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Game Theoretic Treatments of Social Niche Construction: How do the Conditions for Cooperation Evolve?

by

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ABSTRACT

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The presence of cooperation has long puzzled evolutionary biologists; the resolution to this puzzle is often attributed to population structure. While the effects of population structure on cooperation are understood, less is known regarding how population structure is itself subject to evolution. The research program of Social Niche Construction (SNC) explores these issues. This thesis presents three related papers that further our understanding of SNC and addresses a number of issues within the research program.

Firstly, I demonstrate that diploid organisms under the presence of meiotic drive represents an example of SNC; where assortative mating plays the role of the social niche modifier. I thus argue that assortative mating may be an adaptation that overcomes meiotic drive.

Secondly, I present a formal argument for why a gene that causes individuals to assort cannot invade a population of freely-mixed defectors at equilibrium. I present a potential solution to this problem; namely, that if individuals engage in multiple simultaneous cooperative dilemmas, then there may be a continued selection pressure for increased assortment.

Lastly, I present a model for the evolution of a cooperative division of labour. Previous game-theoretic definitions assume cooperation to be a single behaviour. I argue that this is too narrow, as often the benefits of cooperation come about through the interaction of differing types. To address this issue I define a class of games; which I call Division of Labour (DOL) games, that have the property that fitness is maximised by a mixture of different types. I show that DOL games are not resolved by a positive assortment on phenotype; instead mean fitness is maximised by positive assortment on a genotype that can exhibit phenotypic plasticity; i.e. express multiple phenotypes conditionally upon social environment.

Together these models broaden and deepen our understanding of how population structure evolves and how SNC transforms social dilemmas and modifies social outcomes.

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Preface

This thesis is presented in the *three paper* format. The papers are written in such a way that they can be read in isolation from one another. While each paper is a self-contained manuscript, there are recurring themes and methodologies, which offer a number of conclusions when this body of work is interpreted as a whole.

In addition to the three papers that constitute the main body of work, this thesis starts with a brief section that introduces the main themes of the papers; as well as highlighting their commonalities, see section 1. Finally, this thesis ends with a discussion section, the purpose of which is to set the claims of each paper into a broader context and to propose a way forward for further research.

Also note that each paper contains some additional material that does not contribute to the central flow of the argument, but complements or strengthens the central point of the paper. This may include detailed mathematical proofs, which are important, but may otherwise distract the reader from the central point if presented in the body of the text. Each of these appendices are positioned after each respective paper, rather than at the end of the entire thesis.

List of Publications

First Authored Papers

- Game Theoretic Treatments for the Differentiation of Functional Roles in the Transition to Multicellularity, *Journal of Theoretical Biology (2016)*, Simon J. Tudge, Richard A. Watson and Markus Brede
- Game Theory, Meiotic Drive and the Evolution of Assortative Mating, *Journal of Theoretical Biology (2016)*, Simon J. Tudge, Richard A. Watson and Markus Brede (in submission)
- Multiple Games and the Evolution of Assortment, *Journal of Theoretical Biology (2016)*, Simon J. Tudge, Richard A. Watson and Markus Brede (in submission)
- A Tale of Two Theorems: Comment on Universal Scaling for the Dilemma Strength in Evolutionary Games by Z. Wang et al., *Physics of Life Reviews*, Simon J. Tudge and Markus Brede
- Cooperation and the Division of Labour, *Advances in artificial life, ECAL, 2013* , Simon J. Tudge, Richard A. Watson and Markus Brede
- The Evolution of Assortment with Multiple Simultaneous Games, *Advances in artificial life, ECAL, 2015* , Simon J. Tudge, Adam Jackson, Richard A. Watson and Markus Brede

Coauthored Papers

- Replication Strategies and the Evolution of Cooperation by Exploitation, *Advances in artificial life, ECAL, 2013*, Markus Brede and Simon J. Tudge
- Evolutionary Connectionism: Algorithmic Principles Underlying the Evolution of Biological Organisation in Evo-devo, Evo-eco and Evolutionary Transitions, *Evolutionary Biology (2015)*, Richard A. Watson et. al.

Contents

Chapter 1

Introduction

1 Themes of the Thesis

Cooperation is a potentially puzzling phenomenon in nature. We often see members of a species seemingly forgoing reproductive potential in the aiding of those around them. If this behaviour has a genetic component, and is undirected in its delivery, then selection should always act to reduce its frequency until that behaviour reaches extinction. Those individuals who do not forgo reproduction to help others will, by definition, have more offspring than those that do, meaning that their genes will be represented in the next generation at the expense of their more cooperative conspecifics. Despite this convincing argument, cooperation is endemic in the natural world; as can be seen in eusocial insect, such as ants (Hölldobler and Wilson, 1990), blood sharing in vampire bats (Wilkinson, 1984), the cells of a multicellular organism (Buss, 1987; Maynard Smith and Szathmary, 1997; Michod and Roze, 2001), slime moulds (Strassmann et al., 2000), the reproductive and motile cells of volvocine algae (Michod, 2006a,b, 2007), homologous genes in diploid organisms that conform to Mendel's laws (Traulsen and Reed, 2012; Burt and Trivers, 2009) and the maternally derived organelles of a male cell (Xu, 2005).

The resolution to the puzzle of cooperation frequently takes the form of assortment on cooperative behaviours (Eshel and Cavalli-Sforza, 1983; Michod and Sanderson, 1985; Godfrey-Smith, 2008). If there exists a form of population structure whereby cooperators meet other cooperators more often than would otherwise be expected, then the benefits generated by cooperation fall disproportionately on other cooperators; thus, it may be beneficial to the individual to cooperate. If assortment is taken into account then there is no fundamental paradox to the evolution of cooperation.

As well as the biological themes of this thesis, there are also a collection of tools that I use throughout this body of work. I make heavy use of the theory of dynamical systems, see for instance Strogatz (2001). In particular, I use the formalism of the replicator equation (Taylor and Jonker, 1978; Maynard Smith, 1982; Hofbauer and Sigmund, 1998; Weibull, 1997), which

is a dynamical representation of social evolution. I always complement a purely mathematical analysis with a simulation based approach; making use of genetic algorithms, as well as ideas from agent-based modelling.

Although lacking any immediate practical application, a number of insights are presented that could be utilised in the future. One example being an understanding of the competing selection between lower and higher levels of organisation, which has implications for human health. Genetic conflict may cause illness in humans (Haig, 2014; Burt and Trivers, 2009) and, therefore, a better understanding of it will be beneficial. Cooperation, or lack of, between mother and child can cause serious health issues during pregnancy (Haig, 1993). This can only be understood properly through understanding the competing strategic interactions of the paternally and maternally inherited genes present in the offspring.

Cytoplasmic male sterility is caused by competing selection between nuclear and mitochondrial DNA within the sex cells of a plant (Chase, 2007), and has been shown to have an adverse effect on the yield of crops such as corn (Schnable and Wise, 1998; Duvick, 1965). The evolution of cooperation in viruses may be central to understanding the onset of viral infections such as HIV (Nowak and May, 1992b). Cancer may be explicable in terms of the breakdown of cooperation between the cells of a multicellular organism (Nunney, 1999). Thus, understanding cooperation is of importance to human society.

1.1 Cooperation in Evolutionary Biology

The real problem for a theorist is in making sense of the many different formalisms and mechanisms that have been proposed to explain cooperation. Nowak (Nowak, 2006b) claims there are five fundamental mechanisms for cooperation; network reciprocity (Nowak and May, 1992a; Hauert, 2004; Ohtsuki et al., 2006; Santos et al., 2006b), group selection (Maynard Smith, 1964; Wilson, 1975; Borrello, 2005), kin selection (Hamilton, 1964a,b; Maynard Smith, 1964; Grafen, 1982, 1985; Dawkins, 1979; Gardner et al., 2011), direct reciprocity (Axelrod and Hamilton, 1981; Axelrod, 1988; Nowak and Sigmund, 1993; Nowak, 1990; Imhof and Nowak, 2010) and indirect reciprocity (Nowak and Sigmund, 1998b). Other authors such as D. S. Wilson argue that group selection explains all cooperation (Wilson, 1975), while others claim that it is kin selection that is the fundamental mechanism at play (Gardner et al., 2011; Grafen, 1985; Bourke, 2011). Other authors argue that we can view all of these mechanisms in terms of something more fundamental, such as assortment (Eshel and Cavalli-Sforza, 1983; Sober, 1992; Fletcher and Zwick, 2006; Godfrey-Smith, 2008; Michod and Sanderson, 1985).

Part of the problem stems from the fact that there is no consensus on what constitutes cooperation. West et al. (2007) state that cooperation is any behaviour or feature of an organism that was selected in order to increase the fitness of other individuals. Lehmann and Keller (2006) make an important point, which I follow, by saying that there is a fundamental distinction between the types of cooperation that we might observe. The distinction concerns the nature of the benefits

	Increases Recipient's Fitness	Decreases Recipient's Fitness
Increases Actor's Fitness	(+/+) Mutualism	(+,-) Selfishness
Decreases Actor's Fitness	(-/+) Altruism	(-,-) Spite

TABLE 1.1: Four types of social behaviour.

of cooperation. Individuals may perform costly actions, but in doing so they more than recoup their losses during their lifetime. They are able to receive the help of others by virtue of being a cooperator. This is not strictly altruism, but can be referred to as reciprocity or reciprocal cooperation. This is in contrast to altruism, in which individuals never recoup the costs of cooperating at any point in their life cycle. Altruism can be understood in terms of inclusive fitness and kin selection.

Hamilton categorises social behaviours into four different varieties, depending both upon the effect on the actor and the effect on the recipient. The action may increase (+) or decrease (-) the fitness of the actor or the recipient, the four possible combination of these effects correspond to the four types of social behaviour, which are summarised in table (1.1). Selfishness and Mutualism present no special puzzle to Darwinism as the actor's fitness is increased in each case. An obvious example of selfishness is predation. Mutualism may often be favoured by natural selection, note, however, that if fitness is relative this may not be the case. Giving more benefit to one's competitors than one receives oneself may decrease one's relative fitness. Typical examples of mutualism include all symbiosis, such as between fungi and plants in lichen. Specifically it was the problem of altruism that lead to the formalisation of group selection and kin selection. Less well studied, but still important, is spiteful behaviour, which can also be understood in terms of inclusive fitness, particularly if kin recognition is possible (West and Gardner, 2010). I will follow these naming conventions in this thesis, but note that other conventions do exist.

For altruism, or indeed spite, to be stable or to invade a population some manner of population structuring is required. By population structure I mean any scenario in which the interactions between individuals occur in a non-random fashion. Godfrey Smith neatly summarises the conditions for the evolution of altruism verbally as follows:

"Altruism may be favoured if the benefits of cooperation fall disproportionately on those who are able to pass it on" (Godfrey-Smith, 2009).

Hamilton's rule is a precise mathematical statement of when this effect is strong enough for altruism to evolve.

Lehmann et. al. make another distinction in terms of the mechanisms by which this structuring comes about. In the first case individuals are indiscriminate cooperators, and interaction structure comes about through an aspect of the environment, usually some sort of spatial structure. The second case involves individuals who are able to cooperate upon some condition, either by recognising kin (Tarnita et al., 2009; Jansen and van Baalen, 2006; Riolo et al., 2001;

Traulsen and Schuster, 2003), cooperating on condition of a recipient's reputation (e.g. indirect reciprocity (Nowak and Sigmund, 1998a)) or conditionally upon previous interactions with that individual (e.g. direct reciprocity (Imhof and Nowak, 2010; Axelrod and Hamilton, 1981; Lindgren, 1991)).

1.2 Social Niche Construction

A full explanation of the evolution of cooperation must not only explain how population structure leads to the evolution of cooperation, but must also explain how this population structure came about in the first place. Populations of organisms live in a spatial world; therefore interactions will often be correlated. Because of this many of the current explanations of cooperation take population structure for granted. However, it may be the case that individuals have a genetic component that affects the manner in which they interact with other individuals. It is therefore possible that assortment is itself subject to the principles of evolution and can evolve alongside cooperative behaviours. If assortment can co-evolve with cooperation then it will evolve in such a way as to support cooperation, provided that the population is polymorphic in the cooperative trait. (Powers, 2010; Powers et al., 2011; Ryan et al., 2016). The phenomenon whereby the population structures that support cooperation evolve concurrently with cooperation itself is referred to as *social niche construction* (SNC). The notion of SNC is applied and extended in the papers presented here.

The central notion of *SNC* is that selection may act upon aspects of individuals that affect the social environment, or *social niche*, of an individual. Whether or not cooperation is stable, and whether it is able to invade, depends not only on the cost and benefit of the cooperative act, but on the social environment of the individuals in the population. Generally, this amounts to some form of population structure, whereby individuals interact with a non-random subset of the whole population. The research program of social niche construction is the idea of *endogenising* the population structure into our explanation of cooperation. This involves identifying some aspect of the environment that is conducive to cooperation and asking what would happen if this feature were able to evolve alongside the cooperative trait.

Whilst much progress has been made in investigating and understanding social niche construction, further work needs to be done in order to firmly establish SNC as a solid scientific theory.

Much of the previous work is based largely on simulation models (Powers et al., 2011; Powers, 2010). More rigorous and general mathematical results would greatly add to the field. In particular, it would be beneficial to investigate the outcome of the evolution of assortment over the set of all possible games, under some reasonable set of constraints; such as restricting our attention to two-player, two-strategy symmetric games. In addition, models of social niche construction and the evolution of assortment lack some key parameters that may be important to their outcome. Whilst it is not desirable to include many of the details of a real biological system, it is important to test that claims are robust to variation in key assumptions. To this end the

models of social niche construction should include the addition of a cost to assortment. Whilst the model presented in Powers et al. (2011) did include a cost to assortment, in that individuals were penalised for living in small groups, this cost was left implicit. The evolution of cooperation from the primitive state of all defect relied upon drift, which would be greatly reduces if cost were increased. Therefore, a systematic and explicit investigation of cost in such models would be beneficial. In addition, models of social niche construction could include crossover between the social niche modifier and the social strategy. A key factor in social niche construction is the build-up of linkage disequilibrium, which is often reduced by crossover, therefore, it is important to test the general claims of social niche construction are robust to the inclusion of crossover.

Furthermore, the ideas of social niche construction have not been applied to many real biological systems. This is of central importance, as a theory must ultimately be judged upon the understanding it brings to specific real world systems. Ultimately the theory should make testable predictions about nature. Connection with experimental biology is also important, as often observation can bring to our attention key aspects that we had otherwise neglected.

If positive assortment is an evolvable parameter, then high levels of positive assortment may be stable. However, a freely-mixed population composed of defecting individuals cannot be invaded by an individual with a small value of assortment. Consider a population of freely-mixed defectors playing a prisoner's dilemma, if we introduce a small number of cooperators with a small proclivity to assort, then these cooperators cannot invade such a population. However, if the population is composed entirely of cooperators who assort, then they cannot be invaded by non-assorting defectors. In order to reach the assorted cooperative state the population must cross a fitness valley, therefore the evolution of assortment has difficulty *getting started*. Thus, we cannot give a gradualist account of the evolution of assortment, as, whilst assortment is stable, it cannot invade a population in small quantities. This is an issue that needs to be addressed, and is the subject of the second of the papers of this thesis.

Often the benefits of cooperation come about through the division of labour. Conventional studies of the evolution of cooperation, in the world of evolutionary game theory, label a single strategy as cooperate; in such games the mean fitness of the population is monotonic in the number of cooperators; more is better. However, in many biological systems cooperation is manifest in the interaction of differing types, this can be seen in the interaction between spore and stalk cells in cellular slime moulds (Strassmann et al., 2000), in the differing castes in eusocial insects (Hölldobler and Wilson, 1990) or in the different types of zooids in communal siphonophorae (Dunn et al., 2005; Dunn and Wagner, 2006). In such a scenario we cannot label any one strategy as cooperate, as it is not the case that the mean fitness is monotonic in the number of any one type. A slime mould population composed only of stalk cells would have zero fitness; likewise a population composed only of spores would not be of high fitness. Instead high fitness is obtained by having some intermediate mixture of the two types. *Nonetheless,* there is some manner of cooperative dilemma present here, as one of these two types (the stalk) is more costly for the individual, there is therefore no incentive for an individual to play this

strategy. The typical resolution to cooperative dilemmas is a positive assortment of cooperative strategies. However, assortment, by its very nature, is such that it reduces those interactions that are between unlike types. Therefore, assortment may actually be detrimental to the mean fitness of a population; this is an issue that is addressed at length in the final paper of the thesis.

1.3 The Major Transitions in Evolution

A central application of the concepts of social niche construction is *the major transitions in evolution* (Maynard Smith and Szathmary, 1997; Buss, 1987; Bourke, 2011; Godfrey-Smith, 2009; Jablonka and Lamb, 2006). The thesis of Maynard Smith and Szathmary's book is born from the observation that the biological world is hierarchical in nature. Individuals are formed of collections of cells. Cells are themselves formed of both nuclear DNA and collections of organelles. The nuclear DNA is itself subdivided into chromosomes. These are, in turn, at least roughly speaking, formed of multiple genes. The claim of the major transitions research program is that these lower level entities are the decedents of individuals that were once freely living individuals in their own right. The history of life, at the macro-scale, is characterised by the coming together of individuals into higher level entities. The parts of the higher level entity become co-dependent to such an extent that they are incapable of independent reproduction, we may thus think of these colonies as individuals in their own right.

Selection can act on the lower level entities, which can potentially disrupt the continuity of the evolutionary unit above. This is the problem of "subversion" from within (Queller, 1997, 2000; Bourke, 2011). Inseparable from communal living is the potential for a cooperative dilemma. Individual components always have the option of not contributing to the fitness of the higher level entity and instead gaining a fitness advantage via a more direct route. Many of the characteristics of individuals that we observe in nature can thus be thought of as adaptations that overcome this fundamental problem. Thus, the evolution of higher level entities is actually an example of social niche construction. Understanding SNC is then not simply a topic in ethology (the study of animal behaviour) but is central to understanding the nature of biological individuality (Clarke, 2011).

More generally the central theme of this thesis is in determining which features of the biological world can be understood as adaptations that resolve cooperative dilemmas. This is of interest because it allows us to make some very general statements about evolution and biology. All biological systems are subject to evolution, and all evolutionary systems in which there is social evolution may potentially be subject to the tragedy of the commons (Hardin, 1968) (i.e. a cooperative dilemma). Thus, anything that evolves as a response to the presence of a cooperative dilemma may be thought of as a general biological property, rather than a contingent fact about a particular species in a particular niche.

2 Paper Synopses

Each paper is presented with its own abstract, but here an outline of each is presented with more of an emphasis on their commonalities.

2.1 Paper 1

Game theory, meiotic drive and the evolution of assortative mating

This paper takes the notion of the evolution of cooperation and applies it to a more specific example in biology, namely that of meiotic drive. Meiotic drive is the phenomenon whereby certain alleles cheat Mendel's laws (Lindholm et al., 2016; Sandler and Novitski, 1957; Silver, 1993; Burt and Trivers, 2009). They are thereby present in more than half of the individual's gametes. This is a cooperative dilemma and can be modelled with the machinery of evolutionary game theory (Traulsen and Reed, 2012). We draw on the parallel between assortment in evolutionary game theory and of inbreeding in diploid genetics; showing that these two concepts are isomorphic. The central claim of the paper is that assortative mating can be understood as an adaptation that reduces the efficacy of meiotic drive. Whilst this is an interesting hypothesis in its own right, we are also interested in the fact that there is a complete isomorphism between diploid genetics and evolutionary game theory, and suggest that this would be a fruitful line of inquiry to pursue further.

