1$) and its cost of reproduction ($R_r$) must decrease for there to be an advantage in increasing the reproduction threshold.

Finally, I consider the cost of growth/maintenance $R_c$. Figure 3.2 shows plots of the growth rates for two lineages where all parameters are the same apart from $R_c$. The extra cost has shifted the plot of lineage B to the right on the graph. This means that this lineage has lower growth, and is therefore at a disadvantage, for all values of $u$.
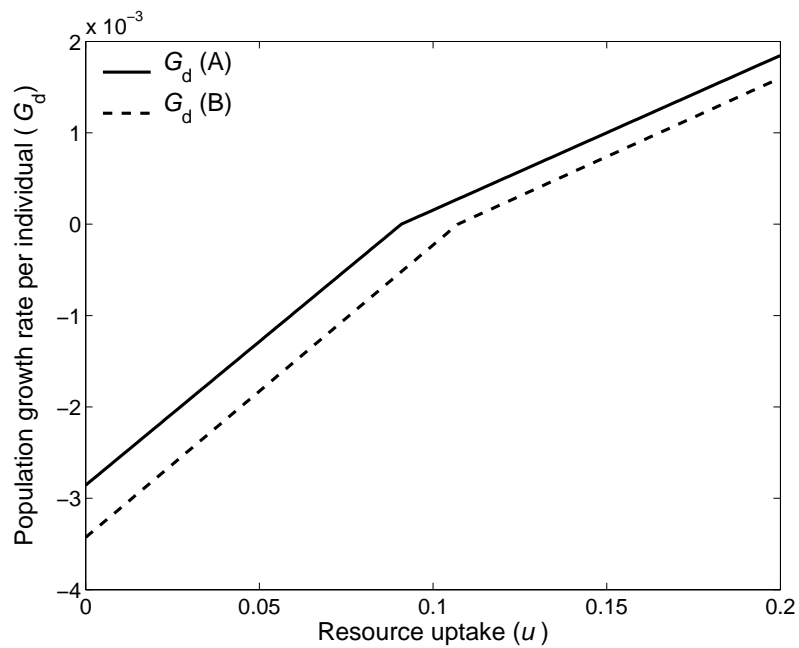


Figure 3.2: Comparison of the growth rates of two lineages A and B with different growth/maintenance costs. Equation (3.5) is with parameters $R_1 = 1.0$, $R_u = 0.01$, $R_r = 0.3$, $\delta t = 1$, $R_c(\text{A}) = 0.001$ and $R_c(\text{B}) = 0.0012$.

I also consider the case where lineage B has increased values of both the reproduction threshold ($R_1$) and the cost of growth/maintenance ($R_c$) compared with lineage A. Figure 3.3 shows plots of the growth rates for the two lineages. As with Figure 3.2, the plot of lineage B is shifted to the right. If the resource uptake is always bounded within $u = 0.079$ (where the plots cross) and $u = 0.11$ (where lineage B has zero growth), then lineage A is always dominant.

It is difficult to cleanly analyse the case shown by Figure 3.3 because it is not compatible with Equations (A.1) and (A.2), used in Theorem 2 in Appendix A. The two lineages (A and B) have different values of $R_c$ and therefore different values of $u_0$. A qualitative
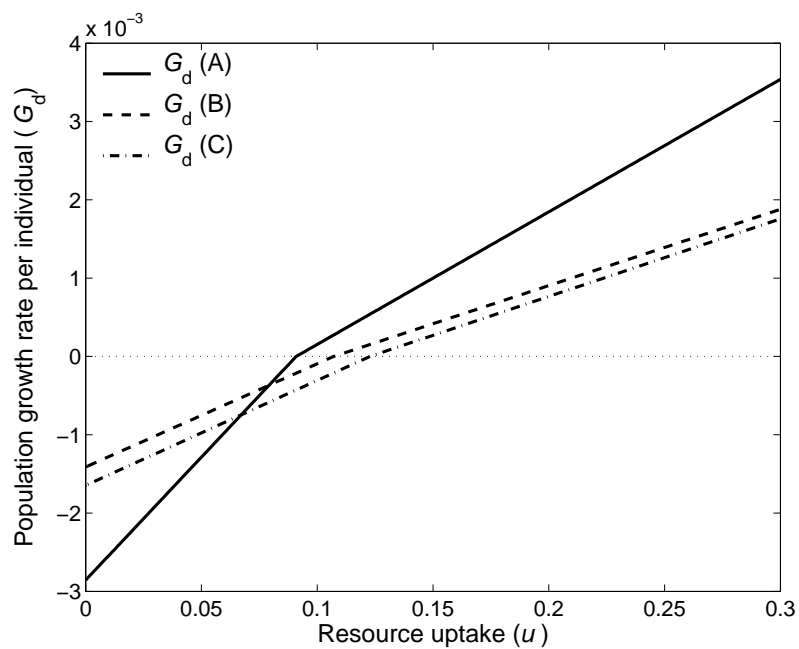
Figure 3.3: Comparison of the growth rates of two lineages A and B with different growth/maintenance costs and reproduction thresholds. Lineage C also shown (it is similar to lineage B but with an even greater value of $R_c$) to illustrate further the effect of increasing $R_c$. Equation (3.5) is with parameters $R_1(A) = 1.0$, $R_1(B) = 2.0$, $R_1(C) = 2.0$, $R_u = 0.01$, $R_r = 0.3$, $\delta t = 1$, $R_c(A) = 0.001$, $R_c(B) = 0.0012$ and $R_c(C) = 0.0014$.

analysis of the figure does however suggest that lineages B and C may be dominant when the amplitude of resource fluctuations is greater.

To investigate the effect of greater resource fluctuation amplitudes further, I have done a simulation the two lineages A and B using Equation (3.3). Lineage population levels are represented by a floating point variable $N(t)$, where $N(t+1) = GN(t)$. For each simulation resource uptake fluctuates between two values ($u^+$ and $u^-$) based on the state of the system which fluctuates between growth and decline periods. Specifically, when the combined population of the two lineages ($N_A + N_B$) goes below a lower threshold (here 1.0) the system goes into a growth phase and $u = u^+$, and when the combined population ($N_A + N_B$) goes above an upper threshold (here 10.0) the system goes into a decline phase and $u = u^-$. Time traces for two simulations are shown in Figure 3.4.

The figure shows how, when the amplitude of fluctuations is low, lineage A is dominant (with its lower values of $R_1$ and $R_c$). Alternatively, when the amplitude of fluctuations is high, lineage B is dominant (with its higher values of $R_1$ and $R_c$). To show this pattern is the case for lineages A and B over a full range of values of $u$, I firstly show how the range of $u$ in which lineage A is dominant over lineage B will increase as $R_c(B)$ increases. Second, I show that there are still ranges for $u$ where lineage B is still dominant.

To show that the range of $u$ in which lineage A is dominant over lineage B will increase as $R_c(B)$ increases, I consider Figure 3.3. The value of $u$ where lineage A crosses lineage B will decrease because the plot of lineage B has a shallower gradient to the plot of lineage A [because $R_1(B) > R_1(A)$, see Equation (3.5)]. If $u$ stays above this bound then the growth rate of lineage A will always be above lineage B. Therefore the range of $u$ in which lineage A is dominant over lineage B will increase as $R_c(B)$ increases.

Second, to show that B may still dominate over A, it is possible to look at the case where $u$ oscillates between only two values, $u^+$ where the population grows and $u^-$ where the population declines. For each value for $u$, the growth rates $G_d(u)$ can be generated. Corollary 3 in Appendix A shows that if

$$\frac{G_d(B, u^+)}{-G_d(B, u^-)} > \frac{G_d(A, u^+)}{-G_d(A, u^-)},$$

then lineage B will dominate. Since all the values in the fractions are positive, this can be rearranged as,

$$\frac{G_d(B, u^+)}{G_d(A, u^+)} > \frac{G_d(B, u^-)}{G_d(A, u^-)}.$$
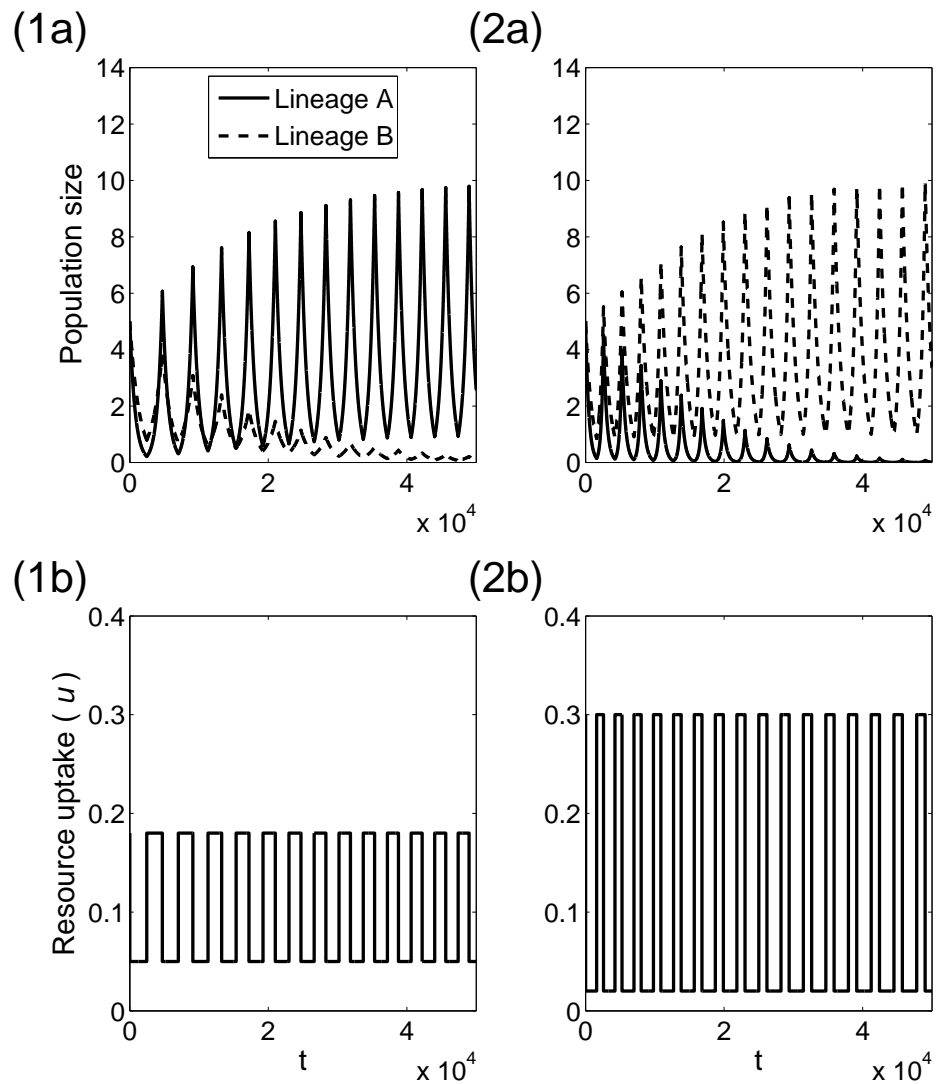
Figure 3.4: A lineage with an increased value of $R_1$ and $R_c$ may invade when the amplitude of resource fluctuations is increased. Panels (1a) and (2a) show population numbers and panels (1b) and (2b) show the corresponding values of resource uptake. Population growth is given by Equation 3.3. Parameters for populations A and B are as given in Figure 3.3. Panels (1a) and (1b) use $u^+ = 0.18$ and $u^- = 0.05$, panels (2a) and (2b) use $u^+ = 0.3$ and $u^- = 0.02$.

In the case shown in Figure 3.3, for $u^+ > 0.25$,

$$G_d(B, u^+)/G_d(A, u^+) > 0.516 \, .$$

Similarly, for $u^- < 0.021$ we have that

$$G_d(B, u^-)/G_d(A, u^-) < 0.516 \, .$$

So if $u$ is outside the range $0.021 > u > 0.25$, lineage B will dominate. One important point to note is that these values for $u^+$ and $u^-$ were chosen arbitrarily. It is possible to choose different values for $u^+$ and $u^-$ (perhaps increasing or decreasing both of them) and still see dominance of lineage B. The distance between $u^+$ and $u^-$ is not fixed either and how it changes can also depend on the choice of the other parameters. This means that more general analysis of this case is difficult within this model. What can be said however, is that the effect of increasing the parameter $R_c$ is always the same, so the basic reasoning that a larger amplitude of resource fluctuations is necessary for increased values of $R_c$ is applicable over sensible parameter ranges.

The significant results from this section are as follows. First, this model predicts that, when all other parameters are held equal, the optimal strategy is to maximise the reproduction threshold $R_1$. Second, when the value of $R_r$ is linked to the value of $R_1$ any increase in $R_1$ may be matched by a corresponding increase in $R_r$ as long as the value of $\alpha = R_1/R_r$ decreases. Finally, when a lineage has increased values of both $R_1$ and $R_c$, I have shown that a larger amplitude of resource fluctuations can be important its viability. However, the model does not yield to simple analysis. This means that I will look to simulations, presented later on in this chapter, to provide further insights into this scenario.

In this model, all individuals receive a continuous level of resources. This makes it very difficult to analyse what will happen when the population is at its carrying capacity. In the next section, I introduce stochasticity into the model which means I may analyse the population in this state.

### 3.2.4 Stochastic model

In this section equations for $W_s$ and $F_s$ are presented for the stochastic model and a growth rate $G_s$ is derived. As outlined above, time is broken up into segments of length $\delta t$. Tithe individual resource level in the stochastic model is given by Equation (3.1) with $U(i, t)$ modelled by white noise with $\Pr(U = 1) = u$ and $\Pr(U = 0) = 1 - u$ for each

time segment. The same variable $u$ is used as in the deterministic model as the average per-timestep resource uptake over realisations is $uR_u$ – in harmony with the deterministic model. Figure 3.5 shows the resource level of a typical individual in the stochastic model.
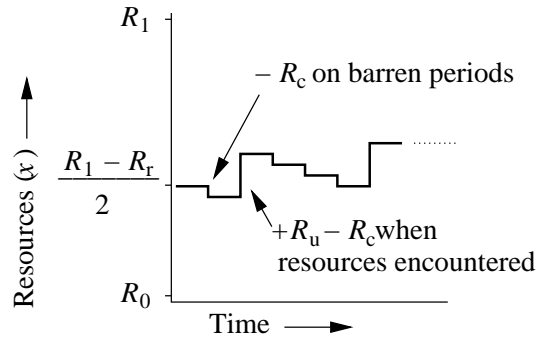


Figure 3.5: The resources ($x$) of individuals in the stochastic model perform a random walk throughout the individual's lifetime. The individual shown starts its walk after reproduction with half the maximum resource ($R_1$) less a cost of reproduction ($R_r$). For each time segment of length $\delta t$ an individual will expend $R_c$ resources and may receive $R_u$ resources with probability $u$.

For simplicity of modelling a variable $m$ is introduced where $m = R_u/R_c - 1$ [so, $R_u = (m+1)R_c$]. Since $R_u > R_c$, and both are positive, $m$ is positive. The expected generation time $W_s(x)$ is modelled using the backward equation (Berg 1993). For an individual starting with energy value $x$:

$$W_s(x) = \delta t + uW_s[x + mR_c] + (1-u)W_d(x - R_c) \ . \tag{3.8}$$

The equation models the fact that during time $\delta t$, an individual will have fed and moved to energy $x + mR_c$ with probability $u$ or will not have fed and therefore moved to energy $x - R_c$ with probability $1 - u$. Here, to harmonise with normal calculus notation, I set $\delta x = R_c$. Dividing Equation (3.8) through by $\delta x$ and moving $W_s(x)$ to the right hand side gives:

$$0 = \frac{\delta t}{\delta x} + mu\frac{[W_s(x + m\delta x) - W_s(x)]}{m\delta x} + (1-u)\frac{[W_s(x - \delta x) - W_s(x)]}{\delta x} \ . \tag{3.9}$$

When $\delta x$ is small the first three terms of a Taylor expansion are considered:

$$W_s(x + m\delta x) \simeq W_s(x) + m\delta x W_s{}'(x) + \frac{(m\delta x)^2}{2!}W_s{}''(x) + ...$$

$$W_s(x - \delta x) \simeq W_s(x) - \delta x W_s{}'(x) + \frac{\delta x^2}{2!}W_s{}''(x) + ... \ . \tag{3.10}$$

In the case where $m\delta x < 1$ (or $mR_c < 1$) it is possible to substitute Equations (3.10) into Equation (3.9) giving a continuous approximation:

$$
\begin{aligned}
0 &\simeq \frac{\delta t}{\delta x} + mp\left[W_s'(x) + \frac{m\delta x}{2}W_s''(x)\right] + (1-u)\left[-W_s'(x) + \frac{\delta x}{2}W_s''(x)\right] \\
&\simeq \frac{\delta t}{\delta x} + (mu + u - 1)W_s'(x) + \frac{\delta x}{2}\left(m^2u + 1 - u\right)W_s''(x)
\end{aligned} \tag{3.11}
$$

Reverting back to using $R_c$ instead of $\delta x$ and setting

$$
\begin{aligned}
\lambda &= (m+1)u - 1 \\
\mu &= \frac{R_c}{2}[(m^2 - 1)u + 1]
\end{aligned} \tag{3.12}
$$

gives

$$
0 \simeq \frac{\delta t}{R_c} + \lambda\frac{dW_s}{dx} + \mu\frac{d^2W_s}{dx^2} . \tag{3.13}
$$

The equation can be solved (by first integrating, then using an integrating factor) with the boundary conditions [$W_s(0) = 0$ and $W_s(R_1) = 0$] to give the equation:

$$
W_s = \frac{\delta t}{R_c\lambda}\left[-x + \frac{R_1\left(1 - e^{-\frac{\lambda x}{\mu}}\right)}{1 - e^{-\frac{\lambda R_1}{\mu}}}\right] . \tag{3.14}
$$

The role of $\lambda$ is interesting, it is at zero when $u = 1/(m+1) = u_0$. When lambda is positive, $u > u_0$, resources are abundant and individuals are more likely to move towards their upper energy limit ($R_1$). When lambda is negative, $u < u_0$, resources are sparse and there is a tendency for individuals to move towards their lower energy limit ($R_0$). There is a special case when $\lambda = 0$, $W_d = \infty$ in Equation 3.14. To look at the case where $\lambda = 0$, i.e., $u = u + 0$ and $\mu = R_cm/2$ , Equation (3.13) is reconsidered:

$$
0 \simeq \frac{\delta t}{R_c} + \frac{R_cm}{2}\frac{d^2W_s}{dx^2} . \tag{3.15}
$$

This can be solved with the same boundary conditions as Equation (3.14) giving:

$$
W_s = \frac{\delta t}{R_c^2m}(-x^2 + R_1x) . \tag{3.16}
$$

When resources are neither abundant nor sparse there is an unbiased random walk. Equation (3.16) demonstrates that the expected lifetime is still finite. This is one significant difference from the deterministic model where the expected lifetime is infinite when re-

source uptake is close to the threshold for population growth.

The ratio $F_s$ of individuals at energy $x$, which reach the upper threshold $R_1$ to those that reach the lower threshold $R_0$ is given by the backwards equation:

$$
\begin{aligned}
F_s(x) &= uF_s(x+R_u-R_c)+(1-u)F_s(x-R_c) \\
&= uF_s(x+mR_c)+(1-u)F_s(x-R_c) \ .
\end{aligned}
\tag{3.17}
$$

Again an individual either will have fed and moved to energy $x+mR_c$ with probability $u$ or will not have fed and therefore moved to energy $x-R_c$ with probability $1-u$. Now move $F_d(x)$ to the right hand side of the equation and replace $R_c$ with $\delta x$.

$$
0 = mu\frac{[F_s(x+m\delta x)-F_s(x)]}{m\delta x}+(1-u)\frac{[F_s(x-\delta x)-F_s(x)]}{\delta x} \ .
\tag{3.18}
$$

Using an approximation of the first three terms of a Taylor expansion:

$$
\begin{aligned}
0 &\simeq mu\left[F_s'(x)+\frac{m\delta x}{2}F_s''(x)\right]+(1-u)\left[-F_s'(x)+\frac{\delta x}{2}F_s''(x)\right] \tag{3.19}\\
&\simeq (mu+u-1)F_s'(x)+\frac{\delta x}{2}\left(m^2p+1-p\right)F_s''(x) \ . \tag{3.20}
\end{aligned}
$$

Reverting back to using $R_c$ instead of $\delta x$ and using the previous definitions of $\lambda$ and $\mu$ gives

$$
0 \simeq \lambda\frac{dF_s}{dx}+\mu\frac{d^2F_s}{dx^2} \ .
\tag{3.21}
$$

The boundary conditions are $F_s(0)=0$ and $F_s(R_1)=1$. Solving as with $W_s$, gives:

$$
F_s = \frac{1-e^{-\frac{\lambda x}{\mu}}}{1-e^{\frac{-\lambda R_1}{\mu}}}
\tag{3.22}
$$

To derive the growth rate, Equation 3.3 is used giving,

$$
G_s(j) = \frac{2F_s(j)-1}{W_s} \ .
\tag{3.23}
$$

The predictions of this equation and Equations (3.14) and (3.22) are compared with the predictions of the deterministic model in the next section.

### 3.2.5 Comparison of stochastic model and deterministic model

The stochastic and deterministic models differ in the way individuals receive resources. To understand under which parameter regimes the deterministic model is a good approximation of the stochastic model, I compared the growth rates of the deterministic model ($G_d$) and the stochastic model ($G_s$). These are calculated using Equation (3.5) and Equation (3.23) over a variety of parameters. The difference between the two ($G_d$ - $G_c$) is plotted in Figure 3.6.
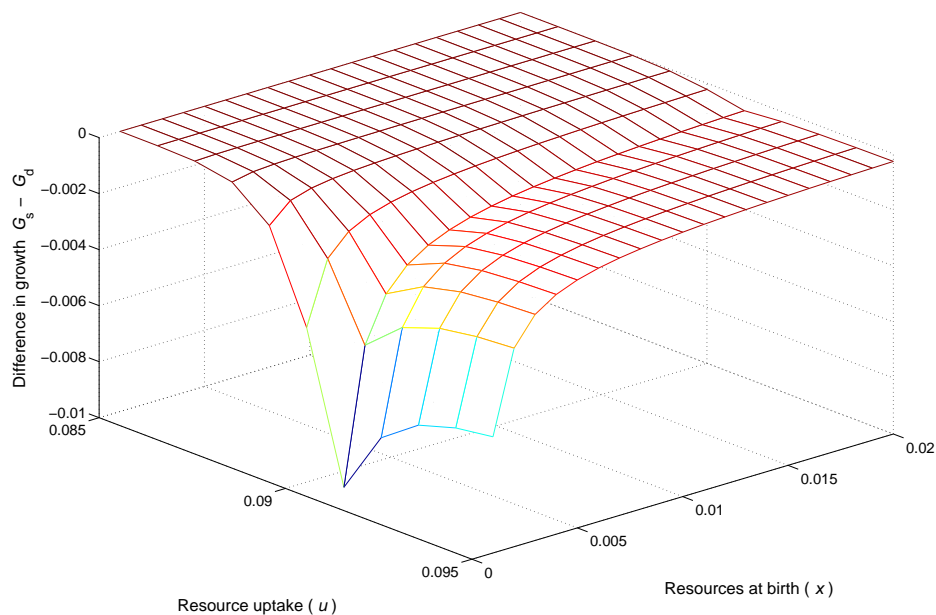


Figure 3.6: A comparison of predicted growth rates [using Equation (3.3)] for the deterministic and stochastic models. The parameters used were $R_c = 0.001$, $R_u = 0.011$ and $R_1 = 1.0$.

In general the deterministic model is a good approximation of the stochastic model, however when growth is low there are some differences. With $R_c = 0.001$ and $R_u = 0.011$, the point where resource uptake per timestep equals resource expenditure in both models is where $u = u_0 = 0.0909$. When $u \approx u_0$ and $x < 0.5$, there is a lower growth rate for the stochastic model than the deterministic model. There is a higher growth rate when $x > 0.5$ (not shown). This is explained by the fact that, at $u = u_0$, the stochastic model models an unbiased random walk so an individual's resource level will eventually reach one of the thresholds. The stochastic individual's resource level is more likely to reach the upper threshold when $x > 0.5$ and the lower threshold when $x < 0.5$. Alternatively, the growth rate of the deterministic model is zero when $u = u_0$, hence the difference in the plot between the deterministic model and stochastic model. This difference was also apparent when different values of $R_1$, $R_c$ and $R_u$ were tested.

When $x$ is close to the low threshold ($x < 0.01$) and $u \approx u_0$ or $u > u_0$, the stochastic model predicts a lower growth rate than the deterministic model. Similarly, when $x$ is close to the upper threshold ($x > 0.997$) and $u \approx u_0$ or $u < u_0$, the stochastic model predicts a higher growth rate than the deterministic model (not shown). In the stochastic model, the value of $x$ may move in both positive and negative directions. Therefore, when the value of $x$ is close to one of the thresholds there is a small likelihood that it will cross that threshold, even if the general trend is for the value to move in the opposite direction. Again, this effect was noticeable when different values of $R_1$, $R_c$ and $R_u$ were tested.

The scenario considered in this chapter considers the case where individuals start life at $x = [R_1 - R_r(j)]/2$. With $x$ starting from this point, the stochastic model only differs significantly from the deterministic model when $u \approx u_0$ (the point where resource uptake equals consumption). This case is therefore considered in the next section.

### 3.2.6 Stochastic model analysis

To analyse the stochastic model, I consider the case where the population is close to equilibrium and there is little or no population growth, i.e., where resource uptake is approximately equal to resource consumption. I therefore assume that resource uptake ($u$) is directly dependent on population density and there are no wild fluctuations. I define a resource uptake value $u_e(j)$, dependent on the lineage, for which there is no growth. Note that it is not necessary that $u_e(j)$ is equal to $u_0$. The value of $u_e(j)$ is also the threshold for population growth so when $u < u_e$ the population level will decrease. Because $u$ is dependent on population density, it will correspondingly increase. Similarly, when $u > u_e$ the population level will decrease and $u$ will increase. When $u = u_e$ there will be no growth. I envisage a scenario where the value of $u$ will rapidly converge to $u_e$ and zero population growth. To compare two lineages with different values of $u_e$, I consider the cartoon in Figure 3.7.

The cartoon shows how a lineage which has a low value of $u_e$ will invade all lineages which have higher values of $u_e$. This is because the dominant lineage will grow when the other lineages are at equilibrium and the other lineages will decline when the dominant lineage is at equilibrium.

To find the value of $u_e(j)$, I solve Equation 3.23 for $G_s(j) = 0$. This gives $F_s = 1/2$. The Equation (3.22) is unfortunately too hard to solve for $u$, so the only approach is to find numerical solutions. This is done by iterating through values of $u$ until $F_s$ is within a tolerance ($10^{-7}$) of 0.5.

As with in Section 3.2.3, I first looked at the case where all individuals are identical
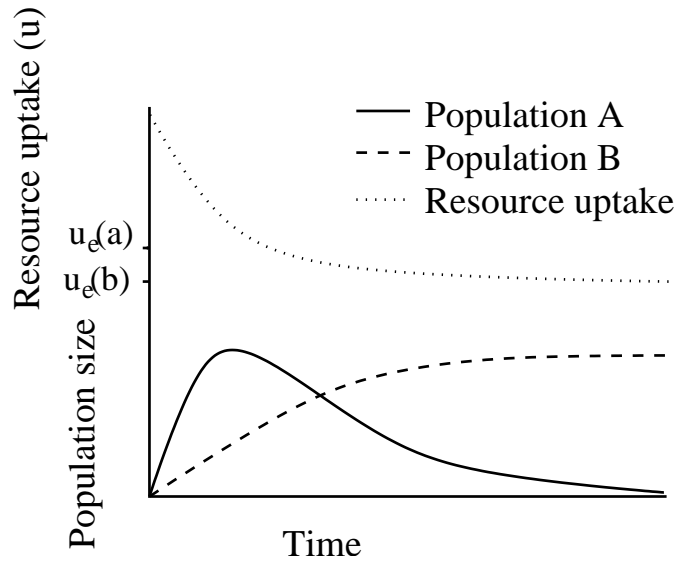
Figure 3.7: The population with the lower value of $u_e$ will dominate. Here lineage A increases more quickly at first, but the increase in overall population density means that $u$ will decrease. When $u$ reaches $u_e(A)$ population A starts to decline. As lineage B can continue growing, $u$ must decrease further until it reaches $u_e(B)$ where lineage B has a stable population level but lineage A is declining.

apart from their reproduction threshold ($R_1$). The value of $u_e$ was calculated for each value of $R_1$ and is plotted in Figure 3.8.

As with the deterministic model [see Equation (3.6)], lineages with higher values of $R_1$ dominate lineages with lower values of $R_1$. This is shown by the fact that as the value of $R_1$ increases in the figure, the resource uptake required for population equilibrium [$u_e(j)$] decreases. Any lineage that needs a higher resource uptake for equilibrium will be dominated by those that can survive on a lower resource uptake. There is a very steep decrease at low values of $R_1 < 1$, with the gradient becoming shallower as $R_1$ increases. Varying the parameters ($R_r$, $R_u$ and $R_c$) did not change the basic shape of the graph. I also experimented with very large values of $R_1$: this indicated that as $R_1 \to \infty, (u - u_0) \to 0^{+ve}$.

To look at the situation where there is a trade-off between the value of $R_r(j)$ and $R_1(j)$, I again consider the case $R_r(j) = \alpha R_1(j)$. The value of $u_e$ was calculated for values of $R_1$ and $R_r$ and is plotted (with both $u_e$ and $R_1$ on logarithmic scales) in Figure 3.9.

As with Figure 3.8, the value of resource uptake required for population equilibrium ($u_e$) decreases as the reproduction threshold ($R_1$) increases, even when the reproduction cost ($R_r$) increases as well. Again there is a very steep decrease at low values of $R_1 < 1$, with the gradient becoming shallower as $R_1$ increases. As the value of $\alpha$ was increased, the steepness of the descent of the curve increased.

Varying the other parameters ($R_u$ and $R_c$) did not change the basic shape of the graph.
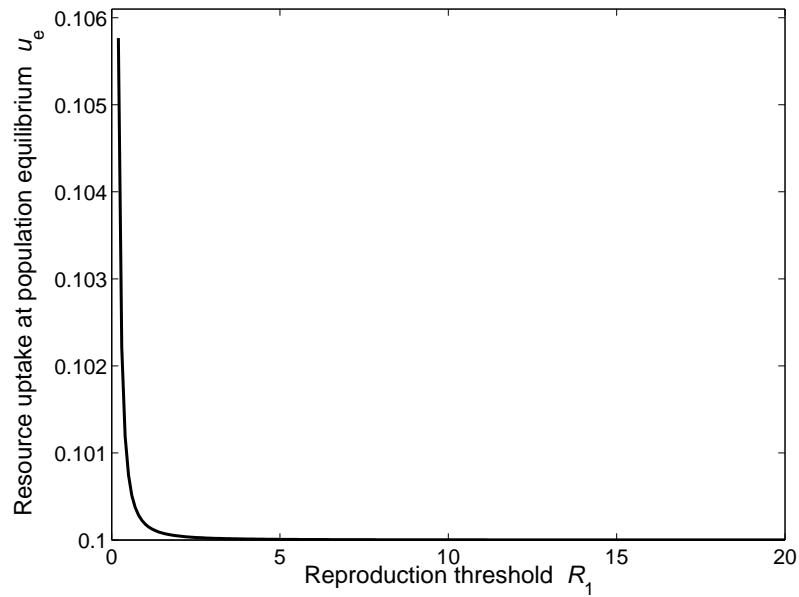
Figure 3.8: The resource uptake required for population equilibrium ($u_e$) decreases as $R_1$ is increased. The value of $u$ for which $F_s = 1/2$ is plotted. Other parameters for Equation (3.22) were $R_r = 0.1$, $R_u = 0.01$ and $R_c = 0.001$.
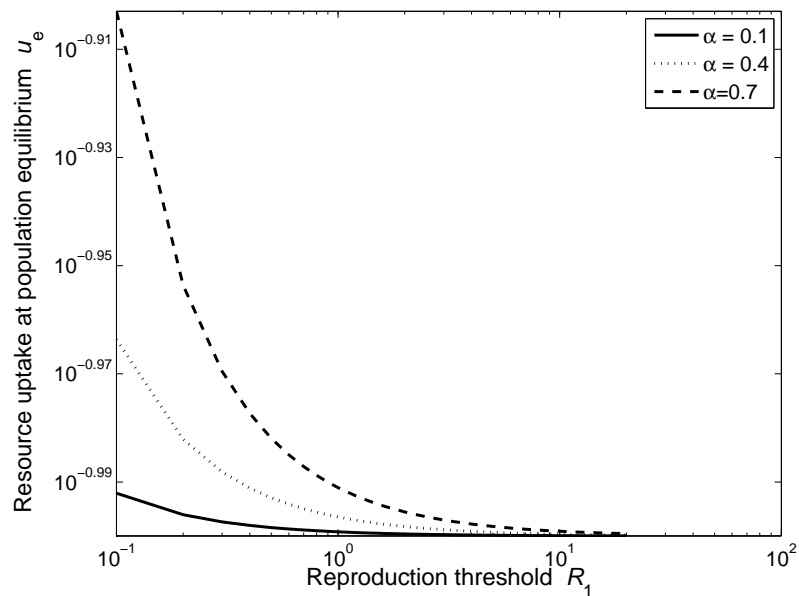


Figure 3.9: The resource uptake required for population equilibrium ($u_e$) decreases as $R_1$ is increased and $R_r = \alpha R_1$. The value of $\alpha$ is as shown. The value of $u$ for which $F_s = 1/2$ is plotted. Parameters for Equation (3.22) were $R_u = 0.01$ and $R_c = 0.001$.

Again, very large values of $R_1$ indicated that, for all values of $\alpha$, as $R_1 \to \infty, (u - u_0) \to 0^{+\text{ve}}$.

This result is not consistent with the result from the deterministic model [see Equation (3.7)]. Here, when $\alpha$ is constant, increasing $R_1$ is advantageous. In the deterministic model, when $\alpha$ is constant, there is no advantage to increasing or decreasing $R_1$.

Finally, I consider the scenario where $R_c$ may vary with $R_1$ and $R_r$ is fixed: $R_c = \beta R_1$, $\beta$ is a constant. The value of $u_e$ was calculated for each value of $R_1$ and $R_c$ and is plotted in Figure 3.10.
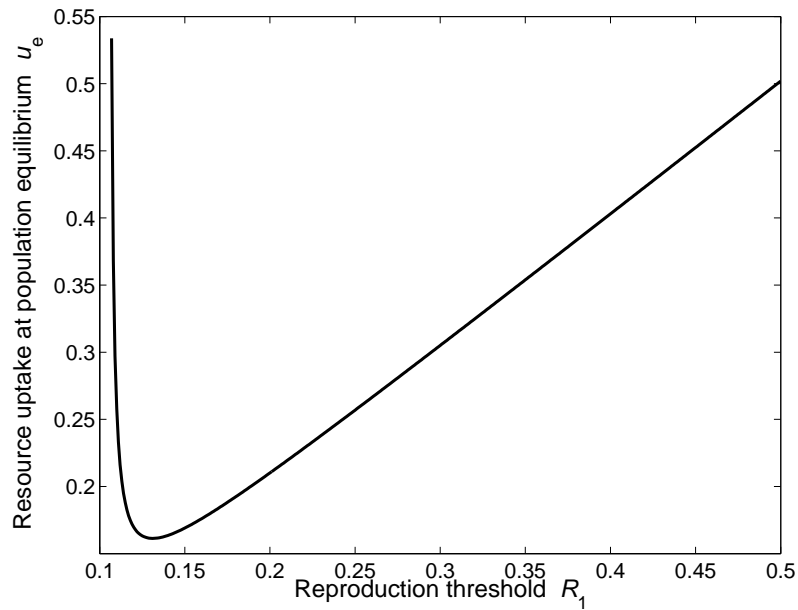


Figure 3.10: The resource uptake required for population equilibrium ($u_e$) decreases, then increases, as $R_1$ is increased and $R_c = \beta R_1$. The value of $u$ for which $F_s = 1/2$ is plotted. Here $\beta = 0.01$, other parameters for Equation (3.22) were $R_u = 0.01$ and $R_r = 0.1$.

At first, as $R_1$ is increased, there is a steep decrease in the value of resource uptake required for population equilibrium ($u_e$). The curve reaches its nadir at $R_1 = 0.131$ and $u_e = 0.161$. After this point the value of resource uptake starts to increase in a linear fashion approximately tracing the function $u_e = R_1$. The value of $R_1$ at the nadir of the curve is plotted for different values of $\beta$ in Figure 3.11.

The figure shows how the optimum reproduction threshold decreases as the ratio of maintenance cost to reproduction threshold increases. The value at $\beta = 0$ is not present because with $R_c = 0$ no individuals will reach the lower threshold so $F_s = 1$. All values of $\beta > 0.1$ are also not present because with $R_c > R_u$ no individuals will reach the upper threshold. Increasing $R_r$ or $R_u$ increases the value of $R_1$. The downward slope of the graph changes approximately proportionally to $R_r$.
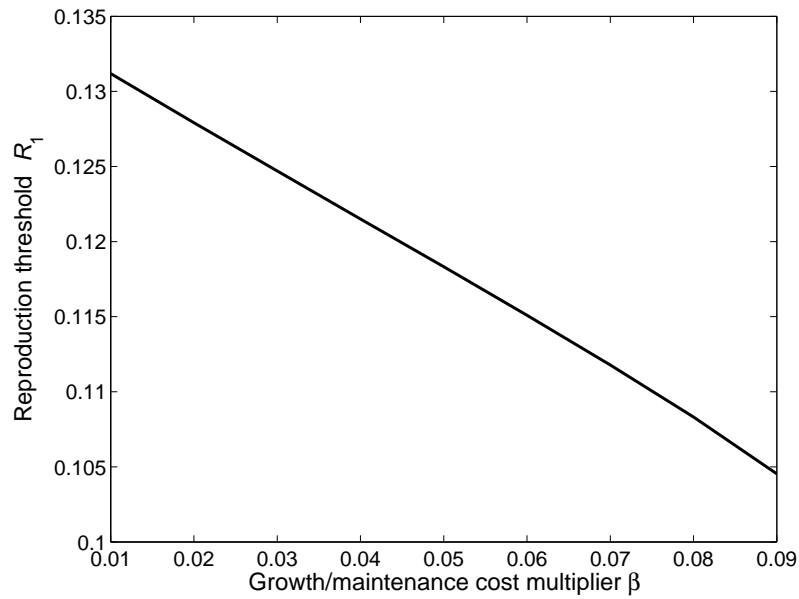
Figure 3.11: As the ratio of maintenance cost to reproduction threshold increases, the optimum reproduction threshold decreases. The value of $R_1$ for the minimum value of $u_e$ [calculated using Equation (3.22), see Figure 3.10] is plotted over several values of $\beta$. Other parameters for Equation (3.22) were $R_u = 0.01$ and $R_r = 0.1$.

## 3.3 Simulations

The simulations are run for two reasons. First, to see how accurate the mathematical models are, simulation models were performed of the lifetime of individuals and the ratio of individuals that reach the upper threshold to those that reach the lower threshold. Second, to validate and extend the mathematical analysis, evolutionary simulations were performed.

The simulations are based on the stochastic model. In all simulations, each individual is represented as a resource level $x$. The evolution of $x$ is modelled by an Euler approximation of Equation (3.1):

$$x(i, t + \tau) = x(i, t) + U(i, t)R_u - R_c(j) . \tag{3.24}$$

As with the stochastic model, see Section 3.2.4, $U(i, t)$ was modelled by white noise: for each time segment of length $\delta t$, $\Pr(U = 1) = u$ and $\Pr(U = 0) = 1 - u$. Usually the value $\tau$ was set to $\delta t$, but tests were made for the simulation runs with smaller values of $\tau$ to ensure no artifacts were introduced due to the size of $\tau$.

### 3.3.1 Testing the accuracy of the deterministic/stochastic models

To test the diffusion approximation made for the stochastic model equations [Equations (3.14) and (3.22)], simulations are run of the individual generation cycle. The individual generation cycle is the period of time while an individual's resources move from a starting position $x_0$ to either the upper ($R_1$) or lower ($R_0$) threshold. Once an individual has reached the lower threshold ($R_0$) or the upper threshold ($R_1$), which threshold reached and the time taken to reach it (the generation time) is recorded. Pseudo-code for the simulations is presented in Figure 3.12. For each simulation run this is done a number of times, with different random seeds, and mean values are taken. This gives a mean value for the lifetime $\bar{W}_{\text{sim}}$ and a mean value for the birth-to-death ratio $\bar{F}_{\text{sim}}$.

```
0:  Require t = 0
1:  for x_0 is 0 to 1 in steps of 0.01
                | x_0 |
2:       x =    | ... |
                | x_0 |
3:       while |x| < 0
4:           increment t
5:           for x is each element of x
6:               x = x − R_c
7:               with probability u, x = x + R_u
8:           end for
9:           Remove elements of x that are < 0 or > R_1,
             and record lifetime (=t) and whether at 0 or R_1
10:      end while
11:      Calculate and record mean lifetime and the ratio that reach R_1
12: end for
```

Figure 3.12: Pseudo-code for the simulations to test the diffusion approximation made for the stochastic model equations [Equations (3.14) and (3.22)].

To test the accuracy of Equations (3.14) and (3.4), simulations were run 1,000 times for each different value of $x$ to generate the mean value for the lifetime $\bar{W}_{\text{sim}}$. This is plotted alongside values of $W$ given by Equations (3.14) and (3.4) in Figure 3.13.