The paper presents a model of the co-evolution of assortment and cooperation, but with an emphasis on a particular biological system. We recognise that meiotic drive is a cooperative dilemma, which is solved by assortative mating and thereby ask what would happen if meiotic drive were to co-evolve with a preference to mate assortatively. This model is therefore an application of the idea of social niche construction.

2.2 Paper 2

Multiple games and the evolution of assortment

This paper presents a model of the evolution of cooperation, with a particular focus on the co-evolution of assortment and cooperation. This is done in an abstract sense in order to have as wide an applicability as possible.

Assortment is observed in nature. Furthermore, this assortment often comes at a cost to the individual and therefore, I argue, represents an adaptation; i.e. there is, or was, an active selective pressure towards increased positive assortment. However, I show that the concurrent evolution of assortment with cooperation has difficulty "getting started"; in that there is no selective pressure for the evolution of positive assortment unless cooperation already exists in non-zero

frequencies. The problem is particularly apparent when there is a cost to assortment. We present a possible solution to this problem, namely, that the "getting started problem" can be resolved if the individuals engage in multiple simultaneous games.

To investigate this we develop a model involving the concurrent evolution of multiple games being played simultaneously. The notion of multiple games has not been studied in an evolutionary context before. Moreover, we show how the presence of multiple games can lead to the fixation of cooperation even in those games in which cooperation is not present in the well-mixed case. This is a feature that previous models of the co-evolution of cooperation and assortment do not take into account.

The contribution of this paper towards the ideas of social niche construction is in formalising a particular problem with social niche construction, namely the getting started problem, and presenting a potential resolution to this issue.

2.3 Paper 3

Game theoretic treatments for the differentiation of functional roles in the transition to multicellularity

This paper presents an abstract model for the evolution of functional specialisation. It is intended to be a general model for any such system, but, for concreteness, particular reference is made to the early stages of the evolution of multicellularity. This paper argues that the conventional interpretation of cooperation, based largely on Dawesian cooperative dilemmas (Dawes, 1980), in which there is a single cooperative strategy, is too narrow in scope. Particularly the benefits of cooperation come about through the interaction of multiple types, rather than in simply having a large number of cooperators. Thus, as well as presenting a model of the evolution of functional specialisation, this paper attempts to broaden our definition of the notion of a cooperative dilemma by defining a new type of game; which I call division of labour games. Division of labour games are those games in which fitness is maximised by a mixture of complementary types.

In such games interactions between individuals that are of differing strategies are beneficial. This is an issue when one considers the evolution of assortment, as assortment leads to a reduction in heterogeneous interactions. However, phenotypic plasticity is able to resolve the tension between the need for assortment and heterogeneity. Phenotypic plasticity means that individuals can be genetically assorted whilst playing different roles. Understanding phenotypic plasticity in detail is important for our account of the evolution of assortment, particularly as applied to the major transitions in evolution. Phenotypic plasticity is essential for the evolution of cooperation when considering division of labour games and hence the model in this paper can be viewed as a manner of social niche construction, where it is the plasticity that is evolving alongside cooperation, rather than assortment.

Chapter 2

Game Theory, Meiotic Drive and the Evolution of Assortative Mating

Abstract

There exists an exact correspondence between the game theoretic study of two-player games and the population genetics of diploid individuals. This isomorphism is particularly useful when one considers the dynamics of meiotic drive; in which case the population genetics is described by a family of well-known cooperative dilemmas. Cooperative dilemmas are typically resolved by positive assortment on cooperative strategies. We extend upon previous studies of the isomorphism between evolutionary game theory and diploid genetics by showing that the coefficient of inbreeding, F is equivalent to α, the value of assortment typically used in evolutionary game theory. We thus show that populations with assortative mating will be less susceptible to meiotic drive. We take recent models of the concurrent evolution of assortment and cooperation in game theory and apply them here, asking whether inbreeding can be partially understood as an adaptation that mitigates the effects of meiotic drive. We solve the evolutionary dynamics of such a co-evolutionary situation and find that assortative mating preferences can indeed evolve as a response to the presence of meiotic drive. This effect is strongest at intermediate levels of drive efficacy and low levels of dominance and is robust to varying level of crossover. However, assortative mating preferences can never reach fixation if they come at a cost to the individual and will always coexist with some non-zero level of meiotic drive. We discuss the hypothesis that such dynamics are one of a number of reasons for the wide spread prevalence of assortative mating in nature and the relative scarcity of meiotic drive.

1 Introduction

Evolutionary game theory is the study of the evolutionary dynamics of individuals who engage in strategic interactions (Weibull, 1997; Hofbauer and Sigmund, 1998). Typically it is the phenotype of the individual that constitutes the strategy of the game. Population genetics, on the other hand, is the study of the change in frequency of genes within a population in terms of the fitness effects they have on the individual in which they reside (Hartl and Clark, 1998). Recently, however, a number of authors have noted that there exists a complete isomorphism between the study of two-player evolutionary games and the population genetics of diploid individuals (Traulsen and Reed, 2012; Bohl et al., 2014; Gardner et al., 2007; Chastain et al., 2014). The correspondence between these two theories exists only if we consider the alleles, not the organism in which they reside, as the strategies in the game. This analogy is useful in so far as it allows us to transfer results and intuitions from one field into the other.

Cooperative dilemmas have been a major area of study in evolutionary game theory (Nowak and May, 1992a; Nowak and Sigmund, 1998a; Lindgren, 1991; Santos et al., 2006b; Hauert, 2004). A game is considered a cooperative dilemma if there is a strategy, cooperate, for which the population's welfare would be maximised if every agent performed this strategy, but for which, at the individual level, there is nonetheless an incentive not to perform the cooperative action (Dawes, 1980) (however see Tudge et al. (2016b) and Stark (2010) for an important caveat). There is thus conflict between what is good for the population, and what is good for the individual. Positive assortment of strategy is one mechanism by which the interests of the individual may become aligned with that of the population (Eshel and Cavalli-Sforza, 1983; Sober, 1992; Fletcher and Zwick, 2006; Godfrey-Smith, 2008; Michod and Sanderson, 1985). Here positive assortment means that each phenotype is more likely to meet another individual of its own type than would be expected if individuals were to meet at random.

In fair (i.e. Mendelian) meiosis, (see inset below), each of the two copies of a gene has an equal chance of transmission to the gametes. In a diploid individual, if meiosis is fair, then the game that the alleles play can never be a cooperative dilemma. The only way in which an allele can increase its representation in the next generation is by contributing to the fitness of the individual in which it resides. The appropriate parallel to cooperative dilemmas in diploid genetics is the study of meiotic drive. If an allele is able to affect its probability of transmission to the gametes then the allele is said to be a meiotic distorter. If, in addition to this, it has a detrimental effect on the fitness of the individual, perhaps only when the individual is a homozygote for the meiotic distorter, then the allele can be thought of as a defector in a cooperative dilemma. The allele that does not attempt to over-represent itself in the gametes can be viewed as the cooperative strategy. Clearly the individual itself is better off in the absence of meiotic distorters, but the meiotic distorter alleles will still increase in frequency, hence the situation fits the definition of a cooperative dilemma, see also table 2.1.

Meiotic Drive

Meiosis is said to be *fair* whenever Mendel's laws are obeyed, that is when both of the homologous alleles of a diploid organism are present in equal ratios in the gametes. An allele that violates these laws is said to be a meiotic distorter; these alleles are present in more than fifty percent of the gametes of the individual. This can happen in both males and females and across many varying taxa (Lindholm et al., 2016). Meiotic distorters (Sandler and Novitski, 1957), or more generally selfish DNA, are genetic elements that are able to increase in frequency in a more direct way, which is often detrimental to the fitness of the organism (Fishman and Kelly, 2015; Lewontin and Dunn, 1960). This may be by killing sperm that do not contain the allele in question and thereby reaching the egg more than half of the time (Presgraves, 2009), by being copied into the corresponding locus on a partner chromosome (e.g. homing endonuclease genes) (Gimble and Thorner, 1992), by moving directly to the germ line of the individual, or through over replication, such as in B-chromosomes and transposable elements (Lynch and Walsh, 2007), as well as in organelles such as mitochondria, see (Rice, 2013). Two well-studied examples are: segregation distorters in drosophila (Larracuente and Presgraves, 2012) and the *t-haplotype* in mice (Silver, 1993), see Burt and Trivers for an extensive review of meiotic drive (Burt and Trivers, 2009) and (Lindholm et al., 2016) for a briefer and more recent review. Meiotic distorters are interesting to evolutionary biology because they represent a situation in which natural selection inarguably operates on something other than the individual. Once one realises that selection acts not only on individuals, but also directly on genes, then the logic of meiotic distorters becomes clear. A meiotic distorter may make the individual in which it resides less fit. This loss is more than made up for by the additional relative increase in the gametes of the offspring, the precise dynamics of the situation depend upon the exact ratio of these quantities. (See also Okasha (2015) for a discussion of meiotic drive in the context of multi-level selection and kin selection.)

The major problem in understanding meiotic drive lies not in explaining its existence, but in explaining its scarcity. Why are individuals able to exist at all given the constant potential for disruptive selection at the lower level? Selection can potentially act at other loci in favour of other genes that suppress meiotic distorters (Leigh, 1971). This is thus an example of cooperation enforced through policing mechanisms (see for example Frank (1995)). However, less well understood is the role that inbreeding has on the reduction of meiotic distorters. A number of authors have noted the importance of sexual systems on the prevalence of meiotic distorters (Giraldo-Perez and Goddard, 2013; Weissing and van Boven, 2001; Lenington et al., 1994). Burt and Trivers (1998) demonstrate, through a population genetics model, how inbreeding reduces meiotic drive. What is less clear, however, is an actual conceptual understanding of this result. In re-deriving this result through game theory we are able to massively reduce its mathematical complexity. More importantly, we give the following conceptual understanding and connect the result to existing theory in social evolution. Both Traulsen and Reed (2012) and Sarkar (2016) have studied the evolutionary dynamics of meiotic drive through the lens of evolutionary game

theory. However, neither have made the connection between inbreeding in diploid individuals and the role that assortment has on overcoming cooperative dilemmas.

Assortment can remove or lessen the severity of a cooperative dilemma. In the analogy we are drawing here assortment corresponds to an allele being paired with a like allele more often than would be expected if pairings were random. The amount of assortment at the allelic level is exactly the inbreeding coefficient (see for example Hartl and Clark (1998)). It is determined by measuring the extent to which a population deviates from the Hardy-Weinberg frequency. One way in which inbreeding may come about is the violation of the assumption of random mating, which is the main mechanism we consider here. However, note that there are many other ways in which a population can exhibit non-zero inbreeding, such as non-addative mortality rates in heterozgotes (Hartl and Clark, 1998). At this point the power of the analogy between diploid population genetics and two-player game theory becomes apparent. It is clear that inbreeding should lower the prevalence of meiotic distorters in a population in the same manner as the imposition of assortment of strategies lowers the level of defection in a population facing a cooperative dilemma.

If one assumes the presence of assortment, *a priori*, one has only partially explained the presence of cooperation. A full explanation would give an account of why assortment came about in the first place. Some more recent studies in evolutionary game theory have looked at the concurrent evolution of social strategies and properties that affect the assortment of those strategies. They find that, at least in some games, population structure will co-evolve in a way that facilitates the evolution of cooperation (Powers et al., 2011; Jackson and Watson, 2015; Fort, 2008; Akçay and Roughgarden, 2011). Given that assortative mating can reduce the efficacy of meiotic drive, i.e. lead to cooperation at the level of the gene, this then presents us with the hypothesis that assortative mating is an adaptation that reduces the effects of meiotic drive. That is, if genetic preferences for assortative mating were able to co-evolve with a gene that may or may not cheat meiosis, then selection will act in a way that leads to an increase in assortative mating and hence a reduction in meiotic drive.

Breden and Wade (1991) show, through a two locus model, that selection will favour an allele that causes a tendency to inbreed when coupled with a kin selection model of the evolution of cooperation. Peck and Feldman (1988) show that a tendency to mate monogamously will also coevolve with altruistic behaviours. In both cases these models show that when a population structuring trait can coevolve with a trait for altruism that is polymorphic, then selection will favour the trait that increases population structure, in these cases embodied through relatedness. The present study concurs with these two studies, but the focus here is on intra-genomic cooperation, rather than on cooperation between differing biological individuals.

Assortative mating is any non-random pattern of mating between members of a species (Lewontin et al., 1968). Random mating occurs whenever the genetic covariance of mating pairs is zero, and can be reached by pairing a random male with a random female. We employ the more specific use of the term, however, that implies that assortative mating is the phenomenon whereby

individuals mate with other individuals who are statistically more similar to themselves than a random individual would be. Note that we assume no conscious effort on the part of the individual, and are agnostic as to the method by which this may come about. Assortative mating leads to a positive coefficient of inbreeding (Hartl and Clark, 1998; Lynch and Walsh, 1998), which simply measures the excess of homozygotes relative to that expected by random chance alone. Assortative mating is common in nature across a wide range of *taxa* (see Jiang et al. (2013) and references therein), including humans (Guo et al., 2014; Bittles et al., 1993). A recent meta-analysis suggests that positive assortative mating is much more common than negative or *disassortative* mating, which may be almost non-existent in animals (Jiang et al., 2013). Assortative mating may be non-adaptive in many cases. Structure in populations may come about through spatial segregation, which would likely lead to assortative mating as a bi-product (Crespi, 1989; Arnqvist et al., 1996; Helfenstein et al., 2004). Alternatively, competition for mates could lead to the fittest females selecting the fittest male, and thus there would be assortative mating as a bi-product, when what is really being selected is simply a mating preference for inherently fit individuals (Crespi, 1989; Henry, 2008). A number of other hypotheses exist for how assortative mating may be an adaptation. All such explanations rely on there being some form of disruptive selection in action (Kirkpatrick, 2000; Gavrilets, 2004; Arnqvist, 2011), whereby extreme phenotypes are fit, but intermediate ones are less so. Assortative mating maintains variance and therefore decreases the production of intermediate phenotypes that are less fit than either extreme (Lynch and Walsh, 1998). Whilst in some cases assortative mating may come about as a bi-product, it has been shown that it is at least sometimes adaptive (Bonneaud et al., 2006; Ortego et al., 2009). We suggest a hypothesis for why this might be the case, namely that if genetic outlaws exist it pays to create assortment in order to mitigate the effects of meiotic distortion. The hypothesis that we propose is in no way mutually exclusive with other explanations, it simply states that this is an additional force for the evolution of assortative mating.

The structure of the rest of the paper is as follows: we begin by reviewing the salient points regarding evolutionary game theory. We then briefly review the notion of meiotic drive and go on to show how this can be framed as an evolutionary game. We demonstrate that the inbreeding coefficient is equivalent to the measure of assortment, α, that is typically used in evolutionary game theory (see van Veelen (2011)). Finally, we present a model of the concurrent evolution of assortative mating and fair meiosis, this is split into two separate cases: the tightly linked case and the case in which there is crossover.

2 Evolutionary Game Theory and Diploid Genetics

In this section we briefly review the evolutionary game theory of two-player cooperative dilemmas and the notion of assortment.

The TS-plane

Any two-player, two-strategy, symmetric game can be represented by the payoffs given by:

$$M = \begin{pmatrix} a & b \\ c & d \end{pmatrix} \tag{2.1}$$

which represents the payoff the column-player receives on meeting the row-player. If the game is well mixed, i.e. players meet at random, "A" players receive an average payoff given by: $\pi_A = ax_A + bx_B$, where x_A/x_B are the frequencies of As/Bs. Likewise Bs receive a payoff given by: $\pi_B = cx_A + dx_B$. The mean payoff, $\bar{\pi}$, is given by: $\bar{\pi} = x_A\pi_A + x_B\pi_B$. Typically, it is assumed that the total size of the population is fixed and one may set $x_A = x$ and $x_B = 1 - x$. The change in frequency of type A in the population is given by the replicator equation (Taylor and Jonker, 1978):

$$\dot{x} = x\left(\pi_A - \bar{\pi}\right) \tag{2.2}$$

A standard result from evolutionary game theory is that the replicator dynamics remain unchanged upon addition to the payoff matrix by a constant and by multiplication by a positive constant (Nowak, 2006a; Tudge and Brede, 2015), in other words an affine transformation of the payoff matrix. Thus, if $a > d$ we can transform the above matrix into a standard form, removing two arbitrary degrees of freedom:

$$\begin{pmatrix} a & b \\ c & d \end{pmatrix} \Rightarrow \begin{pmatrix} \frac{a-d}{a-d} & \frac{b-d}{a-d} \\ \frac{c-d}{a-d} & \frac{d-d}{a-d} \end{pmatrix} \Rightarrow \begin{pmatrix} 1 & S \\ T & 0 \end{pmatrix} \tag{2.3}$$

where $S = \frac{b-d}{a-d}$ and $T = \frac{c-d}{a-d}$. Thus, any two-player game can be represented as a point on the TS-plane (Santos et al., 2006b). If $S + T < 2$ then the population's mean fitness will be maximised if every individual is an A. If in addition either $S < 0$ or $T > 1$ then there is some incentive to play B. In which case the game is a cooperative dilemma and strategy A is labelled C for cooperate and B is labelled D for defect.

Analysis of the properties of these games comes from analysing the fixed points of the replicator equation. Cooperation is stable against invasion by defection if $T < 1$, and defection can be invaded by cooperation if $S > 0$. Four fundamental games are represented in the TS-plane, they are:

1. $T > 1$ and $S > 0$: the snowdrift game; the ESS contains a mixture of cooperate and defect. The single stable fixed point, x^*, is given by:

$$x^* = \frac{S}{(S + T - 1)} \tag{2.4}$$

2. $T >= 1$ and $S <= 0$: the prisoner's dilemma; total defection is the only stable fixed point ($x^* = 0$).

3. $T < 1$ and $S < 0$: the stag-hunt game; both defection and cooperation are stable ($x^* = \{0, 1\}$). Which one is reached depends on the initial conditions. The tipping point, x_T, is given by:
$$x_T = \frac{S}{(S + T - 1)} \tag{2.5}$$

4. $T <= 1$ and $S >= 0$: the harmony game; only total cooperation is stable ($x^* = 1$). This is not a cooperative dilemma.

Assortment

In evolutionary game theory we can mathematically model the role of assortment by noting that: a game given by payoff matrix, M, under a level of assortment, α, is dynamically equivalent to a payoff matrix, M', under no assortment, such that the elements of M' are given by:

$$M'_{ij} = \alpha M_{ii} + (1 - \alpha) M_{ij} \tag{2.6}$$

We can thus understand the effects of assortment as an effective transformation of the underlying game (van Veelen, 2011). In a more general formulation we can specify the payoff of an individual i in terms of the payoff matrix M and a matrix of conditional probabilities P, where P_{ij} specifies the probability of meeting an individual of type i given that one is of type j (van Veelen, 2011). The payoff to an individual is thus:

$$\pi_i = \sum_j M_{ij} P_{ij} \tag{2.7}$$

For two-player games we can define the matrix P with a single parameter α, as above. The rows and columns of P must sum to one, meaning there is only one degree of freedom. In which case:

$$P_{ij} = \alpha \delta_{ij} + (1 - \alpha) x_j \tag{2.8}$$

Here δ is the Kronecker delta matrix, the values of which are equal to one when the indices are equal and zero otherwise. The matrices M and P completely define the dynamics of a game with population structure.

2.1 Diploid Genetics as a Two-Player Game

In this section we illustrate how the population genetics of meiotic drive can be construed as an evolutionary game and show that the inbreeding coefficient, I, is isomorphic to assortment, α.

Consider two alleles, C and D, which may occupy a particular locus in a genome. Here D, for defect, represents the meiotic drive allele and C, for cooperate, the *fair* allele, i.e. one that does not distort meiosis. There are thus 3 potential genotypes: DD, DC and CC. The respective fitnesses are labelled by: ω_{00}, ω_{01} and ω_{11}. (Note, we are arbitrarily labelling defect

as zero and cooperate as one for convenience). Following Traulsen and Reed (2012), we do not consider fitness at the level of the individual, but at the level of the allele. In each generation an allele is paired with another allele to form a diploid organism, syngamy is thus the equivalent of the meeting of two agents in a game. Traulsen and Reed (2012) show that the standard Fisher-Wright process used to model this situation is equivalent to the replicator dynamics, if we take the allele's marginal fitness as the fitness that appears in the replicator equation (see also Bohl et al. (2014)). The fitness of an allele is the fitness of the individual in which it resides, multiplied by the probability of being represented in the gametes of the individual. Under fair meiosis this probability is always one half. However, meiotic distorters, by definition, are present in gametes with a frequency greater than one half. We use the parameter δ, which we call *drive efficacy*, to quantify the effectiveness of the meiotic distorter allele. δ linearly interpolates between the two extremes in which meiosis is fair ($\delta = 0$) and in which cheating meiosis is one hundred percent effective ($\delta = 1$), i.e. an allele that, in heterozygotic form, is present in all gametes. We assume that $\omega_{11} > \omega_{00}$, that is an individual composed of two meiotic distorters is less fit than one which is free of meiotic distorters. We assume also that $\omega_{11} \geq \omega_{01} \geq \omega_{00}$, that is the heterozygote can be as fit as either homozygote, or anywhere in-between. Typically in population genetics the three genotype fitnesses are parametrised as follows: $\omega_{11} = 1$, $\omega_{01} = 1 - hs$ and $\omega_{00} = 1 - s$. Where s is the *selection coefficient* and parametrises the difference in fitness of the two homozygotes and h is the *degree of dominance* (Hartl and Clark, 1998). h defines the degree to which the heterozygotic fitness differs from the mean fitness of the two homozygotes. $h > 1/2$ means that the heterozygote is more fit than the mean of the two homozygotes, and conversely $h < 1/2$ refers to the case in which the heterozygote is less fit that the mean of the two homozygotes. The alleles engage in the game shown in table 2.1.

	C	D
C	1	$(1-\delta)(1-hs)$
D	$(1+\delta)(1-hs)$	$1-s$

TABLE 2.1: The matrix of diploid games under standard population genetics parametrisation.

We can perform the standard normalisation of games onto the TS-plane:

$$S = \frac{(1-\delta)(1-hs) - (1-s)}{s} \tag{2.9}$$

$$T = \frac{(1+\delta)(1-hs) - (1-s)}{s} \tag{2.10}$$

Note that if $\delta = 0$ then $S = T$. This is a so called partnership game (Weissing and van Boven, 2001), in which there is no conflict, and represents a line on the TS-plane. We can thus represent

all possible two-player games as points in the $h\delta$-plane, in an equivalent manner to the TS-plane. The equilibria of these games are shown in figure 2.1. Note that the parameter s sets the scale of the game, and represents a simple linear transform of the space. Thus, we may understand the full set of dynamic possibilities without varying s.

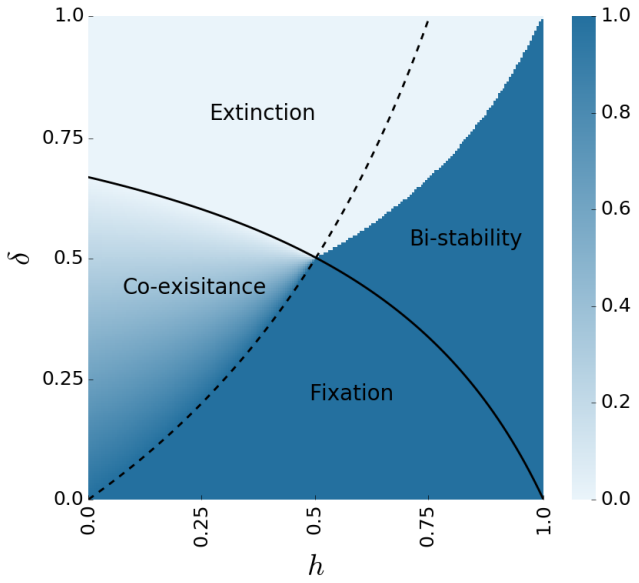


FIGURE 2.1: The diploid universe. The outcome of selection for two alleles, one of which may be a meiotic distorter. Either the 'fair' allele goes extinct (equivalent to the prisoner's dilemma game), reaches fixation (harmony game), coexists (snowdrift game), or the system is bi-stable, whereby one of the alleles will reach fixation, but which one depends upon the initial conditions (stag-hunt game). This is in exact correspondence to the space of all possible two-player cooperative dilemmas. Note that, here, $s = 2/3$.

As previously noted, the action of assortment can be seen as an effective transformation of the underlying game. This transformation is shown schematically in figure 2.2, and can be mathematically represented as the following transformation equations:

$$h' = (1-\alpha)\,h + \alpha/2 \tag{2.11}$$

$$\delta' = \frac{2\delta\,(1-\alpha)\,(1-hs)\,\alpha s}{2\,(1-hs) - \alpha\,(1-2h)\,s} \tag{2.12}$$

The action of assortment is to move games towards the game $h = 1/2$, and towards the *fair* game in which $\delta = 0$.

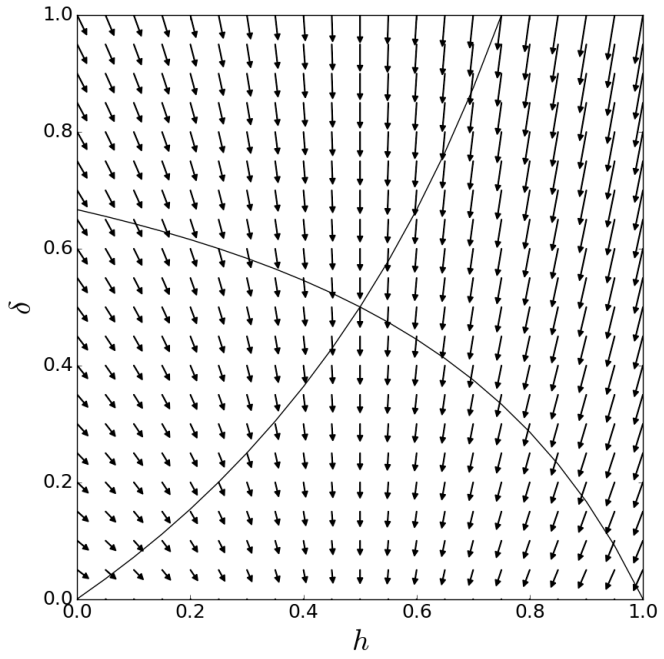


FIGURE 2.2: The effective transformation of h and δ when one considers the action that assortment has on the underlying game. The tail of each arrow lies on a particular game given by h and δ, and the head of the arrow is the effect of the transformation given by equations (2.11) and (2.12), with $\alpha = 0.025$.

Inbreeding and Assortment

In population genetics the coefficient F measures the extent to which a population is inbred. If there are two alleles in a population, A and B, with frequencies given by x_A and x_B respectively, then the Hardy-Weinberg frequency for heterozygotes is given by $2x_Ax_B$ (see any text on population genetics, e.g. Hartl and Clark (1998)). F measures the extent to which the population deviates from this frequency. If the measured number of heterozygotes is given by f_{AB} then:

$$F = 1 - \frac{f_{AB}}{2x_Ax_B} \tag{2.13}$$

On the level of description that takes the allele as the agent in a game F plays the role of assortment. It is the extent to which alleles meet alleles of the same type above which would be predicted at random. In a population of interacting agents the frequency of interactions that are between unlike types is $f_{AB} = x_AP_{AB} + x_BP_{BA}$, using equation (2.7) it follows that:

$$\begin{aligned} f_{AB} &= x_A\left(1-\alpha\right)x_B + x_B\left(1-\alpha\right)x_A & (2.14)\\ &= 2\left(1-\alpha\right)x_Ax_B & (2.15) \end{aligned}$$

and hence that:

$$\alpha = 1 - \frac{f_{AB}}{2x_Ax_B} \tag{2.16}$$

It is clear that $\alpha = F$. Given what is known about the effects of assortment on cooperative dilemmas it should then be immediately clear that inbreeding acts to suppress meiotic distorters.

Using equation (2.6) and (2.1) one can write down the effects a level of inbreeding F has on the game, see table (2.2).

	C	D
C	1	$(1-\delta)\,y + F$
D	$(1+\delta)\,y + F$	$1 - s$

TABLE 2.2: Diploid game under the effects of inbreeding, where $y = (1-F)(1-hs)$.

Again, this can be transformed to the ST parametrisation following the standard procedure:

$$S = \frac{(1-\delta)(1-F)(1-hs) + F - (1-s)}{s} \tag{2.17}$$

$$T = \frac{(1+\delta)(1-F)(1-hs) + F - (1-s)}{s} \tag{2.18}$$

In order for cooperation to be stable, that is for a meiotic driver allele to be unable to invade, we require that $T < 1$. We can write this condition in terms of a minimum level of inbreeding needed to suppress a given strength of meiotic drive:

$$F > \frac{\delta}{\delta + s} \tag{2.19}$$

Likewise, for cooperators to be able to invade, that is for a non-meiotic distorter to invade a population of meiotic distorters, we require that $S > 0$ and hence:

$$F > \frac{1 - s - (1-\delta)\,(1-hs)}{(1-\delta)\,(1-hs) + 1} \tag{2.20}$$

Equations (2.19) and (2.20) together summarise the key results for the evolution of meiotic drive with inbreeding, by stipulating the minimum level of inbreeding that is needed in order that: (A) meiotic drive cannot invade a population and; (B) that a population of meiotic distorters can be invaded by a fair allele.

3 The Evolution of Assortative Mating

We have established that a given level of inbreeding will lead to a reduction in the level of meiotic drive. We now go on to investigate whether the presence of meiotic drive can cause an adaptive pressure towards an increase in the levels of inbreeding via a preference for assortative mating. For this purpose we present an abstract model, which is analysed both mathematically and via simulation.

We consider a population of diploid individuals with two loci, one of which potentially cheats meiosis, and another that determines the tendency to mate assortatively. These can be interpreted as a social trait and a social niche modifying trait respectively, see Powers et al. (2011); Powers (2010); Ryan et al. (2016). We analyse the evolutionary dynamics of the underlying game via the corresponding replicator equation. This is analysed in two ways, firstly through numerical integration and secondly through stability analysis of the fixed points. These two approaches show near perfect agreement. The exact mathematical form of these solutions are complex and the details are left to appendix A. We also complement this with an agent-based simulation model, which supports these results, the details of which are left to appendix C.

Assortment could be modelled as a continuous parameter or a discrete choice. However, the simulation model with continuous strategies shows that this choice is unimportant. Furthermore, this result is backed up by a result from evolutionary game theory that states that a bi-population model will never show polymorphism if the fitness of members of population A are only determined by their interaction with members of population B (see Hofbauer and Sigmund (1998) for a proof). This case pertains here, as we may think of a bi-locus model as selection occurring between two interacting populations (see also Gardner et al. (2007)), and furthermore the allele for assortment only affects the fitness of the individual via interactions with the other locus. We therefore restrict our attention to discrete values of assortment.

The model proceeds as follows. We initialise a population of diploid individuals. These individuals have two loci, one of which, a, controls the tendency to mate assortatively, this allele may take the value of either 0 (i.e. mate at random), or 1, (i.e. always attempt to find a mate with the same genetic value as oneself). The second gene, m, determines whether or not the allele distorts meiosis. This may either take on the value of 0 (i.e. defect, try to cheat meiosis) or 1 (cooperate, don't cheat meiosis). The fitness of the individual is determined by the locus m, such that the homozygote with $m = 1$ has a fitness of 1. The heterozygote has a fitness of $1 - hs$, and the homozygote with $m = 0$ has fitness $1 - s$. In addition, individuals incur a cost for having a tendency to assort. There are many reasons why assortative mating may incur a cost to the individual, such as an energetic cost to finding a suitable mate, or inbreeding suppression (Charlesworth and Charlesworth, 1987). Here we include a generic cost to assortment in order to account for all such possibilities. The cost incurred to an individual is $1/2c\,(a_1 + a_2)$, where 1 and 2 index the homologous (i.e. the maternal and paternal) alleles of the individual.

We then proceed by selecting individuals for the next generation by fitness proportionate selection. The total number of individuals in the population is N. We select $2N$ individuals with replacement. Each of these individuals produce one gamete, so that the total size of the population is conserved. The gametes are selected not uniformly at random (i.e. according to Mendelian laws), but according to the rules of meiotic distortion. A heterozygotic individual will produce a gamete with $m = 0$ with probability $1/2(1 + \delta)$ (and therefore a gamete with $m = 1$ with probability $1/2(1 - \delta)$).

The extent to which crossover occurs is parametrised via k, where $k = 0$ corresponds to the complete absence of crossover, and $k = 1$ corresponds to the case where the two genes are inherited statistically independently. If the allele m_1 is selected, then with probability $1 - k$ allele a_1 is selected, and with probability k the allele at the a locus is selected at random.

Now that $2N$ alleles have been selected they are paired to form the next generation of individuals. For this purpose we split the population into two equal halves at random. We call the sub-populations, somewhat arbitrarily, male and female. We go through each female in turn, if the value of $a = 1$ then we pair that gamete with a male gamete that has the same value of m. Note that we do not specifically pair the gamete with another male with the same value of a, so that assortment is purely on the meiotic drive locus. If there are no males left with the necessary value of m we pair the female with a random individual. If the female gamete has a value of $a = 0$ then we pair it with a male with a random value of m.

Note that our model concerns the maintenance of fair meiosis, rather than its origins. We are studying the case in which fair meiosis has already been established, but is susceptible to the invasion of cheating mutant alleles. Tudge et al. (2016a) study the conditions under which assorting cooperators can invade a primitive population of freely mixed defectors.

This entire process of individual formation, selection, and gamete production is repeated for a predetermined number of generations. We present a mathematical model for the situation described, and complement this with a full simulation model in appendix C. The next two sections look at, respectively: the case of zero crossover, for which there is an analytical solution and the more general case of non-zero crossover, for which we present numeric solutions.

3.1 Tightly Linked Model

If the two genes a and m are tightly linked, and therefore do not undergo crossover, then we can model the situation simply by considering the haplotype as the agent in the game. There are thus four strategies, which we label by the values of their genes at loci a and m. The four individuals are thus: $(0, 0)$, $(0, 1)$ $(1, 0)$ and $(1, 1)$. The state of the population is specified by the frequencies of each of the four strategies, which we denote with $X_{i,j}$. Note also that the sum of all frequencies must equal one, effectively reducing our system to three variables.

We define $P(i|a, m)$ to be the conditional probability of meeting an individual with $m = i$, given an individual of type (a, m). The exact manner in which $P(i|a, m)$ is calculated is left to appendix A. With an expression for $P(i|a, m)$ we can construct a replicator equation. We numerically integrate the replicator equation until a stationary state is reached. We start the population from random initial conditions when performing the numerical integration, and find that in all cases the integration reaches the unique stable points of the replicator equation. In addition, we perform a stability analysis, the details of which are left to appendix A. Both approaches show excellent agreement.

In the absence of crossover, depending upon the game parameters h and δ, there are four qualitatively different evolutionary outcomes. In addition, the location of these regions on the $h\delta$-plane depend upon both s and c. These four regions are illustrated in figure 2.3. Here we show both the fairness (which we define simply as the mean value of m in the population, that is the extent to which the population does not cheat meiosis) and the mean level of assortative mating in the population. This is further illustrated in figure 2.4 in which we fix the value of h ($= 0.2$) and sweep through values of δ and show the equilibrium level of each of the four strategies. In both figures we use the standard parameter values of cost (c) of 0.1 and $s = 2/3$.

The strategy $(1, 0)$ is dominated in all games, as it is a defector that pays a cost to seek out other defectors. It thus does not feature in the equilibrium in any case. The space can be divided into four different regions of behaviour, which are described below (with reference to figure 2.3):

1. Region D, which covers the harmony and stag-hunt games. Cooperation dominates even in the well-mixed case. There is thus no evolution of assortative mating. Note also that cooperation fixates even in the case of the stag-hunt game, this is unlike the well-mixed case. This is because the strategy $(1, 1)$ initially increases in frequency, this in turn allows the strategy $(0, 1)$ to invade. This strategy does better than the $(1, 1)$ when defectors are scarce, as it does not incur the cost of assortment. Once $(0, 1)$ begins to invade it replaces $(1, 1)$ and thus reaches fixation. At this point defect cannot invade. The strategy $(1, 1)$ does not appear in the equilibrium, but it paves the way for co-operation, and makes defection unstable.

2. Region C, which exists in the lower region of the snowdrift game. Defection and cooperation coexist. Defection exists in sufficiently low frequencies that it does not pay to assort (as this comes at a cost) and this solution is in fact identical to the well-mixed case.

3. Region B, which corresponds to the upper part of the snowdrift game and the lower part of the prisoner's dilemma. Defection is at higher frequencies in the well-mixed case, thus assortative cooperators can invade. However, as cooperation increases so does the temptation to defect. We thus see the coexistence of all three strategies (excluding the dominated strategy $(1, 0)$).

4. Region A, which exists in the upper left section of the prisoner's dilemma. The temptation to defect is greater here so that the strategy to cooperate without assorting is dominated by the strategy to cooperate and assort. We thus have a coexistence of the strategies $(1, 1)$ and $(0, 0)$.

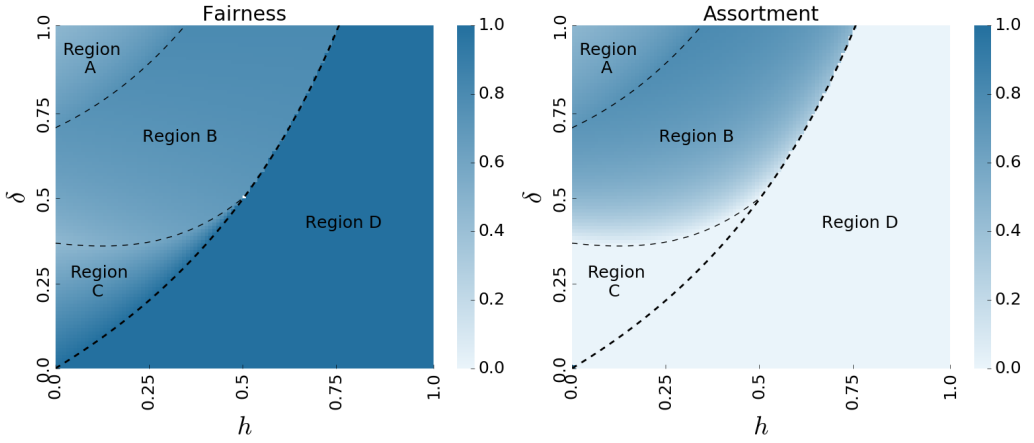


FIGURE 2.3: The $h\delta$-plane for the case of no crossover, in which four regions of different evolutionary outcomes are indicated. The panels show both the fairness (i.e. mean value of m, left) and the assortment (mean value of a, right).