At low to medium values of $m$ and $R_c$ ($R_c < 0.01$ or $m < 100$), Equation (3.14) gives a good approximation to the simulation (Panels A to C of Figure 3.13). The deterministic model is generally a good approximation except when $u \approx u_0$ (Panels C and D). Panel D shows that neither approximation is good with high values of $m$. Similarly, neither approximation is good at higher values $R_c$ (not shown). At the special case where $u = u_0$, the accuracy of Equation (3.16) also depends on $m$ and/or $R_c$. Similarly to Equation (3.14),
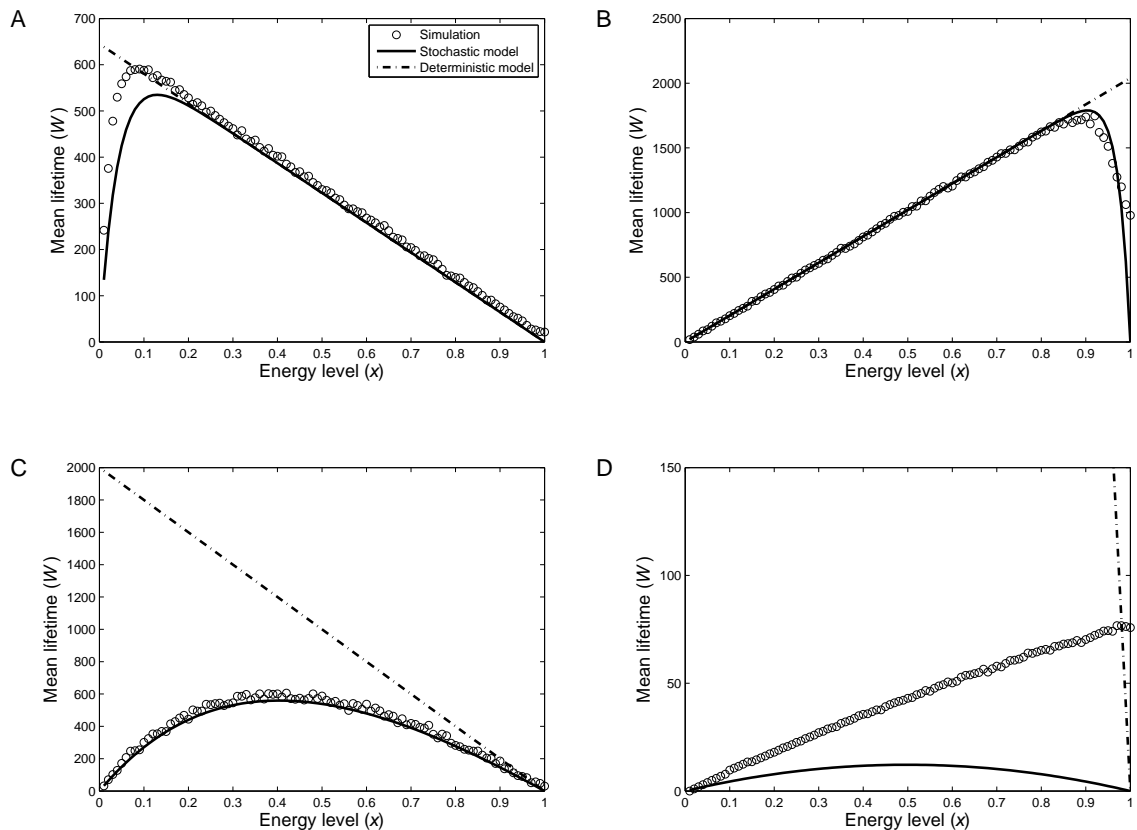
Figure 3.13: A comparison of the lifetime simulation results (signified by circles) with Equations (3.14) (line) and (3.4) (dots). The values $\delta t = 1$ and $R_1 = 1.0$ were used in all Panels. To model a value of $u$ where the population is growing, the other values used in Panel A were $R_c = 0.001$, $m = 50$ and $u = 0.05$. Similarly, to model a value of $u$ where the population is declining, the other values used in Panel B were $R_c = 0.001$, $m = 50$ and $u = 0.01$. To model a value of $u$ close to $u_0$ the values used in Panel C are $R_c = 0.01$, $m = 4$, $u = 0.21$ and $R_1 = 1.0$. Finally, when $m$ is very large, the values used in Panel D are $R_c = 0.01$, $m = 200$, $u = 0.0051$ and $R_1 = 1.0$. Panel D is cropped meaning the continuous solution is not clearly shown. It decreases from $W = 4000$ at $x = 0$ to $W = 0$ at $x = 1$.

Equation (3.16) is only accurate at low to medium values of $m < 100$ and $R_c < 0.01$ (not shown).

Simulations were also done to test Equation 3.22. Each simulation was run 1,000 times for each different value of $x$ to generate the mean value for the birth-to-death ratio $\bar{F}_{\text{sim}}$. This is plotted alongside the value of $F$ given by Equation (3.22) in Figure 3.14.
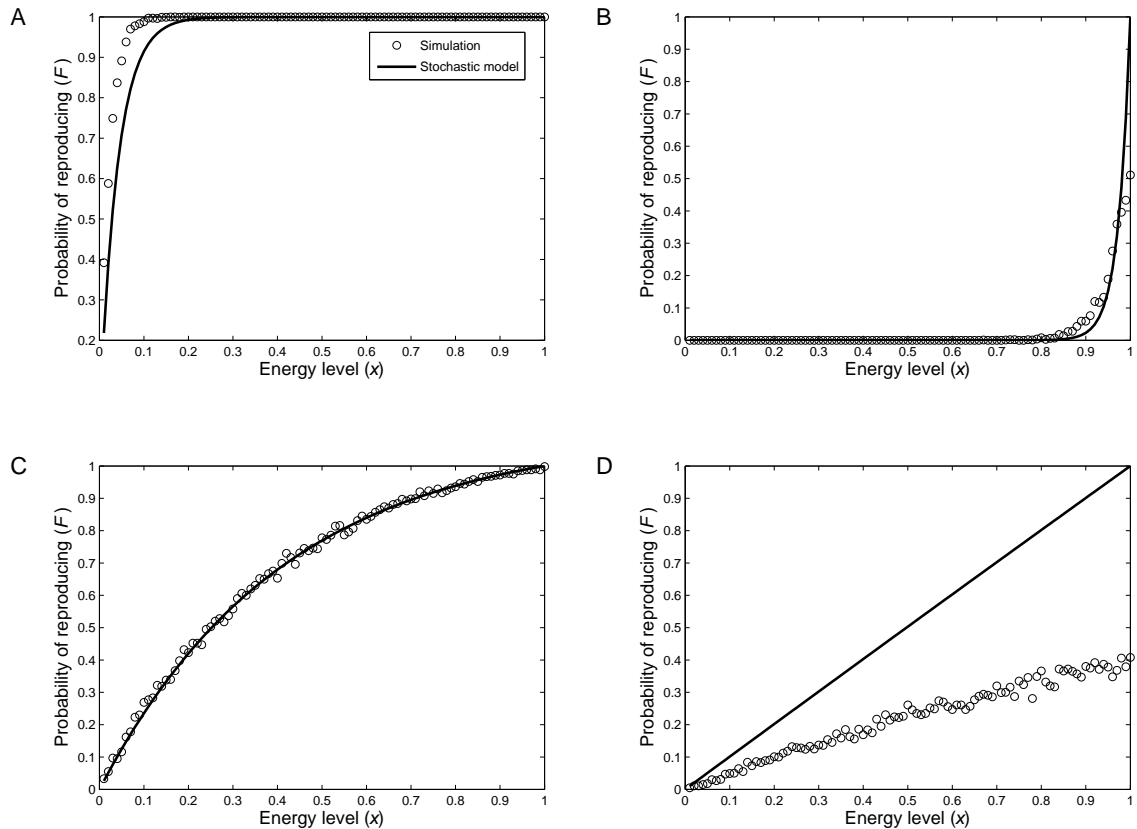
Figure 3.14: A comparison of simulation predictions (signified by circles) with Equation (3.22) (line) for the birth-to-death ratio. The values used in Panels A-D are as in Figure 3.13.

At low to medium values of $m$ and $R_c$ ($R_c < 0.01$ or $m < 100$), Equation (3.22) gives a good approximation to the simulation (Panels A to C of Figure3.14). The deterministic model (not plotted, but $F = 1$ in Panels A,C and D, and $F = 0$ in Panel B) is generally a good approximation except when $u \approx u_0$ (Panels C and D). Panel D shows that neither approximation is good with high values of $m$. Similarly, neither approximation is good at higher values of $R_c$ (not shown).

At this point, the predictions of the deterministic lifetime and reproduction models seem to be quite inaccurate. However, the results given in Section 3.2.5 indicate that the growth rate ($G_d$) predictions are quite accurate. When the lifetime is quite high, the popu-

lation growth is likely to be very small. It is therefore possible that when the deterministic model is used with the population growth equation, Equation (3.3), that the model is quite accurate. This is shown by the comparison with the stochastic model in Figure 3.6. I will compare both the deterministic model and the stochastic model with the results from the simulations in the next section.

### 3.3.2 Evolutionary simulations

Given the results found by the two mathematical models, simulations are done to consider trade-offs between reproduction threshold ($R_1$), and reproduction cost ($R_r$) or cost of growth/maintenance ($R_c$).

The evolutionary simulation models are based on the stochastic model. To allow evolution to occur, software agents are assigned a digital gene which is a floating point number representing the reproduction (or upper) threshold $R_1(i)$ of an agent $i$. As with the mathematical models, when the agent's resource level increases above its reproduction threshold $R_1(i)$ then it will reproduce, paying the cost of reproduction $R_r(i)$, and then share the remainder of its resources with an offspring. A copy of the digital gene is passed on to the offspring. There is a probability of 0.1 of a mutation being applied to the offspring's gene after reproduction: point mutations are made to the value of $R_1(i)$ over a Gaussian distribution with standard deviation of 1%.

Simulations were run under two main scenarios. Scenario (i) models a population that is close to its equilibrium level. This is achieved by giving a static input of resources to the system: the availability of resources per individual therefore depends on population density. To model resource fluctuations in scenario (ii), the probability for each individual to receive resources each timestep is modelled by a system variable. This variable switches between two levels (higher and lower) when the population level passes outside one of the two thresholds (respectively, the lower and upper population thresholds).

Tests were done with various upper and lower population thresholds to make sure they are effective: the thresholds were too close to each other when they were within 33% of the population size, this had the same effect of reducing the fluctuation coefficient. The upper threshold was therefore set to 400 and the lower threshold was set to 200 in all simulations.

In scenario (i), the availability of resources to individuals is dependent on population density, meaning that the population level will quickly converge to a static equilibrium level. Just as with Section 3.2.6, when the population is above the equilibrium level there will not be enough resources in the system to support that number of individuals – and

the population will decrease. Alternatively, when the population is below the equilibrium there will be a surplus of resources and the population will increase. Based loosely on the chemostat, a number of resource units (normally 50, each of value $R_u$) are randomly scattered on a spatial grid (of $50 \times 50$ squares) each timestep. Agents located on the grid move to a random new cell each turn and consume a resource unit if they encounter any on the square they are on[2]. Pseudo-code for the simulation can be seen in Figure 3.15.

```
0:  Define agent as having resources x and reproduction threshold R₁
1:  Initialise x and R₁ for new members of the population agents
2:  for t = 1 to simulation_time
3:      Scatter new resources on the grid
4:      Move each agent to a random grid-square
5:      for each agent (picked in random order) in agents
6:          x = x − Rc
7:          if there are resources on the grid-square
8:              x = x + Ru
9:              remove one resource from the grid-square
10:         end if
11:         if x < 0, remove agent from simulation
12:         if x > R₁
13:             x = x − Rr (pay the cost of reproduction)
14:             Create a new agent (the offspring)
15:             Share the parents resources (x) evenly between the two agents
16:             Mutate the new agent's value of R₁
17:         end if
18:     end for
19: end for
```

Figure 3.15: Pseudo-code for the evolutionary simulations of scenario (i) where availability of resources depends on population density.

In scenario (ii) the agents receive discrete resources [of value $R_u$] with probability $u$. The value of $u$ depends on the state of the system, which fluctuates between two states (abundant, using $u = u^+$, and sparse, using $u = u^-$), and on the *resource fluctuation coefficient* $\eta$. When the system is in the abundant state, $u^+ = u_e \eta$. When the system is in the sparse state, $u^- = u_e / \eta$. The value of $u_e$ was chosen for each simulation based on the results from Section 3.2.6. The system switches between its two states depending on whether the population level is above an upper threshold or below a lower threshold.

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[2]The results presented by Bryden (2005b) show that agents receive resources with between-resource intervals on a geometric distribution when they move to random grid squares each timestep. This is equivalent to a population which receives resources with a probability proportional to the ratio of the population density and the resources in the environment

When the system in in the sparse state and the population goes below the lower threshold, the system switches to the abundant state. Conversely, when the system is in the abundant state and the population goes above the upper threshold, the system switches to the sparse state. Pseudo-code for the simulation can be seen in Figure 3.16.

```
 0:  Define agent as having resources x and reproduction threshold R₁
 1:  Initialise x and R₁ for new members of the population agents
 2:  season = high
 3:  for t = 1 to simulation_time
 4:      if |agents| > upper_threshold, season = low
 5:      if |agents| < lower_threshold, season = high
 6:      for each agent (picked in random order) in agents
 7:          x = x − R_c
 8:          if season = high
 9:              with probability u⁺, x = x + R_u
10:          end if
11:          if season = low
12:              with probability u⁻, x = x + R_u
13:          end if
14:          if x < 0, remove agent from simulation
15:          if x > R₁
16:              x = x − R_r (pay the cost of reproduction)
17:              Create a new agent (the offspring)
18:              Share the parents resources (x) evenly between the two agents
19:              Mutate the new agent's value of R₁
20:          end if
21:      end for
22: end for
```

Figure 3.16: Pseudo-code for the evolutionary simulations of scenario (i) where availability of resources depends on population density.

### 3.3.2.1  Varying reproduction cost

The two mathematical models make predictions about the trade-off between reproduction threshold ($R_1$) and reproduction cost ($R_r$). The deterministic model predicts that, if the population level fluctuates about an equilibrium, then an individual with a greater reproduction threshold will dominate as long as the relative increase in reproduction threshold is greater than the corresponding relative increase in reproduction cost [see Equation (3.7) and accompanying text]. Alternatively, when population levels are static, the stochastic model predicts that individuals with a greater reproduction threshold will dominate even

when the relative increase in reproduction threshold is equal to the corresponding relative increase in reproduction cost.

To test these two predictions, evolutionary simulations were run with the cost of reproduction set to be a proportion of the reproduction threshold ($R_r = \alpha R_1$) – here $\alpha$ is fixed. All individuals started with $R_1 = 7$.[3] Simulations were run under static and fluctuating conditions [scenarios (i) and (ii)] for $10^8$ timesteps. One simulation was run for each set of parameters. Time traces for simulations run with $\eta = 1.2$ are shown in Figure 3.17
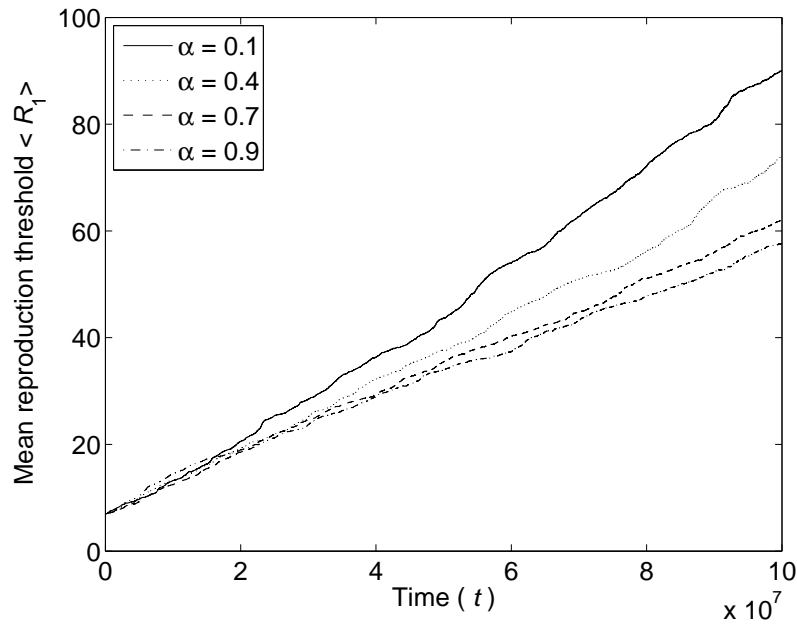


Figure 3.17: The results of evolutionary simulations run where $R_1$ is freely allowed to evolve but $R_r = \alpha R_1$ are plotted for different values of $\alpha$ (shown on the graph). Other parameters were: $R_c = 0.001$ and $R_u = 0.01$.

The figure shows that the value of $R_1$ increases approximately linearly over time. They were still rising at the end of the simulations. One simulation was run for each set of parameters. For all simulation runs, the final value of $R_1$ (at $t = 10^8$) was recorded and the results are plotted in Figure 3.18.

The figure shows how greater values of $R_1$ will dominate over lesser values of $R_1$ for all values of $\alpha$. As with those values shown in Figure 3.17, the mean value of $R_1$ was rising at the end of the simulation in all cases. The differences observed in the figure between the different values of $\eta$ are due to differences in the rate of increase of $R_1$. As the value of $\eta$ increases, so does the rate of increase of $R_1$.

I compare the results in this figure with those produced by the mathematical treatment.

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[3]The value of $R_1$ was set at 7 because, below this value, some populations were seen to show negative growth at the value of $u^+$ – i.e., $u_e > u_0 \eta$
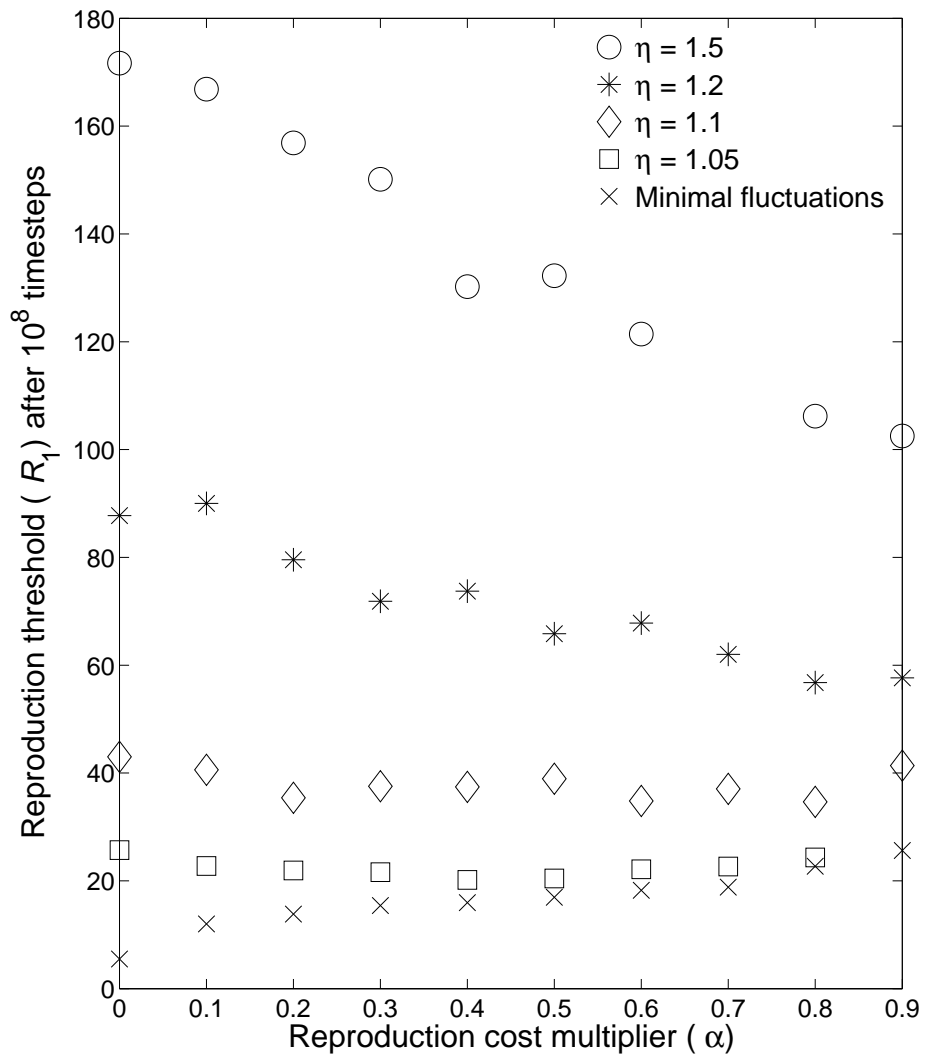
Figure 3.18: Greater population fluctuations mean a greater pressure to increase the reproduction threshold ($R_1$). The results of evolutionary simulations run where $R_1$ is freely allowed to evolve but $R_r = \alpha R_1$ are plotted for different values of $\alpha$ and different fluctuation coefficients. The two missing points ($\eta = 1.5, \alpha = 0.7$ and $\eta = 1.05, \alpha = 0.9$) are due to computer failure. Other parameters were: $R_c = 0.001$ and $R_u = 0.01$.

The stochastic model (see Figure 3.9) predicts that greater values of $\alpha$ will increase the distance between $u_e$ values for two corresponding values of $R_1$. This indicates that at greater values of $\alpha$, $R_1$ will increase at a greater rate. The figure shows that, at lower amplitudes of fluctuation, greater values of $\alpha$ do in fact mean that there is a higher rate of increase of $R_1$. Alternatively, at higher amplitudes of fluctuation, greater values of $\alpha$ mean a lower rate of increase of $R_1$.

### 3.3.2.2 Varying the cost of maintenance

The two mathematical models also make predictions about the impact of varying the cost of maintenance. The deterministic model predicts that a lineage that has an increased cost of maintenance when it increases its value of $R_1$ needs a corresponding increase in the amplitude of resource fluctuations to be viable. To explore this, simulation models were performed as in the previous section, however this time the cost of reproduction was fixed and the cost of maintenance was variable.

Two models for the cost of maintenance were considered. First the cost of maintenance for each individual was dependent on the reproduction threshold $[R_c(i) = R_c + \beta R_1(i)]$. [4] The second model considers the case where the cost of maintenance is dependent on the number of resources an individual has: $R_c(i,x) = R_c + \beta x(i)$.

Simulations were run for both models under static and fluctuating conditions [scenarios (i) and (ii)] for $10^8$ timesteps. One simulation was run for each set of parameters. Considering the first model, where $R_c$ is static throughout the lives of individuals, time traces are plotted for simulations run with $\eta = 1.5$ in Figure 3.19.

The figure shows that there is a a stable equilibrium position for $R_1$. This is dependent on the value of $\beta$: for greater values of $\beta$ the value of $R_1$ is lower. In the cases shown, trace of $R_1$ takes approximately $2 \times 10^7$ timesteps to reach its equilibrium value. Simulations were also run for $\eta = 1.05$ and with no fluctuations [scenario (i)] and the mean value of $R_1$ was calculated for all individuals after $4 \times 10^7$ timesteps. One simulation was run for each set of parameters. The results are plotted in Figure 3.20.

The figure shows that, as predicted by the stochastic model, the equilibrium value of $R_1$ does indeed decrease as the value of $\beta$ is increased. The deterministic model also indicates that large fluctuations in resource availability are necessary for invasion of lineages with increased values of $R_1$ and $R_c$.

Simulations were also run of the second model for $R_c(i)$; again under static and fluctuating conditions. The time traces were similar to those show in Figure 3.19, with the

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[4] When the simpler case of $R_c(i) = \beta R_1(i)$ was considered, this led to very low values of $R_1$ which meant that population extinction events could easily occur without very large populations.
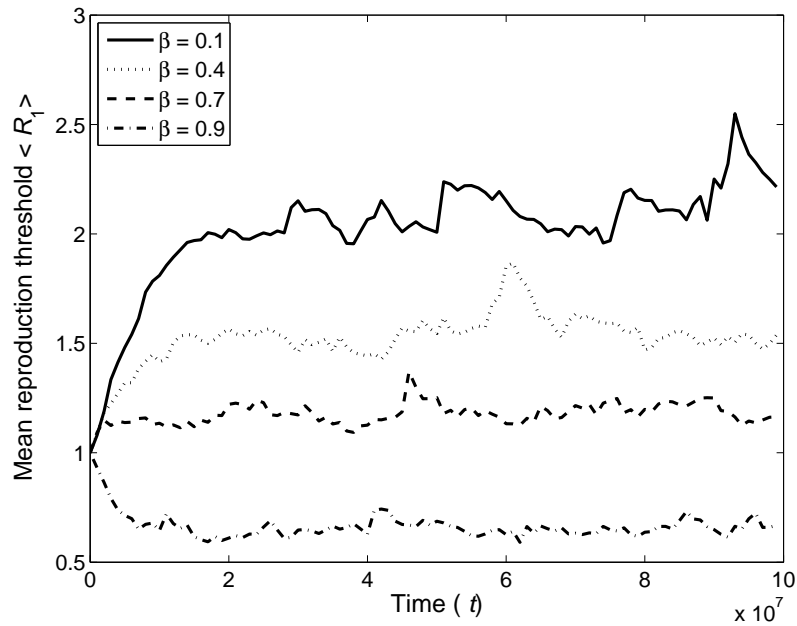
Figure 3.19: The results of evolutionary simulations run where $R_1$ is freely allowed to evolve but $R_c(i) = R_c + \beta R_1$ are plotted for different values of $\beta$ (shown on the graph). Other parameters were: $R_c = 0.001$ and $R_u = 0.01$.

value of $R_1$ reaching an equilibrium value after approximately $4 \times 10^7$ timesteps. Again, the mean value of $R_1$ was calculated for all individuals after $4 \times 10^7$ timesteps. One simulation was run for each set of parameters. The results are plotted in Figure 3.21

The figure shows how the increased amplitude of fluctuation means an increased level of the reproduction threshold $R_1$. In all cases, as $\beta$ is increased, so the level of $R_1$ decreases. This is consistent with the results shown in Figure 3.20 – where the value of $R_1$ also converges on a stable value.

## 3.4  Discussion

This chapter describes the application of a physiological approach to modelling growth rates of individuals with different reproduction strategies under density dependence. The results show the significance of different physiological parameters – the resources accumulated at reproduction, the up-front cost of reproduction and the cost of maintenance – to delayed reproduction. These results outline a different mechanism for delayed reproduction than that presented by Tuljapurkar (1990a) which considers dormant immature individuals. I also show that delayed reproduction can occur under density dependent conditions, even when there are minimal extrinsic fluctuations.

A simple picture of the model presented is that individuals that wait and accumulate
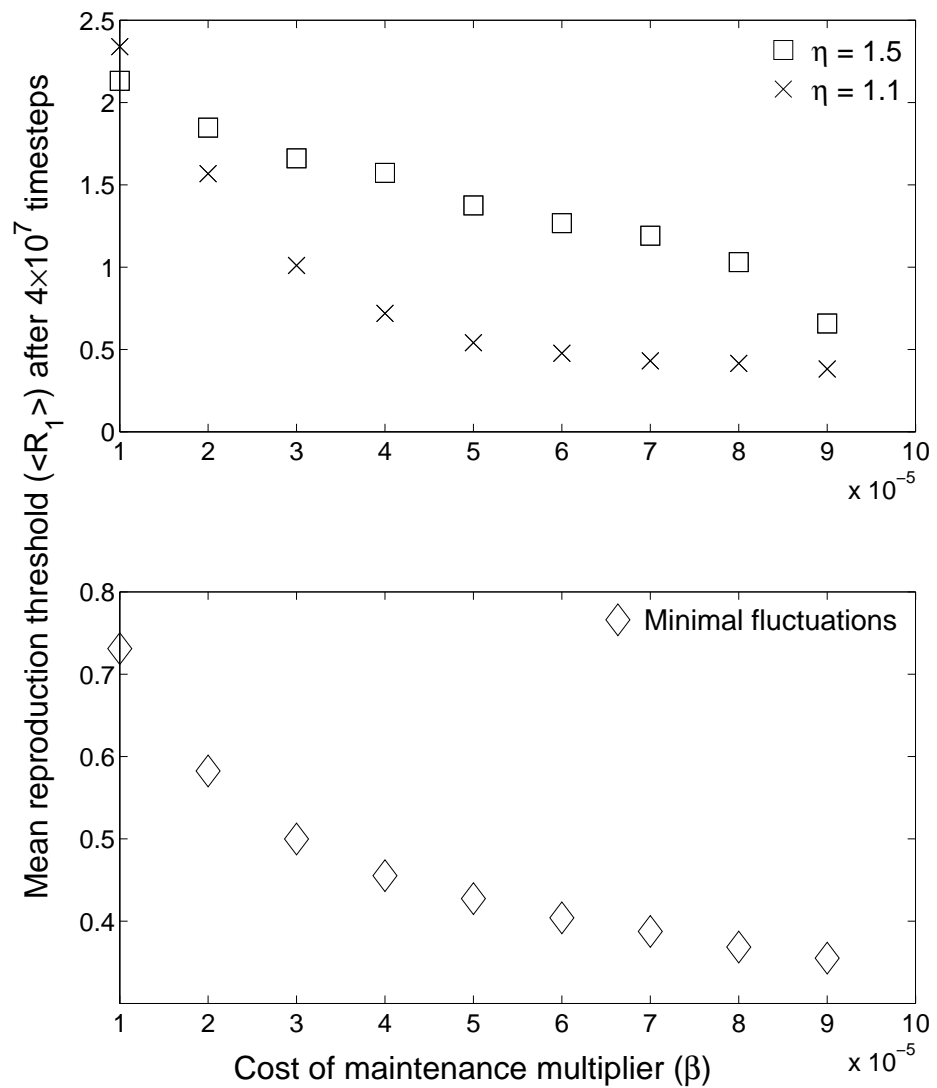
Figure 3.20: The amplitude of resource fluctuations effects the equilibrium value of $R_1$ when $R_c(i) = R_c + \beta R_1$. Other parameters were: $R_c = 0.001$ and $R_u = 0.01$.
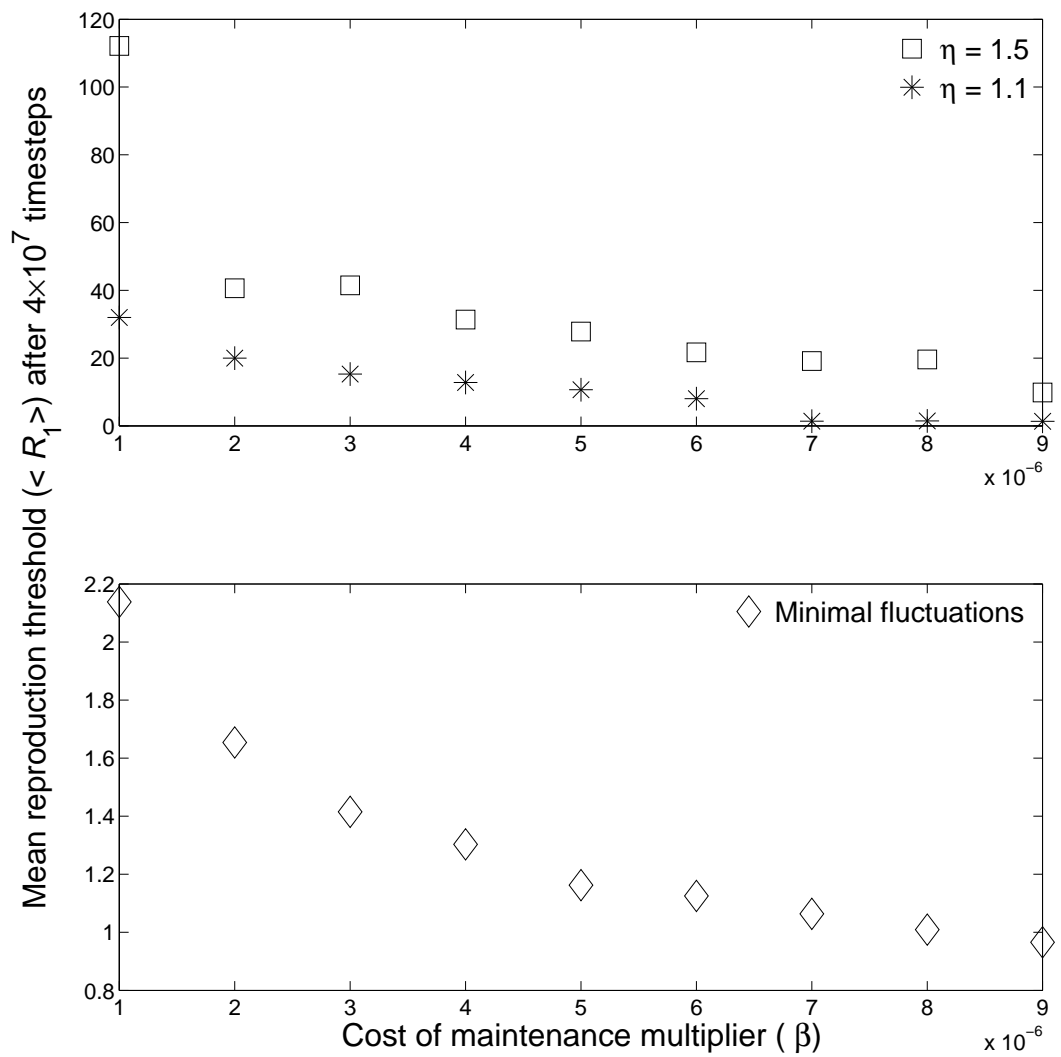
Figure 3.21: The amplitude of resource fluctuations effects the equilibrium value of $R_1$ when $R_c(i,x) = R_c + \beta x$. Other parameters were: $R_c = 0.001$ and $R_u = 0.01$.

more resources, before reproducing, can have an advantage when compared with individuals that reproduce more quickly with less accumulated resources. Naively, the basic reason behind this is that even though the fast reproducing subpopulation is dominant during a growth phase, the slow reproducing subpopulation is less vulnerable during the corresponding decline phase – see Figure 3.1. That said, accumulating more resources does not come for free and slower reproduction will be at a cost. The models predict that any advantage of slower reproduction can potentially be traded off against extra costs of reproduction and extra costs of maintenance which are likely to be incurred.

The work is also relevant to the challenge set down by Heino and Kaitala (1999) for modelling resource allocation between growth and reproduction under environmental feedback. The evolutionary models presented here consider mutations that change the number of resources at maturity – and thus the number of resources allocated to growth. The number of resources allocated to offspring is set at half of this for this model. In these terms, I then consider the optimal allocation of resources to growth under varying environmental conditions. The results concerning the trade-offs between the cost of reproduction and the costs of maintenance apply. The model may be extended to considering conditions where allocation of resources to offspring is variable (see Chapter 6 for an example of such an extension).

The discussion continues with an overview of the model and its results. The results, and the theorem, are then compared with the previous theories on delayed reproduction presented in Section 3.1.1 which use the geometric mean to predict the long term growth rate. The simplicity of the model allows me to consider the significance of the results in light of the experimental results discussed in Section 3.1.2. I then discuss the importance of the results in light of the literature on *r*- and *K*- selection and group selection. Finally I look at possible future directions.

### 3.4.1 Model overview

The two modelling approaches presented in this chapter (mathematical modelling and computer simulation modelling) contribute in different ways toward the question presented in Section 3.1.3. The mathematical model gives clear insights into the questions posed. Then, the approximations of the mathematical models are confirmed in the simulation models. However, since assumptions of the mathematical models are relaxed in the simulation models, new results are presented. These in turn raise questions about the mathematical approach and the insights found through that method. This section therefore comprises of a technical discussion of the merits of the results generated by the models,

and their relationship to each other and the topics presented in Section 3.1.3.

The insights of the mathematical model are mainly derived through analysis of the two growth equations: Equations (3.5) and (3.23). Simple analysis of the deterministic growth equation (Section 3.2.3) shows that while the growth rate of a fast reproducing individual is improved, it is also more vulnerable during declining periods. The application of Theorem 2 in Section 3.2.3 formalises that analysis and generates a simple rule which shows how the ratio between an individual's reproduction threshold ($R_1$) and its cost of reproduction ($R_r$) must decrease for there to be an advantage in increasing the reproduction threshold.

Concerning the importance of fluctuating resources, the mathematical models also give two insights. First, a simple insight can be seen by looking at the effects of an increase in the cost of maintenance (see Figure 3.3). At lower amplitudes of resource fluctuation the faster reproduction model is clearly dominant; but as the amplitude of fluctuations increases, the ratio between the growth to decline rates of the slower population becomes greater than that of the faster population.

Considering the stochastic model, this considers a case where individuals receive resources intermittently in discrete amounts. A comparison of the two models shows that the deterministic model has a good approximation of the growth rate of the stochastic model. This is a little surprising given the comparisons presented in Figures 3.13 and 3.14 where they are also compared with simulations. In fact, both the stochastic and the deterministic models give accurate approximations of simulations of the stochastic model under similar parameter ranges for $R_c$, $m$, $R_1$ and $R_r$. The deterministic model can be a good approximation of the stochastic model, however, when growth is small, there are some important differences (see Figure 3.6). These differences are important when the population is near equilibrium.

The stochastic model considers the case where the population is at equilibrium in more detail. The analysis broadly agrees with that of the deterministic model, however in this model the ratio between the reproduction threshold ($R_1$) and the cost of reproduction ($R_r$) does not need to decrease for there to be an advantage in increasing the reproduction threshold when the ration of $R_1$ to $R_c$ is considered. Analysis, shown in Figure 3.11, shows how $R_1$ will move to an optimum value which is dependent on its ratio to the maintenance cost.

The evolutionary simulation models produce similar results to the predictions of the stochastic and deterministic models. When considering the cost of reproduction, the simulations agree with the stochastic model – the value of $R_1$ increases even when its ratio to $R_r$ stays the same (see Figure 3.18). One interesting point is that at higher amplitudes of

resource fluctuation, greater values of $\alpha$ mean that the reproduction threshold increases more slowly in the simulations. Conversely, at lower amplitudes of resource fluctuation, greater values of $\alpha$ mean that the reproduction threshold increases more quickly.

The evolutionary simulation models also look at a scenario not considered in the mathematical treatment. This is where the cost of maintenance depends on the number of resources an individual has. The simulations (see Figure 3.21) show that there is an optimal value of the reproduction threshold. This value increases when the amplitude of resource fluctuations increases.

The simulation modelling approach relaxes some of the assumptions made in the mathematical approach. The main assumption relaxed is that, in the simulation models, individuals can experience changes in resource availability during their lifetimes. The mathematical models assume that all individuals start their lives at resource level $x = (R_1 - R_r)/2$. In the simulations, individuals that survive declining phases will start their growth phase from low resource levels.

The other assumption (taken for Equation 3.3) is that, given a number of resources $x$ at the start, the mean lifetime of individuals that grow to reproduce is the same as those that die. This assumption is used to generate growth rates for both the stochastic and deterministic models. The results of the deterministic model are qualitatively similar to those produced by the simulations. The main difference is that delayed reproduction is not predicted when the $R_r(j) = \alpha R_1(j)$, however the simulations do show delayed reproduction in all the cases shown by the deterministic model. This indicates that the deterministic model is still valid as an approximation and is therefore instructive as an explanation for the more general stochastic and computer simulation modelling.

### 3.4.2 Comparison with geometric mean predictions

The analysis of the deterministic model is done using Theorem 2 from Appendix A. This theorem states that the ratio of growth to death rates can be used to predict which lineage will dominate. This contrasts with the geometric mean theory which multiplies the rates together.

What is important about Theorem 2 is that it models a density dependent population. When there is a mixed population of two competing phenotypes under density dependence but in fluctuating environments, one will grow relative to the other. To predict which will dominate it is necessary to calculate the growth rates. To do this however, it is necessary to know the times spent under each growth rate ($T_A$ and $T_B$). Instead, by using Theorem 2, these may be ignored as it shows the relative growth rates are important. However this is

only shown to be valid when the growth rates are linear functions of $u$ (see Equation A.1).

### 3.4.3 Comparison with experimental results

The important difference between the results of the models presented here and the experimental results discussed in Section 3.1.2 is the mortality of the individuals. The bacteria analysed by Vasi et al. (1994) and Velicer and Lenski (1999) were not selected under conditions in which they would demonstrate significant population decline. Considering Figure 3.1, this negligible mortality rate would imply that the bacteria existed in the regime to the right of the figure ($u \geq u_0$), i.e., they are either in a state of growth or at least a state of equilibrium. The prediction of my model is that a faster growth rate will always dominate under such a regime – in line with the experimental results.

Focusing on the work by Velicer and Lenski (1999), the bacteria in the two experiments were selected under either slow or fast growth conditions. They were tested against ancestors to ascertain whether adaptation to one condition hampered performance in the other condition. According to the model presented here, all the bacteria were selected under conditions which would promote faster growth. It is therefore unsurprising that individuals adapted to one condition were not at a disadvantage in the other condition – in concordance with the results presented by Velicer and Lenski (1999).

In the other set of experiments, bacteria were exposed to fluctuations between fast growth conditions and conditions with no new resources (zero medium concentration) (Vasi et al. 1994). As the population was effectively only stationary during the periods of zero medium concentration, this experiment selected for individuals with a faster growth rate. One interesting point to consider is the fact that the ancestral bacteria were still growing during the periods of zero medium concentration. After selection, the numbers of bacteria decreased in size. As simple analysis of Equation (3.5) shows, decreasing the threshold of reproduction $R_1$ will lead to an increased growth rate (during growth conditions, $u > u_0$) but this is also accompanied by a corresponding increase in the decline rate (during declining conditions $u < u_0$). This would correspond to the results presented by Vasi et al. (1994). The indication from this is that the regime the ancestral bacteria are adapted to was harsher than the regime in the experiment.

Unfortunately, it is not possible to compare experimental results from either work (Vasi et al. 1994, Velicer and Lenski 1999) with the prediction of Theorem 2 from Appendix A. The theorem requires that the population number must start at some value and eventually return at some point to the same value. In both of the experiments, the bacterial populations essentially grew exponentially during the experiments, never returning to a

previous equilibrium point. Any design of an experiment to explicitly test the model presented here should take that into account. This aspect is also important when the model is compared with traditional theory of $r$- and $K$- selection in the next subsection.

### 3.4.4   $r$- and $K$- selection

On the face of it, the model presented here has much in common with the dichotomy presented for $r/K$ selection. There is a comparison between fast and slow growing populations in both approaches. However there are two reasons why the approach here is different from $r/K$ selection: I discuss these in the following paragraphs.

First, I consider the issue of density dependent growth. In $r/K$ selection theory, fast-growing $r$-selected organisms experience density-independent growth while slow-growing $K$- selected organisms experience density-dependent growth. The model presented here acknowledges that density-independent growth will yield fast-growing organisms. However, the simulations and analysis also predict that environmental variability is important – low variability leads to fast growth and high variability leads to slow growth (see Figures 3.18, 3.20 and 3.21). The model predicts that populations must experience periods of decline before slow-growth becomes viable. This is in contrast to $r/K$ selection theory which suggests that variable environments, especially with catastrophic mortality, can lead to fast growth (Pianka 1970).

Second, the model presented here assumes that offspring are of fixed size. Each parent, after paying a cost of reproduction, shares it resources equally with its offspring. No consideration is given here as to how much or how little a parent should contribute to each offspring. This is an important consideration for $r/K$ selection theory with $r$-selected organisms having many small offspring against $K$-selected organisms having few numbers of large offspring. Further analysis would be needed to see if the amount a parent contributes to its offspring is important in the model presented here.

### 3.4.5   Group selection

The primary purpose of the toy-model presented in this chapter is to consider why an individual might choose a reproductive strategy of lower fecundity. This issue was an important one in the group selection literature discussed in Section 3.1. It was argued by Williams that individuals would not reduce their fitness for the benefit of the group. This, however, does not consider the case where the existence of the group is the reason why an individual may reproduce more slowly.

When a population grows it will start to compete within itself for resources. Such competition within a population will mean that it will eventually stop growing and may either equilibrate or start to decline. In the case of a population at equilibrium, the deterministic model and simulations have shown that the best strategy (dependent on the costs of reproduction and maintenance) can be to reproduce more slowly. When populations actually start to fluctuate around an equilibrium, the simulations show that these effects can be more pronounced.

So, in the absence of any other mechanisms for control of population size, population density will eventually become so great that resources will not match demand. Therefore, in this case, the existence of the group explains why individuals reproduce more slowly. Of course, it is possible that an individual may develop some way of predicting the future and adjust its reproductive strategy accordingly. In the case of microbial organisms, this seems unlikely. It may be possible that individuals may be able to sense population density through monitoring pheromone levels – this would be an interesting question to follow up.

In fact the idea that population pressure is important is very close to Williams' analysis of clutches of birds (Williams 1966a). Here a bird that reproduces too quickly generates too many fledglings which put pressure on its limited resource providing capabilities. There is a key difference here, Williams presents this adaptation as being the optimal way an individual can maximise its successful offspring, regardless of the future environment the offspring find themselves in. The approach presented in this chapter compares lineages against one another, arguing that since an individual's lineage must eventually compete against itself for resources, the slow reproducing lineage may be optimal under the right conditions.

# Chapter 4

# Modelling collective reproduction

## 4.1 Introduction

One of the main threads in Chapter 1 is concerned with the rise in complexity of the individual. One of the motivations of this chapter is therefore the search for processes or mechanisms that can increase the complexity of an individual. The hierarchical nature (see Chapter 1) of biological organisms is a strong inspiration for the search for models of increasing complexity. The reasoning presented by Maynard Smith and Szathmáry is that this hierarchical system is a result of multiple transitions from units that reproduce on their own to sub-units that only reproduce as part of a super-unit (Szathmáry and Maynard Smith 1995). As outlined in Chapter 1, I am searching for an explanation of why an individual might contribute to a higher reproductive process in this thesis. Notwithstanding the value to biology of such an explanatory model, such a model is likely to have value within the ALife field through providing insights into artificial and open-ended evolution (Bedau, Snyder and Packard. 1998, Bedau et al. 2000).