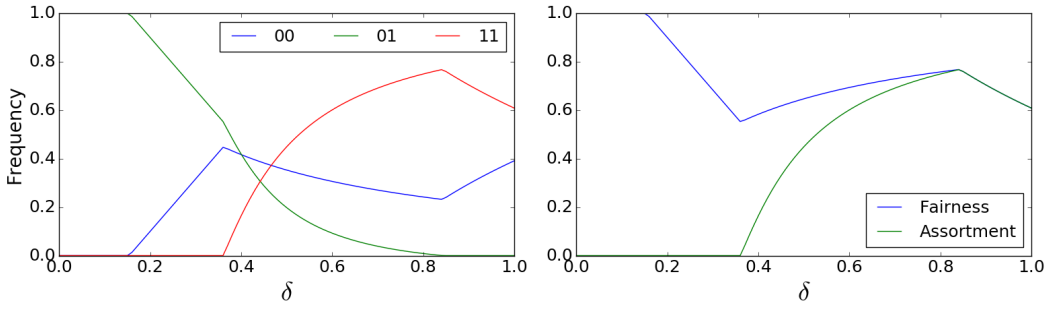


FIGURE 2.4: A sweep across δ, with $h = 0.2$ and the resulting equilibrium of all the strategies involved. The left panel shows the equilibrium frequency of each strategy, whereas the right panel shows the fairness and assortment corresponding to these equilibria. The figures are the result of the stability analysis presented in appendix A. Here cost, $c, = 0.1$ crossover, $k, = 0$ and $s = 2/3$.

Figure 2.4 shows a sweep through δ, for a fixed value of h (0.2). This value of h is chosen in order to pass through all four regions with different evolutionary outcomes. These figures clearly show that a genetic preference for assortative mating may invade in a large region of the game space. This is provided that the efficacy of the meiotic drive allele, δ, is large enough. However, if this assortment comes at a cost a population comprised of only assort-cooperate may always be invaded by the strategy to cooperate without assorting. This in turn means that the defect strategy can invade, leading to the coexistence of all three strategies. Note also that the level of fairness and assortative mating are maximised by intermediate levels of drive efficacy. If efficacy is low then unfair alleles exist in low frequencies, it therefore does not pay to mate assortatively if this comes at a cost to the individual. Likewise, if efficacy is high it is less beneficial to mate assortatively; as there are too few fair alleles for this to be an effective strategy.

3.2 The Effects of Crossover

Next, we analyse the effects that crossover have on the equilibrium levels of fairness and assortative mating. The presence of crossover means that we can't consider the haplotype as the unit of selection, as it is potentially broken up by crossover in each generation. Nonetheless, we proceed by defining the state of the population to be the frequency of each variety of haplotype. We then calculate the expected frequency of each type of individual given the frequencies of each type of haplotype and the previously described assortment mechanism. We then calculate the expected frequencies of each type of individual after one round of selection. Finally, we calculate the expected frequency of each type of haplotype given one round of gamete production. This leads to a form of differential equation that we can integrate through time. The exact details are left to appendix B.

Figure 2.5 is equivalent to figure 2.3 for the case of crossover (k) equal to one. The situation is slightly more complex with crossover, as the previously neglected strategy $(1, 0)$ may be recreated through crossover between $(1, 1)$ and $(0, 0)$. Unlike the case of zero crossover in this model there are only three qualitative regions of differing evolutionary outcomes, see figure 2.6 for a clear depiction of this. These three region are: Region C: the trivial one in which cooperation dominates (i.e. the harmony and stag-hunt game); region B: in which there is no assortative mating and cooperation and defection coexist; and region A: in which all four strategies co-exist. The general statement that assortative mating will only be observed above a critical value of drive efficacy remains true. However, it is no longer the case that assortative mating is maximised for an intermediate level of drive efficacy.

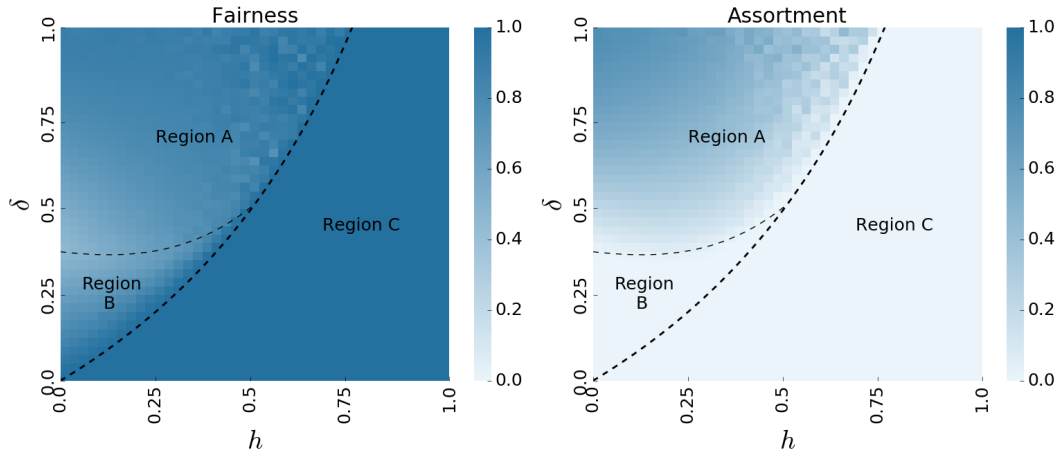


FIGURE 2.5: Phase space with crossover between the two loci set to one, showing both fairness and assortment over the $h\delta$-plane. Here $c = 0.1$, $k = 1$ and $s = 2/3$. Points are the result of numerically integrating the equations of motion from random initial conditions until stability is reached.

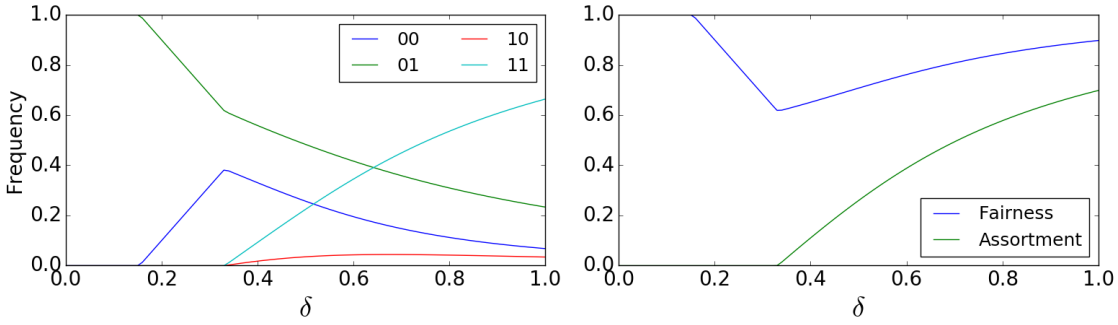


FIGURE 2.6: Sweep through δ with crossover. The lines are the result of numerical integration of the differential equations until fixation is reached. Each run starts from uniform conditions, in which the frequency of each strategy is $1/4$. Here $h = 0.2$, $c = 0.1$, $k = 1$ and $s = 2/3$. The left panel shows the equilibrium value of each of the four possible haplotypes and the right panel shows the corresponding level of fairness and assortment.

Interestingly, crossover can actually increase levels of cooperation; that is suppress meiotic drive. This is surprising as crossover acts to decrease the level of linkage disequilibrium (LD) and the evolution of fairness depends upon the build-up of LD between the assortative mating allele and the fair ($m = 1$) allele. Compared with the case of no crossover the strategy $(0, 1)$ does well. Although this strategy may not be as fit as the other strategies, in the sense of the expected number of offspring, it is more likely to produce a fit offspring. This is because it is the only strategy that cannot have an offspring that is of the worst possible strategy $(1, 0)$ (as it is the opposite of this strategy). Note, that we may think of the reason for the success of the strategy $(0, 1)$ not by virtue of it having more children than the other strategies, but by having more grandchildren.

The two strategies $(1, 1)$ and $(0, 0)$ will sometimes produce an offspring of the dominated $(1, 0)$ strategy via crossover if they are paired with each other. As a consequence the $(1, 0)$ strategy persists as it can be reproduced by crossover in each generation. However, selection acts against it and hence it only exists in small frequencies. The effect of the strategy $(0, 1)$ becoming increasingly viable as crossover increases is evident in figure 2.6. Unlike the case with no crossover this strategy persists even if δ is large.

Despite these differences the overall qualitative results of the model do not change with the introduction of crossover. Costly assortment will evolve provided δ is large enough and h small enough. Despite being able to invade the well-mixed population assortment is unable to reach fixation because, whenever defectors are rare, the strategy $(0, 1)$ will out compete it, which will in turn allow for the introduction of defectors.

The equilibrium in which $(0, 1)$ and $(0, 0)$ coexist is not significantly effected by crossover as there is no variation at the first locus and hence crossover will have little effect on the dominant strategies.

Note that fairness, and hence the fitness of the individuals, increases with increasing crossover. However, fairness does not increase because of an increased level of assortative mating, which actually decreases with increased crossover. This is because, with crossover, the assortment allele is less fit, as it will sometimes be paired with the unfair allele, in which case it is maladaptive. Conversely, a fair allele is more fit if crossover is high, as it may find itself in an organism that mates assortatively, in which case it is beneficial to be fair. An interesting avenue of further study might be to look at how the crossover rate could itself co-evolve with a tendency to assort and fairness, however we leave this study for future investigations.

3.3 Summary of Results

Recent studies have looked at how assortment, and other game changing traits, can co-evolve with social strategies in a cooperative dilemma (Powers et al., 2011; Jackson and Watson, 2015; Fort, 2008). In many cases the outcome of such co-evolutionary models is that selection on traits that modify population structure will do so in such a way as to facilitate the evolution of cooperation (Powers et al., 2011); this effect is often referred to as social niche construction (Ryan et al., 2016). This comes about through the build-up of linkage disequilibrium between the cooperative strategy and the gene for creating an environment conducive to cooperation. This is exactly the mechanism for the results presented in this paper. We extended models of social niche construction to include a possible cost for assortment and the inclusion of crossover. In the former case cost is found to affect the model in such a way that the assortative cooperative strategy will never reach fixation, as it can always be invaded by an unconditional cooperative strategy that does not bear the cost of assortment. This in turn opens the door for the defector, and we thus have the possibility for the coexistence of all three strategies. Which of these three strategies coexist depends upon the cost of assortative mating, the selection coefficient, the degree of dominance and the drive efficacy. But for low values of the degree of dominance and intermediate values of drive efficacy we should see a coexistence of all three strategies. With the addition of crossover we also see the lowest fitness strategy, assort defect, being created by crossover, but persisting only in low frequencies. In addition, crossover increases the fitness of the strategy $(0, 1)$, not because it has more children than the other strategies, but because it has more grandchildren, as it will never create an offspring of the type $(1, 0)$.

In the absence of crossover the level of fairness and of assortative mating is maximised for intermediate levels of drive efficacy. It is only worthwhile to bear the cost of assortment if there is a certain level of defectors in the population, otherwise the cost of assorting outweighs the additional benefit gained from avoiding defectors. However, if drive efficacy becomes too high, then the non-assorting cooperators become extinct, in which case defectors are at a relative advantage, even over the cooperative assorters who then decrease in frequency.

The addition of crossover does not greatly affect the qualitative results. However, and perhaps surprisingly, crossover can actually lead to a slight increase in the level of fairness, as a drive

allele may be paired with an assorting allele due to crossover, in which case meiotic distortion is maladaptive.

4 Discussion

This paper has translated a specific result from evolutionary game theory into the language of population genetics. The result that assortment facilitates the evolution of cooperation is well known in the game theory literature. The analogue of this result in diploid genetics is that inbreeding facilitates the suppression of meiotic distorters. This is a result which is difficult to derive from standard population genetics models (Burt and Trivers, 1998), yet the result follows immediately from the translation between the two fields in question.

Models of this type are important for a greater understanding of evolutionary biology. Conventional theories of evolutionary biology take the integrity of the individual for granted, however, there has been a trend more recently to try to give an adaptive account of the evolution of individuality (Buss, 1987; Maynard Smith and Szathmary, 1997; Jablonka and Lamb, 2006; Ryan et al., 2016). A particular question of interest is why is it that selfish genetic elements are relatively scarce, given the logical fitness advantage they enjoy. The analogy with evolutionary game theory shows that this is precisely the same problem as the evolution of cooperation, albeit manifest at a lower level than conventionally considered. Typical explanations for the evolution of cooperation include: fairness enforcing mechanisms, such as the fairness of meiosis (Frank, 2003); punishment of uncooperative strategies, or policing mechanisms (Boyd et al., 2003; Cant, 2011); or the evolution of cooperation through the positive assortment of cooperative strategies (Eshel and Cavalli-Sforza, 1983; Fletcher and Doebeli, 2009; Godfrey-Smith, 2008), see also Lehmann and Keller (2006) for a review of these ideas. The logic of how these mechanisms lead to cooperation is clear; however, all of these mechanisms are themselves the outcome of selection of other genes; a full explanation of cooperation or fairness cannot take the presence of fair meiosis or of population structure for granted. The features of the biological world that ensure the evolution of cooperation may themselves be the outcome of selection and therefore deserve an explanation in their own right. Ultimately we want to know why it is that genes are able to form stable coalitions, why it is that selfish genetic elements are relatively scarce and what features of the biological world can be explained as adaptations to overcome the social dilemmas present at the genetic level.

Turning this story on its head our model offers a possible explanation for the evolution of assortative mating. Assortative mating is very common in nature. However, there is no consensus on whether assortative mating is an adaptation, or a by-product of another process. Furthermore, there are many potential causes of adaptive assortative mating (see Jiang et al. (2013) and the introduction of this paper for brief summaries of the current explanations). One potential reason is that assortative mating is a form of social niche construction that facilitates the evolution of

fairness at the genetic level. Thus, we can think of a preference for assortative mating as an adaptation to mitigate the effects of meiotic drive. Note that we do not claim that this is the exclusive, or even the principal, reason for the presence of assortative mating in nature. This explanation is in no way mutually exclusive with other explanations, see introduction. Nonetheless, we have shown that this mechanism leads to the evolution of an assortative mating preference for a wide range of parameters, both for tightly linked genes, and for genes residing on different chromosomes.

Future studies of this nature could look at the co-evolution of the crossover rate between the mating preference gene and the meiotic distorter gene. Our models show that a high level of crossover is beneficial for the evolution of fairness. A somewhat surprising result, as social niche construction depends upon linkage disequilibrium.

It is possible that another allele controlling crossover would find a rate of crossover that was optimal for the individual as a whole, and lead to an increased level of fairness at the genetic level. This is reminiscent of one of the major theories for the presence of crossover, which states that crossover evolved in order to break up tightly linked genetic cartels (Haig and Grafen, 1991), and so promote harmony at the genetic level.

Drawing analogies and mathematical equivalences between two existing fields is useful to the extent to which it provides a cross-fertilisation of ideas and tools between the two disciplines. Both population genetics and game theory are active and fruitful areas of research. The analogy could be taken further by, for instance, studying polyploidy via the formalism of multi-player game theory, by looking further at multi-locus models through the multi-population replicator equation, or studying imprinting through asymmetric games. As well as the equivalence between genetics and game theory suggesting hypotheses, the analogy is useful in that it allows us a much broader and general understanding of evolution and selection.

A Appendix: Closed form solutions using stability analysis in the absence of crossover

This appendix describes the closed form solutions for the case of replication without crossover. The details of the algebra are omitted, they are long-winded, but essentially trivial.

The matrix of conditional probabilities, P, (see equation (2.7) and section 3.1) is calculated as follows: we decompose $P(i|a,m)$ into the female and male portions and note that all individuals are either male or female with probability one half, therefore:

$$P(i|a,m) = \frac{1}{2}\left(P_F(i|a,m) + P_M(i|a,m)\right) \tag{A.1}$$

where:

$$P_F(i|a,m) = (1-a)\left(X_{0,i} + X_{1,i}\right) + a\delta_{i,m} \tag{A.2}$$

In order to specify the male portion consider a focal male agent. Each time a female selects a mate one of three things can happen. Either the male is selected by a female of the same value of m as the male, the male is selected by a female of a different value of m, or the male is not selected at that time. The male must ultimately be selected at some point, so we are only interested in the relative probability of being selected by a like and an unlike type at any one point in time. The overall probability of being selected by a like type can be found from the ratio of these two quantities. Let P_{same} be the relative probability (up to multiplicative constant) of being selected by a like type, and likewise P_{dif} the probability of being selected by an unlike type. To be selected by a like type either a female can select assortatively, in which case the pool of males that can be selected is only those that have the same value of m as the female, or the female can select at random. Being selected by an unlike type can only happen un-assortatively. Thus:

$$P_{\text{same}}(m) = \frac{X_{1,m}}{X_{0,m} + X_{1,m}} + X_{0,m} \tag{A.3}$$

$$P_{\text{dif}}(m) = x_{0,1-m} \tag{A.4}$$

$$P_{\text{total}}(m) = P_{\text{same}}(m) + P_{\text{dif}}(m) \tag{A.5}$$

where P_{total} is, by definition, the total relative probability of being selected. Then:

$$P_M(i|a,m) = \frac{\delta_{i,m} P_{\text{same}}(m) + (1-\delta_{i,m}) P_{\text{dif}}(m)}{P_{total}(m)} \tag{A.6}$$

The fitness of each individual is given by:

$$\pi(a,m) = P(0|a,m) M_{m,0} + P(1|a,m) M_{m,1} - ac \tag{A.7}$$

where M is the payoff matrix given by table (2.1). These expressions can be substituted into the replicator equation.

We now describe all of the fixed points of the resulting dynamics. Firstly, the region in which the strategy $(0,1)$ dominates. This is the region in which:

$$\delta < \frac{hs}{1-hs} \tag{A.8}$$

This solution is exactly as in the well-mixed case, it corresponds to the region for which $S < 0$, which can be found from equations (2.9) and (2.10).

Secondly, the region in which the strategies $(0,0)$ and $(0,1)$ coexist. Again the solution is identical to the well-mixed case. We can find it by setting: $\pi_{0,0} = \pi_{0,1}$. The equilibrium thus occurs at:

$$x_{01} = \frac{s - \delta - hs\,(1-\delta)}{s\,(1-2h)} \tag{A.9}$$

which can alternatively be found from combining equations (2.4), (2.9) and (2.10). The lower boundary is given by equation (A.8). The upper boundary is derived by finding the point at

which $\pi_{1,1}$ is equal to $\pi_{0,1}$ (or equivalently $\pi_{0,0}$) provided $(1,1)$ exists in infinitesimal quantities. This is thus the point at which $(1,1)$ is able to invade. One can think of this as the point at which defection has become sufficiently common that paying the cost of assortment becomes worthwhile. This occurs when:

$$\delta = \frac{\sqrt{s\left(c(2-4h)+h^2 s\right)}}{(1-hs)} \tag{A.10}$$

The next region is the one for which the three strategies $(0,0)$, $(0,1)$ and $(1,1)$ all coexist. This can be found directly from the replicator equation. It is the solution of a third order polynomial, and is found using a computer algebra package:

$$x_{01} = \frac{8c^3(2h-1)s + 2c^2((\delta-1)hs-\delta)(-4\delta+4\delta hs+2hs+s) + ((\delta-1)hs-\delta)\sqrt{Z}+W}{4c((\delta-1)hs-\delta)\left(\delta^2(hs-1)^2 - s\left(-2ch+c+h^2 s\right)\right)} \tag{A.11}$$

where:

$$W = -((\delta+1)hs-\delta)(\delta+h(s-\delta s))^2 \tag{A.12}$$

and:

$$\begin{aligned} Z \;=\; & (4c^4(1-2h)^2 s^2 - 4c^3(2h-1)s((\delta-1)hs-\delta)(3\delta(hs-1) \\ & -(h+1)s) - c^2(\delta-\delta hs+hs)^2 \\ & \left(((2-9h)h-1)s^2 + 7\delta^2(hs-1)^2 + 2\delta(7h-3)s(hs-1)\right) + 2\mathrm{c}(\delta-\delta hs+hs)^3 \\ & (-\delta+\delta hs-3hs+s)((\delta+1)hs-\delta) + (\delta-\delta hs+hs)^4(\delta-(\delta+1)hs)^2 \\ & -((\delta+1)hs-\delta)(\delta+h(s-\delta s))^2 \end{aligned} \tag{A.13}$$

and the frequency of $(1,1)$ is given by:

$$x_{11} = \frac{-\sqrt{E} + 6c^2(2h-1)s + cY + ((\delta+1)hs-\delta)(\delta+h(s-\delta s))^2}{4c\left(\delta^2(hs-1)^2 - s\left(-2ch+c+h^2 s\right)\right)} \tag{A.14}$$

where:

$$Y = ((\delta-1)hs-\delta)(s((3\delta+7)h-1) - 3\delta \tag{A.15}$$

$$\begin{aligned} E \;=\; & \left(L + ((\delta+1)hs-\delta)(\delta+h(s-\delta s))^2\right)^2 \\ & -8c(2c-\delta+(\delta-1)hs)\left(\delta^2(hs-1)^2 - s\left(c(2-4h)+h^2 s\right)\right) \\ & \left(\delta^2(hs-1)^2 - s\left(-2ch+c+h^2 s\right)\right) \end{aligned} \tag{A.16}$$

and:

$$L = 6c^2(2h-1)s + c((\delta-1)hs-\delta)(s(3\delta h+7h-1)-3\delta) \tag{A.17}$$

These solutions afford little intuition, they do however provide a very efficient way to solve the model when compared to an agent based simulation.

The final region is the one in which the strategies $(0,0)$ and $(1,1)$ coexist. This can be found simply by setting the payoff of these two strategies to be equal. This leads to the following expression for the equilibrium value.

$$x_{11} = \frac{\sqrt{Q} + 2c - \delta + \delta hs + 3hs - 4s}{2s(\delta h + h - 1) - 2\delta} \tag{A.18}$$

where:

$$Q = (2c-\delta+s((\delta+3)h-4))^2 - 4(\delta - s(\delta h+h-1))(-4c-\delta+s((\delta-1)h+4)) \tag{A.19}$$

The boundary between these two regions has no closed form solution that we were able to find, and is instead calculated by numerical means. The most straight forward way to do this is by solving $x_{01} = 0$ from equation (A.11).

B Appendix: Differential equations for the crossover model

The more general case in which there is an arbitrary level of crossover is modelled through a population dynamics model. We consider the expected change due to one round of selection in order to form difference equations. We then translate these into differential equations for the expected rate of change of each haplotype. (Note that we cannot simply write down the replicator equation, as haplotypes may combine with other haplotypes to form individuals of different types).

The state of the population at time t is given by $X_{a,m}$, which denotes the frequency of every possible haplotype. After one generation of selection the frequency changes to $X'_{a,m}$. We seek equations for X' in term of X, in order to define the evolutionary dynamics in the form of difference equations. The difference equations are found by splitting the life cycle into three distinct phases: firstly, haplotypes combine to form individuals, we thus calculate the expected frequency of each type of individual given the frequencies of each haplotype; then selection on individuals occurs, we thus calculate the expected change in frequency of individuals due to selection; finally, new gametes are produced, we then calculate the expected frequency of gametes produced given frequencies of each type of individual. These three steps are sufficient to formulate the difference equations. The difference equations form a very efficient way of modelling this situation.

An individual is defined by four alleles, a_1, a_2, m_1 and m_2. Each of these may take on one of two values, and there are thus 16 types of individual.

Firstly we consider the expected frequency of individual (a_1, m_1, a_2, m_2), given by $Y_{a_1m_1,a_2m_2}$. This is:

$$Y_{a_1m_1,a_2m_2} = \frac{1}{2}\left(X_{a_1m_1}P(a_2, m_2|a_1, m_1) + X_{a_2m_2}P(a_1, m_1|a_2, m_2)\right) \tag{B.1}$$

where $P(a_2, m_2|a_1, m_1)$ denotes the conditional probability of meeting an a_2, m_2 given that one is of type a_1, m_1.

As before, we split this into a male and a female portion as follows:

$$P(a_2, m_2|a_1, m_1) = \frac{1}{2}\left(P_F(a_2, m_2|a_1, m_1) + P_M(a_2, m_2|a_1, m_1)\right) \tag{B.2}$$

Where:

$$P_F(a_2, m_2|a_1, m_1) = (1 - a_1)\, X_{a_2m_2} + \frac{a_1\delta_{m_1m_2}X_{a_2,m_2}}{X_{0,m_2} + X_{1,m_2}} \tag{B.3}$$

We further break down the formulae for the probability of being selected by a certain type when one is a male into four cases, based upon whether one is selected assortatively and whether one is selected by an individual of the same value of m. These are denoted by subscripts a (for assort) and n for not assort, as well as s for same and d for different. These four quantities are given as follows:

$$p_{s,a} = \frac{X_{1,m_1}}{X_{0,m_1} + X_{1,m_1}} \tag{B.4}$$

$$p_{s,n} = X_{0,m_1} \tag{B.5}$$

$$p_{d,a} = 0 \tag{B.6}$$

$$p_{d,n} = X_{0,1-m_1} \tag{B.7}$$

$P_M(a_2, m_2|a_1, m_1)$ is then computed by selecting the appropriate formula from above, i.e. a if $a_2 = 1$ and n otherwise. And s if $m_2 = m_1$ and d otherwise. More formally this can be expressed as:

$$P_M(a_2, m_2|a_1, m_1) = \frac{\delta_{m_1,m_2}a_2p_{s,a} + \delta_{m_1,m_2}\left(1 - a_2\right)p_{s,n} + \left(1 - \delta_{m_1,m_2}\right)\left(1 - a_2\right)p_{s,a}}{p_{\text{total}}} \tag{B.8}$$

where:

$$p_{\text{total}} = p_{s,a} + p_{s,n} + p_{d,a} + p_{d,n} \tag{B.9}$$

For these equations we can thus formulate the expected number of each type of individual for given frequencies of each haplotype. We then compute the expected frequency of the individuals after one round of selection, and denote this by $Y'_{a_1m_1,a_2m_2}$. Where:

$$Y'_{a_1m_1,a_2m_2} = Y_{a_1m_1,a_2m_2}\frac{\pi_{a_1m_1,a_2m_2}}{\bar{\pi}} \tag{B.10}$$

where π is the fitness of a given type of individual, this can be calculated from the payoff matrix:

$$M = \begin{pmatrix} 1-s & 1-hs \\ 1-hs & 1 \end{pmatrix} \tag{B.11}$$

and then:

$$\pi_{a_1 m_1, a_2, m_2} = M_{m_1, m_2} - c/2\,(a_1 + a_2) \tag{B.12}$$

where c is the cost of assortment. $\bar{\pi}$ is simply the mean fitness, given by:

$$\bar{\pi} = \sum_{ijkl} \pi_{ijkl} Y_{ijkl} \tag{B.13}$$

The expected frequency of each type of gamete after the selected individuals have produced another set of gametes is given by $X'_{a,m}$. We can calculate the expected frequency of a certain gamete being produced from a given individual as follows. Let:

$$D = \begin{pmatrix} 1/2 & 1/2\,(1+\delta) \\ 1/2\,(1-\delta) & 1/2 \end{pmatrix} \tag{B.14}$$

thus, the indices of this matrix correspond to the probabilities of each value of m being transmitted.

Finally, we define the value $G(a_1, m_1, a_2, m_2, a, m)$ as the expected frequency of gametes of the type (a, m) produced from the individual (a_1, m_1, a_2, m_2). This is:

$$\begin{aligned} G(a_1, m_1, a_2, m_2, a, m) &= \delta_{m,m_1}\delta_{a,a_1}\,(1 - k/2)\,D_{m_1,m_2} \\ &+ \delta_{m,m_1}\delta_{a,a_2}\,k/2 D_{m_1,m_2} \\ &+ \delta_{m,m_2}\delta_{a,a_2}\,(1 - k/2)\,D_{m_2,m_1} \\ &+ \delta_{m,m_2}\delta_{a,a_1}\,k/2 D_{m_2,m_1} \end{aligned} \tag{B.15}$$

The expected frequency of each gamete in the next generation is then given by:

$$X'_{a,m} = \sum_{ijkl} Y'_{ijkl} G\,(i, j, k, l, a, m) \tag{B.16}$$

We can then construct an equation for the expected frequency of each type after one round of selection by combining equations (B.10) and (B.16):

$$X'_{a,m} = \frac{1}{\bar{\pi}} \sum_{ijkl} \pi_{i,j,k,l} Y_{i,j,k,l} G\,(i, j, k, l, a, m) \tag{B.17}$$

Without loss of generality we may set a time step of one, as we are making no assumptions about the time scales of evolution in our models. We can thus formulate a differential equation

for the expected rate of change of each haplotype:

$$\frac{dX_{a,m}}{dt} = X'_{a,m} - X_{a,m} \tag{B.18}$$

C Appendix: Simulation Model with Continuous Assortment

In order to complement the purely mathematical model presented in the main body of the paper we briefly discuss an agent based model and present the results in this appendix. The model differs from the mathematical model in three ways: firstly the tendency to mate assortatively can take on any intermediate level, and is not confined to be either zero or one. Secondly, as this is a simulation there will be stochastic finite population effects. Finally, we introduce a small mutation probability for each locus.

The simulation model follows the mathematical model as closely as possible. We initialise a population of random diploid individuals which have two loci a and m; a controls the tendency to mate assortatively. However, in this model a may take on any random real number between zero and one. Initial values are from the uniform distribution. As before m is a binary number, zero for defect, i.e. cheat meiosis, and one for cooperate, i.e. don't cheat meiosis. $2N$ individuals are selected via fitness proportionate selection with replacement, where N is the population size. Each individual then creates one gamete. We select one of the m alleles at random, according to the definition of δ, see section 2.1. We then perform crossover with probability k. The gametes are portioned into two equal sub-populations, which we label male and female. The female gametes are selected in a random order. For each female gamete with a value of assortment a: with probability a we pair the female gamete with a male gamete that has a value of m equal to that of the female gamete. With probability $1 - a$ we pair it with a male gamete chosen at random. If no male gametes of the right phenotype are available then the female is paired with a random male. Note that it is then possible to be paired with an individual of a different phenotype to oneself even if one has a value of $a = 1$. This can happen if a female gamete runs out of male gametes with the correct phenotype, or if a male gamete is chosen at random by a female gamete. We repeat the whole process for a pre-determined number of generations.

At each generation there is a small probability of mutation. With probability μ_m the value of m is switched to its complement. a is handled differently as it is a continuous parameter; with probability μ_a, a is changed randomly. The amount it changes is drawn from the normal distribution with mean zero and standard deviation of 0.05. (Note that the simulation results are not qualitatively effected by this choice.)

Figure 2.7 shows the sweep through δ in the same manner as figures 2.4 and 2.6. The dotted lines show the results of the stability analysis of the replicator equation and the solid lines the results of the simulation. We perform this simulation for $k = 0$ and $k = 1$.

The simulation model shows qualitatively similar results to the mathematical approach over all explored combinations of parameters. However, the correspondence is not exact. The most probable cause is that the mathematical expectations for conditional probabilities are approximate. When a female assorter chooses a mate it may be the case that there are no more males of the correct phenotype to choose. In this instance, in the agent based model, we have no choice but to pair the individuals with another random individual. This effect will become more pronounced when strategies go to smaller frequencies. This effect is not taken into account in the mathematical formalism of the model. Nonetheless, all the *qualitative* remarks made about the mathematical model hold equally well for the simulation. In particular, assortative mating will evolve, and hence increase fairness, for high levels of drive efficacy, δ, but assortment and fairness will always co-exist with distorting non-assorting haplotypes. This general statement is robust to the presence of cost and crossover and the mathematical approximations that we made.

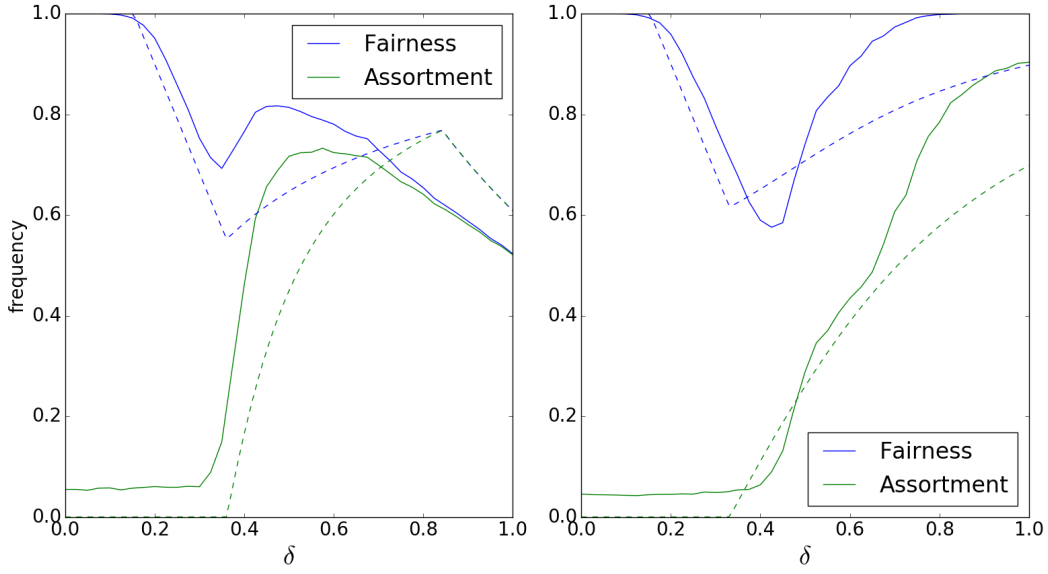


FIGURE 2.7: A sweep through δ with the simulation model. Dotted lines are the fixed points of the replicator equation, see appendix A, and solid lines that of the simulation. Left panel is for $k = 0$ and right for $k = 1$. The simulation is averaged over twelve independent runs. The population size was 5000 and the number of generations was 2500. The mutations rates were $\mu_m = 0.01$ and $\mu_a = 0.02$. Otherwise parameters were the same as they are in figure 2.4.

Chapter 3

Multiple Games and the Evolution of Assortment

Abstract

Cooperation has been a persistent subject of debate in the field of evolutionary biology. Many of the explanations for its existence involve positive assortment between cooperators. Current theories of social evolution, such as kin- and multilevel-selection, predict the direction of selection for a given level of assortment. What remains unclear, however, is how to determine the direction of selection on assortment, if this were itself subject to individual selection. We analyse a simple model of the evolution of assortment and find that assortment will be favoured, and thus increase levels of cooperation, in games of weak altruism (such as the snow-drift game), but in cases of strong altruism (such as the prisoner's dilemma), where all cooperation is lost at equilibrium, assortment necessary to support cooperation cannot evolve under individual selection, in the absence of noise. We further show that if individuals engage in multiple cooperative dilemmas simultaneously then there may be a continued selection on increased assortment, which is ultimately sufficient to resolve the prisoner's dilemma. We find that the final value of evolved assortment is, on average, monotonic in the number of randomly-chosen, two-player cooperative-dilemmas in which individuals engage. We argue that understanding the evolution of assortment is a vital, and largely overlooked, step towards a complete account of the evolution of cooperation.

1 Introduction

The evolution of cooperation was a problem that Darwin labelled his *one special difficulty*. In a nave interpretation of Darwinism, cooperation (and particularly altruism) present a fundamental challenge to the theory of evolution by natural selection. Why would individuals be selected to

perform actions that are beneficial to others at a cost to themselves? The two major attempts at answers to this question come in the form of inclusive fitness theory (Hamilton, 1964a; Gardner et al., 2011; Queller, 1985; Maynard Smith, 1964) and multi-level selection (Borrello, 2005; Eldakar and Wilson, 2011; Maynard Smith, 1964; Nunney, 1985; Wilson, 1975). These two formalisms have been shown to be mathematically equivalent (Lehmann et al., 2007b; Foster, 2006; Queller, 1992), as they both depend on population structure giving rise to assorted interactions. Here assortment means that like individuals will interact with each other more often than would be expected from random interactions. Self-interested individuals will cooperate in an assorted population because, by virtue of being a cooperator, they are more likely to receive the benefits of other co-operators; assortment is thus a key factor in the evolution of cooperation (Eshel and Cavalli-Sforza, 1983; Fletcher and Zwick, 2006; Godfrey-Smith, 2008; Michod and Sanderson, 1985; Sober, 1992; Rosas, 2010).

Current explanations of the evolution of cooperation take the presence of assortment as an assumption, and do not attempt to explain how assortment might arise (Ryan et al., 2016). In many instances in nature individuals may have traits that in effect modify which other members of the population they interact with. For instance: kin-groups are often created by limited dispersal (Smaldino and Schank, 2012; Pepper and Smuts, 2002; Lehmann et al., 2007a; Penn et al., 2012), which may have a genetic component and hence be subject to selection; birds may alter the time at which they leave their natal group (Bulmer, 1994); social wasps may alter the number of eggs that they lay in one host (Ode and Strand, 1995) or bacteria in biofilms may produce an intercellular matrix, which determines population structure (Crespi, 1989; Strassmann et al., 2011; Shapiro, 1998). Furthermore, multicellular organisms undergo a unicellular bottleneck, which may be an adaptation that increases the genetic homogeneity of the organism (Ryan and Watson, 2015; Michod and Herron, 2006). Thus, understanding in detail the evolution of individual traits that affect assortment is of vital importance to our understanding of cooperation, as assortment is often not an exogenous parameter, but a variable that is influenced by natural selection. A number of authors have looked at the evolution of population structure from the point of view of kin recognition (Tang-Martinez, 2001; Waldman et al., 1988; Giron and Strand, 2004; Rousset and Roze, 2007; Schausberger and Croft, 2001), from the perspective of mating systems (Peck and Feldman, 1988; Breden and Wade, 1991) or from the perspective of the evolution of limited dispersal (Johnson and Gaines, 1990; Le Galliard et al., 2005; Hochberg et al., 2008). Each of these studies investigates how specific mechanisms of assortment may evolve. However, what is lacking is a general treatment of the evolution of assortment that is agnostic as to the exact mechanism by which assortment comes about, and therefore has a more general applicability to a wider range of biological phenomena.