The results from Chapter 3 have shown that individuals may achieve a long term gain from reproducing more slowly. Here I consider whether there is any long term gain from sharing reproductive effort – contributing to a higher reproductive process (see Section 1.2). Here I look at collective reproduction where an individual shares resources, and genes, with another or others to generate a new offspring. Each parent has an equal stake in the offspring's genes.

In order to understand why an individual may share its resources with others, the standard approach is to consider social evolution (see Section 1.3) – especially the evolution of altruism (Section 1.3.1). On the face of it, altruism looks like it may be a good explanation as to why an individual may invest in a higher reproductive process. It may explain why an individual would take a reduction in their own reproductive success so

that the reproductive success of the group may increase. It has long been rejected that an individual may reduce its fitness (lifetime reproductive success) to benefit its local group (Williams 1966a). However, it has been shown how an individual may take a reduction in personal fitness to benefit related organisms (Hamilton 1964, Frank 1998) through *kin selection*.

The kin selection perspective has shown some value in explaining the maintenance of eusociality (social insects), however it is not clear that it (or altruism in general) explains the origins of this transition (Maynard Smith and Szathmáry 1995, Wilson and Hölldobler 2005). Indeed one problem with kin selection is that its benefits can be negated by increased local competition for food (West et al. 2002). One question, considered here, is therefore whether altruism actually is crucial for explaining the origins of collective reproduction. It may be possible to find explanations that are mutualistic: i.e., the outcome for both the individual and the group is beneficial (see Table 1.2).

A simple biological example of collective reproduction is sex. On the face of it, using sex as an example of a two parent collective reproductive effort, it looks unlikely that a mutualistic explanation may be found: there is a cost of sex (Maynard Smith 1976, Bell 1982). Put simply, the cost of sex means that individuals which reproduce sexually will grow at a slower rate than those that reproduce individually. Rather than directly considering Maynard Smith's model of the cost of sex, I illustrate the problem by reformulating it within the abstract terms used in this chapter. Here, I make a simple comparison of the growth rates of an individual strategy and a collective reproduction strategy (with two parents) based on the way resources are allocated to offspring, as shown in Figure 4.1.

Since individuals with the collective strategy share contributions to offspring, they contribute less than those with the individual strategy. This means that the average level of resources per individual in the collectively reproducing population will be higher than those in the individually reproducing population. For a fixed input of resources to the system, this means that the collectively reproducing population will grow more slowly than the individually reproducing population. If we look at cases where there are increasingly larger numbers of parents, then the same reasoning can be used to show that the growth rate will be increasingly slower (e.g., when there are 3 parents, this population will have an average resource level of $3R_1/4$ after reproduction).

Given this cost of collective reproduction, it seems unlikely that there is any advantage to collective reproduction. However, this analysis only looks at the growth phase. Any population that grows will eventually exhaust the resources in its environment and the population will either decline or reach a static level. Because collectively reproducing individuals have, on average, greater resources after reproduction they are less vulnerable
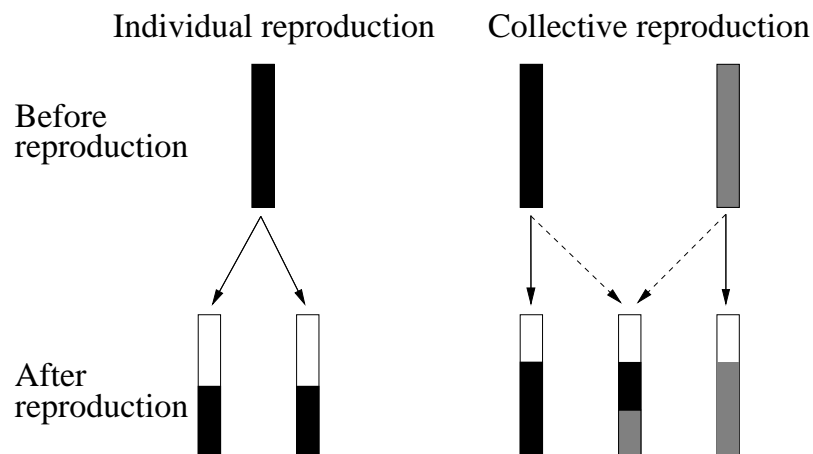
Figure 4.1: Individual reproducers will grow more quickly than collective reproducers. Individuals are represented as resource containers of size $R_1$, with the level of resources represented by how full the containers are. They are shown just before, and immediately after reproduction. Each new individual from the sexually reproducing population will have an average resource level of $2R_1/3$ so, for the same resource input, this population will grow more slowly than the individually reproduction population which starts with an average resource level of $R_1/2$.

during times of population decline.

## 4.1.1   Scope of the model

To approach modelling collective reproduction, I take an abstract perspective in order to produce a toy model. This approach, based on the common factors of the subunits identified above, simplifies the world to resources, individuals and individual resource allocation strategies. Individuals accumulate resources, expend resources, make copies of themselves and share resources with those copies. Given that individuals live within an environment with factors outside of their control, I assume that they have little control over the way they accumulate or expend resources. The space of strategies that they may adopt is therefore concerned with their reproduction strategy.

The model simply approaches the question as to whether it is a better strategy, for the individual, to reproduce collectively or to reproduce clonally. By modelling populations of individuals under density dependence, it questions whether the strategy of sharing resources with other reproducing individuals may dominate the strategy of producing an offspring alone. In context of the introduction, this would provide an explanation for collective reproduction that is mutualistic, not altruistic. In the next section I present the model and its results.

## 4.2 The model

Two modelling approaches are taken in this section. After presenting details common to both approaches, I outline a mathematical model and its predictions. The predictions of the mathematical model are tested by simulation models in Section 4.2.2.

Here each individual $i$ is modelled as a resource level $x(i,t)$ by the equation,

$$\frac{dx}{dt} = U(i,t)R_{\mathrm{u}} - R_{\mathrm{c}} \, , \tag{4.1}$$

where $R_{\mathrm{u}}$ and $R_{\mathrm{c}}$ $[0 < R_{\mathrm{c}} < R_{\mathrm{u}}]$ are positive constants ($R_{\mathrm{u}}$ is the maximum resources available for uptake and $R_{\mathrm{c}}$ is the cost of growth/maintenance . *Resource uptake* (the level of resources received from the environment) is variable and modelled by $u(t) \in [0, 1]$. The variable $u$ here is used as a surrogate for competition: population fluctuations will lead to increasing and declining phases, modelled by changes in the behaviour of $u$. Equation 4.1 can thus be rewritten:

$$\frac{dx}{dt} = uR_{\mathrm{u}} - R_{\mathrm{c}} \, , \tag{4.2}$$

If an individual's resource level decreases below the lower threshold $R_0$ (fixed for all individuals) it will die. Without losing generality, $R_0$ is set to 0 as an *ad hoc* simplification. If an individual's resource level increases above the reproduction threshold ($R_1$) it will reproduce. All individuals pay a cost of reproduction $R_{\mathrm{r}}(n)$ which is dependent on the number of parents $n$.

### 4.2.1 Mathematical treatment

The mathematical treatment assumes that $u$ is static over the lifetime of individuals. The value of $u$ where $dx/dt = 0$ is defined as $u_0$: $u_0 = R_{\mathrm{c}}/R_{\mathrm{u}}$. During reproduction all parents pay the cost of reproduction $R_{\mathrm{r}}(n)$. After this the remaining resources are shared equally between the $n$ parents and the offspring. All individuals therefore start their lives, just after reproduction, with $x = n[R_1 - R_{\mathrm{r}}(n)]/(n+1)$. Two cases for $u$ can now be considered: $u > u_0$ and $u < u_0$. In the first case the individual resource level will increase until it reaches the upper threshold $R_1$, taking an expected time $W$ where,

$$
\begin{aligned}
W_{u>u_0} &= \left[ R_1 - \frac{n\left(R_1 - R_{\mathrm{r}}(n)\right)}{n+1} \right] \frac{dt}{dx} \\
&= \frac{\left[R_1 + nR_{\mathrm{r}}(n)\right]}{(n+1)} \frac{dt}{dx} \, .
\end{aligned}
\tag{4.3}
$$

In the second case individual resources will decrease until it reaches the lower threshold at resource level $R_0$. The expected time $W$ is,

$$W_{u<u_0} = \frac{n\left[R_1 - R_r(n)\right]}{(n+1)} \frac{dt}{dx} .$$ (4.4)

In both cases, as $u \to u_0, W \to \infty$.

The expected population growth rate of a homogeneous population of individuals can be estimated for the two regimes ($u > u_0$ and $u < u_0$). The expected population growth rate per individual $G$ is equal to the reciprocal of the time taken for resources to grow for reproduction during population growth ($1/W_{u>u_0}$) and reciprocal of the time taken for resources to decline for death during population decline ($1/W_{u<u_0}$), or:

$$G = \begin{cases} \dfrac{(n+1)}{n\left[R_1 + nR_r(n)\right]} \dfrac{dx}{dt} & ,u > u_0 \\[3mm] 0 & ,u = u_0 \\[3mm] \dfrac{(n+1)}{n\left[R_1 - R_r(n)\right]} \dfrac{dx}{dt} & ,u < u_0 . \end{cases}$$

substituting Equation (4.2) gives:

$$G = \begin{cases} \dfrac{(n+1)(uR_u - R_c)}{n\left(R_1 + nR_r(j)\right)} & ,u > u_0 \\[3mm] 0 & ,u = u_0 \\[3mm] \dfrac{(n+1)(uR_u - R_c)}{n\left(R_1 - R_r(j)\right)} & ,u < u_0 . \end{cases}$$ (4.5)

The growth rate ($G$) is plotted against different values of $u$ for one [$n = 1$, $R_r(1) = 0.1$] and two [$n = 2$, $R_r(2) = 0.05$] parents in Figure 4.2. In this case, the two parents share the cost of reproduction born by the single parent – i.e., the cost per offspring is the same.

The figure shows that the sexually reproducing population does in fact grow more slowly during population growth ($u > u_0$). This is in line with the reasoning presented in Section 4.1 and Figure 4.1. What is also evident from the figure is that the decline rate of the sexually reproducing population is lower in magnitude than that of the individually reproducing population. Furthermore, the two graphs diverge more as $u$ gets further from $u_0$ indicating that fluctuations may be significant; however the ratio between the two plots
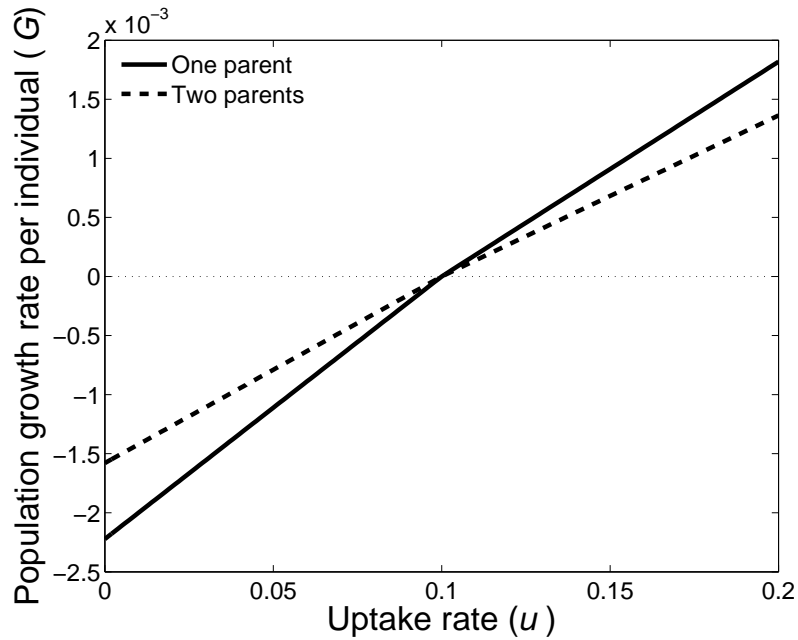
Figure 4.2: Growth rates of individually and sexually reproducing populations. The parameters used were: $R_1 = 1.0$, $R_r(1) = 0.1$, $R_r(2) = 0.05$, $R_c = 0.001$, and $R_u = 0.01$.

stays constant. Indeed, in this example, the growth rate of the sexually reproducing population is 0.75 that of the individually reproducing population. However, the decline rate of the sexually reproducing population is only 0.71 that of the individually reproducing population. Interestingly, the sexually reproducing population has a greater advantage during decline periods than the individual strategy has during growth periods.

This analysis indicates that, similarly to the case in Section 3.2.3, the ratio of the slope in the growth region ($u > u_0$) to the slope in the decline region ($u < u_0$) will determine which lineage will dominate. This is given by (taking $G' = dG/du$):

$$\Psi(n) = \frac{G'_{u>u_0}}{G'_{u<u_0}} = \frac{R_1 - R_r(n)}{R_1 + nR_r(n)} \ . \tag{4.6}$$

Assuming that the growth equation [Equation (4.5)] is a good approximation, Theorem 2 (in Appendix A) shows that, if there are no trends in overall population, then the population with a higher value of $\Psi$ will dominate one with a lower value of $\Psi$. In this model, the behaviour of $\Psi$ depends on the way $R_r(n)$ is determined.

I look at three scenarios for determining $R_r(n)$. These consider the offspring cost which is defined as the total reproduction cost spent on each offspring ($nR_r$). The scenarios are:

(i). The total cost of each offspring is not affected by the number of parents: $R_r(n) =$

$R_{\mathrm{r}}(1)/n$. In this case, Equation (4.6) is increasing (the denominator is constant and the numerator increases).

(ii). The total cost of each offspring increases linearly with each extra parent: $R_{\mathrm{r}}(n) = R_{\mathrm{r}}(1)[1 + c(n - 1)]/n$ ($c$ is a constant).

(iii). In this scenario, $\Psi$ is constant – i.e., there is no advantage or disadvantage to reproducing with more parents. Simple manipulation of Equation (4.6) shows that if $\forall n : \Psi(n) = \Psi(1)$ then,

$$R_{\mathrm{r}}(n) = \frac{2R_{\mathrm{r}}(1)R_1}{R_1 + R_{\mathrm{r}}(1) + n[R_1 - R_{\mathrm{r}}(1)]} \ . \tag{4.7}$$

For any multi-parent strategy to dominate individual reproduction, the cost of reproduction must be below this value. That is, if $R_{\mathrm{r}}(n)$ is above this value, then $\Psi(n) < \Psi(1)$, if $R_{\mathrm{r}}(n)$ is below this value then $\Psi(n) > \Psi(1)$.

The offspring cost is plotted in panel A of Figure 4.3 for different numbers of parents in the three cases. Given the corresponding reproduction costs associated with these offspring costs, panel B shows the value of $\Psi$ calculated by Equation (4.6).

As expected, the plot in Panel B of the figure shows how $\Psi$ increases when the offspring cost is not dependent on the number of parents. The difference in values of $\Psi$ is relatively high when the number of parents is low – the largest increase being the difference between individual and sexual reproduction. When the offspring cost increases linearly with the number of parents, $\Psi$ reaches a maximum at 2 parents (sexual reproduction) and declines thereafter. Considering Panel A of the figure, this indicates that, as the number of parents increases, the total cost of reproduction spent (the cost per offspring) may increase. Again, this increases sharply as the number of parents increase from 1 to about 5 and then levels out. For all graphs, changes in parameters $R_1$ and $R_{\mathrm{r}}$ did not change the shape of the graphs significantly, however, as the ratio $(R_1/R_{\mathrm{r}})$ decreases, the values generated by Equation (4.7) increase (not shown).

## 4.2.2   Simulation models

The predictions of the mathematical treatment in the previous section indicate two things that may be tested with simulation models. Firstly whether the value of $\Psi$ is a good predictor for which strategy is optimal and secondly that fluctuations may also be significant.

Simulations are done with agents modelled as resource levels, based on Equation (4.1). At each timestep an agent pays a growth/maintenance cost $R_{\mathrm{c}}$. When its resources
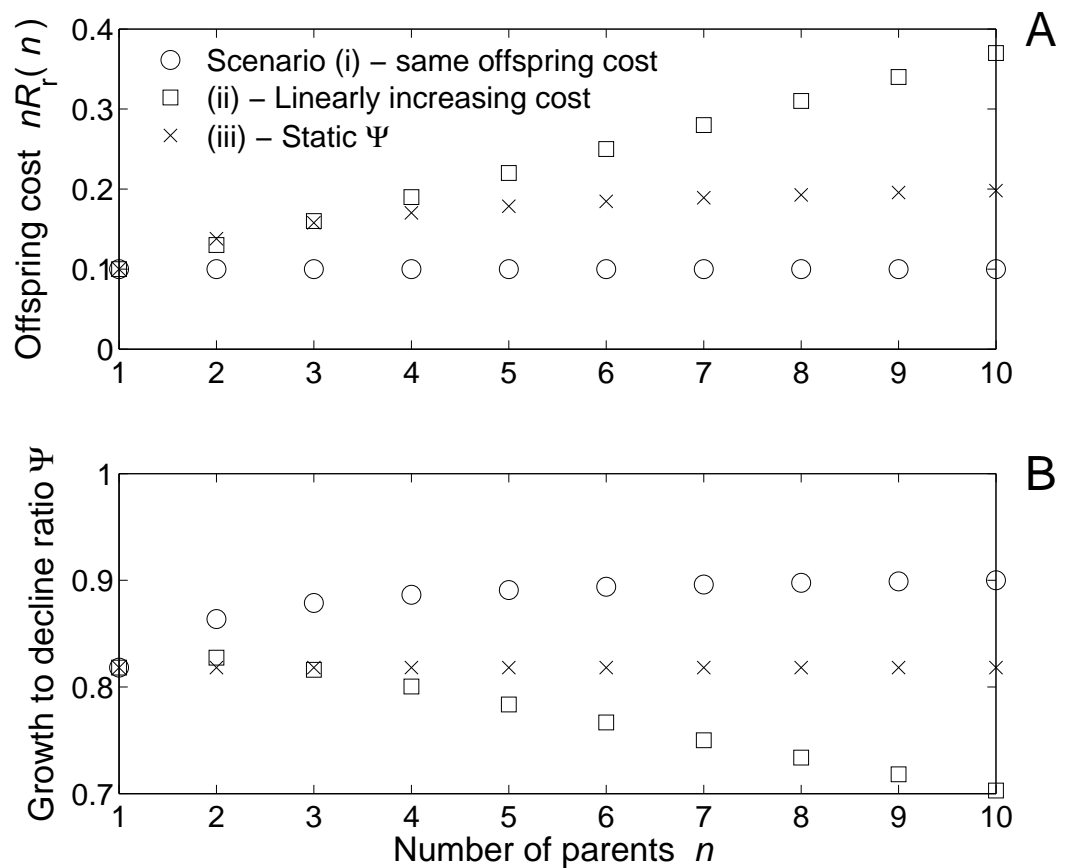
Figure 4.3: The behaviour of the growth/decline ratio changes with different functions for $R_r(n)$. Panel A shows the cost per offspring generated by the three different functions of $R_r(n)$ and panel B shows the corresponding predicted growth to decline ratio. The parameters used were: $R_1 = 1.0$, $R_r(1) = 0.1$, $R_c = 0.001$, $R_u = 0.01$, and $c = 0.3$.

are below zero, an agent will die. Each agent has a reproduction strategy which is defined by the number of parents (from 1 to 10) the agent will reproduce with. Those with the same reproduction strategy will reproduce when enough agents (depending on the number of parents defined by their common strategy) have resource levels that are above $R_1$ (resource levels may go above $R_1$ without penalty). Each parent pays a cost of reproduction $R_r(n)$ and all parents share their energy with the new offspring. Agents were initialised with a random level of resources and a random reproduction strategy. The reproduction strategy was fixed for all agents – there was no mutation in the simulations.

The simulation models are essentially non-spatial, with individuals located on a grid but moving to a random new cell each timestep.[1] Agents consume a resource unit if they encounter any on the square they are on. A number of resource units, each of value $R_u$ are randomly scattered on a spatial grid (of $50 \times 50$ squares) each timestep. The number of units is either static (set to 200) throughout the simulation, or fluctuated between two values (100 and 200) changing every 1,000 timesteps. Pseudo-code for the simulation can be seen in Figure 4.4.

The simulations were run with the three scenarios for determining $R_r(n)$ presented in Section 4.2.1. All scenarios were tested with a static resource input to the system and fluctuating resources. Each simulation was run ten times, with each run initialised with a different random seed. After 1,000,000 timesteps I recorded the number of agents with each reproduction strategy and this was averaged over all the simulations. The results are plotted in Figure 4.5.

The figure shows that, when the offspring cost does not increase with the number of parents, reproduction strategies with higher numbers of parents will dominate. In fact, strategies with less than 7 parents are completely dominated in this scenario with static resource input. When resource input fluctuated similar results were seen (not shown). When the cost per offspring increases linearly, the sexual strategy is dominant over the other strategies (see panel B) – as predicted by the shape of $\Psi$ in Figure 4.3 (panel B, squares). Again, the results were similar with and without fluctuating resource input.

When the cost per offspring increases in line with the upper limit predicted by Equation (4.7) (see Figure 4.3, Panel A, crosses), the viability of collective reproduction depends on fluctuations in resource availability. With no fluctuations, individual reproduction is the most frequent (panel C of Figure 4.5), however a range of strategies are found. When the resources do fluctuate, collective reproduction is dominant (panel D of Figure 4.5).

---

[1] The results presented by Bryden (2005b) showed that agents receive resources with between-resource intervals on a geometric distribution when they move to random grid squares each timestep

```
 0:  Define agent as having resources x and number of co-reproducers n
 1:  Initialise x and n for new members of the population agents
 2:  season = high
 3:  for t = 1 to simulation_time
 4:      if |agents| > upper_threshold, season = low
 5:      if |agents| < lower_threshold, season = high
 6:      if fluctuating_resources and season = low
 7:          Scatter 100 resources on the grid
 8:      else
 9:          Scatter 200 resources on the grid
10:      end if
11:      Move each agent to a random grid-square
12:      for each agent (picked in random order) in agents
13:          x = x − R_c
14:          if there are resources on the grid-square
15:              x = x + R_u
16:              Remove one resource from the grid-square
17:          end if
18:          if x < 0, remove agent from simulation
19:      end for
20:      for i is each number of co-reproducers (1 to 10)
21:          pick agents where n = i and x > R_l
22:              for each group of i agents
23:                  x = x − R_r(i) (pay the cost of reproduction)
24:                  Create a new agent (the offspring)
25:                  Share all resources (x) evenly between all (i+1) agents
26:              end for
27:              # remaining agents do not reproduce this turn
28:          end pick
29:      end for
30: end for
```

Figure 4.4: Pseudo-code for the evolutionary simulations.
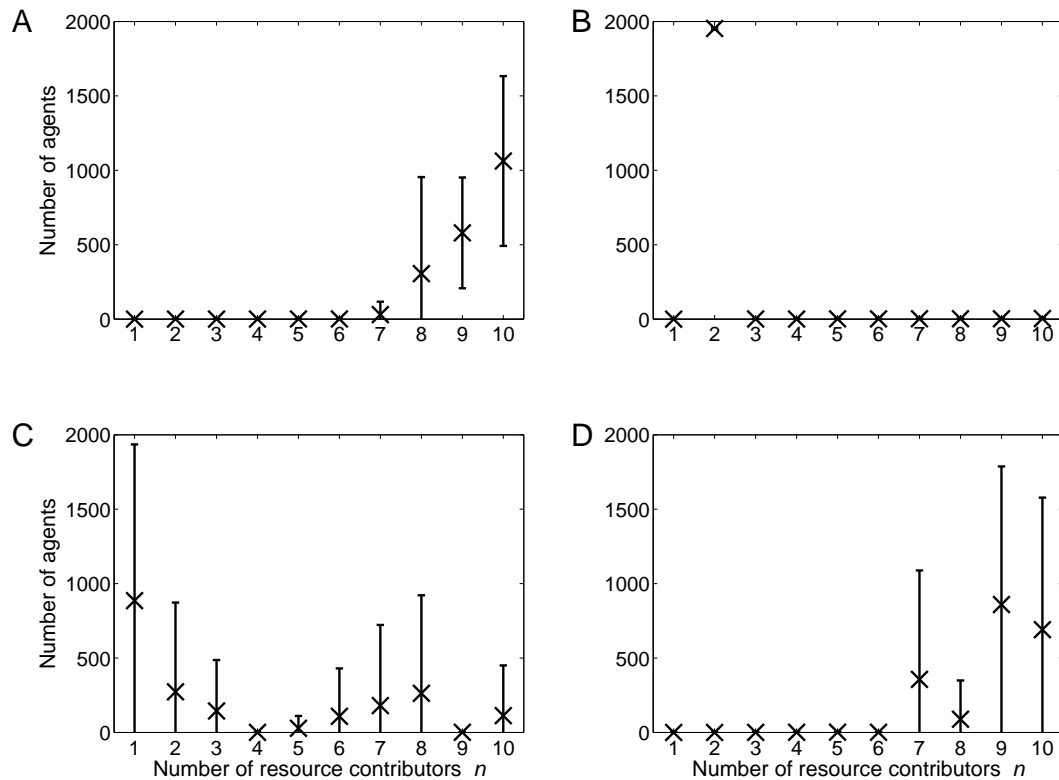
Figure 4.5: Collective reproduction depends on offspring cost and resource fluctuations. Four scenarios were tested: panel A shows scenario (i) with static resource input; panel B shows scenario (ii) with static resource input; panel C shows scenario (iii) with static resource input; and panel D shows scenario (iii) with fluctuating resource input. Each result is plotted with error bars showing the standard deviation over the simulations. Parameters for the simulations were: $R_1 = 1.0$, $R_r(1) = 0.1$, $R_c = 0.001$, and $R_u = 0.01$.

## 4.3   Discussion

The mathematical model and simulations presented in this chapter demonstrate collective reproduction. Individuals that may reproduce on their own, instead reproduce as part of a collective. Collective reproduction here is done by sharing resources contributed to a shared offspring. The modelling work shows that the cost of this process (the cost of reproduction) can affect the viability of agents that reproduce collectively and how fluctuations in environmental resource levels can be significant. The model also presents two different scenarios which predict conditions for when sexual reproduction is optimal and other conditions for when reproduction in larger groups is optimal: this may help to explain why sexual reproduction is dominant in some animals and eusociality is dominant in others.

It is interesting to consider the results of the model with the types of reproduction seen amongst the animal kingdom. When the number of parents increases, there is an additional increase in the amount they may spend per offspring [see Figure 4.3, Panel A, scenario (iii)]. The largest increase is between one parent and two parents. Perhaps when there are more than two contributors, the only cost effective strategy (or perhaps technically possible (Whitfield 2004) that works is to continue to only have two parents – a male and the queen – but have other kin-related workers which have an indirect genetic contribution to the offspring. Since each worker barely increases the cost of reproduction, the larger number of contributors is advantageous [see Figure 4.3, Panel A, scenario (i)] and the colony will grow.

The mathematical predictions presented in Figure 4.3 are concordant with the results in Figure 4.5, both predicting when collective reproduction is viable. This includes subtle effects such as the dominance of individuals that reproduce with one other parent. Since the results are so similar, the simulation models show that $\Psi$ is good predictor for which reproduction strategies will competitively exclude others. This indicates that the growth equation [Equation (4.5)] is a good approximation. The mathematical treatment is therefore instructive (in line with Chapter 2) as to why there is a long term growth benefit to lineages that reproduce in this way: the collectively reproducing individuals have greater resources and are therefore less vulnerable to resource fluctuations.

The work in this chapter gives some insights into how the complexity of the individual may increase. First, it demonstrates how collective reproduction can benefit both partners: when more than one parent contributes resources to the production of an offspring, the combined reproductive expenditure can be significantly larger than with individual parents (see Figure 4.3 panel A, and corroborating simulation results in Figure 4.5). This

extra resource is available for the increased complexity needed for the facilitation of collective reproduction.

Indeed, it is plausible that collective reproduction may happen on many levels in the same class of individual. Some examples of collective reproduction may only be viable when the conditions are right, so different mechanisms for collective reproduction may happen under different conditions. Each may have different optimal numbers of parents. As well as this, collective reproduction may happen at different levels at the same time. With some organisms making direct genetic contributions, others making indirect genetic contributions [through kin-relatedness (Hamilton 1964)] and others perhaps gambling their genetic contribution [see (Bryden 2005a), or Chapter 5, for an example]. There can therefore be many differing mechanisms of collective reproduction taking place within a population at the same time. As such, this is an accretive process and as new viable mechanisms increase complexity, a rich social fabric should emerge.

Secondly, the model, and its insights, implies a potentially fruitful approach to modelling the major evolutionary transitions. Rather than invoking altruism or group selection, the model of collective reproduction presented here shows mutual benefits to reproducing collectively: i.e., that it is in an individual's selfish interest to reproduce collectively. There is no need for the individual to reduce its fitness for the benefit of its kin or its group. Altruism may therefore not be an essential feature in explaining the major transitions.

That said, this does not imply that altruism is not important in collective reproduction. Altruism can happen and will act as an evolutionary force when appropriate. One major assumption of the model is that the resources of all parents are shared out equally between the parents and offspring. Clearly, a parent that does not contribute in this way may be able to disrupt the process by contributing less resources than other parents. It could be argued that a parent that doesn't do this is acting altruistically, however by contributing less resources it will also be harming its own representation in the collective reproductive effort.

Study of such cheating behaviour is outside the scope of this chapter, the case where two individuals are reproducing is discussed in Chapter 6. Further studies considering cases with more than two individuals will also be of interest.

# Chapter 5

# Slime mould and the transition to multicellularity

## 5.1 Introduction

Given the results in the previous chapter show the viability of collective reproduction, I investigate a biological case study in this chapter. When considering reproduction strategy, one of the most striking cases in evolution is that of the transition to multicellularity (Buss 1987, Maynard Smith and Szathmáry 1995). This is a transition between unicellular organisms, that reproduce on their own, to a multicellular organism, in which one germ-line cell reproduces on behalf of the other cells.

This transition is especially interesting to artificial life research where the quest to synthesise hierarchical levels of organisation is a significant open problem (Bedau et al. 2000, Stewart 1997). The transition from unicellular to multicellular organisms is a good example of such hierarchical organisation. A multicellular organism is made up of individual cells. Cells are made up of proteins. Proteins are made up of molecules, and so on... How, and why, individual cells might come together to form a multicellular organism is therefore an interesting question.

To put the transition into a paleontological context, fossil evidence (Maynard Smith and Szathmáry 1995) indicates that multicellular life did not exist for 2,500 million years after the dawn of life. The first examples found were in the Cambrian period (approxi-

mately 540 million years ago). One interesting fact is that all the multicellular phyla are represented in the Cambrian fossil evidence (Maynard Smith and Szathmáry 1995). It is likely that the transition occurred several times (Bonner 1999, Brooke and Holland 2003). However, phylogenetic evidence (Baldauf, Roger, Wenk-Siefert and Doolittle 2000) suggests that multicellular organisms, especially metazoa, share a common ancestor. If it is true that only one mechanism was responsible for the transition, then the fact that it took such a long time to evolve implies that a certain specific set of conditions needed to be present for it to happen.

For now, we may only speculate on what mechanisms or conditions may well have led to the transition. This said, modelling can enable us to develop mechanisms that are at least plausible. Since it is also important to ground such models within current biological knowledge, such models are often based on specific biological systems. These models can also aid our understanding of those systems as well as their evolution. This section therefore introduces background literature on the transition to multicellularity. It then goes on to discuss how a model organism, *Dictyostelium discoideum* (commonly known as slime mould), may shed some light on the transition.

### 5.1.1   The transition to multicellularity

For the purposes of this chapter it is useful to have definitions of both multicellular organisms and the transition to multicellularity. I define a multicellular organism as an aggregation of genetically homogeneous cells. In fact there are more complex processes in most multicellular organisms. They require gene-regulatory mechanisms for differentiating cells (with differentiations being passed from parent cell to offspring), cell adhesion and spatial patterning of cells (Maynard Smith and Szathmáry 1995). But to keep things simple I assume that the most essential requirement is aggregated, genetically homogeneous, cells. The transition is therefore from unaggregated genetically heterogeneous unicellular organisms to aggregated genetically homogeneous multicellular organisms.

This definition raises the question as to why a multicellular organism must have a homogeneous genetic code. A great amount of coordination is required in a multicellular organism for all the different processes and organs to function properly. Differences of foreign, or mutant, cells are therefore most likely to be damaging. Thus, it is best to keep an organism genetically homogeneous.

What is even more damaging is when these harmful traits are passed on to offspring through infection of the germ line. To solve this problem, an organism is generated from one single germ line cell (Buss 1987). Germ line cells are differentiated and isolated from

the soma (or body) cells. This means that any damaging mutant or foreign cells do not have their traits passed on to the next generation.

What is puzzling then, is how differentiation between soma and germ line cells evolved and has remained stable. Any invasion of these germ line cells by other could potentially have harmful consequences as the invaders will pass on their code to all cells in any future offspring. While cell policing (Michod 1999) and early sequestration (Buss 1987) may be valid mechanisms in higher metazoa, it is unlikely that these mechanisms evolved at the same time as germ-line soma differentiation. Any mechanism which describes the evolution of germ-line soma differentiation must describe why the soma cells will cooperate and not invade the germ-line cells. This can be put another way: it must explain how an individual that reproduces individually might instead come to start reproducing as part of a group, with another individual reproducing on its behalf. This question is addressed by the model in this chapter.

I therefore consider evolutionary mechanisms that will explain a transition between unicellular organisms, which compete within their populations and compete with predators and prey, and early multicellular organisms which are clustered together and exhibit germ-line/soma differentiation. In other words, there is a transition from unicellular organisms which are optimised to maximise their own *direct* fitness to cells that must, on the other hand, maximise their *inclusive* fitness at the expense of their direct fitness (i.e., their ability to contribute their fitness to other cells that are highly related must be more important than their own replication chances). [See Frank (1998) for precise definitions of *direct* and *inclusive* fitness.]

Many models of the transition argue that an early stage involved cells clustering together. Wolpert has presented a model where individual cells split to produce a somatic body cell that sticks to its parent and is unable to reproduce (Wolpert 1990). Other work (Pfeiffer and Bonhoeffer 2003) presents some benefits to clustering, arguing that the benefits clustered individuals receive from collective metabolisation may outweigh the costs of increased competition. Another perspective (Michod 1999) argues that the clustered cells were differentiated and received some benefit from this division of labour. A problem with such clustering is that local competition can become a problem, specifically competition over food (Queller 1994, West et al. 2002, Marshall and Rowe 2003b).

While these perspectives may explain why cells might cluster together, no mechanisms are proposed as to how these clusters may become genetically homogeneous. Furthermore, the presence of clustering does not yield an explanation as to why germ-line cells are differentiated from soma cells: or, why an individual might stop reproducing so that another individual may reproduce on its behalf.

A different perspective considers multicellularity through aggregation (Maynard Smith and Szathmáry 1995). Here cells either vegetate and reproduce individually, or aggregate to reproduce collectively. One advantage to this perspective is that, because cells vegetate individually, this reduces competition between cells for food – one of the problems with the early models with clustered organisms. A further advantage of this perspective is that there are model organisms we can study to approach the questions set out above. The model organism chosen for study in more detail in this chapter is (*Dictyostelium*) which is outlined in the next section. If the collective reproduction exhibited by cells that aggregate can identify some kind of differentiation between germ line and soma cells then this may shed some light on the transition to multicellularity.

### 5.1.2 Biological background on *Dictyostelium*

*Dictyostelium* (more commonly known as slime mould) is a model organism for multicellularity through aggregation (Maynard Smith and Szathmáry 1995, Queller 2000). Individual cells mostly vegetate and reproduce asexually on their own. However, under different environmental conditions they also demonstrate collective reproduction behaviour, characterised by individual cells making sacrifices for the benefit of other cells' reproductive chances. This organism therefore demonstrates both the germ-line/soma differentiation (Buss 1982) and clustering that is important for the transition. Biological evidence is now presented concerning *D. discoideum*, one of the more studied species of the genus.

When there is a shortage of food and *D. discoideum* cells begin to starve, they aggregate and one of the two collective reproductive stages commences (Raper 1984). The more well known reproductive stage of *D. discoideum* is shown in Figure 5.1. Here the cells form a slug which collectively migrates. Once the cells find an advantageous location they form a *fruiting body*: cells at the front of the slug (20%) form a stalk and the rest form spore cells at the top of the stalk which are dispersed by the wind. Interestingly, the stalk cells die after the stalk is built. This differentiation between spore and stalk cells is arguably a germ-line/soma distinction (Buss 1982). Since cells that produce stalks do not pass on their genetic code, it is hard to see how this trait is selected for and maintained. Indeed there are examples of slime moulds strains that do not produce stalks (Buss 1982). Computer simulations addressing this question (Armstrong 1984) have indicated that high dispersal of spores can lead to more stability in the stalk producing behaviour.

The second, less well known, collective reproduction stage in *D. discoideum* involves the formation of the *macrocyst* (Raper 1984). Again, when the cells are starving they
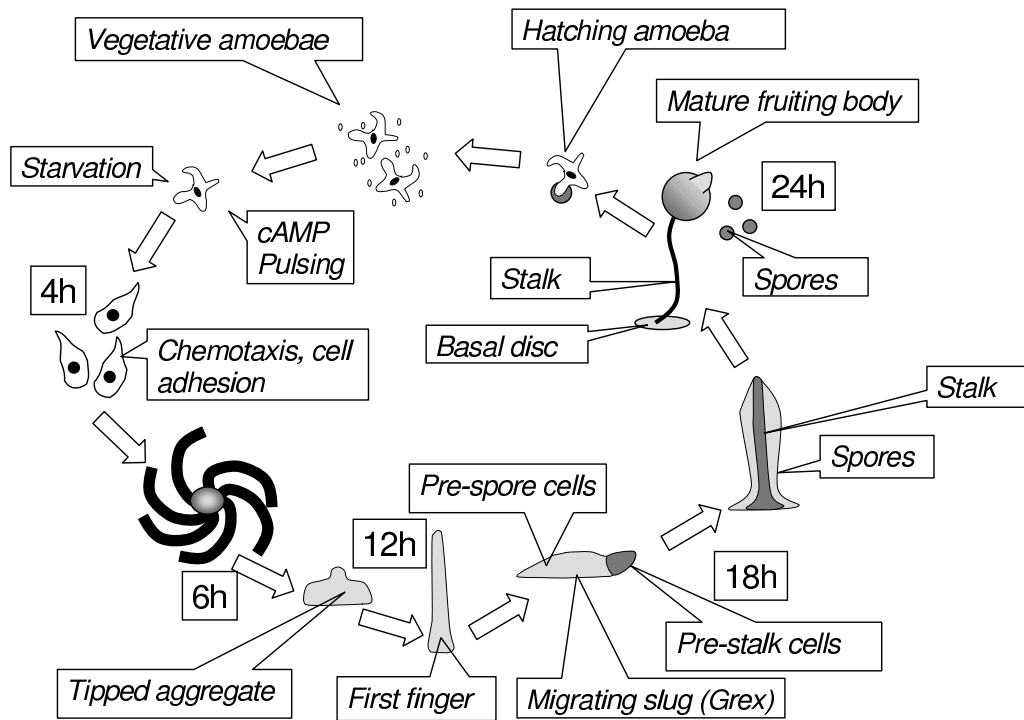
Figure 5.1: The asexual and mitotic life cycles of *Dictyostelium* [data from Raper (1984)].

aggregate. However instead of forming a slug, two cells merge to form a large *Zygote* cell which eats other aggregating cells. The resulting giant cell forms a hard cellulose outer wall and this macrocyst germinates after a few weeks. See Figure 5.2 for a diagram.

The macrocyst stage is thought to be a precursor to the slug/stalk reproductive stage. Kessin (Kessin 2001) argues that evolution generally occurs in incremental stages. He notes that the previous stage to macrocyst development would be the microcyst stage (not observed in *D. discoideum*), where individuals form outer walls on their own. After the evolution of chemotaxis, aggregation could occur giving the right conditions for the evolution of the macrocyst. With added cell adhesion and cell type differentiation into stalks and spores, fruiting body and slug behaviour would then become plausible.

The genetic makeup of the offspring of the macrocyst is an important question. The macrocyst is generally accepted to be the sexual phase of *D. discoideum's* development (Raper 1984), two different *mating types* are generally seen to aggregate. However, experiments do demonstrate that macrocysts can form from only one mating type (Bozzone and Bonner 1982). Typically, the progeny of a macrocyst is observed to be of one genotype (Wallace and Raper 1979). Only one nucleus remains in the zygote (or giant cell) after other ingested nuclei disappear (Okada, Hirota, Moriyama, Saga and Yanagisawa 1986).

The biological evidence therefore points to a picture showing that the macrocyst stage contains a differentiation between a germ line cell and soma cells. With the germ-line cell
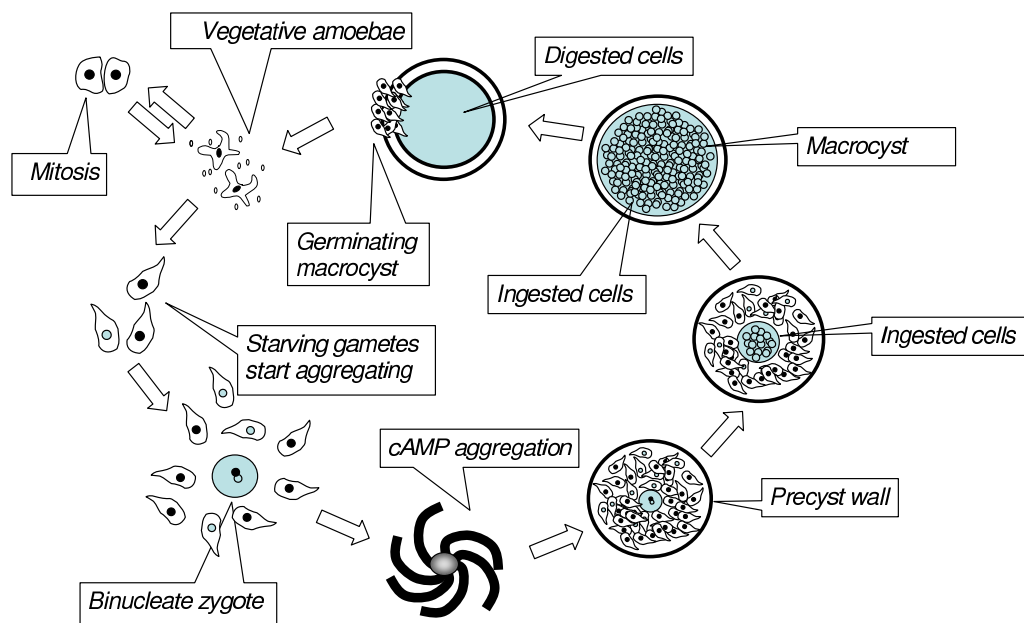
Figure 5.2: The sexual and mitotic life cycles of *Dictyostelium* [data from Raper (1984)].

being the zygotic cell which ingests all the other somatic cells. The cells use chemotaxis to move closer to other aggregating cells. It is unlikely that a cell will have any way of distinguishing between the zygote and any other cell so it will just aggregate towards it. The first cells that produce a zygote can stop moving and generate a powerful chemotactic signal to attract all the other cells. Based on this, the work in this chapter assumes that the central zygotic cell is chosen at random from all those aggregating.

Given that aggregating cells have a random chance of being the zygote, the point at which they aggregate is therefore crucial. Recent evidence implies that the cell may have genetic control over this event. Research (Chae, Inazu, Amagai and Maeda 1998, Hirose, Inazu, Chae and Maeda 2000) identifies genes that can control or delay when or whether a cell will continue to grow or start aggregation. These findings indicate that the cell is capable of turning on or off aggregation to the macrocyst stage which can ultimately lead to cells being eaten by the zygote. This emphasises a need for an explanation as to why an individual might make the 'choice' to aggregate and almost certainly die.

### 5.1.3 Scope of the model

The model in this chapter is an abstract model of the *D. discoideum* macrocyst stage. As with the models in previous chapters, it simplifies the world to resources, individuals and individual strategies. Based on the behaviour of *D. discoideum*, individuals accumulate resources, expend resources, make copies of themselves and may join the macrocyst. The

macrocyst also expends resources and will germinate when the conditions are right.

The model is intended to approach several questions:

(i). To confirm that individuals that normally reproduce on their own are indeed prepared to gamble their own reproductive chances against the 'pot' of reproductive material contained in the macrocyst.

(ii). To confirm the intuition that fluctuations in food availability are important to the viability of the macrocyst.

(iii). To question the role individual mitotic split rates might play in the stability of the macrocyst.

(iv). To make predictions of the effect of different manipulations on the organism.

(v). To speculate on the role the macrocyst might play in the evolution of other altruistic behaviour (such as stalk/spore differentiation) and collective behaviour.

## 5.2   Methods

To investigate the questions outlined in the previous Section, I have built a computer simulation model of the macrocyst stage of *D. discoideum*. Assumptions in the model are based on the biological evidence presented. Notably I have assumed that all the offspring of a macrocyst are of the same genotype. Since sexual fusion does not seem to be necessary, I chose (on parsimonious as well as biological grounds) to model the macrocyst with no sexual recombination. Individual vegetative behaviour was modelled with individuals having a genetically encoded resource threshold above which they mitotically reproduce.

*D. discoideum* cells are modelled as individuals in a non-spatial environment. Each individual has an resource level $x$. At each time step, a number of individuals ($N$) are selected at random, each receives ($R_u = 0.5$) units of resource (representing food) with probability $u$. One cycle in the model contains two seasons. The amount and probability of receiving a resource unit ($N$ and $u$) changes value according to whether the season is 'high' ($N = 100, u = 0.6$) or 'low' ($N = 20, u = 0.3$). Each season lasts 200 timesteps. All individuals pay a resource cost ($R_c = 0.2$) per timestep irrespective of season. If an individual's resource level falls below zero ($x < 0$), it will die.

Each individual cell is modelled with two genes.[1] The genes model resource thresholds which determine the behaviour of the cell. Cells will join the macrocyst when

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[1]Genes are represented as floating-point numbers in the simulation, point mutations occur at each timestep over a Gaussian distribution with standard deviation of 1% of the gene space

their resource level is *below* the value of the first gene, the *macrocyst join threshold* $(-2.0 < R_{\text{join}} < 2.0)$.[2] When a cell's resource level is *above* the value of the second gene, the *split threshold* $(5.0 < R_{\text{split}} < 20.0)$, the cell will pay a resource cost to split mitotically and produce a new cell (sharing resources equally between itself and its offspring).

In the model, there is only one macrocyst which is assumed to be immobile and therefore does not receive resource from the environment. When cells join it, they contribute their own resources ($x$) plus a residual resource amount (equal to the cost of splitting, $R_{\text{r}} = 1.0$) to the macrocyst's 'pot' ($X$). Before closing the macrocyst pays a cost $R_{\text{m}} = 0.05$ per individual joined every timestep to reflect metabolisation and building of cellulose. If the macrocyst resource level falls below zero ($X < 0$) then it (and all its joining cells) will die. When the macrocyst reaches a predetermined resource threshold (30.0), it closes and no other cells may join.

The macrocyst will germinate on the first day of the high season. When it germinates, the resources are divided up into new cells with each cell receiving $x = 2.5$ resource units. All new cells will have the same genotype: a complete genotype (no recombination) is picked at random from all the cells that originally joined the macrocyst.

Simulations were run over 100,000 timesteps. Each simulation started with 100 individuals, each individual having a random genotype and a random resource level between 0.0 and 5.0. Pseudo-code for the simulations is shown in Figure 5.3.

## 5.3   Results

To understand how the harshness of the low season can affect the viability of the macrocyst, simulations were run varying the probability of receiving resources in the low season. Interesting population dynamics, with macrocyst offspring out-competing the non-joining population, were observed and these are presented in this section.

The average percentage of individuals which germinated from the macrocyst is plotted against the probability of receiving resources in the low season in Figure 5.4. When the probability of receiving resources is higher, few individuals ($\approx 20\%$) join the macrocyst. When there is a lower probability of receiving resources, more individuals join the macrocyst. However the rogue data points at the bottom left of the graph are of interest.

To investigate this disparity with some populations producing macrocysts and others not, the probability of receiving resources and seed value were selected from one of the rogue data points. The simulation was run over a longer (150,000) number of timesteps. A

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[2]A negative macrocyst join threshold means that an individual will die before it joins the macrocyst

```
 0:  Define agent as having resources x, split threshold R_split and join threshold R_join
 1:  Define macrocyst as having X resources and n agents
 2:  Initialise x, R_split and R_join for new members of the population agents
 3:  season = high
 4:  for t = 1 to simulation_time
 5:      if time_since_last_season_change = 200
 6:          if season = high
 7:              N = 100, u = 0.6, season = low
 8:          end if
 9:          if season = low
10:              N = 20, u = 0.3, season = high
11:              if there is a macrocyst,
12:                  Create X/2.5 new agents with x = 2.5 for each agent
13:                  Remove macrocyst from simulation
14:              end if
15:          end if
16:      end if
17:      for macrocyst (if there is one)
18:          X = X − nR_m
19:          if X < 0, remove macrocyst from simulation
20:      end for
21:      pick N random agents
22:          with probability u, x = x + R_u
23:      end pick
24:      for each agent (picked in random order) in agents
25:          x = x − R_c
26:          if x < 0, remove agent from simulation
27:          Mutate R_split and R_join
28:          if x > R_split
29:              x = x − R_r (pay the cost of reproduction)
30:              Create a new agent (the offspring)
31:              Share the parents resources (x) evenly between the two agents
32:          end if
33:          if x < R_join
34:              if there is no macrocyst, create one (X = 0, n = 0)
35:              for macrocyst
36:                  n = n + 1
37:                  X = X + x + R_r
38:              end for
39:          end if
40:      end for
41: end for
```

Figure 5.3: Pseudo-code for the macrocyst simulations.
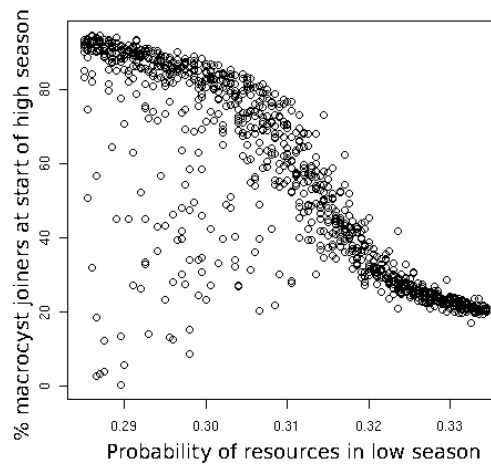
Figure 5.4: Graph showing the percentage of individuals which germinated from a macrocyst at the start of the high season against the probability of receiving resources in the low season. Each data point (ten data points, each generated with different random seeds, per resource-probability value) represents an average over a complete simulation run.

histogram was generated for the macrocyst join threshold at the start of each high season and the results are shown as a 3D mesh in Figure 5.5.

In the figure, the presence of macrocysts can be seen as spikes on the right hand side. An early tendency towards macrocyst joining is evident (far right of graph) but these genotypes die out after $\approx 25$ cycles. A population which does not produce germinating macrocysts immediately flourishes. After $\approx 150$ more cycles there are enough individuals to successfully produce a germinating macrocyst which survives to the end of the low season. Interestingly once this has happened the macrocyst very quickly wipes out the non joiners from the population. The offspring from the macrocyst must have some sort of competitive advantage over the non-joining population.

A closer look at Figure 5.5 indicates that when there are not enough individuals joining the macrocysts to make them germinate, there is only a small tendency toward individuals that will not join the macrocyst when their resource level is very low. Between cycle 25 and cycle 175, the histogram shows a larger proportion of individuals having a join threshold below zero; however some still remain with a threshold above zero. There is clearly little selection pressure against individuals sacrificing small amounts of resources when near to death.

A second 3D histogram was generated for the split thresholds of the population at the start of the high season and can be seen in Figure 5.6. There is a clear disparity of the split thresholds between the macrocyst joining population and the non joiners. Again, in
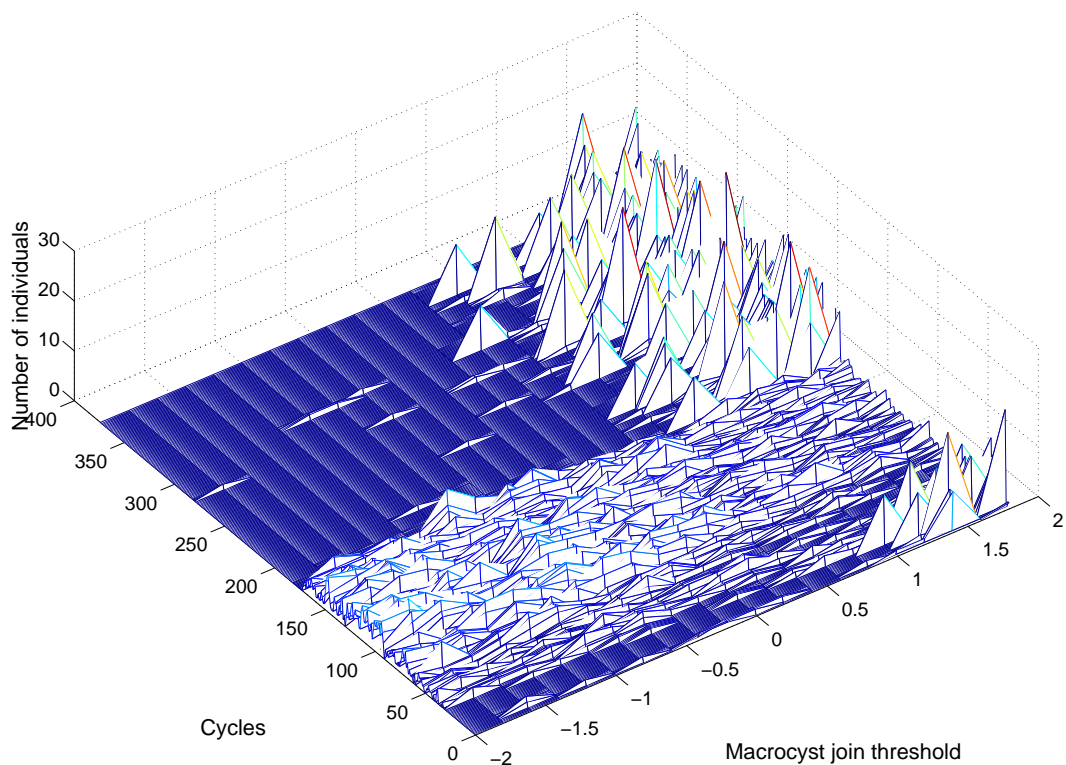
Figure 5.5: 3D histograms of macrocyst join thresholds of the population at the start of each high season.

the first few cycles of the simulation (where the macrocyst joiners were predominant in Figure 5.5), the population has a low split threshold – individuals will split as quickly as possible. After $\approx 25$ cycles the macrocysts die out. There is now a clear tendency for dominance in the population for individuals that split more slowly. Once the macrocysts return (after $\approx 160$ cycles), the split thresholds of the population immediately return to lower values $(< 7)$.

In the simulation shown, the offspring of the last macrocyst of the early period of macrocyst dominance have a higher split threshold than the rest of the macrocysts. This was confirmed in all other observed runs, however no experiment has categorically confirmed this to always be the case.

Simulations run with all individuals having the same, fixed, split threshold resulted in either the individuals all dyeing, through starvation in the low season, or a small percentage joining the macrocyst when resources are more plentiful (data not shown). The competitive advantage of the macrocyst joining population was no longer effective and macrocysts were only formed through enough individuals sacrificing their resources in a similar way to the non-joining population in Figure 5.5.

Other simulations have been run with variable split thresholds and the low season
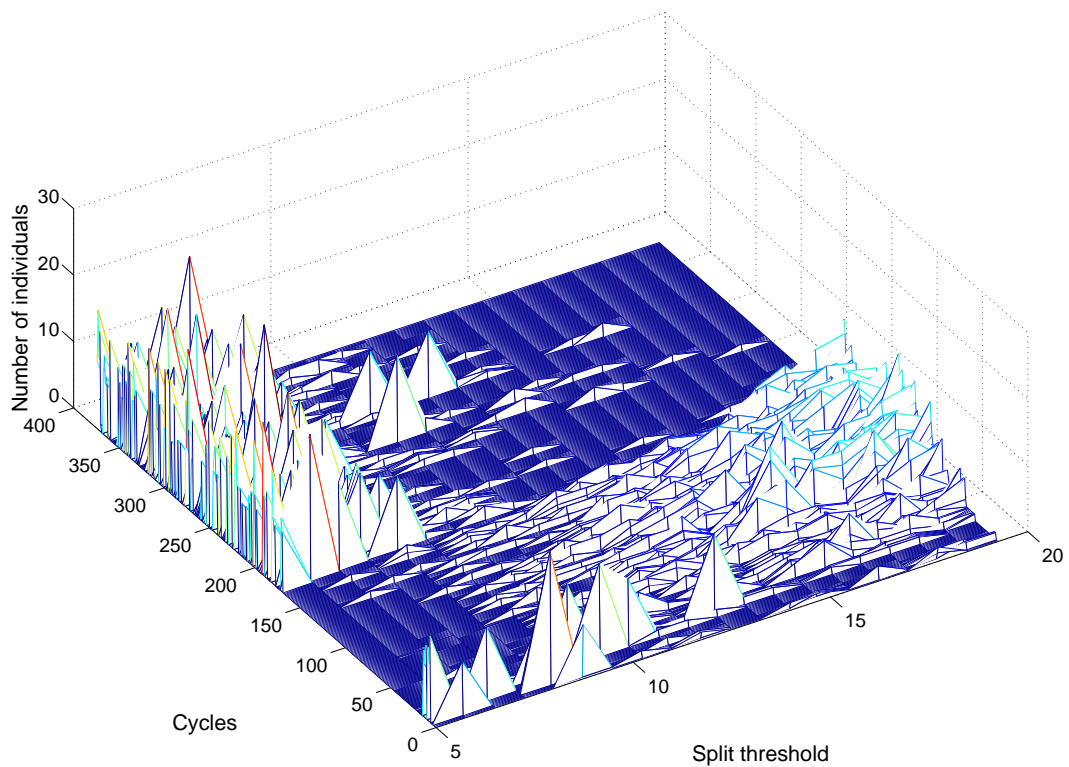
Figure 5.6: 3D histograms of individual split thresholds of the population at the start of each high season.

completely removed to see if parameters exist where a macrocyst can form and dominate the rest of the population. Simulations were run with varying parameters of $N$ and $u$, both seasons having the same values. While some macrocyst production was observed it was only at the beginning of simulations where the random starting population allowed for enough individuals that joined the macrocyst and made it viable for a few cycles (data not shown).

## 5.4  Discussion

This chapter presents a novel perspective on the transition to multicellularity. In Section 5.1 I have justified of the need for a model that demonstrates the transition between individual cells that ordinarily reproduce on their own and cells that become part of a super-organism, with only one genotype of the participating cells being passed on to future generations. The model in this chapter, of the evolution of the *D. discoideum* macrocyst stage, demonstrates a plausible mechanism through which individuals may start to reproduce as part of a group. I investigate this model now within the context of the above requirements, and other questions and literature concerning the transition to multicellu-

larity presented in Section 5.1.

For the macrocyst model to successfully meet the requirements of the transition to multicellularity, it requires that all individual cells must be able to reproduce on their own. It also requires that individual cells must be clustered and that only one of the individual cells reproduces on behalf of the cluster. The model presented meets these requirements. Simulated cells that have the freedom to evolve a strategy in which they will not join macrocyst organisations (where their genes are highly likely to be destroyed) do not evolve this strategy under certain conditions.

The model does however stop short of demonstrating the type of germ-line/soma differentiation and clustering apparent in the metazoa where there is permanent clustering [as in other models, e.g., (Wolpert 1990, Pfeiffer and Bonhoeffer 2003)] and differentiation of the germ line early in development (Michod 1999). The macrocyst's germ-line cell is instead chosen at random with little or any predetermination. The macrocyst cells are only clustered at one point of the life cycle. However, the fact that the macrocyst's offspring are of only one genotype and that they out-compete individuals that do not join the macrocyst is of some significance.

The fact that the macrocyst produces offspring of a single genotype is important in three ways. Firstly it has the effect of producing several genetically homogeneous offspring which are all 'pre-programmed' to join the macrocyst at the start of the next low season. These offspring have a competitive advantage over individuals that do not join the macrocyst. The macrocyst therefore contributes to its future success. Since microbes can evolve many 'policing' mechanisms (Travisano and Velicer 2004), it is not inconceivable that after several generations, the macrocyst way well have become established in the organism without the need for a harsh low season each cycle.

Secondly, the high relatedness of the offspring can be seen to promote other social behaviours. Relatedness is crucial for any traits that require many coordinated individuals or altruism to be successful. The aclonal nature of the macrocyst offspring means that it is highly likely that the next aggregation event will be aclonal or at least highly related. If these individuals, perhaps due to some mutation, no longer fuse to form a zygote then other interesting collective behaviour may occur instead. These behaviours could include the slug behaviour of *D. discoideum* which requires many coordinated individuals (Marée, Panfilov and Hogeweg 1999), and the stalk behaviour of *D. discoideum* which requires altruism from many cells (Armstrong 1984). The combination of the macrocyst model with one of the stalk/spore behaviour [based on the work of Armstrong (1984)] will hopefully confirm how important the population homogenisation effects of the macrocyst were for the evolution and maintenance of stalk/spore behaviour in *D. discoideum*.

The homogeneous macrocyst offspring are important in a third way. By picking the genotype of its offspring from one individual at random, this can restrict the potential for cheating. For example, if an individual were to evolve a cheating trait so that its genes were most likely to be picked, the next population would all have that same trait - with no individual having any advantage. That said, it is conceivable that a cheating trait may in fact be disruptive to the macrocyst. Further modelling is required to investigate the potential effects of disruptive cheating.

To consider how split thresholds are important I analyse a complete cycle (of a high and a low season). In one cycle of the model presented here there are four phases for non macrocyst joining amoebae:

  (i). Early high season exponential growth;

 (ii). Population equilibrium at high season;

(iii). Early low season exponential decimation of the population;

(iv). Population equilibrium at low season.

While it is easy to see that fast (low threshold) splitting amoebae would flourish during phase (i), these same amoebae will be closer to dying during phase (iii). The results suggest that a slow (high threshold) splitting strategy is more profitable, not only in phase (iii) but in phase (iv) as well. In phase (iv) individuals receive food with a low probability, those with a fast (low) split threshold are less adapted to survive fluctuations in food availability.

The macrocyst allows individuals to avoid phases (iii) and (iv) and hence fast splitting individuals that germinate from it at the start of the high season are very well adapted to phase (i). This ability to perform well during circumstances of diminishing populations has already been observed as an important feature of early multicellular organisms (Kerszberg and Wolpert 1998).

While I have attempted to be faithful to biological evidence, the model presented here has made some assumptions and has some limitations. Further analysis and research is required into the biological plausibility of the split thresholds in the model. The question as to what might happen if individuals have a seasonally varying split threshold is also important. The model is undimensional and therefore lacks spatial effects (though the way the organisms are fed is set up to mimic a spatial environment): a spatial model would allow for analysis as to what might happen if individuals could affect their chances of being the chosen genotype. The mutation rate in the model is unnaturally fast, however slower mutation rates provided similar results over longer periods. Finally, there is

only one macrocyst in the current model; future simulations could model more than one macrocyst.

The evolution of collective, coordinated and altruistic behaviour is of some significance within ALife modelling. The homogenisation effects of the macrocyst and its likely relevance in the evolution of coordinated and altruistic effects means that this effect is likely to be of some use in the design of evolutionary algorithms for multi-agent systems. Further investigation into this stage may well reveal its significance in the evolution of sophisticated collective behaviour in artificial and natural organisms.

The model and results presented in this chapter demonstrate that, given the assumptions outlined, the *D. discoideum* macrocyst stage is plausible under the large fluctuations in food in the model. The results and analysis lead me to hypothesise that the model of the macrocyst presented in this chapter, where individuals gamble their genes to become the germ line of a super-organism, may well have been a crucial stage in the transition to multicellularity. It must be noted that it is only a stage in the evolution of *D. discoideum* and may only be relevant to this organism. Though mere speculation, it is intriguing to consider that if the macrocyst was an important stage in the transition to multicellularity, then the harsh and specific environmental conditions needed may explain why the transition only happened once and took such a long time to arise.

# Chapter 6

# The origins of sexual reproduction

## 6.1 Introduction

In Chapter 4 I look at a general model of collective reproduction. That work does not consider how collective reproduction is affected when individuals cheat by giving less resources than they should. This is considered in the present chapter, looking specifically at the origins of sexual reproduction.

The origin of sexual reproduction is an important question in evolutionary biology. Sexual reproduction is very common amongst multicellular organisms where, in general, individuals produce gametes that fuse together to form a zygote. In fact there are many modes of sexual reproduction (Bell 1982, Whitfield 2004); for example it is also observed in unicellular organisms where the organism fuses with another organism. For simplicity, this chapter looks at sexual reproduction from an abstract perspective: two individuals both contribute genetic material and a proportion of their resources to an offspring. This is contrasted with asexual reproduction where only one individual contributes genetic material and resources to its offspring.

Given its prevalence in the animal kingdom, it seems odd that there isn't a simple explanation of its benefits. In fact many models (Williams 1975, Maynard Smith 1978) have found that there is a cost to sex. From a population growth rate perspective (Maynard Smith 1978), production of non-egg-producing male individuals implies a cost in population growth – Maynard Smith dubbed this the "two-fold cost of sex" (Maynard Smith 1971). At its very extreme, males may contribute no resources toward the production of its offspring; thus leaving a two-fold cost of producing males that seemingly have little or no actual benefits. The alternative perspective considers the "cost of meiosis" (Williams 1975) pointing out that, in meiosis, the diploid zygote only contributes half its genes to the haploid gamete. Both these cases have been argued to be equivalent

(Bell 1982), basically each sexually reproducing parent will only receive half an offspring each time it reproduces rather than the whole offspring produced by an asexually reproducing parent. It is therefore argued that an individual which produces asexual offspring would invade a sexually reproducing population. Sexual reproduction appears to be a paradox: the question as to how sexual reproduction originated and persists is a puzzle.

I have reformulated the notion of the cost of sex into the abstract terms used within this chapter. I make a simple comparison of the growth rates of an individual strategy and a collective reproduction strategy (with two parents) based on the way resources are allocated to offspring. This is shown by Figure 4.1 (see Chapter 4).

Since individuals with the sexual strategy share contributions to offspring, they contribute less than those with the asexual strategy. This means that the average level of resources per individual in the sexually reproducing population will be higher than those in the asexually reproducing population. For a fixed input of resources to the system, this means that the sexually reproducing population will grow more slowly than the asexually reproducing population.

There have been many theories as to the advantages of sexual reproduction. Since sexual reproduction generally results in two parents contributing half their genes to each offspring, *recombination* (genetic mixing) can occur. There are many advantages of genetic mixing including resistance against deleterious mutations (Muller 1964) and the greater ability to incorporate advantageous mutations (Fisher 1958).

One problem with this approach is that the benefits from this type of reproduction are generally quite slow to evolve (with an evolutionary time-scale), too slow to counteract population invasion (with an ecological time-scale) of parthenogenetic mutants (Maynard Smith 1978). The genetic mixing arguments are therefore open to the same types of criticism applied to group selection arguments (see Section 3.1 for more information).

Given these criticisms, recent models [see reviews from Otto and Lenormand (2002) and Agrawal (2006)] still view the cost of sex as problematic and have therefore argued that genetic mixing could be advantageous enough to explain why sexual reproduction can be maintained in light of the cost of sex. For instance, sexual selection can accentuate the effects of resistance against deleterious mutations(Agrawal 2001), though this does require a significant mutation rate in a population. Other models consider the advantages that recombination may give as protection against parasites (Hamilton, Axelrod and Tanase 1990). Recent work (Hakoyama and Iwasa 2003) demonstrates that coexistence is possible between sexual and unisexual populations when the unisexual population is more than twice as susceptible to parasites as the sexual population. While these models do go quite far in explaining the advantages of genetic mixing, the argument for using the

advantages of genetic mixing to explain sexual reproduction does still raise some questions.

The main problem with the argument that genetic mixing may explain the cost of sex is concerned with the type and ubiquity of sexual reproduction seen in nature. Surely if the parthenogenetic strategy is the better strategy for reproduction, then some other mechanism that allows for occasional genetic mixing with parthenogenesis being the norm should prevail? There are a few examples of androdiecious organisms (Weeks, Benvenuto and Reed 2006) where the populations consist of males and hermaphrodites, an example being *C. elegans* where the males are as rare as 0.1% of the population (Chasnov and Chow 2002). Interestingly, deleterious mutation rates do not seem to be a likely explanation for difference between the selfing behaviour in *C. elegans* and the obligately outcrossing behaviour of another *Caenorhabditis* species, *Caenorhabditis remanei* (Cutter and Payseur 2003). Perhaps sexual reproduction (as defined earlier) and genetic mixing are two separate issues and should be explained separately.

In fact, modelling work has been done that does not consider the advantages of genetic mixing (Doncaster, Pound and Cox 2000). This considers that if a population of sexually reproducing individuals has a resource uptake advantage (and thus a higher carrying capacity) over an asexual population then the two populations may coexist. While this model is certainly convincing regarding how sex may be maintained, when it comes to explaining the origins of sex the model is not so convincing. It requires that an advantage (in this case the resource uptake advantage) evolved concurrently to a disadvantage (in this case, it is assumed that sexual reproduction is at a disadvantage) – this is generally not thought to be good reasoning (Maynard Smith and Szathmáry 1995).

In contrast, the model I shall present in this chapter does not confer any explicit advantages or differences on either the asexually or sexually reproducing individuals. It takes the approach that a precursor to sexual reproduction may have existed where two individuals reproduced collectively, contributing a proportion of their resources to a shared offspring. I will compare this *two-parent* resource allocation strategy with the *one-parent* (i.e., clonal) strategy. I avoid using the terms sexual and asexual to describe these strategies as sexual reproduction includes genetic mixing which is not specifically modelled. In the two-parent strategy, each parent has an equal stake in the genetic makeup of the offspring, whether through one parent being selected as the genotype by random chance (with equal probabilities) or there being a mix of the two parents' genes.

There are quite a few examples in the biological literature [e.g, see (Glesener and Tilman 1978, Bell 1982)] of related species where one species reproduces sexually and the other reproduces parthenogenetically. There is a repeating pattern which has been ob-

served which links geographical characteristics of an area with the reproduction strategies of its inhabitants. Sexually reproducing species are more likely to be found in mainland areas where there is assumed to be a higher level of biotic stress. Conversely, asexual organisms tend to be found in more extreme conditions where there is assumed to be less biotic stress but more abiotic stress. The higher biotic stress is due to the greater diversity shown in the surrounding ecosystem of the areas with lower abiotic stress. It is suggested that this higher level of biotic stress can lead to greater uncertainty (due to inter-specific interactions) than abiotic stress (Glesener and Tilman 1978). I therefore consider whether the amplitude of unpredictable resource fluctuations is an important factor in the model presented in this chapter.

### 6.1.1 Scope of the model

To investigate the questions raised in the previous section, the work here reconsiders the original arguments presented by Maynard Smith and Williams. Rather than consider a population of males and females being invaded by parthenogenetic individuals I consider the opposite: a population of clonal (one-parent) reproducers being invaded by two-parent reproducers. I ask which is the stable strategy and under what conditions.

I approach this question by simplifying the world to resources, individuals and individual reproduction strategies. Individuals accumulate resources, expend resources, make copies of themselves and share resources with those copies.

Given that individuals live within an environment with factors outside of their control, I assume that they have little control over the way they accumulate or expend resources. The space of strategies that individuals may adopt is therefore only concerned with their reproduction strategy. One factor that is important however is the size of the population will affect the availability of resources. This means that I am able to compare one- and two-parent resource allocation strategies under density dependent conditions.

The model is an extension of the one presented in Chapter 4, which also compared one- and two-parent reproduction. In the previous model, individuals shared their resources equally with their offspring, whereas here individuals may selfishly contribute differing amounts of resources. In line with the biological data, the individuals are tested under varying resource availability conditions.

In the next section I outline the methods I used to model the different reproductive strategies. This includes deriving the growth equations which will allow me to compare the different reproduction strategies. I also present methods for the simulation models which will be used to test and expand the mathematical predictions. The results start by

considering what level of resources an individual may contribute (Section 6.3.1) before considering whether this level is high enough for the two-parent strategy to dominate an optimal one-parent strategy. I discuss the results in Section 6.4.

## 6.2   Methods

The model presented here considers individuals receiving stochastically varying resources from the environment. When their resource level ascends to a high threshold then they reproduce either clonally or share the production of an offspring with another parent. All parents pay a cost of reproduction and then share some of their resources with the new offspring. The reproduction strategy of individuals is therefore defined by the proportion of their resources that they will share with their offspring and whether they have a one- or two-parent strategy for producing offspring.

The amount of resources individuals will give their offspring is important because it affects the growth rate. The reason I study it in detail here is because there is a selfish benefit for parents to contribute less resources when they produce an offspring with another parent. Consider a selfish parent that gives less resources to an offspring than an unselfish parent that shares its resources equally. The selfish parent will have sufficient resources to reproduce again more quickly than the unselfish parent. A selfish parent would therefore be expected to invade a population of parents contributing more resources. This would result in a population of individuals with the two-parent strategy contributing very little resources to their offspring.

Two modelling approaches are presented in this section: mathematical modelling and computer simulation models. The mathematical model presents a tractable approach to the problem which is instructive to the simulations.

The strategy space for individuals is defined by how many resources they contribute to their offspring and whether they have a one- or two-parent reproduction strategy. Because I am studying like-for-like, all individuals have the same interaction with their environments. In all models, an individual is modelled as having a resource level $x$, given by,

$$\frac{dx}{dt} = U(i,t)R_{\mathrm{u}} - R_{\mathrm{c}} \, , \tag{6.1}$$

During an average timestep of time length $\delta t$, the individual will mandatorily use $R_{\mathrm{c}}$ resources. *Resource uptake* (the level of resources received from the environment) is variable and modelled by $U(i,t) \in [0,1]$. If an individual reaches its lower resource threshold $R_0$ then it will die. Similarly, if an individual reaches its upper resource threshold $R_1$

then it will reproduce. In reproduction, each parent pays an upfront cost of reproduction $R_r(j)$ dependent on its reproduction strategy and contributes a proportion of the remaining resources $O$ toward its offspring.

## 6.2.1 Mathematical modelling

For the mathematical modelling approach, I derive growth rates for each of the strategies. Growth rates are calculated by assigning individuals with the one-parent strategy one offspring and individuals with the two-parent strategy half of an offspring. I use the growth equations derived in Chapter 3.

In Chapter 3, I considered two different models of growth: a deterministic and a stochastic model. The model used in this work was the stochastic model (derived in Section 3.2.4). This is for two reasons, firstly because this model was shown to be a good approximation of the simulation results in the case where the resource uptake rate is close to population equilibrium levels. The second reason is that it was also shown to be more accurate when $x$ is close to the lower threshold. Since selfish parents may contribute low resource levels to their offspring, this model is needed to give accurate results. The function $U(i,t)$ [see Equation (6.1)] is thus modelled by white noise depending on a variable $u$ and $\delta t$. Time is broken up into segments of length $\delta t$ and for each segment $\Pr(U = 1) = u$ and $\Pr(U = 0) = 1 - u$. The average over realisations ($< U >= u$) is also constant over an individual's lifetime.

As an *ad hoc* simplification, $R_0$ is set to 0. We can study different values of $R_0$ by scaling $R_c$, $R_r$ and $O$ appropriately.

The resource values of individuals after reproduction are important and will be used in the model. Thus, directly after one-parent reproduction, the parent's resource level is given by:

$$x_{\text{parent}} = (R_1 - R_r)(1 - O) .$$

Similarly the offspring's resources are given by:

$$x_{\text{offspring}} = (R_1 - R_r)O .$$

Secondly, after two-parent reproduction, the parents share the cost of reproduction $R_r$ and then each contributes a proportion of its remaining resources $O(q)$ towards the offspring. Thus, directly after two-parent reproduction, the resource level of a parent $i$ is given by:

$$x_{\text{parent}}(i) = (R_1 - R_r)[1 - O(i)] .$$

The resource level of the offspring is given by the sum of the resource contributions of the parents:

$$x_{\text{offspring}}(i) = (R_1 - R_r)\Sigma^i O(i) .$$

The growth rate $G$ for each strategy (in new individuals per individual per time $\delta t$) is given by the number of births minus the number of deaths per individual in time $\delta t$. The birth rate and death rate are calculated using the equations for the expected generation time $W$ (the time taken for an individual starting with $x$ resources to reach either the upper or lower threshold) and the reproduction probability $F$ (the probability of an individual, starting with $x$ resources, reaching its upper threshold before it reaches its lower threshold).

The equation for the expected generation time $W$ (the time it takes an individual with resources $x$ to reach either its upper or lower resource threshold) has two solutions which are derived in Section 3.2.4. Again, for simplicity of modelling a variable $m$ is introduced where $m = R_u/R_c - 1$ [so, $R_u = (m+1)R_c$]. Since $R_u > R_c$, and both are positive, $m$ is positive. I consider the general case where the mean resources uptake level is at $u$ [here $u \neq u_0$ where $u_0 = 1/(m+1)$] is given [from Equation (3.14)] by:

$$W_g = \frac{\delta t}{R_c \lambda} \left[ -x + \frac{R_1 [1 - \exp(-\lambda x/\mu)]}{1 - \exp(-\lambda R_1/\mu)} \right] , \tag{6.2}$$

where $\lambda = mu + u - 1$ and $\mu = R_c(m^2 u + 1 - u)/2$. The case where an individual receives on average as many resources as it expends each timestep ($u = u_0$) is given [from Equation (3.16)] by:

$$W_e = \frac{\delta t}{R_c^2 m}(-x^2 + R_1 x) . \tag{6.3}$$

The ratio of individuals that reach the upper threshold to those that reach the lower threshold $F$ was also derived in Section 3.2.4. An individual, starting with $x$ resources, must reach its upper resource threshold ($R_1$) before it reaches the lower resource threshold ($R_0$). Again there is a general case where $u \neq u_0$. This is given [from Equation (3.22)] by:

$$F_g = \frac{1 - \exp(-\lambda x/\mu)}{1 - \exp(-\lambda R_1/\mu)} \tag{6.4}$$

The case where an individual receives on average as many resources as it expends each timestep ($u = u_0$) is given by:

$$F_e = \frac{x}{R_1} . \tag{6.5}$$

To calculate the growth rate I consider new individuals just after reproduction. In the

one-parent reproduction model (see Chapter 3) both offspring after reproduction are identical. In this model they may have different resource levels: after one-parent reproduction the parent will therefore have $x_{\text{parent}}$ resources and the new offspring will have $x_{\text{offspring}}$ resources. I therefore approximate the growth rate of an average individual as follows. The average number of births is given by $\left[F(x_{\text{parent}}) + F(x_{\text{offspring}})\right]/2$ and the average number of deaths is given by $\left[1 - F(x_{\text{parent}}) + 1 - F(x_{\text{offspring}})\right]/2$. The average generation time is given by $\left[W(x_{\text{parent}}) + W(x_{\text{offspring}})\right]/2$. I therefore approximate the growth rate as the average number of births per time $\delta t$ minus the average number of deaths per time $\delta t$, or,

$$
\begin{aligned}
G_{\text{one}} &= \frac{F(x_{\text{parent}}) + F(x_{\text{offspring}})}{W(x_{\text{parent}}) + W(x_{\text{offspring}})} - \frac{2 - F(x_{\text{parent}}) + F(x_{\text{offspring}})}{W(x_{\text{parent}}) + W(x_{\text{offspring}})} \\
&= \frac{2\left[F(x_{\text{parent}}) + F(x_{\text{offspring}}) - 1\right]}{W(x_{\text{parent}}) + W(x_{\text{offspring}})} .
\end{aligned} \tag{6.6}
$$

The two-parent case considers the parent and its offspring in the same way as the one-parent case. The difference here is that each parent only gives birth to half an offspring (so there are 1.5 individuals after reproduction rather than 2). The number of births is therefore given by the average number of births per individual. This is given by the average number of reproduction events of the 1.5 individuals divided by 2 (since these individuals only have half of an offspring): $[2F(x_{\text{parent}})/3 + F(x_{\text{offspring}})/3]/2$. Similarly, the number of deaths is given by $2[1 - F(x_{\text{parent}})]/3 + [1 - F(x_{\text{offspring}})]/3$ and the average generation time is $2W(x_{\text{parent}})/3 + W(x_{\text{offspring}})/3$. Again, I approximate the growth rate as the average number of births per time $\delta t$ minus the average number of deaths per time $\delta t$, giving a growth equation of,

$$
G_{\text{two}} = \frac{3\left[F(x_{\text{parent}}) + F(x_{\text{offspring}})/2 - 1\right]}{2W(x_{\text{parent}}) + W(x_{\text{offspring}})} . \tag{6.7}
$$

If the environment is static ($u$ constant), then the strategy that will dominate is the one which has the larger value of $G$ from Equations (6.6) and (6.7). On the other hand, if resources fluctuate between abundant and sparse periods ($u$ varies between two fixed values $u^+$ and $u^-$), the dominant phenotype can be determined by using Corollary 3 in Appendix A. The corollary states that, if a population is in a steady state (has the same total size at the beginning and end of a long time period), then the strategy that will dominate is the one with the largest ratio of growth during abundant periods ($u = u^+$) to

growth rate during sparse periods ($u = u^-$). Taking $\Psi$ as,

$$\Psi = \frac{G(u^+)}{-G(u^-)} \, , \tag{6.8}$$

the two-parent strategy therefore dominates when:

$$\Psi_{\text{two}} > \Psi_{\text{one}} \, . \tag{6.9}$$

## 6.2.2 Parental contribution

As identified earlier, when one parent contributes less resources to a shared offspring than another, it will be ready to reproduce more quickly and is expected to invade the population. In this section I use the growth equations from the previous section to predict which offspring contribution strategies will dominate over other offspring contribution strategies.

To do this I consider two parents of class Pa and Pb which contribute $O_a$ and $O_b$ resources respectively. The matrix in Table 6.1 shows the resource levels for a parent and the resulting offspring after two-parent reproductive encounters with parents of both classes.

|  | **Pa** | **Pb** |
|---|---|---|
| **Pa** | $x_{\text{parent}} = (R_1 - R_r)(1 - O_a)$ | $x_{\text{parent}} = (R_1 - R_r)(1 - O_a)$ |
|  | $x_{\text{offspring}} = (R_1 - R_r)2O_a$ | $x_{\text{offspring}} = (R_1 - R_r)(O_a + O_b)$ |
| **Pb** | $x_{\text{parent}} = (R_1 - R_r)(1 - O_b)$ | $x_{\text{parent}} = (R_1 - R_r)(1 - O_b)$ |
|  | $x_{\text{offspring}} = (R_1 - R_r)(O_a + O_b)$ | $x_{\text{offspring}} = (R_1 - R_r)2O_b$ |

Table 6.1: Table showing resources of parents and offspring immediately after reproduction, the values for $x_{\text{parent}}$ and $x_{\text{offspring}}$ shown are for when the parent on the left column reproduces with a parent on the top row.

For simplicity, the first case I consider is where resource uptake is static. The growth rate of each strategy when it reproduces with either parent can then be calculated using Equation (6.7). For two parental contribution strategies, Pa and Pb give $O_a$ and $O_b$ to offspring respectively, it is useful to be able to predict which strategy will dominate the other. Taking $G(\text{Pa};\text{Pb})$ to be the growth rate of parent Pa and its half offspring from a reproductive encounter with parent Pb, I use game theory (Maynard Smith 1982) to analyse the outcome of competitions between mixed populations of Pa and Pb. This predicts that if $G_{\text{Pa};\text{Pa}} > G_{\text{Pb};\text{Pa}}$ and $G_{\text{Pa};\text{Pb}} > G_{\text{Pb};\text{Pb}}$ then Pa will be monodominant over

Pb. Alternatively, when $G_{\text{Pb;Pa}} > G_{\text{Pa;Pa}} > G_{\text{Pa;Pb}} > G_{\text{Pb;Pb}}$ then there will be a mixed equilibrium of both Pa and Pb individuals. The hawk-dove model (Maynard Smith 1982) is used to predict that there is an ESS (Evolutionarily Stable Strategy: one that is not invaded by alternative strategies). Therefore the stable relative frequency of Pa to Pb, $\rho(\text{Pa,Pb})$, is given by:

$$\rho(\text{Pa, Pb}) = \begin{cases} 1 & \text{, Pa dominant over Pb} \\ \dfrac{G_{\text{Pa;Pa}} - G_{\text{Pb;Pb}}}{G_{\text{Pa;Pb}} + G_{\text{Pb;Pa}} - G_{\text{Pa;Pa}} - G_{\text{Pb;Pb}}} & \text{, mixed equilibrium} \\ 0 & \text{, Pb dominant over Pa} \end{cases} \qquad (6.10)$$

As with the last section, I consider what happens when the probability of resources fluctuates between abundant and sparse regimes. As in the previous section, the two regimes are considered using $u^+$ for when the population is increasing and $u^-$ for when the population is declining. The respective growth functions are $G(u^+)$ and $G(u^-)$. For the two parents Pa and Pb, I consider the growth during regime $u^+$ relative to the growth during regime $u^-$. To give an example, the relative growth of parent Pa and its half offspring from a reproductive encounter with parent Pb is $G_{\text{Pa;Pb}}(u^+)/G_{\text{Pa;Pb}}(u^-)$. Using Corollary 3 (in Appendix A) it can be seen that if

$$\Psi_{\text{Pa;Pa}} > \Psi_{\text{Pb;Pa}}$$

and

$$\Psi_{\text{Pa;Pb}} > \Psi_{\text{Pb;Pb}}$$

then Pa will be monodominant over Pb. I assume, for simplicity that if this is not true, then Pb will dominate over Pa.[1] The stable relative frequency of Pa to Pb is therefore given by:

$$\rho^*(\text{Pa, Pb, u}^+, \text{u}^-) = \begin{cases} 1 & \text{, Pa dominant over Pb} \\ 0 & \text{, Pa not dominant over Pb .} \end{cases} \qquad (6.11)$$

## 6.2.3  Simulation modelling

To validate and extend the predictions made by the mathematical approach in the previous section, simulation models were built. This section details the functional properties of the simulation models.

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[1] It is not possible to simply follow Maynard Smith's reasoning (Maynard Smith 1982) to calculate a figure for the relative frequency of the two strategies when there is a mixed strategy, i.e., neither Pa nor Pb is predicted to be dominant.

Copying the mathematical approach, the population is modelled at an individual level with discrete timesteps of length $\delta t$. Each individual is represented by a software agent. Each software agent maintains a resource level (represented as a floating point number) and a digital chromosome. Changes in individual resources are given by Equation (6.1) with $U(i,t)$ modelled by white noise [as with the stochastic model, see Section 6.2, $\Pr(U = 1) = u$ and $\Pr(U = 0) = 1 - u$]. Each timestep a mandatory resource cost $R_c$ is deducted from each agent's internal resource level. If an agent's resource level goes below $R_0$ then the agent will die and is removed from the simulation. When an agent's resource level increases above the reproduction threshold $R_1$ then it is ready to reproduce. At the start of all simulations individuals started with a random resource level generated randomly from a flat distribution between $R_0$ and $R_1$.