The vast majority of the studies of the evolution of cooperation take population structure as given; a more complete understanding of the evolution of cooperation would be facilitated by studying how such genetic traits effecting assortment will evolve concurrently with genetic traits that determine social behaviours. A growing number of recent studies have begun to look at such

processes. The concurrent evolution of cooperation and population structures that support cooperation is referred to as *social niche construction* (SNC) (Powers et al., 2011; Ryan et al., 2016), so called because individuals modify the selective pressure they experience on their social behaviour by altering the social niche in which they are evolving. Powers et al. (2011) study a model in which individuals play a public goods game in a group structured population. In addition to a gene controlling social strategy (i.e. cooperate or defect) they also look at the concurrent evolution of a gene that determines the group-size preference of the individuals. Because a population composed of small groups is more highly assorted than a population composed of large groups (because small samples exhibit greater variance in the proportion of types (Wilson, 1975)), this *group-size preference* then has the effect of an assortment parameter. However, in this model the evolution of assortment will only arise if the population supports a non-zero level of cooperation to begin with; it cannot, therefore, give a gradualist account of the evolution of assortment from the starting point of a population of freely-mixed defectors.

Jackson (2016) use the formalism of *meta-games* to investigate the evolutionary dynamics of game-changing behaviours, such as assortment. In their model each agent has a genetically determined payoff matrix representing the parameters of the game that an individual plays, as well as a gene determining their social strategy. They find that a strong linkage disequilibrium emerges, whereby cooperators are selected such as to alter the underlying game in order to favour cooperation and, similarly, defectors are selected such as to alter the game to make it more favourable to defectors. The outcome of the game is determined by the relative frequencies of cooperation and defection. For selection to have any effect on the underlying game there must be some polymorphism in the social strategy, which is not the case in the prisoner's dilemma at equilibrium. Alternatively, assortment will evolve if one pre-supposes some level of pre-existing assortment on the game changing gene (Jackson and Watson, 2013). However, as in Powers et al. (2011), this model does not give an account of the evolution of assortment from the starting point of freely-mixed defectors.

In both of these models assortment has difficulty getting started; a well-mixed infinite population facing a cooperative dilemma, such as the prisoner's dilemma, cannot be invaded by a cooperator with a small value of assortment. Note, however, that finite population effects may lead to the evolution of assortment in prisoner's dilemma games that are close to the boundary with the snowdrift game. Nonetheless, it remains true that assortment will not evolve, even in finite populations, for severe cooperative dilemmas that are not close to the snowdrift game. So, whilst a genetic trait that causes assortment may be stable when at high frequency, it cannot invade. This is an issue for our account of social niche construction, as the prisoner's dilemma represents the biological scenario of strong altruism (West et al., 2007). We can thus give no gradualist account of the evolution of assortment in the face of strong dilemmas. This is at odds with the observation that eusocial insects (Hölldobler and Wilson, 1990; Bourke and Franks, 1995), multicellular organisms (Ryan and Watson, 2015; Michod and Roze, 2001), communal siphonophore (Dunn and Wagner, 2006), and slime moulds (Strassmann et al., 2000) all exhibit strong altruism and have all evolved from free-living ancestors. These are examples of fraternal

transitions in evolution, which are thought to have occurred at least fifteen times in evolutionary history (Grosberg and Strathmann, 2007). Thus, finding a satisfactory solution to the getting started problem is a major obstacle to our understanding of the major transitions in evolution, as well as to our account of the evolution of cooperation more generally.

Note that while we present here an explanation for the gradual evolution of assortment, an alternative explanation could make use of exaptation. i.e. the assortative trait could have evolved for other reasons. We do not discount these alternative explanations, but note that our account of the evolution of assortment does not depend upon coincidental factors, and is therefore more likely to be observed across a wider distribution of organisms. Furthermore, we do not claim that assortment is always an adaptive trait, merely that there are at least some important instances when it is so.

A number of authors have looked at the evolution of population structure from within the field of adaptive networks (Zimmermann et al., 2004; Santos et al., 2006a; Pacheco et al., 2006; Ren et al., 2006; Van Segbroeck et al., 2009; Cao et al., 2011). They investigate a collection of models in which individuals play a social dilemma on a network and may, in addition, adjust their social ties; thereby affecting population structure. These models are attempts at descriptions of human social networks, and not at more basic biological systems, such as bacterial populations. So, whilst of some relevance, they do not serve as a general biological account of the evolution of assortment. Whilst these social dynamics have the effect of altering assortment, this is left implicit; there is therefore, in addition, a need for a more explicit analytical treatment of the evolution of assortment.

This paper presents a formal mathematical model to investigate under what conditions there is a positive selection gradient on assortment. In agreement with previous studies we find that such a gradient only exists if the population is polymorphic in cooperation. In the language of two-player games this situation is represented by the snowdrift game. We find that games, such as the prisoner's dilemma, that do not allow for coexistence of cooperation and defection, do not result in a selection for increased assortment, a result that is investigated in previous models, but is formalised here.

We show a plausible scenario in which assortment can be increased sufficiently by selection to levels high enough to "solve" the prisoner's dilemma (or in other words levels high enough to observe strong altruism). The principal idea is that individuals will be engaged in multiple social interactions at once, assumed to be controlled by genes at different loci. A simple example may be a species of bacteria that may produce a number of public goods, each of which is simply a protein. Each individual bacterium may or may not produce each public good. Thus, the multiple interactions within the species can be represented via a set of games (rather than conventional studies, which consider only a single game taking place). Each individual may be a cooperator or a defector in each game independently of whether or not they cooperate or defect in other games. In such an instance it may be the case that one of these games is a snowdrift game and hence provides a positive selection gradient for increased assortment until

a sufficient level of assortment has arisen to fix cooperation at this locus. As a bi-product of this process other games will become transformed such that they are then polymorphic (i.e. contain a mixture of both cooperators and defectors). Given enough games between individuals there will be a continual selection on increased assortment, so that the population ends up highly assorted and therefore cooperation can evolve, even for much more severe dilemmas.

Very few authors have looked at the possibility of the outcome of multiple games being played at once or sequentially between agents/individuals; those that do, do so from within economics or psychology. In particular a number of authors (Bednar and Page, 2007; Bednar et al., 2012; Grimm and Mengel, 2012) have looked at the consequences of multiple, and qualitatively different, games being played in sequence between subjects. The key themes of these papers tend to be to do with cognitive spill-over, i.e. how the outcome of one game might affect another, or the cognitive load on the individual, i.e. how individuals might use heuristics or rules learnt in one game to reduce the computation needed to solve other games. To the best of our knowledge no authors have looked at the dynamics of multiple games from within evolutionary game theory. One reason for the lack of such a study may be that the basic result, i.e. that each game reaches its own ESS independently (see appendix A), is not of interest unless one allows for some manner of epistatic interaction between games. In our model this epistasis comes via the intermediary of the evolving assortment parameter. With this interdependence the presence of multiple games results in a qualitatively different outcome, as we shall show.

The paper proceeds as follows: we firstly define a mathematical model for the evolution of assortment, allowing for the possibility of multiple games. We then go on to analyse two special cases of this model: firstly, the case in which there is only one game being played. The key result here, which we demonstrate analytically, is that there is a critical minimum initial value of assortment necessary in the prisoner's dilemma, below which the evolution of assortment cannot get started. Secondly, we analyse the special case in which there is no evolution of assortment, but individuals engage in multiple games at once. We show that each social strategy independently finds the stationary state of that game, regardless of other social interactions occurring in parallel. We then go on to present simulation results from the full model, in which we show that multiple games can lead to the evolution of a level of assortment sufficient to resolve the prisoner's dilemma. Finally, we perform some further simulations, complemented with an analytical argument, to show the statistical dependency of the number of randomly-chosen games on the evolution of assortment.

2 Model

We shall restrict our analysis to pair-wise interactions; such interactions can be represented via a two-player game. Santos et al. (2006b) show that the space of all possible two-player, two-strategy symmetric games is two dimensional, the payoff matrix for which can be parameterised

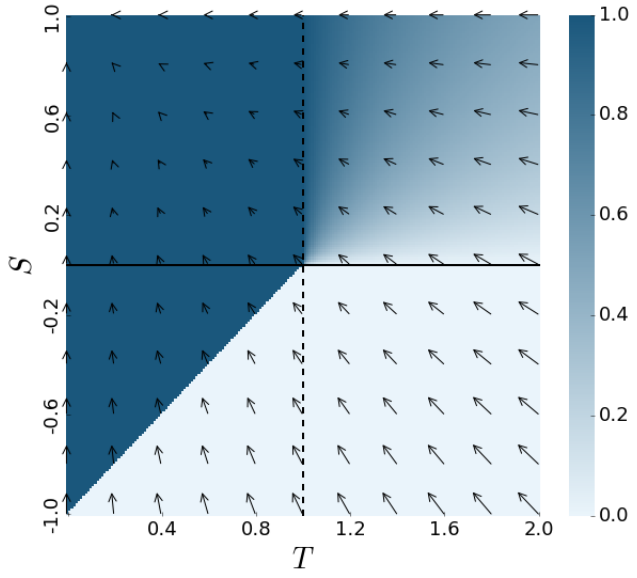


FIGURE 3.1: The space of all two-player, symmetric cooperative dilemmas and the effects of assortment. Each point in the space represents a two-player game parameterised via S and T. The colour represents the equilibrium frequency of cooperation, given initial condition of one half cooperators. (One represents cooperate and zero defect). Each arrow represents the effective transformation of the game under assortment of $\alpha = 0.05$. Assortment has the effect of transforming the game towards the harmony (top left) region of game space, $(S = 1, T = 0)$ thus making it more cooperative. The black lines delimit the four qualitatively different games, which are: top left: the harmony game, top right: the snowdrift game, bottom left: the staghunt game and bottom right: the prisoner's dilemma.

as:

$$M = \begin{pmatrix} 1 & S \\ T & 0 \end{pmatrix} \tag{2.1}$$

The effect of assortment can be modelled via a transformation of the game. A level of assortment, α, is defined as follows: with probability α an individual is paired with another individual of the same strategy and with probability $1 - \alpha$ it is paired with a random individual. It can be shown that the outcome of a game, M, under assortment, α, is equivalent to the outcome of the game, M', with no assortment (van Veelen, 2011) , where the elements of M' are given by:

$$M'_{ij} = \alpha M_{ii} + (1 - \alpha) M_{ij} \tag{2.2}$$

Thus, one can consider assortment as an effective transformation of the game into a more harmonious one, as figure 3.1 illustrates.

Furthermore, we introduce the notion of multiple games; whereby individuals engage in multiple social interactions simultaneously. Each individual may cooperate or defect in each game, independently of their strategy in other games. The strategy in each game is determined at a separate locus. We assume that all individuals play all games with all other individuals and that the overall payoff they receive is the sum of the payoffs from all games.

Let N_G be the number of games being played at once. A sequence of matrices determines all of the games such that the k^{th} game is given by:

$$M_k = \begin{pmatrix} 1 & S_k \\ T_k & 0 \end{pmatrix} \tag{2.3}$$

Each value of S and T is chosen at random from the uniform distributions: $S \in [-1, 1]$ and $T \in [0, 2]$. The social strategy of individual i is represented by a binary string, s_i, of length k. Thus, element s_{ik} determines the strategy played by individual i in the k^{th} game. We arbitrarily label cooperate one and defect zero.

In addition, individuals have a gene for "tendency to assort": α_i. Consider a population of unicellular organisms that, upon division, either stick to their parent, or interact with another non-related individual. This may be the case in the early stages of the evolution of multicellularity for instance, where colonies are formed when individuals fail to separate after division. We construe the gene for the tendency to assort as controlling the probability of sticking with a clonally related individual. With probability α_i individual i interacts with a clonally related individual, i.e. with an individual that has the same value of s at *all* loci. With probability $1 - \alpha_i$ it enters a pool of players and, therefore, interacts with an agent chosen randomly from the subset of those other individuals who have also joined the pool.

Selection proceeds generationally, via fitness proportionate selection. Mutation may occur at a locus controlling social strategy; with probability μ_s a locus mutates to its opposite strategy. The assortment gene is mutated with probability μ_α and consequently changes by an amount drawn from the normal distribution, with mean zero and standard deviation 0.01 (the results do not depend qualitatively on the particular choice of these parameters). If the value mutates outside of the permitted range it is scaled back to zero/one. Crucially, we also assume that the primitive state of the population is freely-mixed; we are primarily interested in the transition of free-living individuals to communally-living individuals. We thus seed our experiments with $\alpha = 0$ for all individuals and allow strategy frequencies to reach equilibrium before "turning on" mutation in α after 100 generations.

In addition, we allow for a small cost to assortment, $k \times \alpha_i$, which increases linearly with the individuals' assortment. This cost is introduced to ensure that all change in assortment is adaptive, rather than being due to drift. When all else is equal then selection will not favour an increase in assortment. Furthermore, it is biologically plausible that assortment may come at a cost to an individual. This may be in the form of the production of an adhesive protein, or in the energetic cost expended in seeking out an individual similar to oneself.

We proceed by deriving a number of analytic results by considering an infinite population and then go on to compare and extend these results with a series of simulations.

3 Results

We proceed by investigating special cases of the more general model. Firstly, we analyse the case in which $N_G = 1$. Secondly, we investigate multiple games, but in the absence of assortment; this can be thought of as the case in which $\mu_\alpha = 0$. Finally, we analyse the full model with both evolvable assortment and multiple games.

3.1 The Evolution of Assortment with a Single Game

For a single game there is no selective pressure for increased assortment unless the well-mixed ESS contains a non-zero level of cooperation. There is a critical value of α, for which a cooperator can invade a freely-mixed population of defectors, which depends on the parameters of the game. We thus argue that there is no gradual adaptive walk towards altruism from a freely-mixed population. We first sketch a mathematical argument of this point and then go on to compare it with a simulation based model.

Let the frequency of cooperators in the pool, i.e. those individuals who have not interacted with clonal partners, be p_C and likewise for defectors p_D. The payoff an individual with strategy i gets (as a function of α) is:

$$\pi_i(\alpha) = \alpha M_{ii} + (1-\alpha)\left(p_C M_{iC} + p_D M_{iD}\right) \tag{3.1}$$

p_C is calculated by taking a mean of the number of cooperators, weighted by the chance that they enter the pool, which is $1-\alpha_i$. E_C, the expected *number* of cooperators in the pool, is given by:

$$E_C = \sum_i s_i(1-\alpha_i) \tag{3.2}$$

We neglect the index k as we are considering only one game. Note, that in general there will be linkage disequilibrium between cooperation and assortment (Powers et al., 2011) and therefore E_c does not represent the frequency of cooperate in the whole population. Similarly, for defectors:

$$E_D = \sum_i (1-s_i)(1-\alpha_i) \tag{3.3}$$

It follows that:

$$p_{C/D} = \frac{E_{C/D}}{E_D + E_C} \tag{3.4}$$

To proceed, we determine when individuals with a slightly larger than normal level of α can invade a population. We consider a population composed of individuals who all have the same value for assortment tendency, α, and then investigate whether a mutant with a slightly larger value of assortment tendency, $\alpha + \delta\alpha$, can invade this population. This is established by determining whether or not payoff is an increasing function of α. The payoff to cooperators is given

by:

$$\pi_c(\alpha) = \alpha + (1-\alpha)(p_C + Sp_D) \tag{3.5}$$
$$= (1 - p_C - Sp_D)\alpha + p_C + Sp_D \tag{3.6}$$

Because $S < 1$ it follows that $(1 - p_C - Sp_D) > 0$ and thus $\pi_i(\alpha)$ is an increasing function of α, which means that there is always a selection pressure for existing cooperators to increase α. This is intuitive, as cooperators should always seek to interact with other cooperators.

Ultimately, we wish to know when sufficient assortment can evolve to lead to altruism. We thus consider a population of freely-mixed defectors and ask: for what value of α will a small frequency of cooperators be able to invade? Because we consider cooperators invading in infinitesimal quantities we assume $p_C = 0$ and also $p_D = 1$, see equations (3.5) and (3.6)). Thus:

$$\pi_c(\alpha) > \pi_d(0) \tag{3.7}$$
$$\alpha + (1-\alpha)S > 0 \tag{3.8}$$

Equation (3.8) reduces to:

$$\alpha > \frac{S}{S-1} \tag{3.9}$$

Therefore, in the limit of infinitesimal increase in α, cooperators can only invade if $S > 0$ (found by setting $\alpha = 0$ in equation (3.9)); that is α will only increase in a snowdrift game. Therefore, there can be no gradual evolution of assortment in the prisoner's dilemma in an infinite population. However, due to finite population effects assortment may evolve in a prisoner's dilemma. This occurs when cooperators with small but finite values of assortment invade a population by chance and subsequently lead to larger values of assortment, until the critical value of α (equation (3.9)) is reached. This becomes increasingly unlikely as mutation rates decrease and S becomes increasingly negative, see figure 3.2.

To investigate the effects of finite populations further we complement this mathematical argument with a simulation. We record the value of mean α and mean cooperation at equilibrium and plot these over the space of all possible games on the TS-plane. The results are presented in figure 3.2.

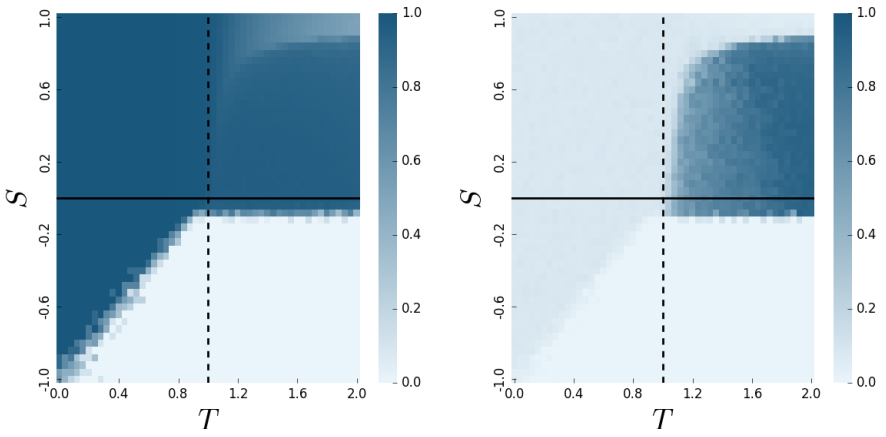


FIGURE 3.2: Equilibrium levels of cooperation (left) and α (right) over the space of all possible two-player games. Only snowdrift games provide a positive selective gradient on α. Each simulation was run with a population size of 1 028 for 10 000 generations. Each data point is the mean of 12 runs. $\mu_s = 0.001$, $\mu_\alpha = 0.1$ and cost $= 0.05$.