The digital chromosome contains numerical values that represent genetically determined factors: the amount of resources an individual contributes to its offspring and whether it reproduces with a one- or two-parent strategy. The digital chromosome is simply passed on from the parent to the offspring in the one-parent case. In the two-parent case, a random parent is selected to pass on the chromosome. Two genes are used in the chromosome, the first gene represents the resource contribution level $O = [0 : 1]$. The second gene $S = [0 : 1]$ determines whether the individual reproduces with a one- or two-parent strategy. It is also represented as floating point number between 0.0 and 1.0, with values over 0.5 representing the two-parent strategy. At the start of all simulations all genes were generated randomly from a flat distribution between 0.0 and 1.0. When new offspring are generated, point mutations are applied to the numerical values in the chromosome. Mutation values were from a flat distribution (between 0.0 and 1.0) and applied with a probability of 1/100 immediately after a new individual is created.

The contribution of resources from parents to offspring depends on whether the individual's chromosome specifies a one- or two-parent strategy. If it is one-parent reproduction then it will simply pay a cost of reproduction $R_r$ and then contribute a proportion of its resources $O$ to a new offspring. For two-parent reproduction, random pairs of agents are selected from those that are ready (any leftover agents must wait for the next timestep). Each parent pays half the cost of reproduction $R_r$ and contributes a proportion of its resources $[O(i)]$ to the new offspring.

The way agents receive resources from the environment is determined by whether I am considering a population close to equilibrium or a population that experiences large fluctuations in resource availability. When the population is at equilibrium, a number of resource units [of value $R_u$] are randomly scattered on a spatial grid each time step.

Agents located on the grid move to a random new cell each timestep[2].

When individuals are exposed to resource fluctuations, their resource uptake rates are dependent on the current state of the system. There are two resource uptake values for $u$ (called $u^+$ and $u^-$) which defined by the resource fluctuation coefficient $\eta$ as follows: $u^+ = u_e\eta$ and $u^- = u_e/\eta$.[3] The system switches between its two states depending on the population level. An upper and a lower population threshold are defined which trigger the two states. The system state using $u = u^+$ is triggered by the lower population threshold and the system state using $u = u^-$ is triggered by the upper population threshold.

Tests were done with various upper and lower population thresholds to make sure they are effective: the thresholds were too close to each other when they were within 33% of the population size, this had the same effect of reducing the fluctuation coefficient (as the resources started to fluctuate within the generation cycle of individuals). The upper population threshold was therefore set to 400 and the lower population threshold was set to 200 in all simulations.

Two simulation scenarios were run depending on whether individuals had fixed offspring resource contributions or not. Simulations in Scenario (i) were run with no mutations to the value of $O$ and with the population close to equilibrium (no fluctuations) in order to validate the game theoretic approach in Section 6.2.2. Each simulation was started with an evenly mixed population of agents with two different offspring resource contribution amounts representing strategies Pa and Pb. All agents used a two-parent strategy and were similar in all other ways. After a suitable amount of time, the proportion of agents with strategy Pb to strategy Pa was recorded. Pseudo-code for the scenario is presented in Figure 6.1.

The simulations in Scenario (ii) allowed mutations to the value of $O$. All agents were exposed to resource fluctuations. To confirm the predictions made by Equations (6.10) and (6.11) I performed simulations with only two-parent agents. I then ran competitions between agents with two- and one-parent strategies to confirm the conditions under which two-parent agents will dominate over one-parent agents. Pseudo-code for the scenario is presented in Figure 6.2.

---

[2]The results in the spatial chapter show that agents receive resources with between-resource intervals on a geometric distribution when they move to random grid squares each timestep

[3]An appropriate value of $u_e$ was chosen for the simulations based on the predicted value of $u_e$ given by the parameters

```
 0:  Define agent as having resources x and resource contribution O
 1:  Initialise x and O for new members of the population agents
 2:  for t = 1 to simulation_time
 3:      Scatter new resources on the grid
 4:      Move each agent to a random grid-square
 5:      for each agent (picked in random order) in agents
 6:          x = x − R_c
 7:          if there are resources on the grid-square
 8:              x = x + R_u
 9:              remove one resource from the grid-square
10:          end if
11:          if x < 0, remove agent from simulation
12:          for random pairs of agents (parents) with x > R_1
13:              for each agent x = x − R_r (pay the cost of reproduction)
14:              Create a new agent (the offspring)
15:              Both parents contribute xO resources to the offspring
16:          end if
17:      end for
18: if all agents have the same value of O stop simulation
19: end for
```

Figure 6.1: Pseudo-code for the evolutionary simulations of scenario (i)

## 6.3 Results

This section consists of results from the mathematical treatments and from the simulation modelling. The results work toward a comparison of one- and two-parent strategies. Chapter 4 has already shown that the two-parent strategy may be dominant in the long term when parents share resources with their offspring. I thus first consider if there is a stable amount of resources that parents may contribute to their offspring. I then determine whether the lower resources contributed by selfish parents is too low for the two-parent strategy to dominate the one-parent strategy.

### 6.3.1 Stable parental resource contribution

In order to predict what level of resources individuals may contribute to offspring, I first consider competitions between two parents that contribute different amounts of resources. I then consider whether there is a stable strategy when there is a fully mixed population.

```
0:  Define agent as having resources x, resource contribution O
        and reproduction strategy S
1:  Initialise x, O and S for new members of the population agents
        (when population is all two-parent, set S = 1)
2:  season = high
3:  for t = 1 to simulation_time
4:      if |agents| > upper_threshold, season = low
5:      if |agents| < lower_threshold, season = high
6:      for each agent (picked in random order) in agents
7:          x = x − R_c
8:          if season = high
9:              with probability u⁺, x = x + R_u
10:         end if
11:         if season = low
12:             with probability u⁻, x = x + R_u
12:         end if
13:         if x < 0, remove agent from simulation
14:         for random pairs of agents (parents) with x > R_1 and S > 0.5
15:             for each agent x = x − R_r (pay the cost of reproduction)
16:             Create a new agent (the offspring)
17:             Both parents contribute xO resources to the offspring
18:             Mutate the new agent's value of O
19:             if including one-parent agents, mutate the new agent's value of S
20:         end if
21:         for agents with x > R_1 and S <= 0.5
22:             x = x − R_r (pay the cost of reproduction)
22:             Create a new agent (the offspring)
23:             The parent contributes Ox resources to the offspring
24:             Mutate the new agent's values of O and S
25:         end for
26:     end for
27: end for
```

Figure 6.2: Pseudo-code for the evolutionary simulations of scenario (ii)

### 6.3.1.1 Two player resource contribution competitions

The stable frequency of two parental resource contribution strategies can be predicted by the game theory outlined in Section 6.2.2 when resources are not fluctuating. Two parents Pa and Pb were assigned higher and lower resource contribution values $O_a$ and $O_b$ respectively. For each resource contribution value assigned to parent Pb, parent Pa is assigned a range of higher values ($O_a > O_b$). The value of $\rho(\text{Pa,Pb})$ is calculated using Equation (6.10) and is plotted in Figure 6.3.
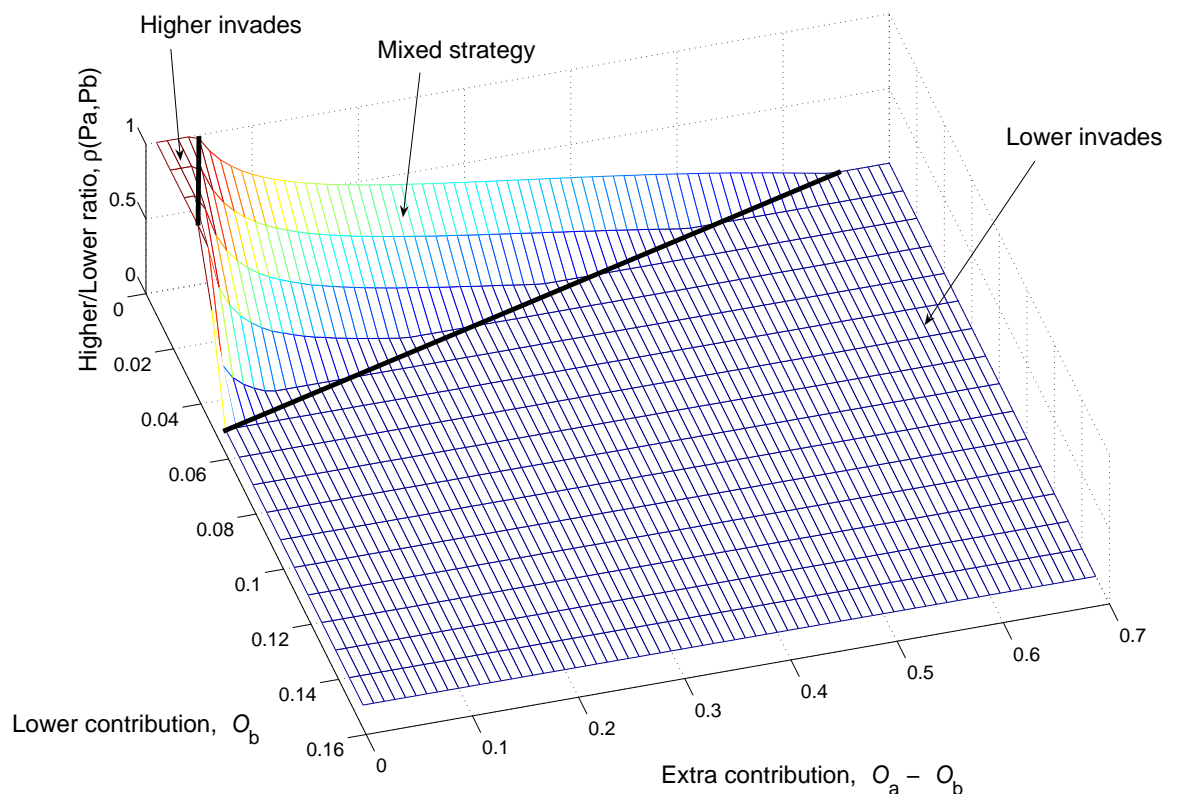


Figure 6.3: Plot of the game theoretic predictions, given by Equation (6.10), for the outcome of mixed populations of individuals with offspring resource contribution strategies Pa and Pb. Parents Pb donate lower resource contributions and parents Pa donate an extra contribution. Values used were: $R_c = 0.01$, $R_1 = 1$, $R_r = 0.1$, $m = 2$ and $u = u_0$.

The figure shows three regimes for Pa and Pb. As expected there is a large regime, covering most of the figure from the bottom right, where the lower resource contribution invades the higher one (Pb invades Pa). In the top left of the figure there is a region where the higher resource contribution is dominant. The rest of the figure shows a mixed strategy where there is a stable equilibrium between the two strategies – for example, when $O_b = 0$ and $O_a = 0.2$ there is a 50:50 mix of Pa and Pb..

One interesting prediction made by the graph is that the highest combined contribution, which is not invaded by a lower value, is a mixed strategy. Indeed, there are stable mixed equilibria when parents with strategy Pa contribute a large number of their resources, even as many as 0.6 or beyond.

To test the mathematical predictions, simulation runs were done starting with mixed populations of individuals which all had a two-parent strategy. Each simulation run took a pair of resource contribution values $(O_a, O_b)$ from Figure 6.3 (all other parameters were the same) with respective individuals from the two halves of the population taking one of the two values at the start of the simulation. There were no mutations to any values in the simulation. Resource units were scattered on a 100x100 grid at a rate of 100 each timestep maintaining a population of approximately 300 individuals. The fraction of individuals with each strategy was recorded at each timestep. Each simulation was run until either one strategy was completely dominant in the simulation, or 2 million timesteps had elapsed. The final fraction was recorded and is presented in Figure 6.4.

The figure shows that the results were very similar to those made by the mathematical prediction. However there are two obvious differences. Firstly at very low extra contribution values $(O_a - O_b < 0.01)$ the simulations do not show the complete invasion of Pa by Pb shown by the mathematical prediction. This may be because the relative growth rate was too small for the simulations to converge. The second difference was observed when $O_b = 0$: much higher values of $O_a$ were stable to invasion.

### 6.3.1.2 Evolutionarily stable strategy

The predictions from competitions between two parents from the previous section indicate that there may be a resource contribution strategy which is stable to invasion. There are two ways of calculating this value. First, one can calculate it by considering the outcomes of two player competitions between parents of all strategies. The second way is to use evolutionary simulations of populations of individuals with mutating resource contribution values.

Since I am only looking for the value of resource contribution which is stable to invasion, I do not need to be able to predict mixed strategies. This means I am able to consider the case where resources are fluctuating [Equation (6.11) doesn't give a prediction for mixed strategies]. Again, two parents Pa and Pb were assigned resource contribution values $O_a$ and $O_b$. Equation (6.11) can be used to calculate whether population Pa will dominate Pb over the values of $u^+$ and $u^-$. The value of $\rho^*$ is calculated over various values of $O_a$ and $O_b$ and the results are shown in Figure 6.5.

The figure shows that there is one value of $O$ that is stable to invasion from other
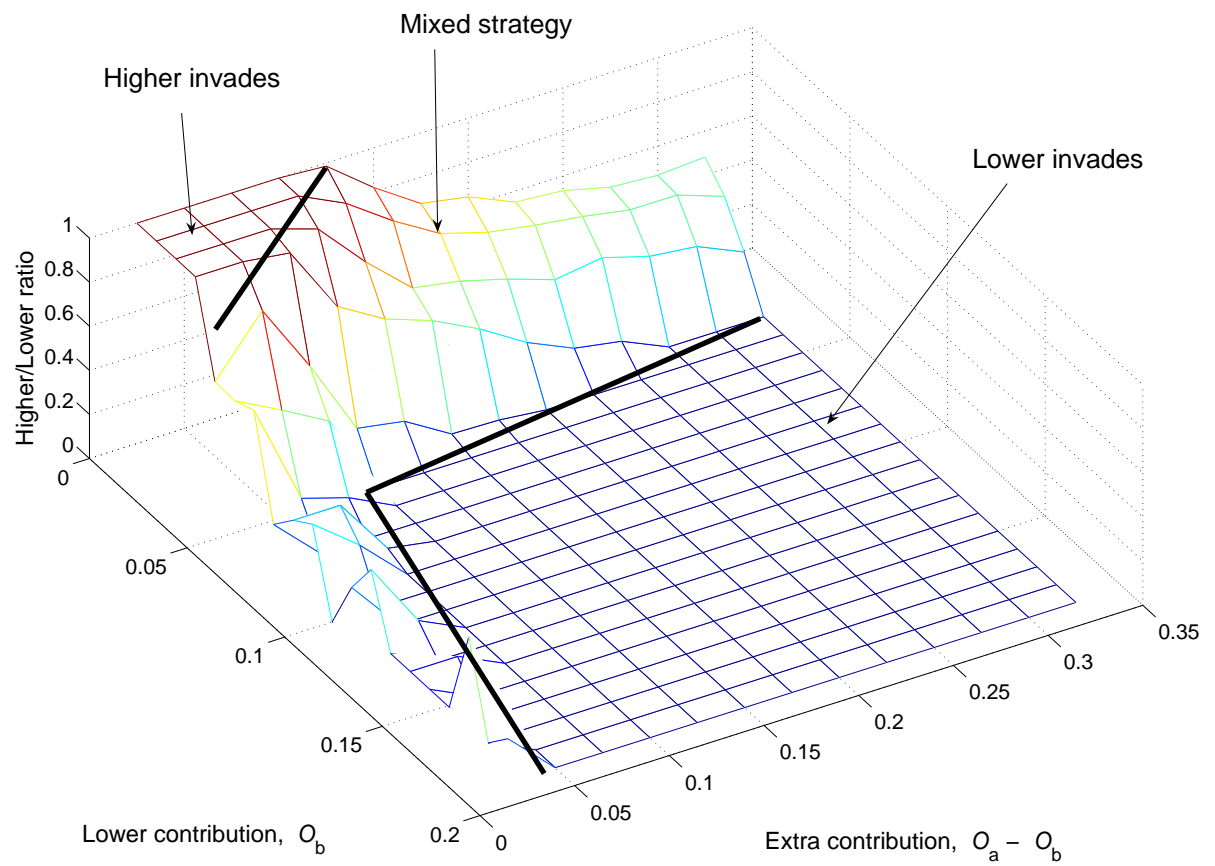
Figure 6.4: Plot of simulations to test the game theoretic predictions, given by Equation (6.10), for the outcome of mixed populations of individuals with offspring resource contribution strategies Pa and Pb. Parents Pb donate lower resource contributions and parents Pa donate an extra contribution. Values used were: $R_c = 0.01$, $R_1 = 1$, $R_r = 0.1$ and, $m = 2$.

values, I dub this value $O_{ess}$. When a population has a value of $O$ which is lower than $O_{ess}$ (to the right of $O_{ess}$ on the graph), it is invaded by individuals with higher values of $O$. Similarly, when a population has a value of $O$ which is higher than this value (to the left of $O_{ess}$ on the graph) it is invaded by individuals with lower values of $O$. Tests over a broad range of parameters confirmed that there was a stable value of $O_{ess}$ for each parameter set tested. However this method is quite slow so an exhaustive test was not done.

I use a faster algorithm for predicting $O_{ess}$ which can then be compared with the results from evolutionary simulations. This scans along the graph from right to left until it reaches $O_{ess}$. To do this I iterate through progressively larger values of $O$ (in steps of $\delta O = 0.001$). Each value of $O$ is compared using Equation (6.11) with the previous value until the higher value is no longer predicted to invade the lower value. The largest value of $O$ to invade $O - \delta O$ is the predicted value of $O_{ess}$. The predictions made were
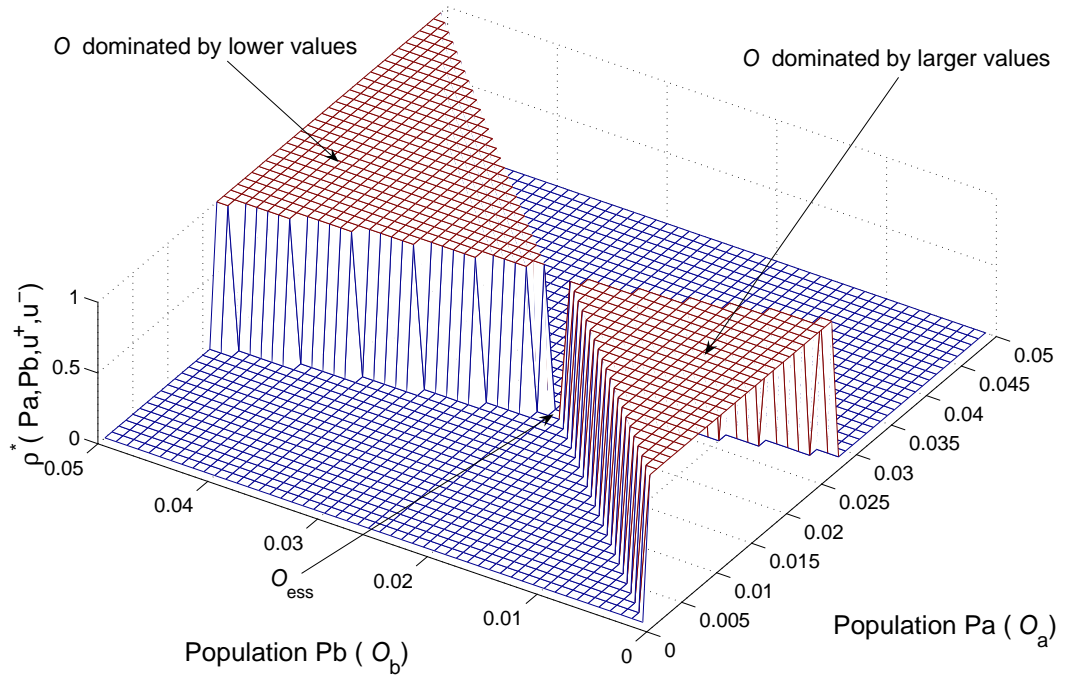
Figure 6.5: There is a stable resource contribution strategy at value $O_{\text{ess}} = 0.0220$. The predictions of Equation (6.11) are plotted for varying values of $O_a$ and $O_b$. Values used were: $R_c = 0.001, R_1 = 1, R_r = 0.1, m = 7, u^+ = 0.1375$ and $u^- = 0.1136$.

compared with evolutionary simulations using different parameter values of $R_c, R_r$ and $m$, over differently fluctuating resources.

Each simulation started with a population of 300 two-parent reproducing agents, the sparse resource level ($u = u^-$) was triggered by a population level above 400 and the abundant resource level ($u = u^+$) was triggered by population levels below 200. There was no one-parent reproduction at all in these simulations. Ten simulations were each run over 20 million timesteps. Two values of $R_c$ were tested, Figure 6.6 plots the predicted value of $O_{\text{ess}}$ together with the mean value of parental resource contribution $O$ (averaged over all simulations) for various different values of $m$ and resource fluctuation coefficients for $R_c = 0.001$ and Figure 6.7 plots the same for $R_c = 0.005$.

With this range of parameters, both figures show that $O_{\text{ess}}$ is not always accurate as a predictor for the mean value of $O$ in the simulated population. However the value of $O_{\text{ess}}$ is generally a good lower bound for the parental resource contribution. In Figure 6.6, out of the 55 points plotted, the mean value of $O$ from the simulation runs was less than $O_{\text{ess}}$ at only 6 points. A further 25 simulations run on each of these points showed that the erroneous simulation values were within the bounds of stochastic error. With the
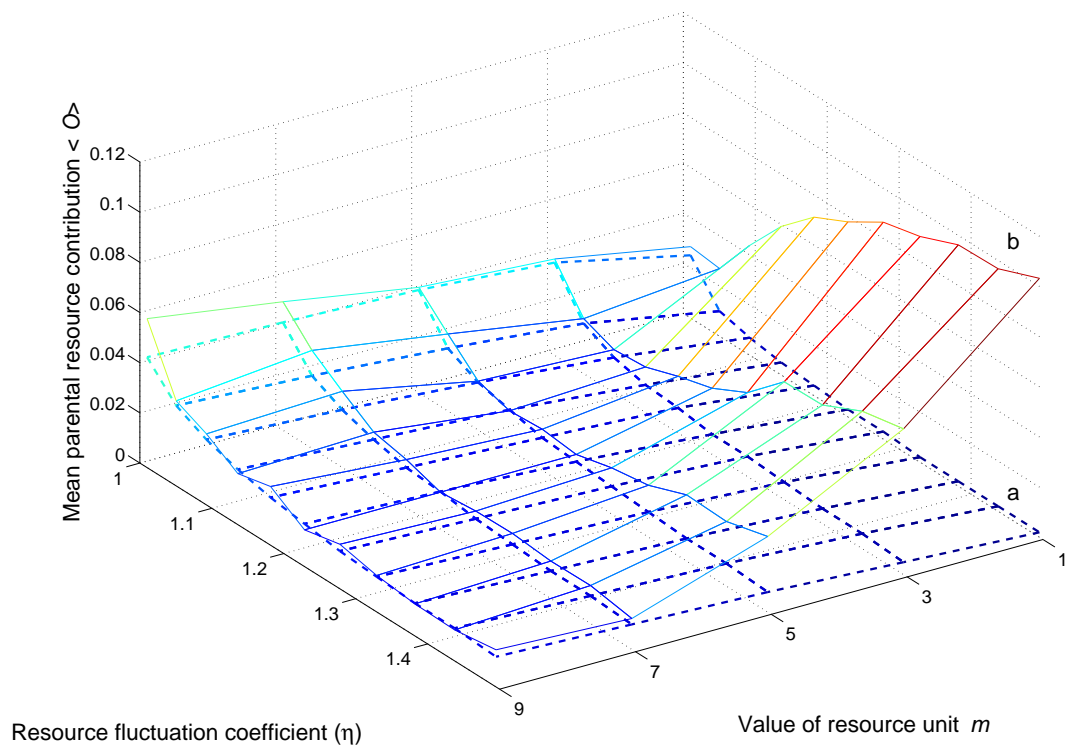
Figure 6.6: Parental resource contributions to offspring over different values of resource uptake units ($m$) and different resource fluctuation coefficients. Mesh **a** (dashed lines) shows the predicted value of $O_{ess}$ using Equation (6.11), mesh **b** shows the corresponding values found by the simulation runs. Other values used were: $R_c = 0.001, R_1 = 1$ and $R_r = 0.1$.

greater value of $R_c$, Figure 6.7 shows that $O_{ess}$ is still a good minimum value for the parental resource contribution at higher values of resource fluctuation. At lower values of resource fluctuation ($< 3\%$) and higher values of $m$ the simulation runs show markedly lower contribution levels.

Several simulations (normally 10) were run for each point in both of the figures and the standard deviation was calculated over these simulations for each point. The mean standard deviation for Figure 6.6 was 0.0057. The mean standard deviation for Figure 6.7 was 0.0076. Generally, the standard deviation was below 0.01 for all of the points, except when the resource fluctuation coefficient was at $\eta = 1.01$. For this case, the standard deviation was below 0.016 in Figure 6.6 and below 0.03 in Figure 6.7.

Within the simulations, the distribution (and the mean) of individuals with different resource contribution strategies depended on the mutation rate. With a higher mutation rate the mean increased. This was due to there being a fixed boundary at $O = 0$ so all perturbations increased the mean. Inspection of a histogram of the values of $O$ of individuals from many simulations (with different random seeds) of the same parameter set
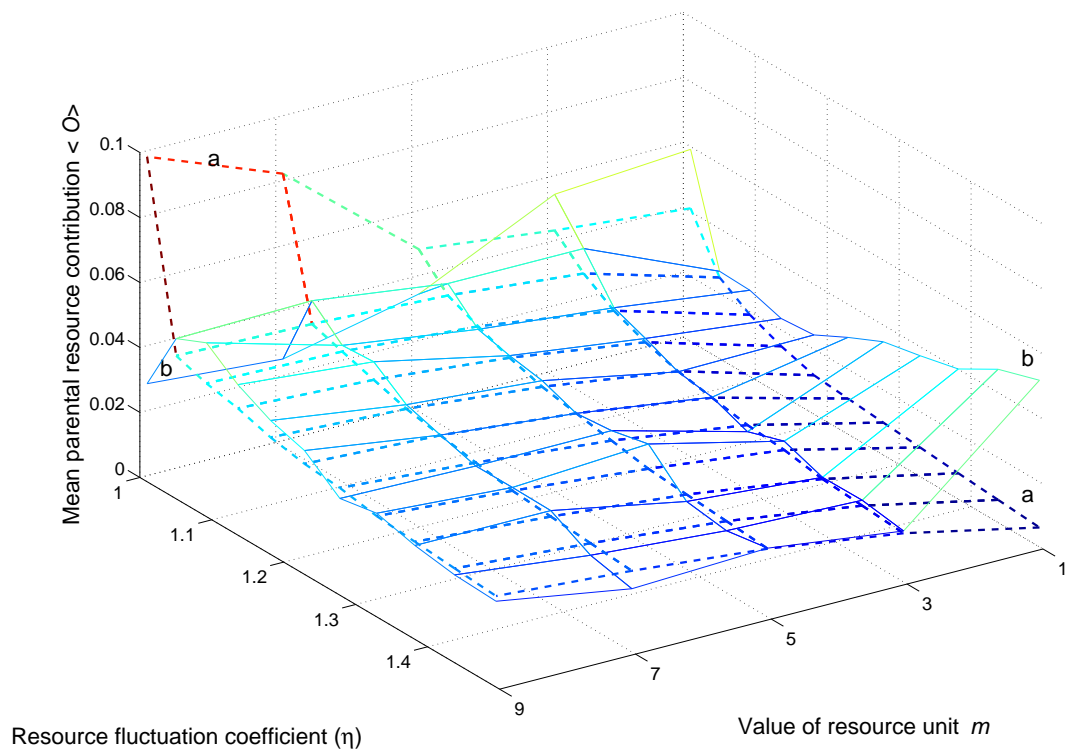
Figure 6.7: Parental resource contributions to offspring over different values of resource uptake units ($m$) and different resource fluctuation coefficients. Mesh **a** (dashed lines) shows the predicted value of $O_{\mathrm{ess}}$ using Equation (6.11), mesh **b** shows the corresponding values found by the simulation runs. Other values used were: $R_{\mathrm{c}} = 0.005, R_1 = 1$ and $R_{\mathrm{r}} = 0.1$.

($R_{\mathrm{c}} = 0.001$, $R_{\mathrm{r}} = 0.1$, $m = 5$, $\eta = 1.3$) revealed an exponential distribution (not shown).

While the mathematical prediction gives a good minimum value, when resource unit size is at a lower value ($m < 5$ in Figure 6.6 and $m < 2$ in Figure 6.7) and resource fluctuations are relatively high ($> 10\%$) then the simulations give significantly greater values. To check this wasn't due to the simulations taking a long time to converge, simulations were run on sample points over much longer time periods. These simulations showed little or no difference indicating that the values shown had fully converged.

Considering the impact of resource fluctuations both figures give similar results. The values of $O_{\mathrm{ess}}$ generated through mathematical prediction (mesh **a** in Figure 6.6) are higher when the amplitude of resource fluctuation is lower and decrease as the amplitude of resource fluctuation increases: the mean contribution in (over all $m$) decreases from 0.038 at low fluctuation values to 0.0074 at high fluctuation values. In the simulations the decrease of $O_{\mathrm{ess}}$ is evident as fluctuations increase, unless the value of $m$ is low (as already discussed).

## 6.3.2 One vs two-parent competitions

In the previous section I have outlined a method for predicting the stable amount of resources ($O_{ess}$) that parents may contribute to offspring. In this section I will look more closely at the number of resources a population with the two-parent strategy must contribute to offspring to dominate an optimal population with the one-parent strategy (which shares all its resources with its offspring).

### 6.3.2.1 Stable resources

To show how the growth rates of both populations are affected by changed values of $O$, I consider the growth rates of monomorphic one- and two-parent populations with different values of $O$ under stable environmental conditions. Considering the one-parent population, Equation (6.6) was used to generate population growth rates for different parental resource contributions. I look at the case where the population is close to its equilibrium level, i.e., $u = u_0$, so I use Equations (6.3) and (6.5). Different values of $O$ are plotted in Figure 6.8.
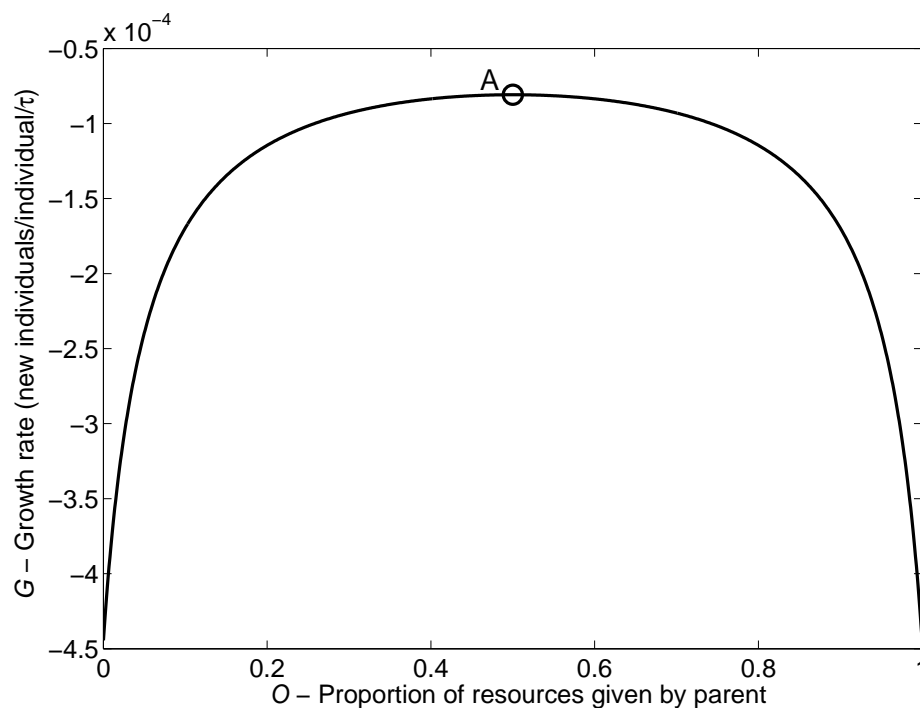


Figure 6.8: The growth rate of the one-parent strategy depends on the resource contribution factor $O$. The graph shows plots of the expected population growth rate $G$ for clonally [Equations (6.6)] reproducing individuals with varying resource contributions. The maximum growth rate of the one-parent population is marked as **A**. Parameters used were: $R_c = 0.01$, $R_1 = 1$, $R_r = 0.1$ and $m = 2$.

The ESS for the one-parent population is the maximum growth rate ($O = 0.5$). This is marked as point **A** ($G = -8.08 \times 10^{-5}$) on the figure. Any two-parent population that has higher growth than this value will dominate a one-parent population. To understand how the growth rate of the two-parent population changes with different values of $O$, I plot the growth rate predicted by Equation (6.7) [again using Equations (6.3) and (6.5)] in Figure 6.9.
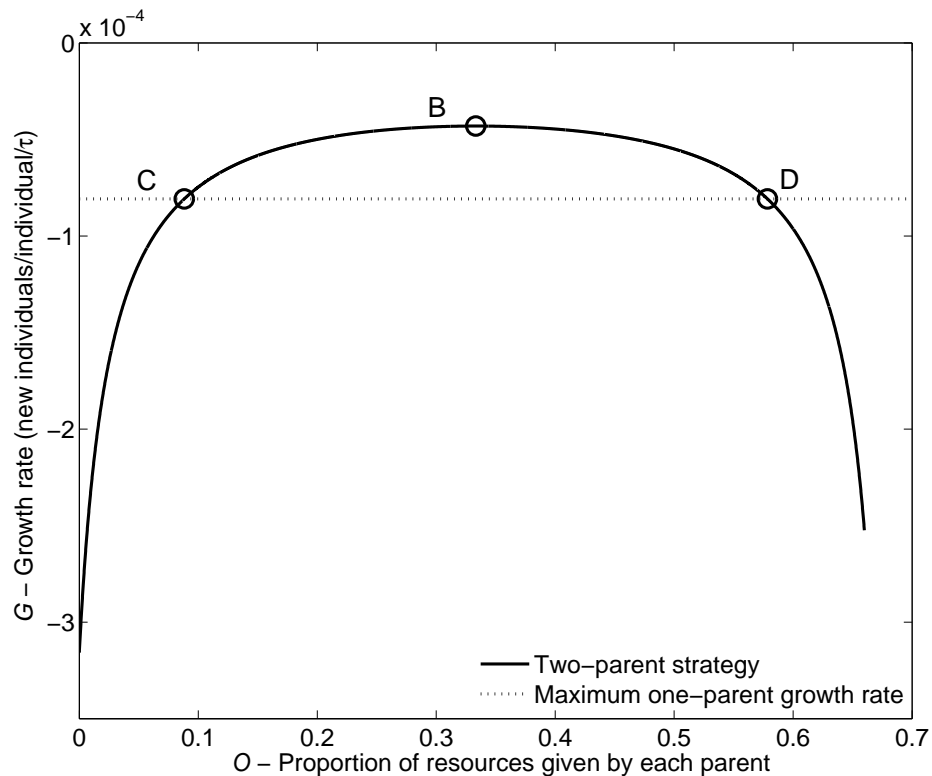


Figure 6.9: The growth rate of the two-parent strategy also depends on the resource contribution factor $O$. The graphs show plots of the expected population growth rate $G$ for two-parent [Equation (6.7)] reproducing individuals with varying resource contributions. The maximum growth rate of the two-parent population is at point **B** and the maximum one-parent growth rate is shown to enable comparison between the two strategies. The two-parent population can dominate if its resource contribution is greater than **C** and less than **D**. Parameters used were: $R_c = 0.01$, $R_1 = 1$, $R_r = 0.1$ and $m = 2$.

The figure shows that the two-parent population will have a greater growth rate than any one-parent population, provided its resource contribution lies between points **C** ($G = -8.08 \times 10^{-5}$, $O = 0.0882$) and **D** ($G = -8.08 \times 10^{-5}$, $O = 0.5784$). The two-parent growth rate function [Equation (6.7)] is only shown between $O = 0.0$ and $O = 0.68$. For values of $O > 0.68$ the total resources contributed to new offspring is greater than $R_1$.

At the value of $u$ used to generate the growth rates in Figures 6.9 and 6.8 all growth

rates are below zero. While individuals receive as many resources per timestep as they expend on living cost ($R_c$), this does not account for the cost of reproduction occasionally expended when an individual reaches its upper threshold. Different values of $u$ were tested using Equations (6.2) and (6.4). Similar results were obtained, however as the value of $u$ was increased the value of $O$ at point **C** (the point above which the individuals with the two-parent strategy must donate to dominate a one-parent population) shifted to the right. When the value of $u$ was decreased, the value of $O$ at point **C** decreased. Since variations in $u$ are important, in the next section I consider how one- and two-parent strategies perform under fluctuating resource conditions.

### 6.3.2.2 Fluctuating resources

The results in the previous section indicate that dominance of the two-parent strategy may be determined by the value of $O_{\text{ess}}$, but this depends on the level of resource availability. When resource availability fluctuates between two values ($u^+$ where the population grows and $u^-$ where the population declines), Corollary 3 from Appendix A can be used to show which strategy will dominate. The two-parent strategy will dominate when

$$\Psi_{\text{two}} > \Psi_{\text{one}} \, .$$

The value of $\Psi_{\text{two}}$ is calculated using the value of $O_{\text{ess}}$ (see Section 6.3.1.2) and this is compared with the maximum value of $\Psi_{\text{one}}$ (tested over various values of $O$ from 0 to 1 in steps of 0.01) in Figure 6.10.

The figure shows that the two-parent strategy will dominate when the amplitude of resource fluctuations is high enough. In this case, the resource fluctuation coefficient must be higher than 1.1 (where the line for the two-parent strategy crosses the line of the one-parent strategy). This indicates that there is a minimum value of resource fluctuations needed for dominance of the two-parent strategy.

To confirm this prediction that higher levels of resource fluctuations are necessary for dominance of the two-parent strategy I have tested it over a wide variety of parameters with both the mathematical analysis used in Figure 6.10 and simulation runs of mixed populations of individuals with the one- and two-parent strategies.

Each simulation started with a population of 300 agents. The resources introduced into the environment flipped between two states: the sparse resource state ($u = u^-$) was triggered when the population level went above 400 and the abundant resource state ($u = u^+$) was triggered when the population levels went above 200. All individuals started with a random resource level generated from a flat distribution between 0 and $R_1$. The repro-
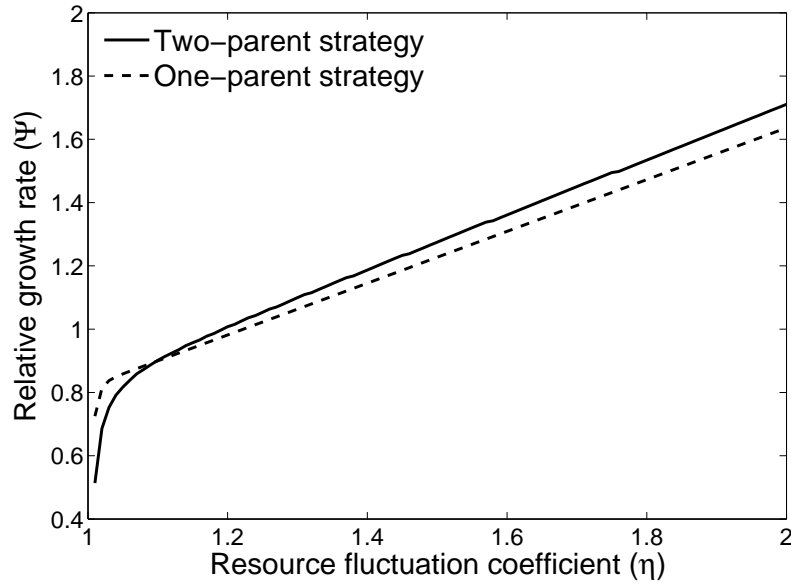
Figure 6.10: As the amplitude of resource fluctuations increases, the two-parent strategy starts to dominate the one-parent strategy. Parameter values used were: $R_c = 0.001, R_1 = 1, R_r = 0.1$ and $m = 7$.

duction strategy of individuals (the resource contribution $O$ and whether the individual reproduced with the one- or two-parent strategy) was defined by its digital chromosome as outlined in Section 6.2.3. At each time step the fraction of individuals which reproduce with another parent to those which reproduce alone was recorded. The simulation was stopped when the population was dominated completely by one of the reproduction strategies, or 10 million timesteps had elapsed. The final fraction of individuals with the two-parent strategy was recorded for 10 simulations run with the same parameters but with different random seeds.

Simulations were run over varying values of $R_r$ and $m$. The resource fluctuation coefficient ($\eta$) was set at one of the values: [1.0010 1.0200 1.0394 1.0592 1.0793 1.0998]. The results all showed that the one-parent strategy was dominant at lower resource fluctuation amplitudes and the two-parent strategy was dominant at higher resource fluctuation amplitudes.

Mathematical predictions were also made by using Equation 6.9 over the same range of values of $\eta$. As with the single case of Figure 6.10, the value of $\Psi_{two}$ was calculated using the value of $O_{ess}$, see Section 6.3.1.2. This was compared with the maximum value of $\Psi_{one}$, calculated over a range of values of $O$ from 0 to 1 in steps of 0.01. In harmony with the results from the simulations, the mathematical model demonstrated that the one-parent strategy would dominate at lower values of $\eta$ while the two-parent strategy dominated at

higher values.

To compare the results of the mathematical treatment with the results of the simulations, I calculate the minimum resource fluctuation amplitude required for dominance of the two-parent strategy – I dub this parameter $\eta_{\min}$. For the mathematical treatment this is the smallest value of $\eta$ where $\Psi_{\text{two}} > \Psi_{\text{one}}$ (calculated over a range of values of $\eta$ from 1.0001 to 2.0 incremented by 0.0001). For each simulation run, $\eta_{\min}$ is the smallest value of $\eta$ where the fraction of individuals with the two-parent strategy was greater than 0.9. Mean values of $\eta_{\min}$ are plotted in Figure 6.11 for the different values of $m$ (each mean is over a range of values of $R_{\text{r}}$). Similarly, mean values of $\eta_{\min}$ are plotted in Figure 6.12 for the different values of $R_{\text{r}}$ (each mean is over a range of values of $m$)
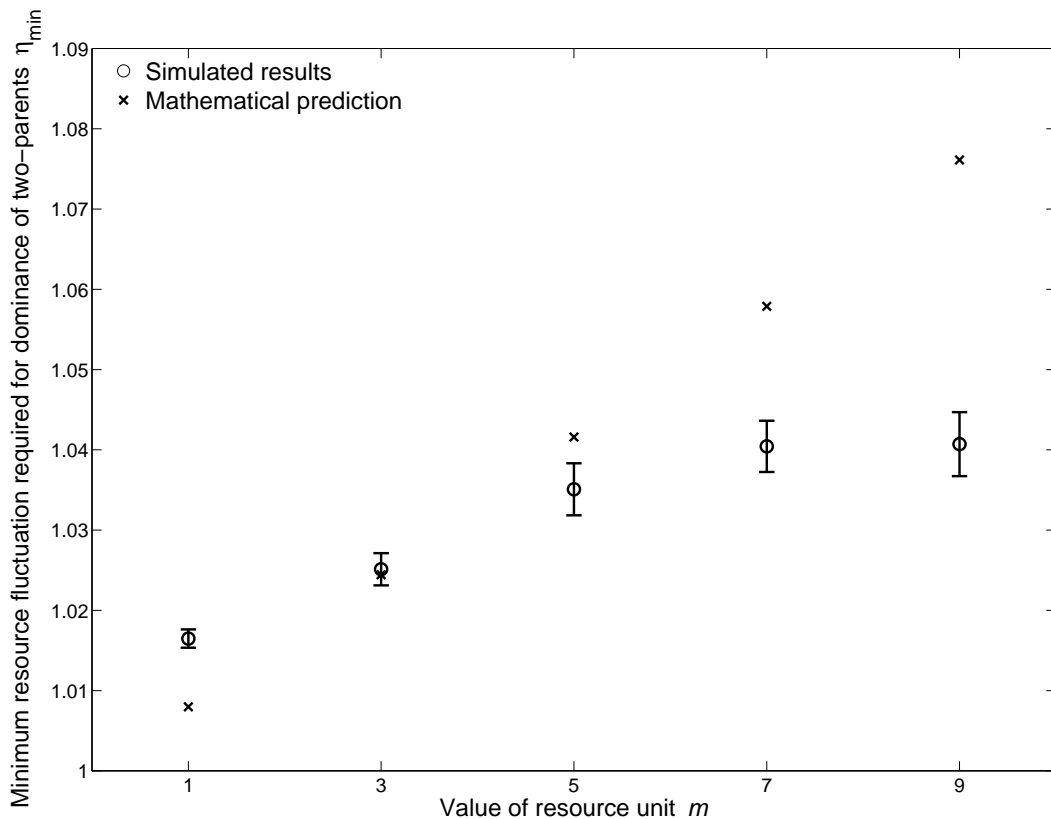


Figure 6.11: Plot of the minimum resource fluctuation coefficient ($\eta_{\min}$) required for dominance of the two-parent strategy over the one-parent strategy. Each point is an average over a range of values of $R_{\text{r}}$, the standard error is shown in the error bars. The cost per timestep $R_{\text{c}}$ was 0.001.