We conclude that a positive selection for assortment may only occur if there exists some preliminary level of cooperation at equilibrium. As there is mutation on strategy there is also a small amount of selection pressure to increase assortment even in games that are dominated by cooperators (as is the case in the harmony game and some of the stag hunt game). This can be understood as protection against the occasional introduction of defectors into the population. In the snowdrift regions in which the population is almost all cooperate and also in the region in which $S \simeq 1$ there is little increase in assortment. Note that assortment will evolve for only slightly negative S, as this requires only small mutations in α.

Note, that our argument does not rely on the fact that the population starts with a zero level of assortment. Instead, it must only be the case that the existing level of assortment, coupled with the underlying game, is not sufficient to lead to the evolution of altruism. There are other reasons for why there may be some pre-existing, and non-adaptive, level of assortment (see for example Ratcliff et al. (2013)). As a general point, we do not claim that assortment is always an adaptation to overcome a cooperative dilemma, merely that this is sometimes the case, and offer an explanation for such cases.

3.2 Multiple Games in the Absence of Evolvable Assortment

Before analysing the full model we briefly look at the special case in which assortment is fixed at zero and evolution acts only upon the social strategy, but individuals engage in multiple games. Each allele represents the social strategy in an independent game. We find that the evolutionary dynamics of each social strategy is independent of the other interactions occurring in parallel; the presence of multiple games does not affect the outcome of selection. This is to be expected, as there is no epistasis. This is in agreement with results from population genetics that state

that individual genes will fixate independently in the absence of epistasis (Hartl and Clark, 1998). A more formal justification is presented in appendix A, in which we show that the replicator dynamics of a single population engaged in multiple games is formally equivalent to the replicator dynamics of multiple populations, each of which plays only one game.

Figure 3.3 shows a typical output of this model. To aid interpretation we create a scatter plot of the games being played on the TS-plane alongside the evolution of strategy frequencies. As expected, each gene fixates at the ESS of the relevant game, indicated by dashed lines.

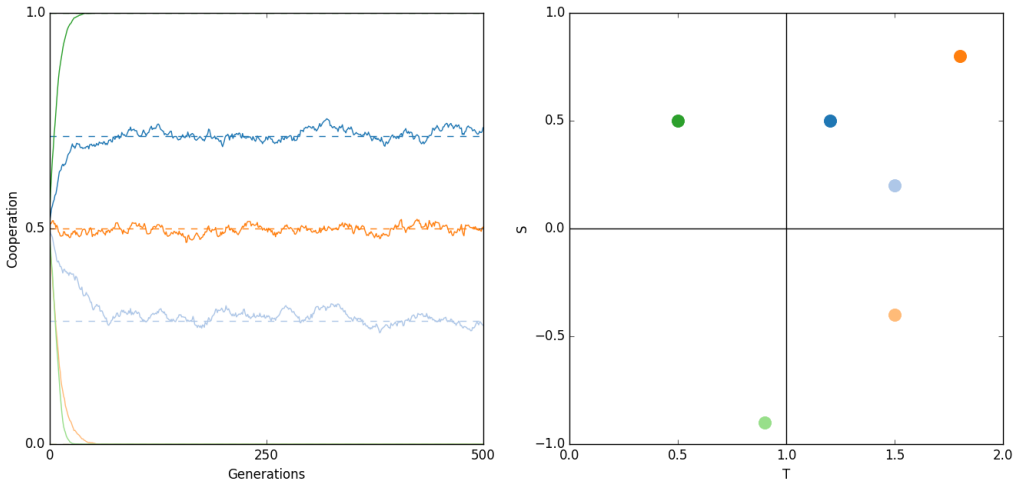


FIGURE 3.3: A selection of randomly chosen games. We record the frequency of the population that plays cooperate at each allele (left). This matches predictions for the ESS of each game independently (dotted lines). Right panel illustrates where each of these games lies in the TS-plane. $\mu_s = \mu_\alpha = 0$ with a population size of 10 000 run for 500 generations. (Note that the corresponding lines and dots are matched by colour.)

3.3 The Evolution of Assortment with Multiple Games

Finally, we present the results of the full version of the model. In addition to genes determining social strategies, individuals also have a gene that determines their level of assortment. In this model there is no explicit epistasis; nonetheless epistasis comes about through the intermediary of the assortment gene. We cannot, therefore, consider each game in isolation.

Recall that for $N_G = 1$ there was no selective pressure for increased assortment unless the population was polymorphic in the cooperative trait (see figure 3.2). However, the selective pressure *opposing* the evolution of assortment in the prisoner's dilemma is small. This comes from the cost to assortment and the small mutation rate in strategy; when cooperators appear it is beneficial for a defector not to assort. Given a relatively small value for the mutation rate and the cost of assortment, then the positive selective pressure for increased assortment in snowdrift games significantly outweighs the negative selective pressure in the prisoner's dilemma.

Consider a situation in which the population engages in two cooperative dilemmas, one of which is a snowdrift game and the other a prisoner's dilemma. The snowdrift game will create a selective gradient on assortment, which will subsequently increase. As a bi-product the gene engaging in the prisoner's dilemma will also become assorted. Thus, the prisoner's dilemma is *dragged* by the snowdrift game into a more cooperative one. This game may itself become polymorphic and introduce further selective pressure for increased assortment. Figure 3.4 illustrates one snowdrift game, in which assortment increases to near unity and one prisoner's dilemma, in which assortment does not increase appreciably. We then perform a simulation in which both of these games are played simultaneously and demonstrate that in this case assortment does evolve. The prisoner's dilemma is dragged by the snowdrift game into a more cooperative region of the TS-plane. We also show an example of how this may work for multiple games. In instances where there are many games occurring at once any number of these could be polymorphic at one time; thus creating an evolutionary pressure for increased assortment.

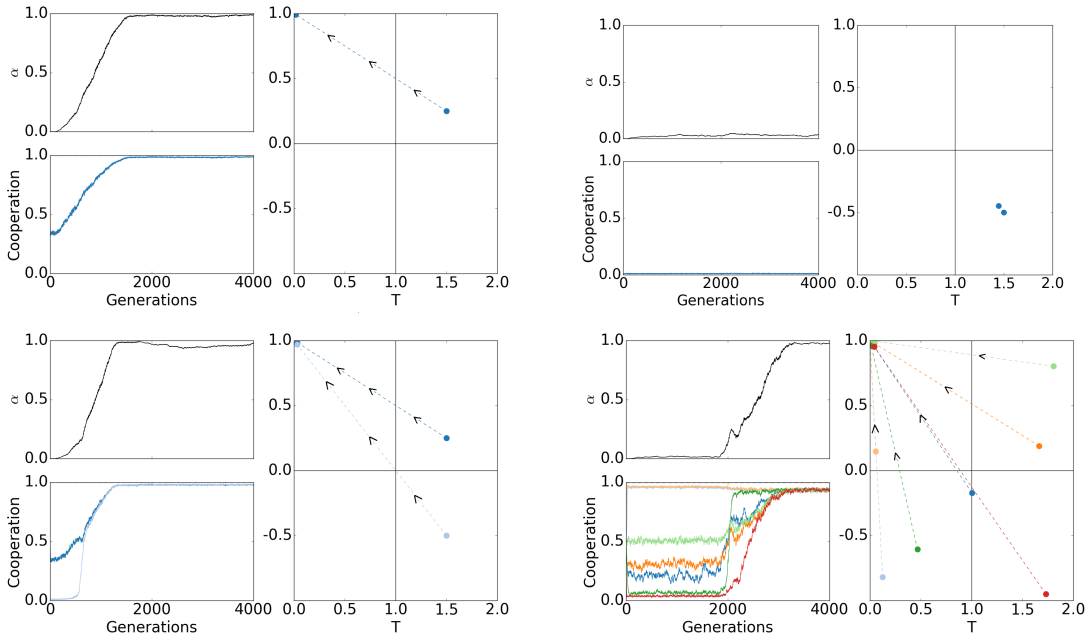


FIGURE 3.4: Four independent realisations of the simulation. Top left: a snowdrift game in which assortment evolves to a high level. Top right: a prisoner's dilemma, in which there is no evolution of assortment. Bottom left: the top two games played together, in which the snowdrift game drags the prisoner's dilemma into the Harmony game. Bottom right: evolutionary dynamics of 7 randomly chosen games. Each panel is composed of three figures, they are: right: the distribution of games on the TS-plane along with their transformation due to the resultant assortment. Top left: the mean frequency of assortment over time, and bottom left: the mean value of each strategy gene. In each case the population size was $4\,096$ and the simulation was run for $4\,000$ generations. $k = 0.01$, $\mu_S = 0.01$ and $\mu_\alpha = 0.01$.

Finally, we investigate the effects of varying the number of games, N_G, on the evolved level of assortment, α. This is illustrated in figure 3.5. We do this by repeatedly generating random games from the uniform distributions: $S \in [-1, 1]$ and $T \in [0, 2]$. Each game is selected

independently and at random. We run the simulation for a predetermined number of generations (10 000) and record the final level of α.

This figure supports the hypothesis that an increase in the number of simultaneous social dilemmas increases the selective pressure on assortment, thus mitigating the *getting started* problem. If individuals engage in multiple interactions at once, then assortment can evolve such as to bring about cooperation; even in severe cooperative dilemmas, such as the prisoner's dilemma.

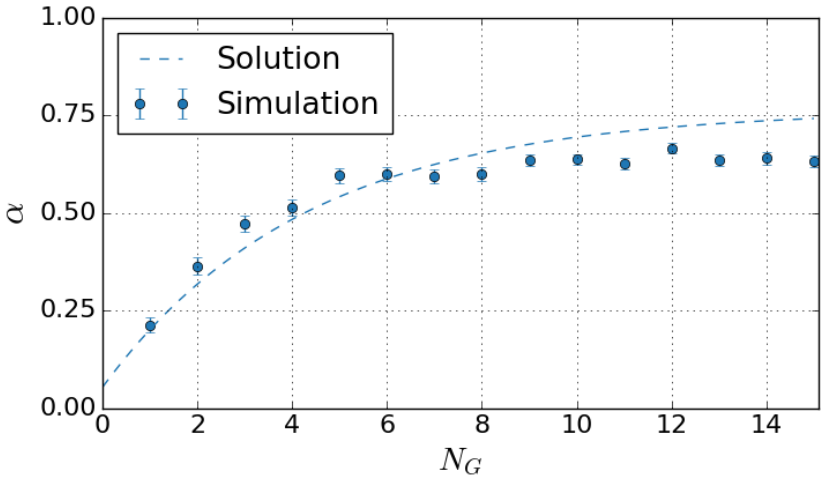


FIGURE 3.5: Evolved assortment vs. number of randomly-chosen games. Points represent the results of simulation with standard errors. The dashed line is the outcome of the estimate given by equation (3.11) with $Z = 0.21$, $Q = 0.76$ and $L = 0.053$. Each simulation was run with a population size of 1 028 for 10 000 generations, with N_G randomly chosen games. Each data point is the mean of 300 runs. $\mu_s = 0.01$, $\mu_\alpha = 0.01$ and cost $= 0.05$.

The underlying distributions for the level of evolved α are bimodal, see figure 3.6. That is, either there is relatively little evolution of assortment, or the evolution of assortment reaches a high level. As game number increases it become increasingly likely that assortment will reach this higher level, see figure 3.5. This is for the simple reason that there is an increasing probability that any one game will lie in the snowdrift quadrant of the TS-plane as game number increases. For a single game this probability is $1/4$ (given our assumptions about the distributions from which we draw S and T). If there are N_G games then the probability that at least one of these lie in the snowdrift region is:

$$N_G(\alpha) = 1 - (3/4)^{N_G} \tag{3.10}$$

This serves as a crude estimate for the probability of assortment increasing for a given number of randomly chosen games. This approximate measure gives a reasonable estimate, but can be improved upon with a slightly more sophisticated argument, which we sketch below.

As distribution for the final level of assortment is bimodal, then either assortment will remain low, or will increase to a much higher level; intermediate outcomes are unlikely. When there is no significant increase in assortment the value of α will still be slightly larger than zero. This is

principally due to finite population effects such as drift. For similar reasons when α increases it will fall slightly short of one. We categorise those outcomes in which assortment stays low and those in which it increases. To do this we define a threshold value, which we set equal to 0.35 (our results are not sensitive to the exact choice of this parameter, see figure 3.6). Let the mean value of α for those games in which assortment does increase be Q and the mean value of α for those games in which assortment does *not* increase be L. To approximate these values we use the data gathered in the one game version of the model, see figure 3.2. We find that $L \approx 0.05$ and $Q \approx 0.8$. Finally, we measure the fraction of those games that reach the high α state over the whole of the TS-plane, the dark blue region of figure 3.2. We denote this fraction Z and find it to be roughly 0.21 (close to the purely theoretical one quarter, however, cost means that assortment does not evolve for all snowdrift games). The evolved assortment as a function of the number of games is then roughly:

$$\alpha\left(N_G\right) = L + \left(Q - L\right)\left(1 - \left(1 - Z\right)^{N_G}\right) \tag{3.11}$$

This more refined estimate is plotted in figure 3.5 and provides a reasonable estimate of the distribution. Figure 3.6 clarifies the meaning of L, Q and Z, by plotting these values on a histogram of final α, for $N_G = 1$.

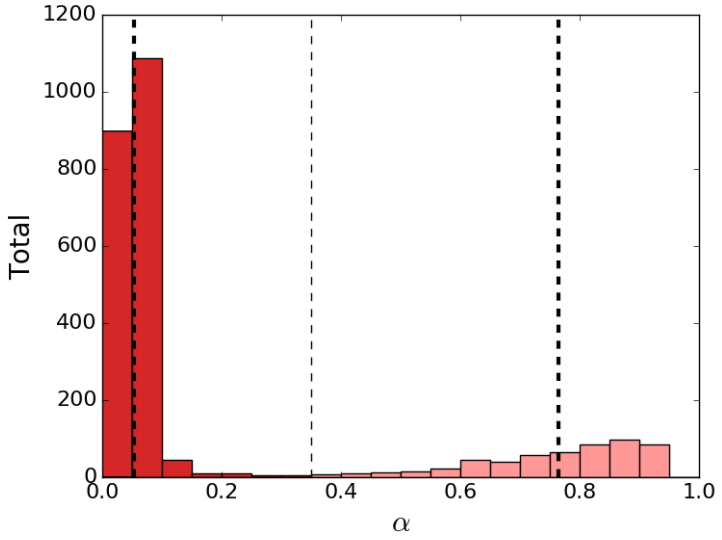


FIGURE 3.6: Histogram illustrating the calculation of L, Q and Z. The histogram depicts the evolved assortment for randomly chosen games, in which $N_G = 1$. The distribution is bimodal, with two clear peaks. We split the distribution into those data points that are less than the threshold (in red), and those that are greater than the threshold (in pink). The threshold is indicated with the middle dashed line. L, the mean of the lower mode, is indicated with the thick dashed line on the left of the figure. Q, the mean of the upper mode, is indicated with the dashed line on the right of the figure. Z is the total area of the right (pink) distribution divided by the total area of the entire distribution.

4 Conclusions and Discussion

In conclusion, we have shown that, given the assumptions of our model, assortment will not evolve for severe prisoner's dilemmas unless the population is polymorphic in the cooperative trait; this is an issue that we have labeled the *getting started* problem. If individuals engage in multiple interactions simultaneously, but in the absence of assortment, then the outcome is equivalent to a series of isolated populations playing each game independently. An evolvable assortment parameter provides an effective epistasis between games at different loci. In this scenario games that are polymorphic for the cooperative trait *drag* those games that are not polymorphic into a cooperative game. Thus, multiple games represent a potential solution to the getting started problem. The final level of assortment that evolves is monotonic in the number of randomly-chosen, two-player games being played; this can be roughly estimated by simple probabilistic arguments concerning whether or not a game lies in the snowdrift quadrant of the TS-plane.

It is well known in evolutionary biology that population structure is important when considering the evolution of social traits (Eshel and Cavalli-Sforza, 1983). Specifically, positive assortment can allow for the evolution of cooperative or altruistic behaviour. Formalisms such as inclusive fitness (Hamilton, 1964a; Grafen, 1982; Maynard Smith, 1964) and multi-level selection (Price, 1970; Okasha, 2009) allow one to make precise calculations of the expected change in the frequency of a cooperative allele due to selection (see for example (Gardner et al., 2011)). Furthermore, the natural world is full of examples of cooperative behaviour, such as the cooperation between cellular slime moulds (Strassmann et al., 2011), eusocial insects (Hölldobler and Wilson, 1990) or the cells of a multicellular organism (Michod and Roze, 2001; Buss, 1987; Queller, 2000). In many cases the population structure of such organisms may have a genetic component, and thus be subject to evolutionary change. Whilst it is plausible that many of these features are adaptations, there lacks a unified theoretical understanding of the conditions under which an increases positive assortment will evolve.

Recently a number of authors (Powers et al., 2011; Jackson, 2016) have begun to address this issue. One point that has emerged from these studies, and is backed up by a formal mathematical argument here, is that selection will not increase assortment unless the underlying game is polymorphic. Games such as the prisoner's dilemma are not polymorphic at equilibrium, and therefore selection on assortment cannot "get started". Furthermore, these games represent the biologically prevalent case of strong altruism (see for example: Doncaster et al. (2013)). We have presented a potential resolution to this problem: if individuals interact in many social dilemmas simultaneously, then there may exist a feedback process whereby the weaker dilemmas transform the stronger dilemmas into weaker ones and, eventually, all dilemmas are resolved.

Biologically models of the evolution of assortment could represent, for example, an evolutionary path towards multicellularity (see also: Michod and Roze (2001); Maynard Smith and Szathmary (1997); Ispolatov et al. (2012); Grosberg and Strathmann (2007); Jablonka and Lamb

(2006)). The cells of a proto-organism will eventually need to cooperate in many different manners, such as by producing different public goods with differing costs or by refraining from different forms of selfish reproduction, each of which with a different benefit. Our model shows how this can be thought of as a continuous process, rather than a binary one (see Godfrey-Smith (2009)). The cells will begin by cooperating in a less severe dilemma, which will create positive selection pressure on assortment, which will in turn create selection pressure to cooperate in another, slightly more severe dilemma, and so on, until all dilemmas have been resolved, and the population will consequently be highly assorted. The likelihood of this happening is greatly increased as the number of dilemmas being played increases. It seems plausible that the case of individuals playing one single social dilemma is an idealisation, and that in reality individuals, having many genes, and many potential social interactions, will usually be engaged in a very large number of social interactions at once, thus making the transition to social living possible.

Okasha (2009) notes that the history of theoretical evolutionary biology, that is the history of our understanding and explanations of evolutionary biology, has been one of increasing *endogenisation*, see also Ryan et al. (2016). Elements of a theory that were previously taken for granted have increasingly themselves come under the umbrella of adaptationist arguments. The current theories of the evolution of cooperation mostly take population structure and the assortment of interactions as given and subsequently give an account of how cooperation thrives in such a niche. This paper, and the associated ideas of social niche construction, attempt to extend this account by explaining how population structure can itself be an adaptation. A full account of cooperation must not only explain why cooperation is stable in a certain environment, but give an account of how cooperation itself feeds back upon the social niche of the population in question. The model in this paper has given an adaptive account of how a positively assorted population of cooperators can evolve from a well-mixed population of defectors, and thus gives a fuller account of the evolution of cooperation.