The results from both the mathematical predictions, and the simulations, show similar trends. The value of $\eta_{\min}$ is positively correlated with the value of $m$ in both the mathematical predictions and the simulations (Figure 6.11). The value of $\eta_{\min}$ is negatively correlated with the value of $R_{\text{r}}$ in both the mathematical predictions and the simulations
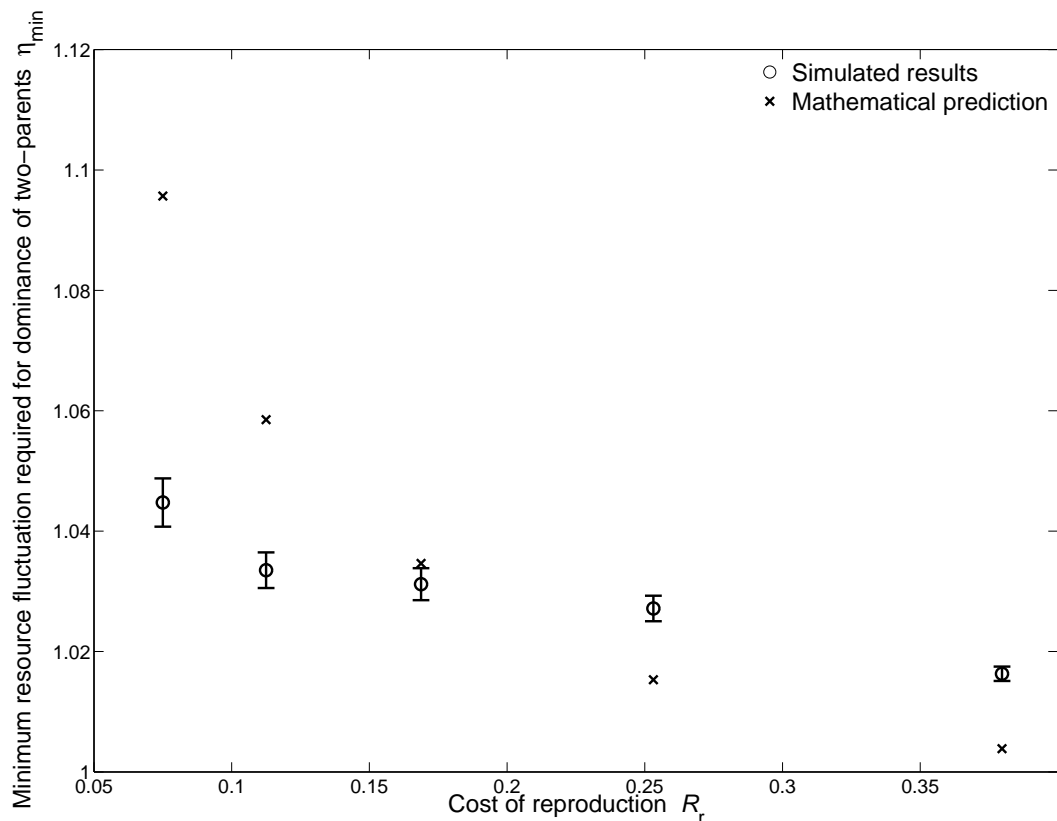
Figure 6.12: Plot of the minimum resource fluctuation coefficient ($\eta_{min}$) required for dominance of the two-parent strategy over the one-parent strategy. Each point is an average over a range of values of $m$, the standard error is shown in the error bars. The cost per timestep $R_c$ was 0.001.

(Figure 6.12). The results from the mathematical treatment diverge strongly from the simulations at higher values of $m (> 7)$ and lower values of $R_r (< 0.15)$.

## 6.4 Discussion

The contribution, toward explaining the origins of sexual reproduction, of the model presented in this chapter is that it demonstrates the plausibility of the theory that two-parent reproduction may dominate due to the way parents allocate resources to their offspring. The model is a simple one, merely considering the most basic aspects of reproduction – individuals take up resources from the environment, produce offspring and share some of their resources with their offspring. The model does not require any other secondary benefits of sexual reproduction such as genetic mixing or division of labour. It compares like for like with all individuals being the same in every respect except for the way they reproduce.

The model presented in this chapter compares the two-parent reproductive strategy with the one-parent reproductive strategy. It demonstrates, in line with arguments based on that evidence from the biological literature (see Section 6.1), that unpredictable resource fluctuations (due to biotic stress) are significant in determining whether an individual should reproduce with another parent or not. If a two-parent strategy will dominate the one-parent strategy then this should explain why hermaphrodites will not invade a population of individuals that selfishly contribute resources to offspring. Why there is an imbalance in resource contributions between males and females is therefore a separate question, but is unlikely to be explained by simple consequences of two-parent reproduction such as genetic mixing.

In the next section I give a summary of the model and its results. I then review the results in light of the argument concerning the cost of sex. The following section includes a discussion of the application and relevance of the model within biology and other fields. Finally, I outline some of the limitations of the model.

### 6.4.1   Summary of the model and results

While the major result of this work does show that the two-parent strategy can dominate, the results also predict that several factors are significant. These are:

(i). Resource fluctuations.

(ii). The cost of reproduction ($R_{\mathrm{r}}$).

(iii). Constraints on resource contributions to offspring; (iv) the value of the resource uptake unit ($m$) .

(iv). The amount of resources used in one timestep ($R_{\mathrm{c}}$).

In this section I consider these factors in more detail.

The first factor identified by the model as significant is resource fluctuations. To explain why they are significant, I turn to Figure 6.10. This demonstrates that the one-parent strategy is more vulnerable to increases in the amplitude of resource fluctuations increases than the two-parent strategy. Since individuals with the two-parent strategy contribute less resources to offspring, they maintain a higher internal level of resources and are less vulnerable to periods of low resources. These mathematical predictions give qualitatively similar results to those generated by simulations (see Figures 6.11 and 6.12).

The cost of reproduction ($R_{\mathrm{r}}$) is an important factor as shown by Figure 6.12. As the cost of reproduction increases, collective reproduction becomes increasingly viable. This

is not surprising as one of the main features of the model is that, in two-parent reproduction, the two parents can share the cost of reproduction. I have considered the introduction of an extra cost of collective reproduction in Chapter 4. This indicates that two parents may still dominate even when the upfront cost of sexual reproduction is greater than the up-front cost of one-parent reproduction. Further modelling will confirm if this is still the case when two parents contribute less resources to their offspring.

The value of the resource uptake unit from the environment ($m$) was shown to be significant in the simulations (see Figures 6.6, 6.7 and 6.11). Since, in this model, resources are encountered in discrete units, in one timestep an individual will either increase its resources by a fixed amount or decrease by a smaller amount. The ratio between these two values is set by $m$. Assuming that the mean level of resources over realisations is the same, a higher value of $m$ will mean there is a greater variance over realisations. This means that strategies that have very low levels of resource are especially vulnerable over a short time-scale. As the value of $m$ is increased, two-parent reproduction therefore becomes less viable (see Figure 6.11).

As far as parental contributions are concerned, the effect of increasing $m$ can mean that resource contributions from two parents can increase (see Figure 6.7). Higher values of $m$ mean that there is an increased variance of resource supply (over a short time-scale) and therefore a harsher environment. This harsher environment means that parents must contribute more to offspring. When the resource fluctuation coefficient ($\eta$) is at a higher value and resource availability fluctuates over a longer time-scale, the opposite is true: lower values of $m$ mean that parents contribute more to their offspring, especially when the cost of maintenance is low (see Figure 6.6). Perhaps when the amplitude of fluctuations is higher, the high variance can be beneficial. Since all offspring are vulnerable, a higher variance of resources over the offspring can mean that one of the offspring may survive – so the higher value of $m$ can make these conditions less harsh. Further analysis should be done to investigate this further.

The final factor of importance was the resources used per timestep ($R_c$). This was shown to be significant in comparisons between the mathematical treatment and simulations in Chapter 3. It effectively describes how discrete or continuous the model is. Only two different values were used in the simulations presented in this chapter – one that is known to be accurate with the ranges of $m$ used ($R_c = 0.001$ and $R_c = 0.005$). Further investigations are needed to simulate over many different values of $R_c$ to fully understand the role of this parameter.

## 6.4.2    The cost of sex?

The traditional argument concerning the cost of sex can be considered in light of the results presented here. The argument, as presented in the introduction, states that each sexually reproducing parent will only receive half an offspring each time it reproduces rather than the whole offspring produced by an asexually reproducing parent. This is reflected in the model in this work, however the model here goes further in that it analyses growth rates during abundant and sparse resource periods. As with the model in the previous chapter, the growth rate of individuals with a one-parent strategy is faster than those with a two-parent strategy during abundant resource periods. However, when resources are sparse, the one-parent strategy declines at a slower rate. The model predicts that, under some circumstances, this slower rate of decline means that the two-parent strategy is advantageous over the one-parent strategy.

At the very least, the model indicates that the traditional arguments concerning the cost of sex need reviewing. The original cost of sex argument is simple. The model presented here takes the same original premise that sexually reproducing individuals receive half as many offspring as asexually reproducing individuals. Equation 6.9 (along with Theorem 2 in Appendix A) can be used to predict when the slower reproducing two-parent reproductive strategy is more advantageous. It shows, by simply analysing population flux rates over both growth and declining phases, that there are benefits to reproducing collectively even when one only receives half of an offspring. This indicates that sexual reproduction may not be that problematic (or even paradoxical) to explain.

The extreme variant of the cost of sex argument argues that when males contribute almost no resources to their offspring, there is an obvious cost to the population to producing them with no obvious benefits. While such cases are not analysed in any detail in this work, two points can still be made. Firstly, the model predicts that the value $O_{ess}$ is a lower limit to the number of resources individual can contribute. When all individuals contribute $O_{ess}$, the lineage of an individual that contributes less resources will be inferior. This indicates that since males contribute their genes to offspring, it must be worthwhile for them to contribute resources as well. The model predicts that in most cases males should make some contribution. The second point is to note that there may be a stable strategy with males that contribute very little but in general the two parents contribute enough to survive: inspection of Figure 6.3 shows that such a strategy may be plausible.

One explanation of the origin of males concentrates on the relative sizes of gametes (Parker, Baker and Smith 1972, Randerson and Hurst 2001). This argues that anisogamy (where gametes are of different sizes) may be explained because there are two extremes for fitness of gametes. There are opposing selection pressures between producing a large

number of small vulnerable gametes or a small number of large gametes which have increased chances of survival. Unfortunately, the models of parental contribution in Section 6.3.1.1 showed an exponential distribution for resource contribution levels. For the model to show different selection pressures for large and small gametes, one would expect two humps in the distribution. Further investigations are needed to develop a model which might reflect better the development of gametes.

Finally, I consider the case where a colony of females that produce non-contributing males (i.e., $O = 0$) has an inferior strategy to an parthenogenetic female. If this colony is being invaded by hermaphrodites, a female that produces contributing males ($O = O_{\text{ess}}$) will resist such an invasion. The contributing males, spreading this 'contributing' gene. Further

While the model does show that it is plausible that two-parent reproduction of the type modelled may well have been significant in the origins of sexual reproduction, there are some caveats and further issues that must be addressed. The model is abstract and does not currently capture many of the features of biological systems, e.g., sexes. Furthermore, it does not show that sexual reproduction is always optimal; the conditions in which it is and is not optimal are discussed in more detail in the next section.

## 6.4.3 Biological applications of the model

The most appropriate biological analogue to the model is sexual reproduction in metazoa. The model is less appropriate for analysing sexual reproduction in protozoa or plants. Protozoa are single celled organisms which, when they sexually reproduce, fuse themselves with their sexual partner. Sexual reproduction in plants is also difficult to explain because it is practically impossible for a male flower to contribute anything more than a small amount of DNA to offspring produced with a female plant. Further modelling is necessary to investigate these two interesting problem domains.

The work identifies several important factors that are significant for the dominance of a two-parent strategy over a one-parent strategy. These are listed in Section 6.4.1. These factors are now discussed in order, with metazoan reproduction in mind.

The causes of resource fluctuations in biological systems are varied. The two general reasons outlined in the introduction are down to biotic and abiotic stress. Abiotic stress is generally caused by environmental conditions such as daily and seasonal cycles. Biotic stress is caused by changes in the environment of prey and competitors. It has been hypothesised (Glesener and Tilman 1978) that biotic stresses are more unpredictable than abiotic stresses and will be more likely to cause periods of abundance and starvation for

the organisms. It is not possible to develop simple behavioural strategies to minimise the effects of unpredictable resources due to biotic stress. This can lead to population increases and declines such as those modelled in the work in this chapter. In line with the biological data outlined in Section 6.1 the model (see Figures 6.10, 6.11 and 6.12) predicts that stresses that can cause large population increases and decreases can lead to two-parent reproduction being an optimal strategy. However, when unpredictable stresses are low, parthenogenesis is more likely to be observed.

The upfront cost of reproduction was found to be a significant factor in the model – a greater upfront cost of reproduction means that two-parent reproduction (where the costs can be shared to some extent) will be more likely to dominate. For a metazoan the cost of reproduction is not merely the cost of producing gametes and fusing them however. The cost of reproduction must include all resources expended toward making that offspring a fully functioning copy of the parents and able to fend for itself. With single celled organisms this process is relatively simple but for multicellular organisms it is complex and costly. Further research is needed to consider whether the cost of reproduction is indeed significant in many sexually reproducing metazoan systems.

In my model, an individual may contribute further resources, on top of the upfront cost of reproduction, toward its offspring. Looking at biological examples, it is hard to see how many metazoa, especially egg laying metazoa, can really find a way to make much extra contribution however. Metazoa generally cannot split their whole body or body parts into two halves in the way single celled organisms can. It is also time consuming and dangerous for parents to spend too long nursing offspring. Therefore, constraints to the levels of resource contributions to offspring seem reasonable. However, further investigation is required to see if this factor is actually important in the origins and maintenance of sex.

The values of the resource uptake unit ($m$) are also shown to be significant. As outlined in Section 6.4.1, this variable affects the variance of resource uptake on a short timescale (within the lifetimes of individuals). The predictions are that parental resource contribution is generally greater when variance is higher, except when there are large fluctuations on a long timescale (outside the lifetimes of individuals). These predictions should be compared with empirical data.

## 6.4.4   Limitations of the model

One limitation of the model is the fact that it does not consider what happens when there is an increased cost of two-parent reproduction. This might represent the costs of mating and locating partners. In fact the relationship between an extra up-front cost of reproduction

and the viability of reproduction between two parents was discussed in Chapter 4. The analysis in that chapter showed that two-parent reproduction can still be viable when this extra cost is as much as 1/3 of the total cost of reproduction. Further work is needed to incorporate an extra upfront cost of reproduction into the model presented in this chapter.

The model does also contain some biologically unrealistic assumptions regarding the life histories of individuals which may be significant. Specifically, the model does not include aging, and reproduction happens instantaneously. Aging individuals might contribute more resources to offspring (since investment in itself has a lower payoff). Conversely, an individual running low on resources may stop a reproductive effort, or restrict resource contributions to offspring. Both of these factors could be considered in future modelling work.

# Chapter 7

# Conclusions

The main topic of this thesis is the explanation of the major evolutionary transitions. I ask why an individual that reproduces on its own might evolve to one that reproduces as part of a larger group. Rather than following the dominant current theoretical approach (for at least the later transitions), inclusive fitness, the models I have presented (in Chapters 3, 4, 5 and 6) show the potential of an alternative theory. This is that an individual placed in an unpredictable and finite environment may hedge its reproductive output by investing in a higher reproductive process.

This principle of hedging one's reproductive output is demonstrated in several models in the thesis. First, in the simplest model, I look at individual reproduction in Chapter 3. This shows how an individual may sacrifice its fecundity (and so reduce its direct fitness) so that it is less vulnerable to fluctuations in resource availability. This recreates results from another model (Tuljapurkar 1990a) using a different mechanism which is relevant to questions concerning the allocation of resources to offspring in variable environments. The chapter shows how there is a trade-off between extra costs of maintenance and reproduction which may be incurred from maintaining higher resource levels.

The model in Chapter 3 is not directly relevant to the major evolutionary transitions, however the basic principle that an individual should reduce its fecundity to make itself less vulnerable is shown to be relevant to the transitions to multicellularity and eusociality in the next chapter, Chapter 4. This shows how an individual can be less vulnerable if it reproduces collectively rather than reproducing on its own. The chapter shows how there is a trade-off between the number of parents and the collective up-front cost of producing new offspring – this may be important in explaining why most multicellular animals are sexual while a few are eusocial.

Most importantly, Chapter 4 provides a proof of concept showing how an individual may benefit from investing in a higher reproductive process. In fact, regarding the dis-

cussion on the increase in individual complexity in Section 1.1, I have argued that such investment is accretive (see Section 4.3) as investment in a higher reproductive process can happen in many different ways. Thus, the models indicate that there may be more than just random drift (as proposed by Gould) at play in the evolution of the complex individual.

The transition to multicellularity is discussed in more detail in Chapter 5. Based on the behaviour of the slime mould *D. Discoideum*, the chapter considers the case where individuals have very low resources and are close to dying. They may then hedge their last few resources by gambling them against a large reproductive pot. Since the offspring are genetically homogeneous, this model shows how there can be a transition from non-aggregating genetically heterogeneous organisms into aggregating genetically homogeneous organisms – I argue that this is an important first step in the transition to multicellularity.

Finally, I consider the case where individuals may be more selfish in the amount of resources they contribute to their offspring. Based on the model in Chapter 4, the model in Chapter 6 addresses the transition to sexual reproduction in more detail. It relaxes the assumption requiring individuals to share resources equally with their offspring. The sexual reproduction strategy is still viable when the amplitude of resource fluctuations is increased.

## 7.1   Social evolution

In this section, I consider how the work I have presented contributes to social evolutionary theory. In general terms, the models presented here all demonstrate cases where individuals hedge their direct fitness for some later benefit. I dub this *hedged fitness*. Since an individual must reduce its fitness, this is a social, or cooperative, act. Table 7.1 has a summary of the models presented in this thesis demonstrating how they can be interpreted in terms of cooperation.

Given that the models here have shown the potential of hedging of reproductive bets, I suggest that social evolutionary modellers should consider whether the more general fitness hedging is an important factor in their models. Further to this, I suggest that the comparison of growth/decline rates using Theorem 2 in Appendix A may be applicable in future models.

Theorem 2 and the more simple Corollary 3 in Appendix A (and illustrated in Figure 1.2) encapsulate the basic principles of hedged fitness. Basically they show that, given that there is no overall growth in population, a lineage with cooperative genes will

| Chapter | Uncooperative individuals | Cooperative benefits |
|---|---|---|
| 3 | Reproduce more quickly (and monopolise resources) | Reserve resources so less vulnerable to low resource periods |
| 4 | Reproduce on their own | Save resources through sharing reproductive investments, also less vulnerable |
| 5 | Doesn't join the macrocyst | Avoids harsh season and has a better chance of reproducing |
| 6 | Very low resources invested in offspring | Increased investment is a better individual strategy when total investment is very low |

Table 7.1: Non cooperative behaviour in the models in this thesis and the advantages of cooperating.

dominate if the ratio of its growth rate to decline rate is greater than that of individuals with uncooperative genes. Thus, an investment in a social institution may be profitable if there is some later payback – even outside the lifetime of the organism making the investment. In the next section I compare the idea of hedged fitness with the current social evolutionary theory of inclusive fitness and the current bet hedging theory.

## 7.2 Comparison with other theory

Two important modelling directions outlined in Chapter 1 were inclusive fitness theory and bet hedging theory. In this section I compare the concept of hedged fitness presented here with those other perspectives.

In inclusive fitness theory (see Section 1.3.1.1), an individual will reduce its (Hamiltonian) fitness if it will benefit another related individual in the same population. As outlined in Section 1.4.3, it seems unlikely that this theory will explain why an individual will invest resources in a related individual's offspring and not its own 100% related offspring. The benefit would have to far outweigh the costs. Conversely, in the hedged fitness models presented in Chapters 4, 5 and 6, individuals don't reduce their fitness so that another individual in the population will benefit. Instead I have shown that if they invest indirectly in their own offspring through a higher reproductive process, then this strategy can be better than reproducing individually.

The perspective outlined in Section 1.5 advocates a different approach to fitness. Since fitness must be calculated over a variety of different environments, it is the geometric mean growth rate over these many environments that is important. Interestingly, this rate can be seen as the fitness of a complete lineage. Thus, lineages should be compared with each other through measuring invasion fitness, not through measuring the growth rate of

individuals in one environment.

From this perspective, combined with the theory put forward in this thesis, we can see how one lineage may dominate another, but yet always have a lower fecundity. If we term describe lineages as groups, this is effectively demonstrates how group selection can work in circumstances position that has long been argued against [e.g., by Williams (1966a)]. The best lineages/groups are selected for based on their performance in many environments.

The long-run growth rate, outlined in Section 1.5.1, of an organism can be calculated by using the geometric mean. Theorem 2 (and Corollary 3) in Appendix A is compared with the geometric mean approach in Section 3.4.2. Essentially the key difference is that Theorem 2 models the population under density dependence – which can have greater biological realism, including when considering the conditions under which the major evolutionary transitions occurred. This means that, rather than picking growth rates from a distribution and multiplying them together, Theorem 2 predicts with certainty that one lineage may dominate another when both are under density dependence.

## 7.3 Future directions

The discussion in the previous section of the differences between hedged fitness and inclusive fitness does not show that the two theories are incompatible. Hedged fitness is concerned with the transfer of fitness to copies of one's genes in the future and inclusive fitness is concerned with the transfer of fitness to copies of genes in the present. Hedged fitness demonstrates why one may invest in a higher reproductive process. A plausible higher reproductive process is investment in a related individual's offspring. Investigations into this would require some unification of inclusive and hedged fitness. It may give important new insights into the origins and maintenance of eusociality and multicellularity and perhaps some of the other major evolutionary transitions.

The discussion of social evolution in Section 7.1 implies that fitness hedging (perhaps allied with inclusive fitness) may have significance in the transition to society (see Table 1.1). It seems plausible that there may be some benefit to investing in social institutions which pay off in the long term. A systems perspective to the modelling of societies has been advocated (Epstein and Axtell 1996, Silverman and Bryden 2007). It was argued that by studying artificially grown societies (*in silico*), we may be able to understand our own social systems better. Fitness hedging may give some important insights into the methods we may require to successfully grow societies *in silico*.

Extensions to the modelling work on individual reproduction in Chapter 3 mainly in-

volve making the models more biologically realistic. This might involve making models of the growth of single celled organisms, perhaps in a chemostat. An obvious further extension would be to investigate the fecundity of multicellular organisms, applying the sexual growth equations from Chapter 6 (and analysing the effect of changing the reproductive threshold $R_1$). Such models would lend themselves well to the inspiration for, analysis of, and comparisons with empirical investigations.

The purpose of Chapter 4 was to show the viability of a model of collective reproduction. To develop this approach, it would be interesting to model how artificial organisms may reproduce collectively and what increases in complexity may be gained from doing this. It may be possible to show a major evolutionary transition *in silico* – where individually reproducing agents come together to form a larger unit and reproduce as part of this larger unit.

One problem with the work modelling *D. Discoideum* in Chapter 5 is that the whole system may collapse due to cheating individuals which, while they broadcast cAMP, do not aggregate and instead wait for others to join. An investigation of this would improve the model. Slime mould is not the only organism to form macrocysts, perhaps a similar modelling approach may be used to investigate the formation of biofilms in bacteria.

The most obvious extension to the work on modelling sexual reproduction (in Chapter 6) is the inclusion of a model of two different sexes. This would question if there was any advantage to having two different mating types which invest different amounts of resources in their offspring. Further to that, I have not investigated, in detail, the case where an asexual parent has an upper limit to the amount of resources it may contribute to its offspring. It is likely that limiting the resources asexual parents may contribute will mean that the sexual strategy is more likely to dominate.

The model of sexual reproduction I have presented is focused mainly on the origin of sex in metazoa. Another interesting extension would be to consider the origins and maintenance of sex in plants and unicellular organisms. The origins of sex in unicellular organisms is close to the macrocyst formation in Chapter 5 – perhaps such a model could resolve why multiple individuals come together to form a macrocyst rather than forming individual microcysts.

## 7.4   Conclusion

For this thesis I set out to develop models that would explain the major evolutionary transitions. While I have fallen short of this extremely ambitious goal, the work I present here does take some important steps. At its core, the work addresses what I have argued

to be a central problem of the major evolutionary transitions – why might an individual contribute toward a higher reproductive process, rather than invest in its own immediate reproductive success?

The answer I have given to that question is that an individual can increase its long term fitness by hedging its short term fitness through investing in a higher reproductive process. I have studied the transitions to sex and multicellularity in more detail here, showing that the collective strategy can be beneficial – especially when fluctuating conditions mean populations can rise and fall.

While I have only considered two transitions in biological detail, it looks promising (see Section 7.3) that this theory may well be applicable to other major evolutionary transitions. Furthermore, this theory may well be of use in the development of computer models that demonstrate artificial transitions.

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# Appendix A

# Hedged fitness theorem

Take two positive functions $a(t)$ and $b(t)$, $a, b \in \mathfrak{R}^+$. These depend on a continuous, but not necessarily differentiable, stochastic function of time $u(t)$ (the dynamic characteristics of $u$ are purposefully left unknown) according to the equations:

$$\frac{da}{dt} = \begin{cases} (u - u_0) g_a^+ a & , u > u_0 \\ 0 & , u = u_0 \\ (u - u_0) g_a^- a & , u < u_0 \end{cases} \tag{A.1}$$

$$\frac{db}{dt} = \begin{cases} (u - u_0) g_b^+ b & , u > u_0 \\ 0 & , u = u_0 \\ (u - u_0) g_b^- b & , u < u_0 . \end{cases} \tag{A.2}$$

Where $u_0$, $g_a^+$, $g_a^-$, $g_b^+$, $g_b^-$ are constants ($g_a^+$, $g_a^-$, $g_b^+$, $g_b^- > 0$).

I assume that $a$ and $b$ are not constant (i.e., there is a period of time where $u \neq u_0$) but that $a + b$ has no overall positive or negative trend. This means that, over time, any increase in $a + b$ will be matched by a corresponding decrease at some later point (and vice versa). Theorem 1 shows that if $g_b^+ / g_b^-$ is greater than $g_a^+ / g_a^-$ then, for each time period where $b + a$ starts and finishes at the same level, the ratio of $b$ to $a$ will increase. Theorem 2 shows that, if $g_b^+ / g_b^-$ is greater than $g_a^+ / g_a^-$ then, as the length of time the functions $a$ and $b$ are dynamic tends to infinity, $b/(a + b) \to 1$.

There is a simpler case where the system simply fluctuates between two regimes with fixed rate of change, a growth regime and a decline regime. Take two positive functions $a^*(t)$ and $b^*(t)$, $a^*, b^* \in \mathfrak{R}^+$.

$$\frac{da^*}{dt} = \begin{cases} g_a^+ a^* & , \text{growth regime} \\ -g_a^- a^* & , \text{decline regime} \end{cases} \tag{A.3}$$

$$\frac{db^*}{dt} = \begin{cases} g_b^+ b^* & \text{, growth regime} \\ -g_b^- b^* & \text{, decline regime.} \end{cases} \tag{A.4}$$

How the system fluctuates is between the two regimes is not determined but I take that $a^* + b^*$ has no overall positive or negative trend. Corollary 3 shows that if $g_b^+/g_b^-$ is greater than $g_a^+/g_a^-$ then, as the length of time the functions $a^*$ and $b^*$ are dynamic tends to infinity, $b^*/(a^* + b^*) \rightarrow 1$.

To prove the theorems a lemma is required. In Lemma 1 two phases are considered which may or may not be connected (see FigureA.1): an increasing phase where both $a$ and $b$ are increasing ($u(t) > u_0$) and a decreasing phase where both $a$ and $b$ are decreasing ($u(t) < u_0$). It doesn't matter which phase comes first, but there are three other conditions: (i) the value of $a + b$ at the beginning of the primary phase is equal to the value of $a + b$ at the end of the secondary phase; (ii) the value of $a + b$ at the end of the primary phase must be equal to the value of $a + b$ at the beginnning of the secondary phase; (iii) at the beginning of the secondary phase the ratio of $b$ to $a$ must not be less than its value at the end of the primary phase. Lemma 1 states that if $g_b^+/g_b^-$ is greater than $g_a^+/g_a^-$ then the ratio of $b$ to $a$ at the end of the secondary phase will be greater than it's value at the beginning of the primary phase.

Theorem 1 shows that for each time period where $b + a$ starts and finishes at the same level, the ratio of $b$ to $a$ will increase. This is done by specifying an algorithm for analysis of a time series of $a + b$. The time series is broken up into segments and subsegments as illustrated by Figure A.2. Each segment has an increasing and a decreasing phase. The theorem shows that all segments ultimately contain subsegments where the end point of the primary phase is the same as the start point of the secondary phase. By recursion, and Lemma 1, it is shown that $b$ must increase relative to $a$ for all segments.

Using similar logic to Theorem 1, Theorem 2 extends the algorithm from Theorem 1 so that the dynamic portion of the time series is covered by an infinite number of segments. Since $b$ increases relative to $a$ in each segment, I therefore only need to prove that the only upper bound to $b/(a + b)$ is 1.

**Lemma 1** *Firstly consider the increasing phase, $u(t) > u_0$. At the beginning of the increasing phase assume $a = a_i$ and $b = b_i$. At the end of the increasing phase, assume both variables have increased to $a = a_j$ and $b = b_j$ (i.e., $a_j > a_i$ and $b_j > b_i$). Secondly, consider the decreasing phase, $u(t) < u_0$. At the beginning of the decreasing phase assume $a = a_\alpha$ and $b = b_\alpha$. At the end of the decreasing phase, assume both variables have decreased to $a = a_\beta$ and $b = b_\beta$ (i.e., $a_\alpha > a_\beta$ and $b_\alpha > b_\beta$). By condition (i), $a_i + b_i = a_\beta + b_\beta$. By condition (ii), $a_j + b_j = a_\alpha + b_\alpha$. If $g_b^+/g_b^- > g_a^+/g_a^-$ then,*
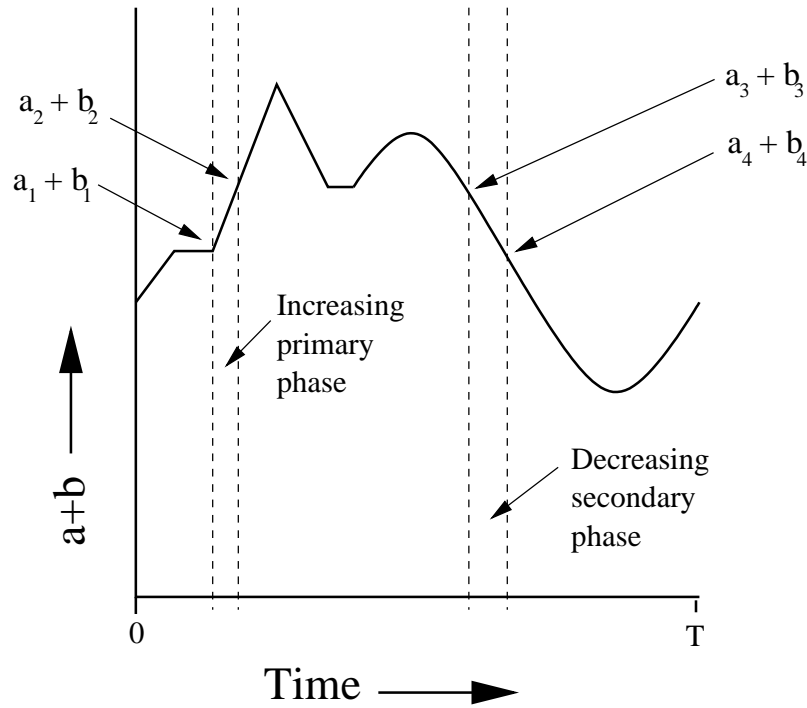
Figure A.1: A trace of $a + b$ showing two phases. The two phases are between the dashed vertical lines shown on the trace. The primary phase is matched by a corresponding posterior secondary phase moving in the opposite direction. The two phases are not connected. In line with conditions (i) and (ii) of Lemma 1, $a_1 + b_1 = a_4 + b_4$ and $a_2 + b_2 = a_3 + b_3$. Condition (iii) for Lemma 1 requires that $b_3/a_3 \geq b_2/a_2$.

**Case 1.** *Wwhere the increasing phase is before the decreasing phase:* $(b_j/a_j) \leq (b_\alpha/a_\alpha)$ *implies* $(b_\beta/a_\beta) > (b_i/a_i)$ *and*
**Case 2.** *Where the decreasing phase is before the increasing phase:* $(b_\beta/a_\beta) \leq (b_i/a_i)$ *implies* $(b_j/a_j) > (b_\alpha/a_\alpha)$.
*(The proposition in both cases corresponds to condition (iii).)*

**Proof** Dividing Equation (A.1) by Equation (A.2) gives:

$$\frac{da}{db} = \begin{cases} \dfrac{ag_a^+}{bg_b^+} & , u > u_0 \\[2em] \dfrac{ag_a^-}{bg_b^-} & , u < u_0 \end{cases} \tag{A.5}$$

First consider the increasing phase where variables $a$ and $b$ start from $a_i$ and $b_i$ and increase to $a_j$ and $b_j$ ($u > u_0$). Integrating Equation (A.5) for $a_i \leq a \leq a_j$ and $b_i \leq b \leq b_j$
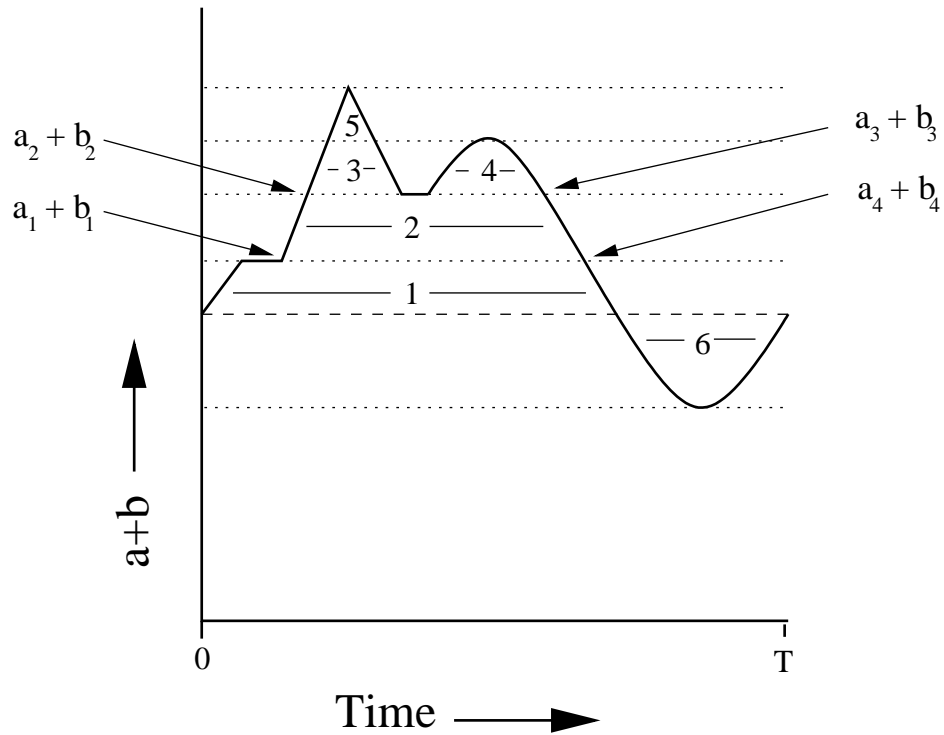
Figure A.2: A trace of $a+b$ broken up into segments with each segment having two phases. Both phases are between the same two horizontal lines. Each primary phase is matched by a corresponding posterior secondary phase moving in the opposite direction. The dashed line shows the value of $(a+b)_{t_1}$. Segments are numbered 1-6, segment 2 is a subsegment of 1, segments 3 & 4 are subsegments of 2, and segment 5 is a subsegment of 3. The four points of segment 2 are marked: the increasing phase from $a_1+b_1$ to $a_2+b_2$ is matched by the decreasing phase from $a_3+b_3$ to $a_4+b_4$.

gives

$$\int_{a_j}^{a_i} \frac{1}{g_a^+ a} da = \int_{b_j}^{b_i} \frac{1}{g_b^+ b} db$$

$$\frac{1}{g_a^+} \ln(a_j/a_i) = \frac{1}{g_b^+} \ln(b_j/b_i) . \qquad (A.6)$$

Secondly consider the decreasing phase where variables $a$ and $b$ start from $a_\alpha$ and $b_\alpha$ and decrease to $a_\beta$ and $b_\beta$ ($u < u_0$). Integrating Equation (A.5) for $a_\alpha \le a \le a_\beta$ and $b_\alpha \le b \le b_\beta$ gives

$$\frac{1}{g_a^-} \ln(a_\beta/a_\alpha) = \frac{1}{g_b^-} \ln(b_\beta/b_\alpha) . \qquad (A.7)$$

Multiplying Equation A.6 by $g_b^+$ and Equation A.7 by $g_b^-$, adding them together and

rearranging (using the property that $\ln \phi / \psi = \ln \phi - \ln \psi$) gives:

$$\ln\left(\frac{b_j}{b_\alpha}\right) + \ln\left(\frac{b_\beta}{b_i}\right) = \frac{g_b^+}{g_a^+}\ln\left(\frac{a_j}{a_i}\right) - \frac{g_b^-}{g_a^-}\ln\left(\frac{a_\alpha}{a_\beta}\right) . \tag{A.8}$$

To prove each case of the lemma, it is assumed that each conclusion is false and then shown that there is a contradiction. The negation of the conclusion of Case 1 is the proposition of Case 2 and vice versa. Therefore, it is only necessary to assume $(b_\beta/a_\beta) \leq (b_i/a_i)$ and $(b_j/a_j) \leq (b_\alpha/a_\alpha)$ and prove there is a contradiction. To prove there is a contradiction I show that, given these assumptions, the left hand side of Equation (A.8) must be less than or equal zero and the right hand side must be greater than zero.

Considering $b_\beta/a_\beta \leq b_i/a_i$, all the variables are positive, so multiplying both sides by $a_i a_\beta / b_i b_\beta$ gives:

$$a_i/b_i \leq a_\beta/b_\beta$$

Adding one to both sides of the equation and then multiplying both sides by $b_i b_\beta$ gives:

$$
\begin{aligned}
b_i b_\beta (a_i/b_i + 1) &\leq b_i b_\beta (a_\beta/b_\beta + 1)\\
b_\beta (a_i + b_i) &\leq b_i (a_\beta + b_\beta)
\end{aligned}
$$

But, by definition, $a_i + b_i = a_\beta + b_\beta$, so:

$$
\begin{aligned}
b_\beta &\leq b_i\\
b_\beta/b_i &\leq 1\\
\ln(b_\beta/b_i) &\leq 0 \tag{A.9}
\end{aligned}
$$

Similarly, consider $(b_j/a_j) \leq (b_\alpha/a_\alpha)$. Again, all the variables are positive, so multiplying both sides by $a_\alpha a_j / b_\alpha b_j$ gives:

$$a_\alpha/b_\alpha \leq a_j/b_j .$$

Adding one to both sides of the equation and then multiplying both sides by $b_\alpha b_j$ gives:

$$
\begin{aligned}
b_\alpha b_j (a_\alpha/b_\alpha + 1) &\leq b_\alpha b_j (a_j/b_j + 1)\\
b_j (a_\alpha + b_\alpha) &\leq b_\alpha (a_j + b_j)
\end{aligned}
$$

Again, by definition, $a_j + b_j = a_\alpha + b_\alpha$, so:

$$b_j \leq b_\alpha$$

$$b_j/b_\alpha \leq 1$$
$$\ln(b_j/b_\alpha) \leq 0. \tag{A.10}$$

Equations (A.9) and (A.10) therefore show that the left hand side of Equation (A.8) is less than or equal to zero.

To show that the right hand side of Equation (A.8) is greater than zero, I show that the first term is larger than the second term. Considering $b_\beta/a_\beta \leq b_i/a_i$, add one to both sides of the equation and multiply both sides by $a_i a_\beta$. This gives:

$$a_i a_\beta (b_\beta/a_\beta + 1) \leq a_i a_\beta (b_i/a_i + 1)$$
$$a_i(b_\beta + a_\beta) \leq a_\beta(b_i + a_i)$$
$$a_i \leq a_\beta, \tag{A.11}$$

using the fact that $a_i + b_i = a_\beta + b_\beta$. Similarly, consider $(b_j/a_j) \leq (b_\alpha/a_\alpha)$. Adding one to both sides of the equation and multiplying both sides by $a_j a_\alpha$ gives:

$$a_j a_\alpha (b_j/a_j + 1) \leq a_j a_\alpha (b_\alpha/a_\alpha + 1)$$
$$a_\alpha(b_j + a_j) \leq a_j(b_\alpha + a_\alpha)$$
$$a_\alpha \leq a_j, \tag{A.12}$$

using the fact that $a_j + b_j = a_\alpha + b_\alpha$. Dividing Equation (A.11) by $a_\alpha$ ($a_\alpha > 0$) gives:

$$a_i/a_\alpha \leq a_\beta/a_\alpha$$
$$a_\alpha/a_i \geq a_\alpha/a_\beta. \tag{A.13}$$

Also, dividing Equation (A.12) by $a_i$ ($a_i > 0$) and reversing it gives:

$$a_j/a_i \geq a_\alpha/a_i$$

which can be combined with Equation (A.13),

$$a_j/a_i \geq a_\alpha/a_\beta$$
$$\ln(a_j/a_i) \geq \ln(a_\alpha/a_\beta). \tag{A.14}$$

By defintion $g_b^+/g_b^- > g_a^+/g_a^-$. Since all these variables are positive this can be written:

$$\frac{g_b^+}{g_a^+} > \frac{g_b^-}{g_a^-} \ . \tag{A.15}$$

Combining Equation (A.14) with Equation (A.15) (by definition $a_j > a_i$ and $a_\alpha > a_\beta$, so both terms in Equation (A.14) are greater than zero) gives:

$$\frac{g_b^+}{g_a^+} \ln(a_j/a_i) > \frac{g_b^-}{g_a^-} \ln(a_\alpha/a_\beta) \ .$$

This shows that the right hand side of Equation (A.8) is positive. Since the left hand side is less than or equal to zero, there is a contradiction. Therefore, the lemma is proved. ∎

The theorems can now be proved.

**Theorem 1** *Over an arbitrary period of time of length $T$, $[t_1 : t_2]$, it is assumed that $a$ and $b$ are not constant (i.e., $\exists t_x, t_y \in [t_1 : t_2] : u(t) = u_0, t \in [t_x : t_y]$) and that there is no overall change in their sum, i.e., $(a+b)_{t_1} = (a+b)_{t_2}$. If $g_b^+/g_b^- > g_a^+/g_a^-$, then $(b/a)_{t_2} > (b/a)_{t_1}$.*

**Proof** The time series is broken up into segments by considering a time trace plot of $a+b$ and specifying horizontal lines on the trace. A horizontal line is added at each point the direction of $a+b$ in the time series changes, i.e. when $\mathrm{d}(a+b)/\mathrm{d}t$ changes between one of three regimes (+ve, 0, -ve) to another. Each segment will have an increasing phase and a decreasing phase.

Segments are formed by following the time series of $(a+b)$ through increasing time. The time series is continuous and the value of $(a+b)$ at the start of the time series is equal to the value of $(a+b)$ at the end. Therefore, for every period of time where the time series moves from one horizontal line to another adjacent line there will be a corresponding posterior period of time where the time series moves in the opposite direction from the second horizontal line to the first. Segments are defined by two phases, a primary and a secondary phase. The primary phase of each segment is the first period of time the time series moves between horizontal lines and the secondary phase is defined by the second period of time the time series moves (in the opposite direction) between horizontal lines. In harmony with the conditions of Lemma 1, the value of $a+b$ at the start point of the primary phase is equal to the value at the end point of the secondary phase. The same is true for the values of $a+b$ at the end point of the primary phase and the start point of the secondary phase.

The segments are then organised into a tree structure – each segment being a node in the tree. Sub-nodes are formed from segments between horizontal lines progressively further away from the starting horizontal line. Sub-nodes are defined by two conditions.

1. Sub-node segments must have their primary and secondary phases within the time period between the inner points of the node segment (i.e, posterior to the end point of the primary phase and prior to the start point of the secondary phase).

2. The value of $a + b$ at the start point of the primary phase of all sub-node segments must be equal to the value at the end point of the primary phase of the node segment.

The base node of the tree has a zero length primary phase which starts and finishes at $t_1$ and a zero length secondary phase which starts and finishes at $t_2$.

Because the time series is continuous and returns to its initial value, all dynamic portions of the time series must be covered by segments and each segment covers a unique part of the time series. This is because every time the time series moves from one horizontal line to another, there must be a corresponding move in the opposite direction (if the time series were to change direction, another line would be added) – forming a unique segment. The theorem can therefore be proved by first showing that if $b$ increases relative to $a$ in all sub-node segments then $b$ must increase relative to $a$ in the node segment; and secondly showing that $b$ increases relative to $a$ in all leaves of the tree (nodes without subnodes).

All segments (apart from the base node segment) must be sub-nodes of other segments in the tree – and ultimately sub-nodes of the base node. For a segment not to be a subnode of another node the time series would have to have a discontinuity. If $b$ increases relative to $a$ in the time period within the primary and secondary phases of a segment then Lemma 1 states that $b$ will increase relative to $a$ over the segment. Since by definition all dynamic portions of a segment are covered by sub-node segments, and $b$ will not change relative to $a$ while $a + b$ is static[1], if $b$ increases relative to $a$ in all sub-node segments then $b$ must increase relative to $a$ in the node segment.

Finally, to prove the theorem, it must be proved that $b$ must increase relative to $a$ in all leaves of the tree. Since $a + b$ is static in the inner period of any leaf node, $b$ will not change relative to $a$ (from footnote 1). Therefore, from Lemma 1 $b$ must increase relative to $a$ in all leaves of the tree. Therefore, $b$ must increase relative to $a$ in all segments including the base node segment. Therefore, the theorem is proved. ∎

---

[1] For $b$ to change relative to $a$ while $a + b$ is static, one would have to increase while the other decreases. But, Equations (A.1) and (A.2) require that if $a$ increases, $b$ must also increase and also that if $a$ decreases, $b$ must also decrease. Therefore it is not possible for $b$ to change relative to $a$ while $a + b$ is static.

**Theorem 2** *Consider a period of time of length T with the same conditions as descibed in Theorem 1: a and b are not constant and that there is no overall change in their sum. Define $\Theta \leq T$ as the length of time where $u \neq u_0$. If $g_b^+ / g_b^- > g_a^+ / g_a^-$, then, as T, $\Theta$ and $t \rightarrow \infty$, $b/(a+b) \rightarrow 1$.*

**Proof** Again, the time series is broken up into segments which form nodes in a tree as specified in Theorem 1. Here, $n$ is the number of nodes. As already shown by Theorem 1, $b$ must increase relative to $a$ in all nodes of the tree and completely cover the dynamic portions of the time series.

New sub-nodes are added by repeating the following procedure:

1. Choose a parent node in the tree: this node has the maximum difference between $a + b$ at the start and end of the primary phase (and, therefore, the start and end of the secondary phase).

2. Insert a new sub-node in the tree below the node: this new sub-node inherits all the sub-nodes of the parent node.

3. The start of the segment of this sub-node is at the point where the value of $a + b$ is half-way between its values at the start and the end of the primary phase. Likewise, the end of the new segment is where $a + b$ is at the same value between the start and the end of the secondary phase.

4. The parent node's primary and secondary phases are shortened accordingly.

New sub-nodes are added with this procedure so that $n \rightarrow \infty$. Since $\Theta \rightarrow \infty$, no new node can be added where the change of $a + b$ between the start and end of the primary phase (and, therefore, the start and end of the secondary phase) is zero. Given Item 2 above, $b$ must increase relative to $a$ in all children of the new sub-node. Therefore, as shown by Lemma 1, as each new sub-node is added, $b$ must increase relative to $a$ in that sub-node and its parent node.

I have now specified an infinite number of nodes for which $b$ must increase relative to $a$. The segments in these nodes completely cover the dynamic portions of the time series. Therefore, $b/(a+b)$ must increase at the end of each segement. I look for an upper bound to which $b/(a+b)$ may tend. Any upper bound must be a stationary point, i.e.,

$$\frac{da}{db} = 0 \ .$$

Equation (A.5) shows that this is only true when $a = 0$, or $b/(a+b) = 1$. Because $a, b$ are both positive, $b/(a+b) \leq 1$. Therefore, the theorem is proved. ∎

I can now show Corollary 3 by choosing appropriate values for $u$.

**Corollary 3** *Consider a period of time of length T with the same conditions as descibed in Theorem 2: $a^*$ and $b^*$ are not constant and that there is no overall change in their sum. Define $\Theta \leq T$ as the length of time where $u \neq u_0$. If $g_b^+/g_b^- > g_a^+/g_a^-$, then, as $T$, $\Theta$ and $t \to \infty$, $b^*/(a^*+b^*) \to 1$.*

**Proof** Define a function $u^*$ so that it is $u_0 + 1$ during the growth regime and $u_0 - 1$ during the decline regime. Setting $u = u*$, gives that [using Equations (A.3) and (A.1)] $a^* = a$ and [using Equations (A.4) and (A.2)] $b^* = b$.

From Theorem 2 the corollary is shown. ∎

# Appendix B

# Sample code listing

Sample code is included for simulations done in Chapter 6.

## B.1   File: sero.cpp

```cpp
#include "sero.h"
#include "environment.h"
#include "time.h"
#include "standarddeviation.h"
#include "random.h"
#include <list>

using namespace std;

CSimulation::CSimulation (REnvironment _env)
  : env(_env)
{
  foodInEnvironment = 0;
  numSteps = env->intParams.entry("NumSteps");
  halfTimeStep =
    env->intParams.entry("HalfTimeStep") ? 1 : 0;

  samplePeriod = env->intParams.entry("SamplePeriod");

  numFood = env->intParams.entry("NumFood");
```

```
mutateAtBirth = env->intParams.entry("MutateAtBirth");

resourceUnit =
  env->doubleParams.entry("ResourceUnit");
reproduceCost =
  env->doubleParams.entry("ReproduceCost");
maintenanceCost =
  env->doubleParams.entry("MaintenanceCost");
reproduceLevel =
  env->doubleParams.entry("ReproduceLevel");
mutationProbability =
  env->doubleParams.entry("MutationProbability");
mutationAmount =
  env->doubleParams.entry("MutationAmount");
baseFoodProbability =
  env->doubleParams.entry("BaseFoodProbability");
fluctuationCoefficient =
  env->doubleParams.entry("FluctuationCoefficient");

includeAsexual = env->intParams.entry("IncludeAsexual");

if (halfTimeStep) {
  resourceUnit = resourceUnit/2.0;
  maintenanceCost = maintenanceCost/2.0;
}

contributionHistogram =
  new C3DHistogram(10000, 0, 1, false);

}


void CSimulation::runSim(string suffix)
{
  // create agents
```

```cpp
agents.clear();

agents.reserve (env->intParams["StartAgents"]);

for (int i = 0; i<env->intParams["StartAgents"]; i++) {
  RAgent agent = new CAgent ();
  agent->energy = randval (reproduceLevel);
  agent->contribution = randval (1.0);
  agent->reproduceSexually = randval(1.0);
  agent->previousTimeStep = -1;
  agents.push_back(agent);
}

RAgent debugAgent = agents[0];


bool highSeason=1;

string datafilename = "data" + suffix + ".m";


ostream & dataout = env->datafiles[datafilename];

for (t = 0; t<numSteps; t++) {

  if (agents.size() > 400)
    highSeason = 0;
  else if (agents.size() < 200)
    highSeason = 1;

  double probabilityOfFood = highSeason ?
    baseFoodProbability * fluctuationCoefficient :
    baseFoodProbability / fluctuationCoefficient;


  agents.shuffle ();
```

```cpp
// FIRST MUTATE AGENTS
if (! mutateAtBirth)
  for (int i = 0; i<(int)agents.size(); i++) {
    RAgent agent = agents[i];
    if (randval(1.0)<mutationProbability) {
      agent->mutate ();
    }
  }


safeVector <RAgent>::iterator ai = agents.begin();
for (; ai!=agents.end(); ai++) {
  RAgent agent = *ai;

  if (halfTimeStep) {
    if (agent->previousTimeStep == -1) {
      if (randval (1.0)<probabilityOfFood) {
        agent->energy += resourceUnit;
        agent->previousTimeStep = 1;
      }
      else {
        agent->previousTimeStep = 0;
      }
    }
    else {
      if (agent->previousTimeStep == 1) {
        agent->energy += resourceUnit;
      }
      agent->previousTimeStep = -1;
    }
  }
  else {
    if (randval (1.0)<probabilityOfFood) {
      agent->energy += resourceUnit;
```

```
        }
      }
    }

    safeVector <RAgent> newAgentsList;
    newAgentsList.reserve (agents.size()*2);

    safeVector <RAgent> sexList;
    sexList.reserve (agents.size());

    safeVector <RAgent> asexList;
    asexList.reserve (agents.size());

    safeVector <RAgent> birthList;

    for (safeVector <RAgent>::iterator ai = agents.begin();
         ai != agents.end(); ai++) {
      RAgent agent = *ai;
      agent->energy -= maintenanceCost;
      if (agent->energy > 0) {
        if (agent->energy > reproduceLevel) {
          if (agent->reproduceSexually >0.5 || !includeAsexual)
            sexList.push_back (agent);
          else
            asexList.push_back (agent);
        }
        newAgentsList.push_back (agent);
      }
    }

    int numnewkids = sexList.size()/2;
    for (int i = 0; i < numnewkids * 2; i+=2){
      // pick a random parent to pass on its genes
      int parent = irand (2);

      RAgent newAgent = new CAgent;
```

```cpp
    newAgent->energy =
      sexList[i]->contribute(reproduceCost/2.0) +
      sexList[i+1]->contribute(reproduceCost/2.0);
    newAgent->contribution =
      sexList[i+parent]->contribution;
    newAgent->reproduceSexually =
      sexList[i+parent]->reproduceSexually;
    newAgent->previousTimeStep = -1;


    newAgentsList.push_back(newAgent);
    birthList.push_back(newAgent);
}

for (unsigned int i = 0; i< asexList.size(); i++) {
  RAgent newAgent = new CAgent;
  newAgent->energy =
    asexList[i]->contribute(reproduceCost);
  newAgent->contribution =
    asexList[i]->contribution;
  newAgent->reproduceSexually =
    asexList[i]->reproduceSexually;
  newAgentsList.push_back(newAgent);
  birthList.push_back(newAgent);
}

if (mutateAtBirth)
  for (int i = 0; i<(int)birthList.size(); i++) {
    RAgent agent = birthList[i];
    if (randval(1.0)< mutationProbability) {
      agent->mutate();
    }
  }


agents = newAgentsList;
```

```cpp
    if (t%samplePeriod == 0) {

      if (includeAsexual) {
        calculateSexAsexRatio ();
        dataout << t
                << "\t" << probabilityOfFood
                << "\t" << agents.size()
                << "\t" << numAsexual
                << "\t" << numSexual
                << "\t" << (double)numSexual /
            (double)(numAsexual + numSexual)
                << endl;



      }
      else {
        calculateContributionStats ();
        dataout << t
                << "\t" << probabilityOfFood
                << "\t" << agents.size()
                << "\t" << contributionStats.mean
                << "\t" << contributionStats.stDev
                << endl;
      }
    }


  string contfilename = "contHist" + suffix + ".m";
  ostream & contout = env->datafiles[contfilename];
  contributionHistogram->outputMatrix (contout);



}
```

```
void CSimulation :: calculateSexAsexRatio ()
{
  numSexual = 0;
  numAsexual = 0;

  for (int i = 0; i <(int )agents.size (); i++) {
    RAgent agent = agents [i];
    if (agent->reproduceSexually) numSexual ++;
    else numAsexual ++;

  }
}

void CSimulation :: calculateContributionStats ()
{
  safeVector<double> contributions (agents.size (), 0.0);

  for (int i = 0; i <(int )agents.size (); i++) {
    RAgent agent = agents [i];
    contributions [i] = agent->contribution ;
  }

  contributionStats =
    getStandardDeviationStats (contributions );

  contributionHistogram ->addDataRow (t, contributions );

}


int main (int argc , char ** argv)
{

  int seed = 0;

  if (argc >1)
```

```cpp
    sscanf (argv[1], "%d", &seed);

  rseed (&seed);

  REnvironment env = new CEnvironment ("params.xml");

  CSimulation sim (env);

  time_t curr=time(0);
  cout << "Simulation started: " << ctime(&curr) << endl
      << "Seed = " << seed << endl
      << endl
      << "Params:" << endl;

  for (stringmap<double>::iterator smi =
        env->doubleParams.begin();
      smi!= env->doubleParams.end(); smi++) {
    cout << smi->first << " = " << smi->second << endl;
  }

  stringstream suffix;
  suffix << seed;

  sim.runSim (suffix.str());

  curr=time(0);
  cout << "Simulation completed: " << ctime(&curr) << endl;


}
```

## B.2  File: sero.h

```cpp
#ifndef _SERO_H
#define _SERO_H 1

#include "counted.h"
#include "environment.h"
#include "standarddeviation.h"
#include "histogram.h"

class CAgent : public PCountedHeapObject
{
 public:

  double energy;

  double contribution;

  double reproduceSexually;

  int previousTimeStep;

  double contribute (double reproduceCost) {
    energy -= reproduceCost;
    double contributeAmount = energy * contribution;
    energy -= contributeAmount;
    return (contributeAmount);
  }

  void mutateGuassian (double SD) {
    double r = (gaussrand<double> ()*SD);
    double newcnt = contribution + r;
    if (newcnt >0.0 && newcnt < 1.0) {
      contribution = newcnt;
    }
    reproduceSexually = randval (1.0);
```

```cpp
  }

  void mutate () {
    reproduceSexually = randval (1.0);
    contribution = randval (1.0);
  }

};


typedef PCountedHolder <CAgent> RAgent;


class CSimulation : public PCountedHeapObject
{
 public:

  CSimulation (REnvironment env);

  REnvironment env;

  int t;

  int samplePeriod;

  safeVector <RAgent> agents;

  int foodInEnvironment;
  int numSteps;
  bool halfTimeStep;

  int numFood;

  bool mutateAtBirth;

  double resourceUnit;
```

```cpp
    double reproduceCost;
    double maintenanceCost;
    double reproduceLevel;
    double mutationProbability;
    double mutationAmount;

    double baseFoodProbability;
    double fluctuationCoefficient;

    bool includeAsexual;

    standardDeviationStats contributionStats;
    R3DHistogram contributionHistogram;

    int numSexual, numAsexual;

    void runSim(string suffix);
    void calculateSexAsexRatio();
    void calculateContributionStats();

};

#endif
```

# Appendix C

# Publications

The following publications, produced from the work in the thesis, are included.

**J. Bryden**, "Slime mould and the transition to multicellularity: the role of the macrocyst stage", In M. S. Capcarrere, A. A. Freitas, P. J. Bentley, C. G. Johnson, and J. Timmis, editors, *Advances in Artificial Life: 8th European Conference, ECAL 2005, Canterbury, UK, September 5-9, 2005. Proceedings.* (2005) 551–561.

**J. Bryden and J. Noble**, "Computational modelling, explicit mathematical treatments, and scientific explanation", *Artificial Life X*. (2006) 520–526.

**J. Bryden**, "The role of collective reproduction in evolution", In: F. Almeida e Costa, L. M. Rocha, E. Costa, I. Harvey, and A. Coutinho, editors, *Advances in Artificial Life, 9th European Conference, ECAL 2007. Proceedings.* (2007) 645–654.

# Slime mould and the transition to multicellularity: the role of the macrocyst stage

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**Abstract.** The transition from unicellular to multicellular organisms is one of the mysteries of evolutionary biology. Individual cells must give up their rights to reproduction and reproduce instead as part of a whole. I review and model the macrocyst stage in slime mould (*Dictyostelium*) evolution to investigate why an organism might have something to gain from joining a collective reproduction strategy. The macrocyst is a reproductive cartel where individual cells aggregate and form a large zygotic cell which then eats the other aggregating cells. The offspring all have the same genetic code. The model is a steady state genetic algorithm at an individual cellular level. An individual's genetic code determines a threshold above which it will reproduce and a threshold below which it will join a macrocyst. I find that cycles in food availability can play an important role in an organism's likelihood of joining the macrocyst. The results also demonstrate how the macrocyst may be an important precursor to other cooperative behaviours.

## 1 Introduction

The quest to synthesise hierarchical levels of organisation in artificial life is a significant open problem [3, 23]. To provide a deeper understanding into how we may be able to use evolutionary algorithms to generate and optimise hierarchical behaviour, we can study the major transitions in evolution [16]. This work focusses on the transition to multicellularity which appears to be one of the most difficult 'bridges' evolution has had to cross. It is unclear whether the transition only occurred once, or several times [4]. Phylogenetic evidence [2] suggests that multicellular organisms, especially metazoa, share a common ancestor. Furthermore, fossil evidence [16] indicates that multicellular life did not exist for 2,500 million years until the Cambrian period (approximately 540 million years ago) where all the multicellular phyla are represented.

Multicellular organisms essentially consist of clusters of individual cells with all cells expressing the same genotype. They therefore require gene-regulatory mechanisms for differentiating cells (with differentiations being passed from parent cell to offspring), cell adhesion and spatial patterning of cells [16]. One particularly crucial cell differentiation stands out: The organism must separate its reproductive (*germ-line*) cells from its body (*soma*) cells [7].

The requirement for isolation of the germ line from the soma was first argued to be necessary by August Weismann [7]. To identify why, we can distinguish the two types of reproduction that are present in metazoan multicellular life and look at the conflicts that arise between them. Firstly, intra-organism reproduction happens when cells replicate within the super-organism, for the good of the super-organism. Conflicts can occur with cells reproducing on their own behalf [17]: mutant cells can disrupt and compete with the super-organism. By generating a whole organism from one initial germ-line cell, it is clear that the vast majority of selfish mutations that disrupt super-organism-level processes will only survive one generation [7]. Therefore, secondly, to solve this problem super-organism reproduction involves the replication of the complete organism through the selection of a germ line cell to reproduce on behalf of the super-organism. However, there is still a conflict over which cell is to be the germ line since selfish mutations that disrupt the super-organism reproductive process will be passed onto the next generation. A stable, policed, germ-line/soma differentiation mechanism must have evolved at some point.

It is unclear where in the evolution of a multicellular lineage, stable, well policed, germ-line/soma differentiation and germ line isolation should occur. However, given the above problems faced with intra-organism conflicts[17], it seems likely that the germ-line/soma differentiation evolved early [7]. Thus, we consider evolutionary mechanisms that will explain a transition between unicellular organisms, which compete within their populations and compete with predators and prey, and early multicellular organisms which are clustered together and exhibit germ-line/soma differentiation. In other words, there is a transition from unicellular organisms which are optimised to maximise their own *direct* fitness to cells that must, on the other hand, maximise their *inclusive* fitness at the expense of their direct fitness (i.e., their ability to contribute their fitness to other cells that are highly related must be more important than their own replication chances). (See [10] for precise definitions of *direct* and *inclusive* fitness.)

Whether the evolutionary transition described above, of organisms clustering and differentiating a germ line, happened in one stage is unclear. Wolpert has presented a model where individual cells may split to produce a somatic body cell that sticks to its parent and is unable to reproduce [27]. What the benefits, through inclusive fitness, are to individual cells and their lineages from doing this is unclear. There is a debate on this subject with some arguing that size is an important reason for multicellularity [4] with undifferentiated population clustering, as modelled in [19] without a germ-line/soma differentiation, being an important first step. Others point out that local competition over food will negate the value of cooperation through relatedness [21, 26, 15]. For this reason Di Paolo warns against relatedness being used as an explanation for cooperative behaviour [9]. There therefore appears to be something of a paradox if we attempt to try to understand the transition to multicellularity with such models of clustering cells. Individuals that cluster compete with each other and may negate the benefits of cooperation through relatedness, yet both clustering and cooperation are needed for the transition to early multicellularity.

A different perspective considers multicellularity through aggregation [16]. Here cells either vegetate and reproduce individually, or aggregate to reproduce collectively. This presents a sort of half way house between the individual and early multicellular behaviour identified above. *Dictyostelium* (more commonly known as slime mould) is a model organism for multicellularity through aggregation [16, 20]. Individual cells can either vegetate and reproduce asexually on their own, or under different environmental conditions they also demonstrate collective reproduction behaviour, characterised by individual cells making sacrifices for the benefit of other cells' reproductive chances. This organism therefore demonstrates both the germ-line/soma differentiation [6] and clustering that is important for the transition. Biological evidence is now presented concerning *Dictyostelium discoideum*, one of the more studied species of the genus.

When there is a shortage of food and *D. discoideum* cells begin to starve, they aggregate and one of the two collective reproductive stages commences [22]. The more well known reproductive stage of *D. discoideum* sees the cells form a slug which collectively migrates. Once the cells find an advantageous location they form a *fruiting body*: cells at the front of the slug (20%) form a stalk and the rest form spore cells at the top of the stalk which are dispersed by the wind. Interestingly, the stalk cells die after the stalk is built. This differentiation between spore and stalk cells is arguably a germ-line/soma distinction [6]. Since cells that produce stalks do not pass on their genetic code, it is hard to see how this trait is selected for and maintained. Indeed there are examples of slime moulds strains that do not produce stalks [6]. Computer simulations addressing this question [1] have indicated that high dispersal of spores can lead to more stability in the stalk producing behaviour.

The second, less well known, collective reproduction stage in *D. discoideum* involves the formation of the *macrocyst* [22]. Again, when the cells are starving they aggregate. However instead of forming a slug, two cells merge to form a large *Zygote* cell which eats other aggregating cells. The resulting giant cell forms a hard cellulose outer wall and this macrocyst germinates after a few weeks. See Fig. 1 for a diagram.

The macrocyst stage is thought to be a precursor to the slug/stalk reproductive stage. Kessin [13] argues that evolution generally occurs in incremental stages. He notes that the previous stage to macrocyst development would be the microcyst stage (not observed in *D. discoideum*), where individuals form outer walls on their own. After the evolution of chemotaxis, aggregation could occur and the macrocyst evolved. With added cell adhesion and cell type differentiation into stalks and spores, fruiting body and slug behaviour would then become plausible.

The genetic makeup of the offspring of the macrocyst is an important question. The macrocyst is generally accepted to be the sexual phase of *D. discoideum's* development [22]. However experiments do demonstrate that Macrocysts can form from only one mating type [5]. The progeny of one macrocyst is observed to be of one genotype [25]. Only one nucleus remains in the zygote (or giant cell) after other ingested nucleii disappear [18].
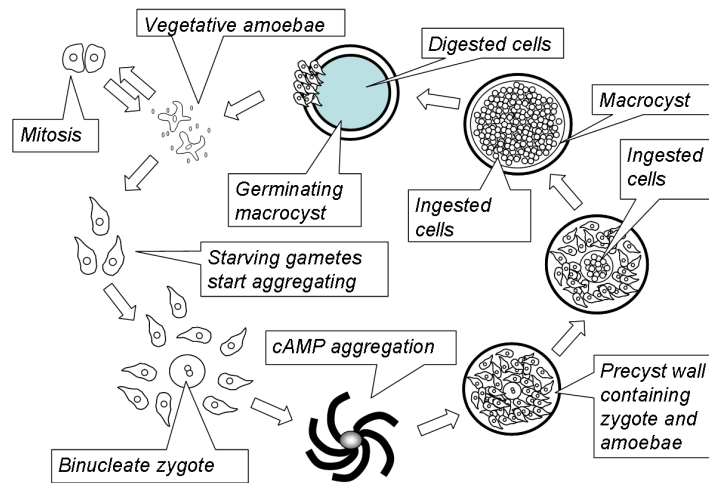
**Fig. 1.** The sexual and mitotic life cycles of *Dictyostelium* (based on [22]).

From Fig. 1 it is clear that differentiation in *D. discoideum* cells occurs when it starts to aggregate. Recent evidence implies that the cell may have genetic control over this event. Research [8, 11] suggests genes that can control or delay when or whether a cell will continue to grow or start aggregation. These findings indicate that the cell is capable of turning on or off aggregation to the macrocyst stage which can ultimately lead to cells being eaten by the zygote. This emphasises a need for an explanation as to why an individual might make the 'choice' to aggregate and almost certainly die.

I have produced a model of the *D. discoideum* macrocyst stage for several reasons: (i) to confirm that individuals that normally reproduce on their own are indeed prepared to gamble their own reproductive chances against the 'pot' of reproductive material contained in the macrocyst; (ii) to confirm my intuition that fluctuations in food availability are important to the viability of the macrocyst; (iii) to question the role individual mitotic split rates might play in the stability of the macrocyst; and (iv) to speculate on the role the macrocyst might play in the evolution of other altruistic behaviour (such as stalk/spore differentiation) and collective behaviour.

## 2   Methods

To investigate the questions in Section 1 I have built a computer simulation model of the macrocyst stage of *D. discoideum*. Assumptions in the model are based on the biological evidence presented. Notably I have assumed that all the offspring of a macrocyst are of the same genotype. Since sexual fusion does not seem to be necessary, I chose (on parsimonious as well as biological grounds) to model the macrocyst with no sexual recombination. Individual vegetative

behaviour was modelled with individuals having a genetically encoded energy threshold above which they mitotically reproduce.

*D. discoideum* cells are modelled as individuals in a non-spatial environment. At each time step, a number of individuals ($N$) are selected at random, each receives a 0.5 units of energy (representing food) with probability $p$. One cycle in the model contains two seasons. The amount and probability of food ($N$ and $p$) changes value according to whether the season is 'high' ($N = 100, p = 0.6$) or 'low' ($N = 20, p = 0.3$). Each season lasts 200 turns. All individuals pay a daily energy cost ($E_c = 1.0$) irrespective of season. If an individual's energy falls below zero ($x < 0$), it will die.

Each individual cell is modelled with two genes[1]. The genes model energy thresholds which determine the behaviour of the cell. Cells will join the macrocyst when their energy level is *below* the first gene, the *macrocyst join threshold* ($-2.0 < G_{\mathrm{join}} < 2.0$). When a cell's energy level is *above* the second gene, the *split threshold* ($5.0 < G_{\mathrm{split}} < 20.0$), the cell will pay an energy cost to split mitotically (see Fig. 1) and produce a new cell (sharing energy equally between itself and its offspring).

There is only one macrocyst in the model it is assumed to be immobile and therefore does not receive food from the environment. When cells join it, they contribute their own energy ($x$) plus a residual energy amount (equal to the cost of splitting) to the macrocyst's 'pot' ($X$). Before closing the macrocyst pays a cost $E_m$ per individual joined every turn to reflect metabolisation and building of cellulose. If the macrocyst energy falls below zero ($X < 0$) then it (and all its joining cells) will die. When the macrocyst reaches a predetermined energy threshold (30.0), it closes and no other cells may join.

The macrocyst will germinate on the first turn of the high season. When it germinates, the energy is divided up into new cells with each cell receiving 2.5 energy units. All new cells will have the same genotype: a complete genotype (no recombination) is picked at random from all the cells that originally joined the macrocyst.

Simulations were run over 100,000 turns. Each simulation started with 100 individuals, each individual having a random genotype and a random energy between 0.0 and 5.0.

## 3   Results

To understand how the harshness of the low season can effect the viability of the macrocyst, simulations were run varying the probability of food in the low season. Interesting population dynamics, with macrocyst offspring outcompeting the non-joining population, were observed and these are presented in this section.

The average percentage of individuals which germinated from the macrocyst is plotted against the probability of food in the low season in Fig. 2. When

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[1] Genes are represented as floating-point numbers in the simulation, point mutations occur at each time step over a gaussian distribution with standard deviation of 1% of the gene space
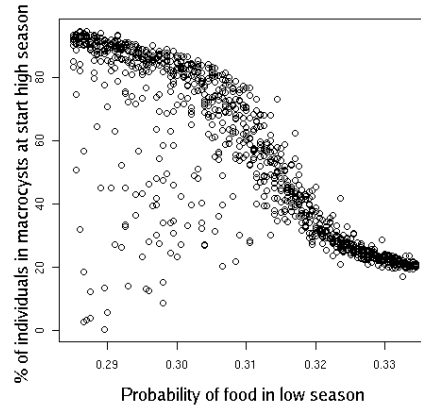
**Fig. 2.** Graph showing the percentage of individuals which germinated from a macrocyst at the start of the high season against the probability of food in the low season. Each data point (ten data points, each generated with different random seeds, per food-probability value) represents an average over a complete simulation run.

the probability of receiving energy is higher, few individuals ($\approx 20\%$) join the macrocyst. When there is a lower probability of food, more individuals join the macrocyst. However the rogue data points at the bottom left of the graph are of interest.

To investigate this disparity with some populations producing macrocysts and others not, the probability of food and seed value were selected from one of the rogue data points. The simulation was run over a longer (150,000) number of turns. A histogram was generated for the macrocyst join threshold at the start of each high season and the results are shown as a 3D mesh in Fig. 3A.

In the figure, the presence of macrocysts can be seen as spikes on the right hand side. An early tendency towards macrocyst joining is evident (far right of graph) but these genotypes die out after $\approx 25$ cycles. A population which does not produce germinating macrocysts immediately flourishes. After $\approx 150$ more cycles there are enough individuals to successfully produce a germinating macrocyst which survives to the end of the low season. Interestingly once this has happened the macrocyst very quickly wipes out the non joiners from the population. The offspring from the macrocyst must have some sort of competitive advantage over the non-joining population.

A closer look at Fig. 3A indicates that when there are not enough individuals joining the macrocysts to make them germinate, there is only a small tendency toward individuals that will not join the macrocyst when their energy is very low. Between cycle 25 and cycle 175, the histogram shows a larger proportion of individuals having a join threshold below zero, however some still remain with a threshold above zero. There is clearly little selection pressure against individuals sacrificing small amounts of energy when near to death.
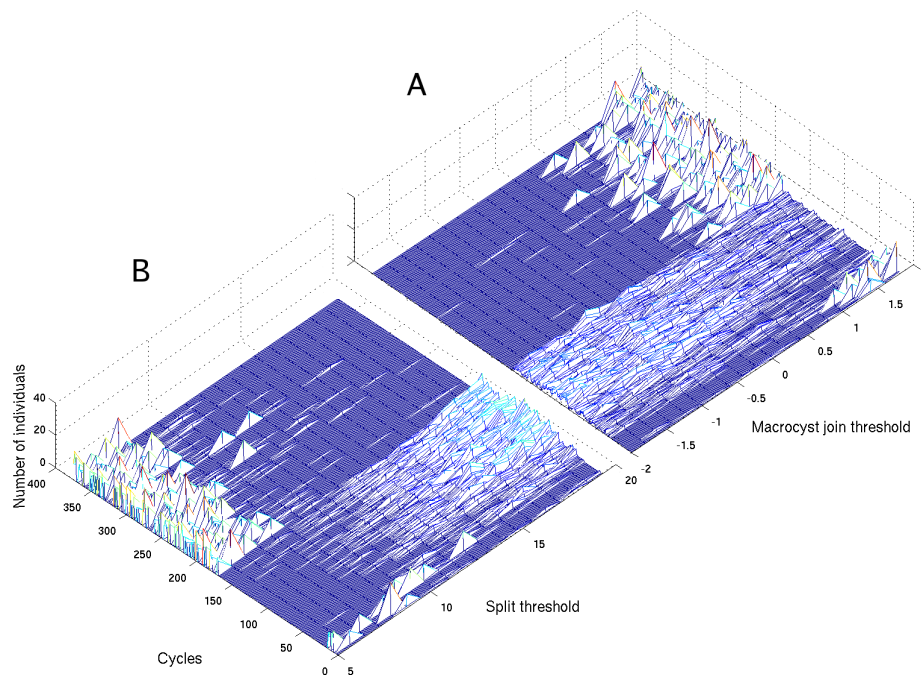
**Fig. 3.** 3D histograms of macrocyst join thresholds (A) and individual split thresholds (B) of the population at the start of each high season.

A second 3D histogram was generated for the split thresholds of the population at the start of the high season and can be seen in Fig. 3B. There is a clear disparity of the split thresholds between the macrocyst joining population and the non joiners. Again, in the first few cycles of the simulation (where the macrocyst joiners were predominant in Fig. 3A), the population has a low split threshold – individuals will split as quickly as possible. After $\approx 25$ cycles the macrocysts die out. There is now a clear tendency for dominance in the population for individuals that split more slowly. Once the macrocysts return (after $\approx 160$ cycles), the split thresholds of the population immediately return to lower values ($< 7$).

Simulations run with all individuals having the same, fixed, split threshold resulted in either the individuals all dieing, through starvation in the low season, or a small percentage joining the macrocyst when food is more plentiful (data not shown). The competitive advantage of the macrocyst joining population was no longer effective and macrocysts were only formed through enough individuals sacrificing their energy in a similar way to the non-joining population in Fig. 3A.

Other simulations have been run with variable split thesholds and the low season completely removed to see if parameters exist where a macrocyst can form and dominate the rest of the population. Simulations were run with vary-

ing parameters of $N$ and $p$, both seasons having the same values. While some macrocyst production was observed it was only at the beginning of simulations where the random starting population allowed for enough individuals that joined the macrocyst and made it viable for a few cycles (data not shown).

## 4    Discussion

In Section 1 I have argued of the need for a model that demonstrates the transition between individual cells that ordinarily reproduce on their own to cells that become part of a super-organism, with only one genotype of the participating cells being passed on to future generations. For the macrocyst model to succesfully meet the requirements of this transition, it requires that all individual cells must be able to reproduce on their own. It also requires that individual cells must be clustered and that only one of the individual cells reproduces on behalf of the cluster. The model presented meets these requirements. Simulated cells that have the freedom to evolve a strategy in which they will not join macrocyst organisations (where their genes are highly likely to be destroyed) do not evolve this strategy under fluctuating environmental conditions.

The model does however stop short of demonstrating the type of germ-line/soma differentiation and clustering apparent in the metazoa where there is differentiation of the germ line early in development [17] and permanent clustering (as in other models, e.g., [27, 19]). The macrocyst's germ-line cell is the zygote which is not differentiated from any other cells in the super-organism. Also, the macrocyst cells are only clustered at one point of the life cycle. However, the fact that the macrocyst's offspring are of only one genotype and that they outcompete individuals that do not join the macrocyst is of some significance.

The fact that the macrocyst produces offspring of a single genotype is important in three ways. Firstly it has the effect of producing several homogenous offpsring which are all 'preprogrammed' to join the macrocyst at the start of the next low season. These offspring have a competitive advantage over individuals that do not join the macrocyst. The macrocyst therefore contributes to its future success. Since microbes can evolve many 'policing' mechanisms [24], it is not inconceivable that after several generations, the macrocyst way well have become established in the organism without the need for a harsh low season each cycle.

Secondly, the high relatedness of the offspring can be seen to promote other social behaviours. Relatedness is crucial for any traits that require many coordinated individuals or altruism to be successful. The aclonal nature of the macrocyst offspring means that it is highly likely that the next aggregation event will also be aclonal or at least highly related. If these individuals have the same mutation which means (perhaps under certain environmental conditions) they no longer fuse to form a zygote then other interesting collective behaviour may occur instead. These behaviours could include, but are not limited to, the slug behaviour of *D. discoideum* which requires many coordinated individuals [14], and the stalk behaviour of *D. discoideum* which requires altru-

ism from many cells [1]. The macrocyst has been argued to be a precursor of these behaviours [13]. The combination of the macrocyst model with one of the stalk/spore behaviour (based on [1]) will hopefully confirm how important the population homogenisation effects of the macrocyst were for the evolution and maintenance of stalk/spore behaviour in *D. discoideum*.

The homogeneous macrocyst offspring are important in a third way: By picking the genotype of its offpsring from one individual at random, the macrocyst stage eradicates the potential for cheating: If an individual were to evolve a 'cheating' trait so that its genes were most likely to be picked, the next population would all have that same trait - with no individual having any advantage.

To consider how split thresholds are important I analyse a complete cycle. In one cycle of the model presented here there are four phases for non macrocyst joining amoebae: (i) Early high season exponential growth; (ii) Population equilibrium at high season; (iii) Early low season exponential decimation of the population; (iv) Population equilibrium at low season. While it is easy to see that fast (low threshold) splitting amoebae would flourish during phase (i), these same amoebae will be closer to dying during phase (iii). The results suggest that a slow (high threshold) splitting strategy is more profitable, not only in phase (iii) but in phase (iv) as well. In phase (iv) individuals receive food with a low probability, those with a fast (low) split threshold are less adapted to survive fluctuations in food availability. The macrocyst allows individuals to avoid phases (iii) and (iv) and hence fast splitting individuals that germinate from it at the start of the high season are very well adapted to phase (i). This ability to perform well during circumstances of diminishing populations has already been observed as an important feature of early multicellular organisms [12].

While I have attempted to be faithful to biological evidence, the model presented here has made some assumptions and has some limitations. Further analysis and research is required into the biological plausibility of the split thresholds in the model. The question as to what might happen if individuals have a seasonally varying split threshold is also important. The model is undimensional and therefore lacks spatial effects (though the way the organisms are fed is set up to mimic a spatial environment): a spatial model would allow us to analyse what might happen if individuals could effect their chances of being the chosen genotype. The mutation rate in the model is unnaturally fast, however slower mutation rates provided similar results over longer periods. Finally there is only one macrocyst in the current model, future simulations will model more than one macrocyst.

The model and results presented in this paper demonstrate that, given the assumptions outlined, the *D. discoideum* macrocyst stage is plausible under the large fluctuations in food in the model. The results and analysis lead me to hypothesise that the model of the macrocyst presented in this paper, where individuals gamble their genes to become the germ line of a super-organism, may well have been a crucial stage in the transition to multicellularity. It must be noted that it is only a stage in the evolution of *D. discoideum* and may be relevant only to this organism. However, the facts that the slug behaviour of *D.*

*discoideum* is reminiscent of other metazoa and that their phylogeny implies a common ancestor imply that slime mould may give some important clues into the evolution of the metazoa and perhaps other multicellular organisms.

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# Computational modelling, explicit mathematical treatments, and scientific explanation

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## Abstract

A *computer simulation model*, can produce some interesting and surprising results which one would not expect from initial analysis of the algorithm and data. We question however, whether the description of such a computer simulation modelling procedure (data + algorithm + results) can constitute an explanation as to why the algorithm produces such an effect. Specifically, in the field of theoretical biology, can such a procedure constitute real scientific explanation of biological phenomena? We compare computer simulation modelling to explicit mathematical treatment concluding that there are fundamental differences between the two. Since computer simulations can model systems that mathematical models can not, we look at ways of improving explanatory power of computer simulations through empirical style study and mechanistic decomposition.

## Introduction

It seems possible that computer simulation modelling could become the new modelling paradigm in biology. As modellers build transparent, tractable, computer simulation models their relaxed assumptions will, in comparison with traditional explicit mathematical treatments, make for considerably more realistic models that are close to the data. The 'Virtual Biology Laboratory' is proposed (Kitano et al., 1997) where a cycle is applied through comparing computer models with empirical evidence: the results from each procedure inspiring the direction of the other. Animals, such as *C. elegans*, have been well studied using computational models, e.g., (Bryden and Cohen, 2004). Indeed the formation of a complete model of the organism has been identified as a potential grand challenge for computing research (Harel, 2002). However, a full exploration of the relationship between mathematical and computational models in biology has not yet been achieved. Questions remain: for instance, whether both forms of modelling can peacefully coexist, whether mathematical models should aspire to the complexity of computational models, and conversely whether computational models can ever be as precise as a mathematical treatment.

In this paper we are mainly concerned with the scientific modelling of biological systems, however we hope that the findings can be applied more generally. Biological systems are made up of many different subsystems at different levels. Alife models often reside in the interface from one level to the next and can become extremely complex, especially as entities from any level can interact with entities from other levels.

The discipline of computer simulation modelling allows modellers previously unheard-of freedom to build and understand systems of many interacting parts. This new expressive freedom appears to have the potential to become the new modelling paradigm in science, perhaps overriding traditional techniques which use explicit mathematical treatments. However, this freedom does not come without a cost: as more and more detail is added computer simulation models can quickly become unwieldy and too complicated to understand.

How then can computer models contribute to the task of producing scientifically acceptable explanations? The use of a complex yet poorly understood model may be acceptable as some sort of loose analogy. However, Di Paolo et al. (2000) have argued that without a proper understanding of the internal workings of a computer simulation model, it can be impossible to say whether such a model makes a valuable contribution to the scientific problem it is addressing. They describe such problematic models as 'opaque thought experiments', arguing the need for explanations of the phenomena modelled. They suggest that modellers should use an 'experimental phase' in which manipulations are made to the computer model, the results of these manipulations hopefully generating insights into the workings of the system. Once the internal mechanisms are understood, the transparent model can then not only give new insights into the system being modelled but can also become a powerful predictive tool.

We question whether a computer simulation model can, in and of itself, constitute a scientific explanation. For example, one might produce a model in which individual organisms are explicitly represented and a particular population-level phenomenon appears to emerge. But this does not constitute an explanation of how entities from one level of a bio-

logical hierarchy produce interesting phenomena at another level. Di Paolo et al. (2000) argue that some explanation is required above a basic description of the model and the system it represents. In this paper we look further into what an adequate explanation of a model's mechanisms should entail. We will compare the account that we construct with the more basic position, sometimes seen in the artificial life literature, that a bare-bones description of a biological system with a computer model that qualitatively produces similar behaviour—with little or no extra analysis or explanation— can constitute a scientific explanation of some phenomena.

Given the above picture we must also consider the traditional methodology of explicit mathematical treatment. By explicit mathematical treatment we mean a model which is complete and contains no implicit steps, the steps can be logical statements and do not need to be formally written using mathematical symbols. While computer simulation models are fundamentally mathematical constructions, they, in the way they are reported, contain *implicit* mathematical steps rather than the *explicit* steps used by formal mathematical models. An explicit mathematical treatment takes logical axioms and specifies a number of clear explicit steps that deductively generate some result. In this paper we compare this traditional treatment with the new computational approach.

Firstly we set the context, we look at a framework for scientific modelling. Then, by looking at two examples of a similar system, we identify some properties that characterise an explicit mathematical treatment and which a computer simulation is unlikely to share. Having established that explicit mathematical treatment is the ultimate goal of any modelling enterprise, we look at how computer simulation models do indeed still have value. We look at how complex and unwieldy computer simulations may be simplified to more easily generate explicit mathematical treatments— proposing that this can be done by decomposition into simpler systems. Finally we set out, in an order of merit, the various different modelling approaches discussed.

## A framework for scientific modelling

To understand how modelling is important and relevant within scientific investigation, we present a framework for scientific investigation with the scientific modelling cycle highlighted. Figure 1 presents a diagram of the framework.

The primary focus of scientific investigation is the building of a good *conceptual model* of the *real world*. Explanations of the real world reside in the conceptual modelling area of the framework, these are recorded in the *scientific corpus*. The basic scientific process involves the submission of concepts to the twin tests of *empirical science* and scientific modelling. The main focus of the framework, however, is on scientific modelling and the interface between a conceptual model and a *working model*.

Both computer simulation models and explicit mathe-
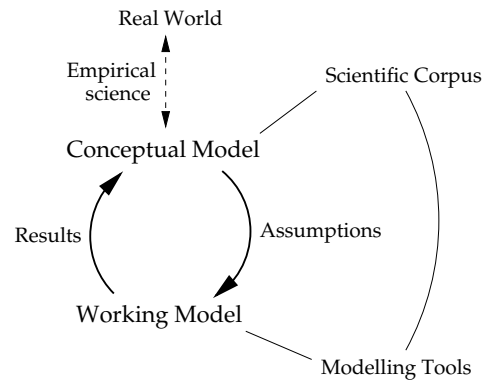


Figure 1: The cycle of enquiry in scientific modelling within the context of scientific investigation.

matical treatments reside in the working model area of the framework. We take a working model to be a deterministic and completely specified model of a system. (Whereas a conceptual model may remain vague in places, a working model must be completely fleshed out.) Logical processes are applied to the axioms and the results of this process are recorded. Logical processes can include mathematical equations, logical deductions and computations. Working models produce *results* which are used to refine and update the conceptual model.

Before we specifically look at the sorts of results that can be generated by explicit mathematical treatments or computer simulation models, we discuss the types of *assumptions* that can be used to generate a working model. An assumption is essentially an abstraction from a more complex system. There will be many abstractions from the real world in the conceptual model (tested by empirical science) and it will normally be necessary to make further abstractions for ease of modelling. One of the main benefits of computer simulation modelling (Di Paolo et al., 2000) is that assumptions can be very easily added to or removed from models to see if they are significant or important. Explicit mathematical treatments tend to be more fixed in their assumptions. The types of abstractions used by either explicit conceptual models or computer simulation models can be distinguished into two groups, *reductionist* and *analogous* abstractions. We take inspiration for this distinction from Bedau's discussion of 'unrealistic' models (Bedau, 1999).

In order to highlight the important differences between the use of computational and mathematical techniques in building a working model, we must first consider the outcomes of a successful working model for the broader scientific project. The more valuable results generated by a working model will form some kind of explanation of why some phenomenon is present in the conceptual model. Other, less valuable, results include those that generate predictions. With an explanation generated by a model to hand, an empirical scientist can easily and quickly generate good empirical

experiments to test whether an explanation is valid or not. A working model may indicate that some factors are more important than others for a particular phenomena. This may direct empirical science toward a more fruitful direction. The value of a result can depend not only on the type of working model used to generate the result, but also the assumptions used to generate the working model in the first place.

## Competence and performance in scientific modelling

The previous section has set out the tasks necessary before embarking on a modelling enterprise: Once a conceptual model has been chosen that builds a picture of what is known about some real-world phenomenon, assumptions are then chosen to simplify this conceptual picture into logical units and axioms that can be built into a model. Up to this point everything is quite similar between the two logical modelling styles. Perhaps it is natural to assume that since both modelling techniques are analytical, the style of the results will also be quite similar?

To answer this question we must consider a thought experiment based on a specific example which can easily be understood and modelled by either a computer simulation model or an explicit mathematical treatment. The Lotka-Volterra system is a mathematical treatment of a predator-prey system. Two equations model the dynamics of the system:

$$\frac{dx}{dt} = Ax - Bxy \tag{1}$$

$$\frac{dy}{dt} = -Cy + Dxy \tag{2}$$

where x is the prey, y is the predator and $A, B, C, D$ are constants. This system famously generates oscillations between the predator and prey populations. This mathematical treatment can be considered alongside an individual based computer simulation model of the same phenomenon.

A typical example system might be as follows. In a computer simulation model, individuals may have a location on a spatial grid moving at random each turn. If a prey individual encounters some food in its square it will receive an energy bonus, if it encounters a predator it will be eaten with the predator receiving an energy bonus. If either a predator or prey individual's energy level goes above a threshold then it will reproduce, and if any individual's energy level goes below a threshold, it will die.

Without wanting to go into too much detail, we assume, for the purposes of argument, that the computer simulation has very similar dynamics to the mathematical system. That is, both systems will make the same predictions about any particular predator-prey system to which they might be applied. The two systems can now be compared against each other and we can review our initial question concerning the nature of the scientific explanation that may be derived from each modelling enterprise.

To answer that question we draw on a distinction introduced by Chomsky between *competence* and *performance* (Chomsky, 1986). Chomsky's approach considers whether the linguistic corpus can be used as a source of empirical evidence for linguistic enquiry. He distinguishes between competence (our internal unconscious capacity for language) and performance (actual instances of language production). Regarding linguistic inquiry, he argues that we should take this distinction into account considering models of linguistic competence above models of linguistic performance.

We use Chomsky's distinction to shed light on the differing styles of scientific explanation that are likely to follow from the use of computational versus mathematical treatments of a particular problem. From this point of view, the computer simulation model must merely be considered as a performance of a scientific explanation, whereas the explicit mathematical treatment can be considered as having competence (an innate capacity) as a scientific explanation.

Simulation runs have the same sorts of problems as those Chomsky identifies for linguistic performances. They are subject to faults (in code as well as in run-time conditions) and each simulation model is merely a single data point and may not reveal the complete potential of a system. In a similar way, it is possible to hide flaws in the performance from the audience. Simulations can be set up so that the data points presented make the best possible case for whatever it is the modeller is trying to argue.

Alternatively, explicit mathematical treatments, assuming they are done correctly, are analytically complete: flaws in the system are immediately obvious. In addition, mathematical treatments are not limited to some narrow range of parameters but provide universal coverage of all variables included in the model. These two properties were identified by Chomsky as arguments in favour of looking at linguistic competence over linguistic performance.

Furthermore, explicit mathematical treatments have more powerful identity conditions than do computational models. By this we mean that one mathematical treatment can automatically be established as the same as, or different to, another treatment, just by comparing the logic. Computer simulation runs, on the other hand, may produce similar results for the same problem, but have very different underlying explanations. The opposite can also occur, in that two computer simulations may be driven by the same underlying process without this being obvious to an observer.

Mathematical treatments are more reusable than computer simulation models. Some give good clean results which can instantly be applied to systems, others benefit from the ease with which they can be written down in full and passed on. Such models can then be used as logical axioms for other models with their competence passed on. In contrast, although computer models can certainly be transferred from

one author to another their results are rarely used, in practice, as axioms for other models.

One might argue at this point that we can distinguish the code for a computer simulation model from an individual execution of the code. The argument continues that a simulation run is merely a performance of the code, the code itself has competence. To answer this point we look at the style of computer model chosen in the Lotka-Volterra example above. It was chosen specifically so that the code would demonstrate an emergent phenomenon (Bedau, 1997). There are only two cases possible here. Either, without an execution of the code its macroscopic function is opaque, or, if the macroscopic function is deducible from the code, then this deductive process would necessarily form an explicit mathematical treatment. If this deductive process is impossible, any explanation generated must be teased out by analysis of simulation runs.

At this point, we are left with a conundrum. If computer simulation models are viewed as mere instances (performances) rather than as systematic explanations (having competence), how can they be of use to science? The answer is that there are many areas, identified especially in the ALife field, which do not yet yield to mathematical modelling but in which simulation models can already be produced. Such simulation models not only have scientific power as proofs of concept and for generation of insights for performing empirical science, but they can also have some explanatory power (Di Paolo et al., 2000).

When considering a complex simulation in which there is no explanation of the effects produced, some explanation can be deduced by performing experiments on the simulated system in the same way that one would do for an empirical investigation. In this mode of enquiry a *control* simulation is generated in which some important phenomenon does not happen. This is normally done through some manipulation of the system. The control simulation is compared with the untampered system and the results are used as evidence that the changes made by the manipulations are part of the explanation of the phenomenon.

The above procedure is very similar to the normal mode of empirical science. A conceptual model can be built of the working model system and this conceptual model acts as an explanation. We will now look further into how this form of explanation relates to an explicit mathematical treatment.

## Analytic explanation versus synthetic explanation

To attempt to understand the difference between an explanation generated through the use of a working model in explicit mathematical form and an explanation generated by experimental manipulations of a computer simulation model, we consider a distinction used by the logical positivists—that of *analytic* and *synthetic* truths.

According to Frege's reworking (Frege, 1980) of Kant's original distinction, an analytic truth is one that can be deduced through logical laws alone. A synthetic truth is one which needs some other means, generally empirical investigation, to establish its truth or falsity.

We use this distinction to identify modes of truth for explanations generated by a working model. As pointed out previously, we assume all working models are using the same assumptions, i.e., they start from the same set of logical axioms. We distinguish between an analytic explanation—one which follows logically from the initial assumptions—and a synthetic explanation—one which must be determined by some other means.

Naturally an explicit mathematical treatment is in itself an analytic explanation. However, empirical experiments done on a computer simulation can only form synthetic explanations. These synthetic explanations require validation in the same way empirical science must be validated. The evidence backing up these validations relies on measurements taken from performances and is thus open to disconfirmation, reproduction and revalidation.

There is an ongoing debate about the analytic/synthetic distinction, some arguing that it is not a black and white distinction but more a question of degree (Quine, 1953). While Quine's arguments are concerned with statements about the real world rather than statements about a closed set of logical axioms, we agree that our distinctions of explanations should not be black and white. A working model can, like a biological system, be large and complex. Some parts of such a system will yield to explicit mathematical treatment, whereas with other parts we may have to rely on empirical-style experiments of the kind discussed by Di Paolo et al. The final explanation generated through such a process will consist of a mixture of analytic and synthetic statements.

In the next section we present an account of how systems can be decomposed into smaller parts to identify explicit mathematical treatments. Successful mathematical treatments will render the resulting explanations more analytic in the way we have just described.

## Decomposition of systems

A system can become hard to analyse when it is made up from many inter-dependent subsystems. In fact, the identification of subsystems is a good first step when tackling such a complicated system. However, this is rarely simple. When subsystems are inter-dependent it is not possible to manipulate one subsystem independently without affecting another: both subsystems, at the same time, affect the overall system. The situation becomes increasingly difficult when the subsystem's components are not mutually exclusive from each other.

Simon (Simon, 1996) describes a 'nearly decomposable system' as being one in which components are independent in the short term, but dependent in the long term. This is a useful way to divide a system up and this has been expanded further (Watson, 2005; Polani et al., 2005) considering mod-

ular dynamical systems. Watson introduces a concept called *modular interdependence* to describe a system with modules that are decomposable but not separable. A hierarchy can be formed from subsystems and it is easy to see how complex behaviour can be generated. This hierarchical perspective is a valuable decomposition of a complex system. If it is possible to divide up a set of microscopic entities into subsets this will allow us first to tackle the mechanisms of the subsets, before understanding how they interact with each other.

In the next section we consider a more general perspective for decomposing systems. Rather than breaking up the set of microscopic entities into subsets, we consider a more arbitrary way of decomposing a system into subsystems that contain a simplified version of the dynamics of the supersystem.

## Mechanistic subsystem

We propose information theoretic definitions of a mechanistic subsystem and interdependence in mechanistic subsystems. This style of definition has been used in (McGregor and Fernando, 2005) to formalise *hyperdescriptions*. We then go on to discuss how these definitions relate to our intuitive notions of these concepts before looking at examples in the next section.

Define a system $S$ as being a set of mathematical entities, their interactions and their parameters. Take a descriptor function $d(S) = M$ that will map the system $S$ to a set of descriptors $M$. Define the *entropy* of a random variable $X$ as $H(X) = -\sum_{x \in X} p(x) \log p(x)$, the *conditional entropy* between two random variables $X$ and $Y$ as $H(Y|X) = -\sum_{x \in X} p(x) \sum_{y \in Y} p(y|x) \log p(y|x)$ and the *mutual information* as $I(X;Y) = H(Y) - H(Y|X)$.

Take a system $S_1$, such that $d(S_1) = M_1$. Then, $S_1$ is a mechanistic subsystem of $S$ if

$$S_1 \subset S \tag{3}$$
$$H(M_1|M) = 0 \tag{4}$$
$$I(M_1;M) > 0 \tag{5}$$
$$I(M_1;M) < H(M). \tag{6}$$

The mechanistic subsystem $S_1$ is a constrained version of its supersystem $S$. The constraints can take place in the parameter space, the number of entities, the nature of the entities, or their interactions. We list the Equations [(3) to (6)] and describe their meaning: (3) $S_1$ is a subset of $S$; (4) all information in $M_1$ is predicted by $M$; (5) $M_1$ and $M$ share some information; (6) there is infomation in $M$ that is not predicted by the information shared by $M_1$ and $M$.

The information theoretic definition presented includes many of the important concepts of a mechanistic subsystem. However a useful mechanistic subsystem should have two further properties. Firstly, it should be transparent, i.e., it is possible to understand why and how it produces its macroscopic effects. Secondly, its macroscopic effects should be

of interest when compared to the macroscopic effects of the main system. We need to avoid specifying macroscopic subsystems that are either equally complex to the main system with only some neglible reduction, or are so simplistic that they are of no analytic value.

Following on from this definition of a mechanistic subsystem, we draw on Polani et al.'s definition (Polani et al., 2005) of a system that is decomposable but not separable to identify how two mechanistic subsystems can be interdependent. Take a system $S$ and two mechanistic subsystems $S_1$ and $S_2$, the subsystems are interdependent if $0 < I(M_1;M_2) < \min[H(M_1), H(M_2)]$. The two subsystems are neither independent nor completely dependent.

With this approach identified, we can see how it is possible to break up a complex system of many interacting parts into simpler mechanistic subsystems.

## Examples of Mechanistic Subsystems

We consider, as an example, the spatial embedding of reproducing agents. Space has been shown to be an important factor in the maintenance of cooperation in a population (Boerlijst and Hogeweg, 1991; Di Paolo, 2000). The common feature of these models is that two regimes are considered. The models are considered and analysed in a non-spatial environment before being placed in a spatial environment. The non-spatial treatment is a mechanistic subsystem of the spatial treatment. In this treatment agents are thought to be in a perfectly mixed spatial environment, a special case of the spatial component. A comparison of the interactions of agents in the spatial and non-spatial environments demonstrates how cooperation is increased. The mechanistic subsystem (the non-spatial model) functions primarily as a control in these experiments.

A different model (Bryden, 2005b) considers collective reproduction in amoebae. This non-spatial model demonstrates that cells that reproduce individually must reproduce more slowly to maintain high energy reserves for periods of low resources. By reproducing collectively during periods of low resources, individuals can avoid the need to reproduce slowly and can dominate periods of high resources by reproducing more quickly. The model is complex and it is not easily apparent why this is occurring. A mathematical treatment (Bryden, 2005a) analyses a mechanistic subsystem of the main model only considering individuals that reproduce individually. This treatment shows that, when there is a greater cost to individual reproduction, the rate of decimation, at times of low resources, will be proportionately greater than the rate of growth at times of high resources. Reproducing more slowly will decrease the cost of reproduction, and so the mathematical analysis explains why this occurs in the full model.

A further model (Bryden, 2005c) considers the effects of space on the individual reproduction mechanistic subsystem: agents in the model live and reproduce on a spatial

grid. This model can be broken up into two mechanistic subsystems, firstly a non-spatial subsystem with individuals reproducing [as treated by (Bryden, 2005a)] and secondly a spatial subsystem without individuals reproducing. Results indicate that the spatial effects increased the frequency of both periods of high resources and periods of low resources. The mathematical model has shown that this would increase the tendency for individuals to conserve resources and reproduce more slowly. This is an example of a system that combines two interdependent mechanistic subsystems (a spatial and a reproductive system) that interact with each other to produce a macroscopic phenomenon.

Away from the field of agent based modelling, we consider models based on neural biological systems. Neural systems have extremely complex dynamics, which are resistant to mathematical analysis. However, the use of linear stability analysis has proved useful in identifying mechanistic subsystems which can be used as building blocks within larger systems. For example, a system of coupled oscillators, based on the FitzHugh-Nagumo model, has been analysed as a mechanistic subsystem (Buckley et al., 2004). This analysis demonstates how, when the oscillators are linked to a simple gas net, the system can produce temporally distinct oscillations. Much other work continues into the identification of simple oscillatory models, such as that done in Central Pattern Generators (CPGs) (Marder and Bucher, 2001). CPGs can work as mechanistic subsystems within models of animal locomotion systems.

In this section we have demonstrated how a complex system that does not yield to explicit mathematical treatment may be simplified into mechanistic subsystems which are more likely to yield to explicit mathematical treatment. We can observe from the examples chosen that the working models arrived at through such a process consist of both synthetic and analytic explanations.

The process of simplification identified above is not the only way of making simpler models. By choosing different assumptions and approaching a conceptual model from a different perspective it is also possible to open up a system to explicit mathematical treatment. With computer simulation it is increasing easy to change the assumptions of a model and get a feel for how the system changes. This sort of approach is invaluable as a tool for the sort of lateral thinking needed when generating an explicit mathematical model.

## Discussion

While this paper has argued that an explicit mathematical treatment will provide a superior explanation of a scientific phenomenon to an equivalent computer simulation, it must be made clear that the overrarching goal of the scientific modeller is to build better models which explain important phenomena which are not as yet understood. To this extent computer simulation is still a crucial part of the modellers toolbox. The ease with which models can be produced with computers is extremely valuable. Furthermore, not only can these early efforts lead to some important scientific results, but they can also point towards new directions for mathematical models. We list below, in increasing order of merit, different styles of working models and explain how valuable each one is in generating scientific explanation. By starting with models at the beginning of the list and progressing up the list, models can become better explanations of scientific phenomenon.

- A description of an opaque computer simulation and some vague rhetorical statements that it consists of an explanation of what it is trying to model. We have argued that this approach is merely setting down a procedure for producing a performance of explanation of some phenomenon. However, this approach can still yield a proof of concept for some topic under debate, or generate insights for empirical experiments.

- The same computer simulation as before, but this time complete with well documented source code, parameters and other data that can easily be tested by other users and reused in new simulations. While this approach does not yet produce a competent explanation, it allows for more simple reproduction of the model which will help others develop it further.

- An opaque computer simulation (with well documented source code) with some manipulations and simulation runs that demonstrate how various attributes of the model explain various phenomena. We have argued that this approach can yield a competent explanation of sorts, but this is merely a synthetic explanation and is not logically grounded.

- An opaque computer simulation (with well documented source code) that has been decomposed into mechanistic subsystems. Some subsystems have been treated mathematically. Such a working model can also yield a competent explanation of sorts, this explanation is more analytic than in the previous case.

- An explicit mathematical treatment. Such a working model yields a competent fully analytic scientific explanation.

As set out above, clearly the best option is to produce an explicit mathematical treatment. However this is rarely simple, and in many cases mathematics is not yet mature enough to approach this goal. Since we must live in the real world, science must answer questions about systems that cannot be yet modelled by mathematical approaches. Computer simulation modelling provides us a working methodology for approaching these complex or complicated systems and making important steps toward understanding them.

Further to this, it is important to note that computer simulation models can extend already established mathematical

treatments. By extending or relaxing the assumptions made in the purely mathematical treatment, the new model will rely on the mathematical treatment as a mechanistic subsystem but may produce new results or important insights on the mathematical model (Harris and Bullock, 2002). Since computer simulation models lend themselves to more accurate, relaxed assumptions, when explanations become available they are more likely to be of value to the conceptual model under question.

What is important is that scientific models progress up the order of merit listed. A novel modelling approach that identifies a new style of working model may have value even if it merely provides a performance of some scientific explanation. Such a system can be experimented with and decomposed into mechanistic subsystems and the standard of explanation will improve. This is one of the benefits of computer simulation modelling in that it gives us tools to break down a problem so that we can get closer to an explicit mathematical treatment through an iterative process. Computer simulation models can be thought of as providing tools for developing imagination and lateral thinking in modelling approaches.

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# The role of collective reproduction in evolution

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**Abstract.** To look for an answer to the puzzle of why complexity may increase, this paper looks to the major evolutionary transitions – a recurring pattern where individuals give up their rights to reproduce individually and instead reproduce as part of a super-organism. A simple model of collective reproduction is presented and discussed in light of this topic. The model finds that collective reproduction is actually to the benefit of the individual, not to the group. The cost of reproduction is shown to be an important factor and different scenarios are presented which show individual, sexual reproduction and collective reproduction (with larger numbers of parents) as optimal.

## 1   Introduction

One of the most striking features of our evolutionary past is the rise of the complex individual. As we replay the timeline of natural history [1], the most complex species has become increasingly more complex. One of the major goals of Artificial life has therefore been to recreate such increases in individual complexity *in-silico* [2]: i.e., within a computer.

The common opinion in biological circles [3] is that core evolutionary theory is all we need to explain the evolution of life and its astounding complexities. However, this does not seem so hopeful when attempts to reproduce such effects in ALife computer models, which implement all the main features of evolutionary theory, have not produced much complexity of interest whatsoever [4]. It seems clear therefore that there is something missing, perhaps just from the models that have attempted to recreate some of the complexity of life, or perhaps even from the evolutionary theory they are based on. This paper looks for a process (or processes) that can increase the complexity of an individual.

A profound theme observed in nature is the hierarchical structures (units made up from sub-units) that can be found [1]. These hierarchical structures are a recurring pattern, and can be seen at all levels of biology. The important feature at every level of these hierarchies is that units are made up of sub-units: proteins are made of chains of amino acids, cells/organelles are made of proteins, eukaryotic cells are made up of organelles, multicellular organisms are made up of eukaryotic cells, and societies are made up of multicellular organisms. Each unit is of greater complexity than each sub-unit.

This repeating pattern implies that there may be some common feature in individual sub-units from every level that leads them to form themselves into

larger units: an evolutionary force that binds these units together. Or, put another way, a mode of interraction that is common to sub-units at every level. In fact, these sub-units have common features at every level. They all make copies of themselves, which may or may not be perfect copies, and they all compete with other similar individuals over resources.

One other feature has been highlighted and is seen in the literature studying the evolution of new super-units from sub-units. These evolutionary steps have been dubbed *major evolutionary transitions* [1]. It has been observed that all of the transitions are characterised by one simple common paradigm: sub-units that could previously reproduce on their own can now only reproduce as part of the super-unit [5]. Since each of the transitions involves an increase in individual complexity, I look for a general model of the transitions which can illuminate processes for increase in individual complexity.

A common approach to explaining some of the individual transitions is to look to altruism and social evolution. The central thesis, here, is that any individual involved in collective reproduction is performing an altruistic act. I.e., taking a reduction in their own reproductive success so that the reproductive success of the group may increase. It has long been rejected that an individual may reduce its fitness (lifetime reproductive success) to benefit its local group [6]. However, it has been shown how an individual may take a reduction in personal fitness to benefit related organisms [7, 8] through *kin selection*.

The kin selection perspective has shown some value in explaining the maintenance of eusociality (social insects), however it is not clear that it (or altruism in general) explains the origins of this transition [1, 9]. Indeed one problem with kin selection is that its benefits can be negated by increased local competition for food [10]. One question, considered here, is therefore whether altruism actually is crucial for explaining the origins of collective reproduction. It may be possible to find explanations that are mutualistic: i.e., the outcome for both the individual and the group is beneficial.

On the face of it, given the literature on the cost of sex (e.g., [11, 12]), it looks unlikely that a mutualistic explanation may be found for collective reproduction. Put simply, the cost of sex means that individuals which reproduce sexually will grow at a slower rate than those that reproduce individually. Rather than directly considering Maynard Smith's model of the cost of sex, I illustrate the problem by reformulating it within the abstract terms used in this paper. Here, I make a simple comparison of the growth rates of an individual strategy and a collective reproduction strategy (with two parents) based on the way resources are allocated to offspring, as shown in Fig. 1.

Since individuals with the collective strategy share contributions to offspring, they contribute less than those with the individual strategy. This means that the average level of resources per individual in the collectively reproducing population will be higher than those in the individually reproducing population. For a fixed input of resources to the system, this means that the collectively reproducing population will grow more slowly than the individually reproducing population. If we look at cases where there are increasingly larger numbers of
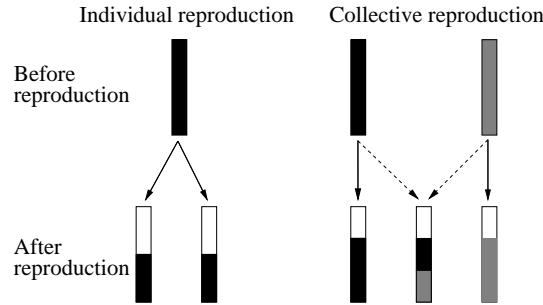
**Fig. 1.** Individual reproducers will grow more quickly than collective reproducers. Individuals are represented as resource containers of size $R_1$, with the level of resources represented by how full the containers are. They are shown just before, and immediately after reproduction. Each new individual from the sexually reproducing population will have an average resource level of $2R_1/3$ so, for the same resource input, this population will grow more slowly than the individually reproduction population which starts with an average resource level of $R_1/2$.

parents, then the same reasoning can be used to show that the growth rate will be increasingly slower (e.g., when there are 3 parents, this population will have an average resource level of $3R_1/4$ after reproduction).

Given this cost of collective reproduction, it seems unlikely that there is any advantage to collective reproduction. However, this analysis only looks at the growth phase. Any population that grows will eventually exhaust the resources in its environment and the population will either decline or reach a static level. Because collectively reproducing individuals have, on average, greater resources after reproduction they are less vulnerable during times of population decline.

To approach modelling the major evolutionary transitions, I take an abstract perspective in order to produce a toy model. This approach, based on the common factors of the subunits identified above, simplifies the world to resources, individuals and individual strategies. Individuals accumulate resources, expend resources, make copies of themselves and share resources with those copies. Given that an individual lives within an environment with factors outside of its control, I assume that they have little control over the way they accumulate or expend resources. The space of strategies that they may adopt is therefore concerned with their reproduction strategy.

The model simply approaches the question as to whether it is a better strategy, for the individual, to reproduce collectively or to reproduce individually. It is looking for an explanation for collective reproduction that is not based on altruism. In the next section I present the model and its results.

## 2 The model

Two modelling approaches are taken in this section. After presenting details common to both approaches, I outline a mathematical model and its predictions.

The predictions of the mathematical model are tested by simulation models in Section 2.2.

Here each individual $i$ is modelled as a resource level $x(i, t)$ by the equation,

$$x(i, t + \delta t) = x(i, t) + u(t)R_{\mathrm{u}} - R_{\mathrm{c}} , \tag{1}$$

where $\delta t$, $R_{\mathrm{u}}$ and $R_{\mathrm{c}}$ $[0 < R_{\mathrm{c}} < R_{\mathrm{u}}]$ are positive constants ($R_{\mathrm{u}}$ is the maximum resources available for uptake and $R_{\mathrm{c}}$ is the cost of growth/maintenance each timestep of length $\delta t$). *Resource uptake* (the level of resources received from the environment) is variable and modelled by $u(t) \in [0 : 1]$. The variable $u$ here is used as a surrogate for competition: population fluctuations will lead to increasing and declining phases, modelled by changes in the behaviour of $u$.

If an individual's resource level decreases below the lower threshold $R_0$ (fixed for all individuals) it will die. Without losing generality, $R_0$ is set to 0 as an *ad hoc* simplification. If an individual's resource level increases above the reproduction threshold ($R_1$) it will reproduce. All individuals pay a cost of reproduction $R_{\mathrm{r}}(n)$ which is dependent on the number of parents $n$.

## 2.1 Mathematical treatment

The mathematical treatment assumes that $u$ is static over the lifetime of individuals. The resource change $\delta x$ over a discrete time interval $\delta t$ is therefore modelled as:

$$\delta x = uR_{\mathrm{u}} - R_{\mathrm{c}} . \tag{2}$$

The value of $u$ where $\delta x = 0$ is defined as $u_0$: $u_0 = R_{\mathrm{c}}/R_{\mathrm{u}}$.

During reproduction all parents pay the cost of reproduction $R_{\mathrm{r}}(n)$. After this the remaining resources are shared equally between the $n$ parents and the offspring. All individuals therefore start their lives, just after reproduction, with $x = n(R_1 - R_{\mathrm{r}})/(n + 1)$. Two cases for $u$ can now be considered: $u > u_0$ and $u < u_0$. In the first case the individual resource level will increase until it reaches the upper threshold $R_1$, taking an expected time $W$ where,

$$
\begin{aligned}
W_{u>u_0} &= \left[ R_1 - \frac{n\left(R_1 - R_{\mathrm{r}}\right)}{n + 1} \right] \frac{\delta t}{\delta x} \\
&= \frac{\left(R_1 + nR_{\mathrm{r}}\right) \delta t}{(n + 1)\delta x} .
\end{aligned} \tag{3}
$$

In the second case individual resources will decrease until it reaches the lower threshold at resource level $R_0$. The expected time $W$ is,

$$W_{u<u_0} = \frac{n\left(R_1 - R_{\mathrm{r}}\right) \delta t}{(n + 1)\delta x} . \tag{4}$$

In both cases, as $u \to u_0, W \to \infty$.

The expected population growth rate of a homogeneous population of individuals can be estimated for the two regimes ($u > u_0$ and $u < u_0$). The

expected population growth rate per individual $G$ is equal to the reciprocal of the time taken for resources to grow for reproduction during population growth $(1/W_{u>u_0})$ and reciprocal of the time taken for resources to decline for death during population decline $(1/W_{u<u_0})$, or [substituting Equation (2) into Equations (3) and (4)]:

$$G = \begin{cases} \dfrac{(n+1)(uR_{\mathrm{u}} - R_{\mathrm{c}})}{n\,(R_1 + nR_{\mathrm{r}})\,\delta t} & ,u > u_0 \\ \\ 0 & ,u = u_0 \\ \\ \dfrac{(n+1)(uR_{\mathrm{u}} - R_{\mathrm{c}})}{n\,(R_1 - R_{\mathrm{r}})\,\delta t} & ,u < u_0\,. \end{cases} \tag{5}$$

The growth rate $(G)$ is plotted against different values of $u$ for one $[n = 1, R_{\mathrm{r}}(1) = 0.1]$ and two $[n = 2, R_{\mathrm{r}}(2) = 0.05]$ parents in Fig. 2. In this case, the two parents share the cost of reproduction born by the single parent – i.e., the cost per offspring is the same.
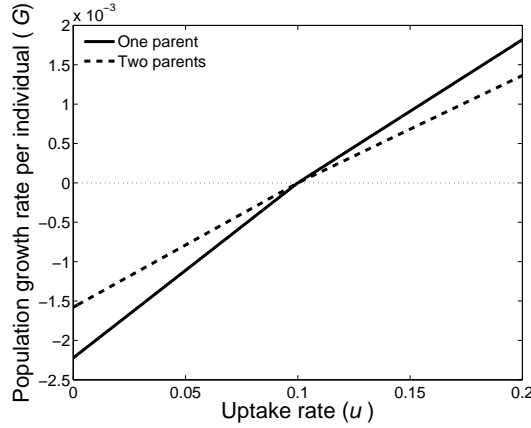


**Fig. 2.** Growth rates of individually and sexually reproducing populations. The parameters used were: $R_1 = 1.0$, $R_{\mathrm{r}}(1) = 0.1$, $R_{\mathrm{r}}(2) = 0.05$, $R_{\mathrm{c}} = 0.001$, and $R_{\mathrm{u}} = 0.01$.

The figure shows that the sexually reproducing population does in fact grow more slowly during population growth $(u > u_0)$. This is in line with the reasoning presented in Section 1 and Fig. 1. What is also evident from the figure is that the decline rate of the sexually reproducing population is lower in magnitude than that of the individually reproducing population. Furthermore, the two graphs diverge more as $u$ gets further from $u_0$ indicating that fluctuations may be important, however the ratio between the two plots stays constant. Indeed, in this example, the growth rate of the sexually reproducing population is 0.75

that of the individually reproducing population. However, the decline rate of the sexually reproducing population is only 0.71 that of the individually reproducing population. Interestingly, the sexually reproducing population has a greater advantage during decline periods than the individual strategy has during growth periods.

This analysis therefore indicates that the ratio of growth and decline rates is important and that fluctuations might also be significant. Simple mathematical analysis can be done on the relative growth to decline rates for populations with different numbers of parents, this is given by:

$$\Psi(n) = \frac{G_{u>u_0}}{G_{u<u_0}} = \frac{R_1 - R_{\mathrm{r}}(n)}{R_1 + nR_{\mathrm{r}}(n)} \; . \tag{6}$$

It seems very likely that populations with larger values of $\Psi$ will competitively exclude populations with lower values: this is tested with simulations in Section 2.2. In this model, the behaviour of $\Psi$ depends on the way $R_{\mathrm{r}}(n)$ is determined.

I look at three scenarios for determining $R_{\mathrm{r}}(n)$. These consider the offspring cost which is defined as the total reproduction cost spent on each offspring $(nR_{\mathrm{r}})$. In scenario (i), I consider the case where the total cost of each offspring is static: $R_{\mathrm{r}}(n) = R_{\mathrm{r}}(1)/n$. In this case, Equation (6) is increasing (the denominator is constant and the numerator increases). Scenario (ii) looks at the case where the cost of total cost of each offspring increases linearly with each extra parent: $R_{\mathrm{r}}(n) = R_{\mathrm{r}}(1)[1+c(n-1)]/n$ ($c$ is a constant). Finally, in scenario (iii) I consider the case where $\Psi$ is constant – i.e. when there is no advantage or disadvantage to reproducing with more parents. Simple manipulation of Equation (6) shows that if $\forall n : \Psi(n) = \Psi(1)$ then,

$$R_{\mathrm{r}}(n) = \frac{2R_{\mathrm{r}}(1)R_1}{R_1 + R_{\mathrm{r}}(1) + n[R_1 - R_{\mathrm{r}}(1)]} \; . \tag{7}$$

This sets an upper limit for the reproduction cost: if $R_{\mathrm{r}}(n)$ is above this value, then $\Psi(n) < \Psi(1)$, if $R_{\mathrm{r}}(n)$ is below this value then $\Psi(n) > \Psi(1)$.

The offspring cost is plotted in panel A of Fig. 3 for different numbers of parents in the three cases. Given the corresponding reproduction costs associated with these offspring costs, panel B shows the value of $\Psi$ calculated by Equation (6).

As expected, the plot in Panel B of the figure shows how $\Psi$ increases when the offspring cost is not dependent on the number of parents. The difference in values of $\Psi$ is relatively high when the number of parents is low – the largest increase being the difference between individual and sexual reproduction. When the offspring cost increases linearly with the number of parents, $\Psi$ reaches a maximum at 2 parents (sexual reproduction) and declines thereafter. Considering Panel A of the figure, this indicates that, as the number of parents increases, the total cost of reproduction spent (the cost per offspring) may increase. Again, this increases sharply as the number of parents increase from 1 to about 5 and then levels out. For all graphs, changes in parameters $R_1$ and $R_{\mathrm{r}}$ did not change the shape of the graphs significantly, however, as the ratio $(R_1/R_{\mathrm{r}})$ decreases, the values generated by Equation (7) increase (not shown).
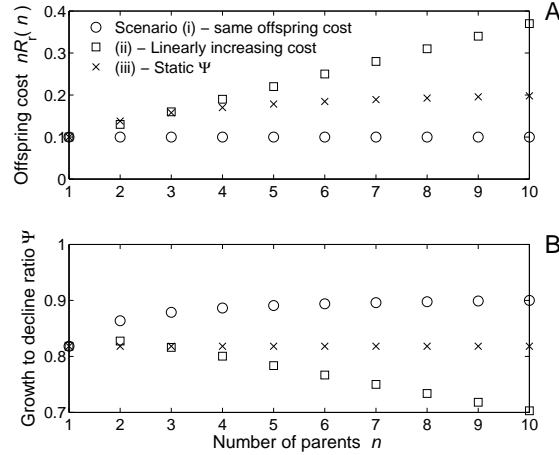
**Fig. 3.** The behaviour of the growth/decline ratio changes with different functions for $R_{\mathrm{r}}(n)$. Panel A shows the cost per offspring generated by the three different functions of $R_{\mathrm{r}}(n)$ and panel B shows the corresponding predicted growth to decline ratio. The parameters used were: $R_1 = 1.0$, $R_{\mathrm{r}}(1) = 0.1$, $R_{\mathrm{c}} = 0.001$, $R_{\mathrm{u}} = 0.01$, and $c = 0.3$.

### 2.2 Simulation models

The predictions of the mathematical treatment in the previous section indicate two things that may be tested with simulation models. Firstly whether the value of $\Psi$ is a good predictor for which strategy is optimal and secondly that fluctuations may also be significant.

Simulations are done with agents modelled as resource levels, based on Equation (1). At each timestep an agent pays a growth/maintenance cost $R_{\mathrm{c}}$. When its resources are below zero, an agent will die. Each agent has a reproduction strategy which is defined by the number of parents (from 1 to 10) the agent will reproduce with. Those with the same reproduction strategy will reproduce when enough agents (depending on the number of parents defined by their common strategy) have resource levels that are above $R_1$ (resource levels may go above $R_1$ without penalty). Each parent pays a cost of reproduction $R_{\mathrm{r}}(n)$ and all parents share their energy with the new offspring.

The simulation models are essentially non-spatial, with individuals located on a grid but moving to a random new cell each timestep.[1] Agents consume a resource unit if they encounter any on the square they are on. A number of resource units, each of value $R_{\mathrm{u}}$ are randomly scattered on a spatial grid (of 50×50 squares) each time step. The number of units is either static (set to 200) throughout the simulation, or fluctuated between two values (100 and 200) changing every 1,000 timesteps.

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[1] The results in [13] showed that agents receive resources with between-resource intervals on a geometric distribution when they move to random grid squares each time step

The simulations were run with the three scenarios for determining $R_r(n)$ presented in Section 2.1. All scenarios were tested with a static resource input to the system and fluctuating resources. The scenarios were run ten times, with each run initialised with a different random seed. After 1,000,000 timesteps the number of agents with each reproduction strategy was averaged over all ten runs. The results are plotted in Fig. 4.
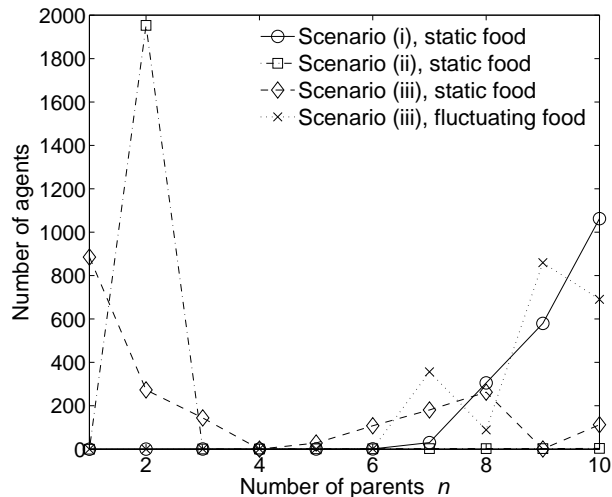


**Fig. 4.** Collective reproduction depends on offspring cost and resource fluctuations. Parameters for the simulations were: $R_1 = 1.0$, $R_r(1) = 0.1$, $R_c = 0.001$, and $R_u = 0.01$.

The figure shows that, when the offpring cost does not increase with the number of parents, reproduction strategies with higher numbers of parents will dominate. In fact any strategy with less than 7 parents has been completely eradicated from the simulations of this scenario. When fluctuations were also introduced, similar results were seen (not shown). When the cost per offspring increases linearly, the sexual strategy is dominant over the other strategies – as predicted by the shape of $\Psi$ in Fig. 3. Again, the results were similar with and without fluctuations. When the cost per offspring increases in line with the upper limit predicted by Equation (7) (see Fig. 3, Panel A), the viability of collective reproduction depends on fluctuations in resource availablity. With no fluctuations, individual reproduction is dominant, but when the resources do fluctuate, collective reproduction is dominant.

## 3 Discussion

The mathematical model and simulations presented in this paper demonstrate collective reproduction. Individuals that may reproduce on their own, instead

reproduce as part of a collective. Collective reproduction here is done by sharing resources contributed to a shared offspring. The modelling work shows that the cost of this process (the cost of reproduction) is important and fluctuations in environmental resource levels can be significant. The model also presents two different scenarios which predict conditions for when sexual reproduction is optimal and other conditions for when reproduction in larger groups is optimal: this may help to explain why sexual reproduction is dominant in some animals and eusociality is dominant in others.

The mathematical predictions presented in Fig. 3 are concordant with the results in Fig. 4, both predicting when collective reproduction is viable. This includes subtle effects such as the dominance of sexually reproducing individuals. Since the results are so similar, the simulation models show that $\Psi$ is good predictor for which reproduction strategies will competitively exclude others. The mathematical treatment is therefore instructive (in line with [14]) as to why there is a long term growth benefit to lineages that reproduce in this way: the collectively reproducing individuals have greater resources and are therefore less vulnerable to resource fluctuations.

The work contributes to explaining the rise in the complexity of the individual in two ways. Firstly, it demonstrates how collective reproduction can benefit both partners: when more than one parent contributes resources to the production of an offspring, the combined reproductive expenditure can be significantly larger than with individual parents (see Fig. 3 panel A, and corroborating simulation results in Fig. 4). This extra resource is available for the increased complexity needed for the facilitation of collective reproduction.

Indeed, it is plausible that collective reproduction may happen on many levels in the same class of individual. Some examples of collective reproduction may only be viable when the conditions are right, so different mechanisms for collective reproduction may happen under different conditions. Each may have different optimal numbers of parents. As well as this, collective reproduction may happen at different levels at the same time. With some organisms making direct genetic contributions, others making indirect genetic contributions (through kin-relatedness [7]) and others perhaps gambling their genetic contribution (see [15] for an example). There can therefore be many differing mechanisms of collective reproduction taking place within a population at the same time. As new viable mechanisms increase complexity, a rich social fabric should emerge.

Secondly, the model, and its insights, implies a potentially fruitful approach to modelling the major evolutionary transitions. Rather than invoking altruism or group selection, the model of collective reproduction presented here shows mutual benefits to reproducing collectively: i.e., that it is in an individual's selfish interest to reproduce collectively. There is no need for the individual to reduce its fitness for the benefit of its kin or its group. Altruism may therefore not be an essential feature in explaining the major transitions.

That said, this does not imply that altruism is not important in collective reproduction. Altruism can happen and will act as an evolutionary force when appropriate. One major assumption of the model is that the resources of all

parents are shared out equally between the parents and offspring. Clearly a parent that does not contribute in this way may be able to disrupt the process by contributing less resources than other parents. It could be argued that a parent that doesn't do this is acting altruistically, however by contributing less resources it will also be harming its own representation in the collective reproductive effort.

Study of such cheating behaviour is outside the scope of this paper but can be addressed in future work. Other future work could also address each of the major evolutionary transitions in more detail. It is to be hoped that the application of the style and approach of modelling in this paper will yield interesting results.

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