A Appendix: Replicator Dynamics of Multigames

In this appendix we prove that the replicator dynamics of a population of individuals playing N multiple games is formally equivalent to the case in which there are N isolated populations, each of which plays one of the games. An individual is represented by a bit string of length N, each bit representing the strategy in the respective game. There are thus 2^N possible types of individual. Let us denote the frequency of each of these individuals with the vector y, of dimension: 2^N. The frequency of each allele is denoted by the vectors x, each of these vectors is of length 2: the number of strategies in the game. There are N such vectors. Let the k^{th} such

vector by denoted by $x^{(k)}$. The vector y can be expressed in terms of the x vectors as follows:

$$\underline{y} = \begin{pmatrix} \underline{x}^{(1)} \\ \underline{x}^{(2)} \\ \vdots \\ \underline{x}^{(N)} \end{pmatrix} \tag{A.1}$$

There are N games being played simultaneously; the k^{th} game given by the matrix $M^{(k)}$. Let the matrix L represent the full payoff matrix. Its elements can be represented in terms of the M matrices as follows:

$$L = \begin{pmatrix} M^{(1)} & 0 & \dots & 0 \\ 0 & M^{(2)} & \dots & 0 \\ \vdots & \vdots & \ddots & \vdots \\ 0 & 0 & \dots & M^{(N)} \end{pmatrix} \tag{A.2}$$

where here zero represents the two-by-two matrix of zeros.

The replicator equation (Taylor and Jonker, 1978; Nowak, 2006a) is:

$$\dot{\underline{y}} = \underline{y}\left(L\underline{y} - \underline{y} \cdot \left(L\underline{y}\right)\right) \tag{A.3}$$

From equations (A.1) and (A.2) it follows that:

$$L\underline{y} = \begin{pmatrix} M^{(1)}\underline{x}^{(1)} \\ M^{(2)}\underline{x}^{(2)} \\ \vdots \\ M^{(N)}\underline{x}^{(N)} \end{pmatrix} \tag{A.4}$$

and that:

$$\underline{y} \cdot \left(L\underline{y}\right) = \begin{pmatrix} \underline{x}^{(1)} \cdot \left(M^{(1)}\underline{x}^{(1)}\right) \\ \underline{x}^{(2)} \cdot \left(M^{(2)}\underline{x}^{(2)}\right) \\ \vdots \\ \underline{x}^{(N)} \cdot \left(M^{(N)}\underline{x}^{(N)}\right) \end{pmatrix} \tag{A.5}$$

Hence the replicator equation is:

$$\begin{pmatrix} \dot{\underline{x}}^{(1)} \\ \dot{\underline{x}}^{(2)} \\ \vdots \\ \dot{\underline{x}}^{(N)} \end{pmatrix} = \begin{pmatrix} \underline{x}^{(1)} \\ \underline{x}^{(2)} \\ \vdots \\ \underline{x}^{(N)} \end{pmatrix} \left(\begin{pmatrix} M^{(1)}\underline{x}^{(1)} \\ M^{(2)}\underline{x}^{(2)} \\ \vdots \\ M^{(N)}\underline{x}^{(N)} \end{pmatrix} - \begin{pmatrix} \underline{x}^{(1)} \cdot \left(M^{(1)}\underline{x}^{(1)}\right) \\ \underline{x}^{(2)} \cdot \left(M^{(2)}\underline{x}^{(2)}\right) \\ \vdots \\ \underline{x}^{(N)} \cdot \left(M^{(N)}\underline{x}^{(N)}\right) \end{pmatrix} \right) \tag{A.6}$$

This is simply N decoupled replicator equations, each of which is the replicator equation for the k^{th} game, namely:

$$\dot{\underline{x}}^{(k)} = \underline{x}^{(k)}\left(M^{(k)}\underline{x}^{(k)} - \underline{x}^{(k)} \cdot \left(M^{(k)}\underline{x}^{(k)}\right)\right) \tag{A.7}$$

It follows then that the replicator dynamics of individuals playing N simultaneous games is formally equivalent to the replicator dynamics of N populations playing one of these games each. Furthermore, nothing in this proof relies on the game being composed of only two strategies, or even each game having the same number of strategies.

Chapter 4

Game Theoretic Treatments for the Differentiation of Functional Roles in the Transition to Multicellularity

Abstract

Multicellular organisms are characterised by role specialisation, brought about by the epigenetic differentiation of their constituent parts. Conventional game theoretic studies of cooperation, based on the prisoner's dilemma and related games, do not account for this division of labour, nor do they allow for the possibility of the plastic expression of phenotype. We extend the definition of cooperative dilemmas to include those games in which fitness is maximised by a mixture of different strategies, and present an extended dynamical model of selection that allows for the possibility of conditional expression of phenotype. We use these models to investigate systematically when selection will favour an adaptive diversification of roles. We find that, if relatedness is high, selection will favour genotypes that are able to develop conditionally upon the social environment in which they find themselves. We argue that such extensions to models and concepts are necessary to understand the origins of multicellularity and development.

1 Introduction

The evolution of cooperation has been a central theme of research within evolutionary biology (Axelrod and Hamilton, 1981; Fletcher and Doebeli, 2009; Lehmann and Keller, 2006). In nearly all such formal studies cooperation is modelled by Dawesian dilemmas, most commonly the Prisoner's dilemma and closely related games (Doebeli and Hauert, 2005). A Dawesian dilemma (Dawes, 1980; Macy and Flache, 2002) is one which satisfies two conditions:

Dawes I There exists a single cooperative strategy whereby mean fitness is maximised if all individuals perform this action.

Dawes II There exists an individual incentive not to perform this action.

Hence, evolution in a freely mixed population will not lead to a cooperative state that maximises mean fitness.

The biological systems in which Dawesian dilemmas are usually applied have, in recent years, greatly expanded from the study of eusocial insects and social vertebrates to a vast number of different cases (Queller, 1997; Bourke, 2011). Of particular interest is the extension of the notion of cooperation to the origin of multicellularity and hence development (Buss, 1987; Maynard Smith and Szathmary, 1997; Michod and Herron, 2006; Grosberg and Strathmann, 2007). The cells of a multicellular organism are often colloquially described as cooperating with one another (Queller, 2000; Lehmann and Keller, 2006; Michod and Roze, 2001). However, the cells cannot be thought of as being engaged in a Dawesian cooperative dilemma, as there is no one phenotype that can be considered as *the* cooperator. Instead the multicellular organism is characterised by having multiple complementary cell types (Bonner, 1993). This diversification of roles is an important detail that conventional studies of the evolution of cooperation do not adequately model. Whilst games such as the snowdrift game are polymorphic at equilibrium, this does not represent a diversification of roles, as the two behaviours do not complement one another, but instead defectors simply exploit cooperators. Proto-multicellular organisms, such as slime moulds (Strassmann et al., 2000) and volvocine algae (Michod, 2007), often have two distinct cell types; both of which are important for the function of the organism; thus no one type should be thought of as the cooperator. To avoid semantic confusion we re-label cooperate as A and defect as B. To reflect better the adaptive nature of role diversification we define a Division Of Labour (DOL) game to be any game that has the property of mean individual fitness being maximised by a polymorphic state. Following Weibull (1997), we call the state that maximises fitness the Socially Efficient State (SES).

In cooperative dilemmas freely-mixed populations will not reach the SES. This, in general, will also be the case for DOL games, and we thus retain the label of cooperative dilemmas in such cases; whilst not adhering to Dawes I. Structuring of interactions, and specifically positive assortment on cooperative phenotypes, is often claimed to be the central resolution to cooperative dilemmas (Eshel and Cavalli-Sforza, 1983; Fletcher and Zwick, 2006; Godfrey-Smith, 2008). However, in the limit, positive assortment of cooperators removes heterogeneity, and thus cannot maximise fitness in a DOL game. Complete positive assortment can only ever lead to interaction between individuals of the same type, whereas fitness in DOL games is maximised if all interactions are between unlike types. This apparent paradox, between the need for heterogeneity of roles to gain from specialisation/complementary functions and homogeneity to resolve the cooperative dilemma can only be overcome if the individuals can express a phenotype conditionally upon their social environment (including, potentially, a phenotype conditioned on the

phenotype of their parent(s)). This is so that individuals can have a positive assortment on genotype, i.e. relatedness, whilst simultaneously creating a negative assortment on phenotype. These are features of biological systems that conventional game theoretic models do not account for. They are, however, crucial for understanding the origins of development and multicellularity; in which the epigenetic determination of phenotype plays a key role (Lachmann and Sella, 2003; Jablonka and Lamb, 2006).

Stark (2010) studies *dilemmas of partial cooperation*, which are mathematically equivalent to DOL games, and concludes that partial levels of cooperation maximise fitness in certain types of dilemma. However, as we shall show, this mixture of strategies is not optimal if one takes into account the structuring of interactions, as fitness can be increased further if one allows for a complete negative assortment on phenotype. Furthermore, Stark concludes that an intermediate level of assortment will allow the population to reach the SES, which we show is not the case (see section 3), as his argument does not account for how the SES is altered by assortment. Other authors (Neill, 2003; Browning and Colman, 2004; Tanimoto and Sagara, 2007) have analysed the *turn-taking* solution to DOL games. They conclude that if individuals alternate sequentially between strategies then the dilemma can be resolved. En route to multicellularity it may be the case that unicellular organisms employ life cycle stages that alternate between roles, but true multicellular organisms have cells that remain specialised for the entirety of their life, as the alternation of cell type is either costly or unfeasible (Michod, 2007). Therefore, we study solutions in which individual cells are constrained to stick to a single phenotype after development.

Archetti (2009) and Boza and Számadó (2010) look at a class of games that they label volunteer dilemmas. In such games groups benefit from individuals performing costly actions. However, the benefits of these actions are reaped if a certain threshold of individuals, less than the number of individuals within a group, perform the cooperative action. As the action is costly, the mean fitness of the group is maximised if only a certain fraction of the individuals cooperate. These are indeed division of labour games by our definition. However, we conceptualise division of labour games more broadly than this. In volunteer dilemmas one of the strategies is simply the absence of action, however, it may be the case that mean fitness is maximised by two, or more, active strategies, in which case it does not make sense to label one of these actions as cooperate. Furthermore, neither of these studies considers the solution in which individuals react conditionally to their social environment, which we show is an important solution to such games.

Previous studies have taken the evolution of specialisation and the evolution of cooperation as separate problems, the latter often modelled via cooperative dilemmas such as the prisoner's dilemma (Doebeli and Hauert, 2005; Fletcher and Doebeli, 2009; Traulsen and Nowak, 2006). These two problems have therefore previously been studied largely in isolation. This paper formulates the issue of specialisation via a simple extension of existing cooperative dilemmas. We thereby relate the relatively understudied problem of specialisation, to a problem that has been studied extensively: cooperation.

A number of papers have looked at models in which individual components can potentially specialise in a number of set tasks and further investigate the conditions under which specialisation may occur. In particular both Gavrilets (2010) and Michod and Herron (2006) look at a model in which individual cells can specialise in one of two tasks relating to fecundity and viability, or alternatively remain as generalists. In these models the groups benefit from having both tasks performed together, but there is an inefficiency cost for individual cells to perform both. If the inefficiency is large enough then specialisation may evolve. Ispolatov et al. (2012) analyse a model in which group structure is not presupposed, and is itself an endogenous parameter of evolution. This is embodied through a stickiness parameter. Again they conclude that specialisation and group structure can evolve in a certain region of parameter space, where here the important parameters are the cost of stickiness and the inefficiency cost for a single cell to perform both actions together. Willensdorfer (2009) presents a similar study, but formulates the model in terms of the fitness of a pre-existing group on which selection acts. The model is phrased in terms of three key parameters: the cost of somatic function, the cost of size (i.e. number of cells) and the benefit of group living. Whilst the authors derive some mathematical results about when specialisation will evolve, and to what extent, they do not address the problem of cooperation *per se*, as selection at the colony level is presupposed. Rueffler et al. (2011) present a model, in which pre-existing colonies begin in an entirely undifferentiated state and then subsequently can evolve specialisation. As in other studies, whether or not this will happen depends upon the detailed relationship between a number of parameters involving the costs and benefits of specialisation. Barker et al. (2015) also construct an abstract model for the evolution of specialisation, in which they study the evolutionary dynamics of groups of related individuals that engage in one of two tasks. See (Hanschen et al., 2015) for a more detailed review.

Whereas in all previous studies (summarised above) groups are essentially undifferentiated bags of cells performing one of two tasks, our model properly considers the internal structure of interactions. We thereby consider an additional step towards the evolution of new levels of individuality. In addition to group formation and specialisation, we recognise that a more detailed and controlled structuring of interaction is necessary for the evolution of multicellularity. Furthermore, we claim that this can only come about through phenotypic plasticity and that mixed strategies that have no context-sensitive expression are insufficient.

In the following section we give a full categorisation of two-player, symmetric DOL games and in section 3 discuss two different notions of social efficiency and how these are affected by population structure. In section 4 we briefly study some biological examples of DOL games from a number of different fields. In section 5 we extend traditional models of evolutionary game theory in order to allow for the possibility of conditional expression of phenotype.

2 Categorisation of DOL games

We restrict our attention to symmetric, two-player cooperative dilemmas, as all our key points can be understood from within this simple framework. All symmetric, two-player cooperative dilemmas can be represented via a two-by-two payoff matrix, M, with four parameters: R, S, T, P (Rapoport and Guyer, 1967) (see equation (2.1)). M can be simplified due to the fact that payoff is relative. One can multiply all payoffs by a positive constant without qualitatively changing the features of the game (although this alters the speed of selection). Likewise, one may also add a constant to every payoff (Weibull, 1997). This permits one to write the payoff matrix as:

$$M = \begin{pmatrix} R & S \\ T & P \end{pmatrix} \Rightarrow \begin{pmatrix} 1 & S \\ T & 0 \end{pmatrix} \tag{2.1}$$

without loss of generality. Thus the space of all possible symmetric, two-player cooperative dilemmas can be represented in the TS-plane (Santos and Pacheco, 2005), in which there are four games with qualitatively different ESSs (see figure 4.1). The state of the population is determined by x, the frequency of the strategy cooperate. The four games are:

Prisoner's dilemma $S <= 0$ and $T >= 1$. Stable/unstable fixed point at $x = 0/x = 1$.

Snowdrift $S > 0$ and $T > 1$. Two unstable fixed points at $x = 0$ and $x = 1$ and a stable fixed point at $x = {}^{S}/_{(S+T-1)}$.

Stag-hunt $S < 0$ and $T < 1$. Two stable fixed points at $x = 0$ and $x = 1$ and an unstable fixed point at $x = {}^{S}/_{(S+T-1)}$.

Harmony $S >= 0$ and $T <= 1$. Stable/unstable fixed point at $x = 1/x = 0$.

The DOL games lie in the region where $S + T > 2R$. These games have previously been excluded from the definition of cooperative dilemmas because they do not meet Dawes first criterion; that fitness is maximised by all individuals cooperating (see for example Macy and Flache (2002)). DOL games have the property that the SES is polymorphic. Furthermore, we distinguish two varieties of SESs: firstly, the unstructured SES is the frequency of strategies that maximises mean fitness given that the interactions between individuals are constrained to be random with respect to phenotype (i.e. there is no phenotypic assortment) and secondly, the structured SES, which is the state of the population (including both strategy frequency, and their pattern of interactions) that maximises mean fitness. The unstructured SES is found from the equation for mean fitness: $\bar{\pi} = x^2 + x(1-x)(S+T)$, which has a maximum at:

$$x_{\text{SES}} = \frac{S+T}{2(S+T-1)} \tag{2.2}$$

Note that this is not equal to the ESS, except when $S = T$. Section 3 discusses the structured SES.

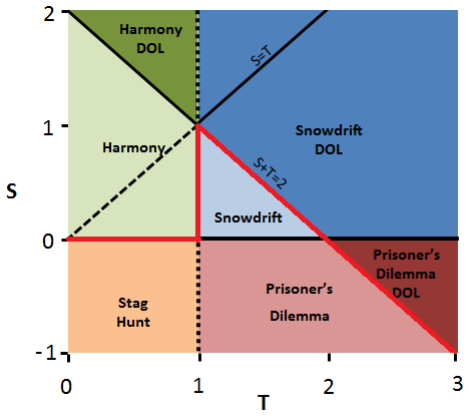


FIGURE 4.1: The TS-plane: all games are categorised according to the nature of their equilibria (the four named games) and on the nature of the state that maximises the mean payoff; either pure cooperate, or a mixed state (appended with DOL). The Dawesian dilemmas are those under the red line.

3 The Role of Assortment

Assortment measures the extent to which the interactions in a population are structured. Given that in a DOL game fitness is maximised by an intermediate level of 'cooperation' it is tempting to reach the conclusion that an intermediate level of assortment will lead to an intermediate level of cooperation, and would thus maximise mean payoff (as claimed by Stark (2010)). This is not the case, however, as we show here.

The state of the population is fully specified by: x_A, the frequency of strategy A, and φ, the frequency of interactions that are between *unlike* types (Pacheco et al., 2006; Van Segbroeck et al., 2009). We define the *population state* to be the pair of variables (x_A, φ). φ lies within the interval $[0, 2 \times \min\{x_A, 1 - x_A\}]$ (the triangular region of figure 4.2).

If the population is well-mixed then the probability that an A meets a B is equal to the probability that a B meets a B and hence:

$$\varphi = \varphi^{(R)} = 2x_A(1 - x_A) \tag{3.1}$$

where R stands for random. Note, that equation (3.1) can alternatively be arrived at by assuming that the probability of an A meeting an A is equal to the probability of a B meeting an A. The essential point is that the probability of meeting a certain type is statistically independent of one's own type.

The SES given in equation (2.2) assumes that interactions are uncorrelated by phenotype. In general the SES is a function of phenotypic assortment. Furthermore, if individuals are able to control both phenotypic assortment and strategy frequency, then there exists an even higher fitness state. Let the *structured* SES be the value of (x_A, φ) that maximises mean payoff. In the

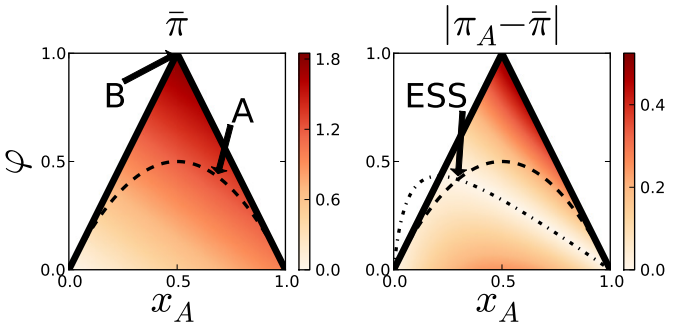


FIGURE 4.2: Left: colour indicates mean payoff $\bar{\pi}(x_A, \varphi)$. The dotted line represents $\varphi^{(R)}$, the unstructured (A) and structured (B) SESs are also indicated. Right: colour is the absolute value of the difference between the fitness of strategy A and the mean fitness ($\pi_a - \bar{\pi}$), thus giving a measure of stability under selection. Dashed dotted line indicates $\pi_a - \bar{\pi} = 0$. In this example $(S, T) = (0.8, 2.9)$.

TS-plane the average payoff, $\bar{\pi}$, is given by:

$$\bar{\pi} = x_A + \frac{1}{2}(S + T - 1)\varphi \tag{3.2}$$

And thus:

$$\text{structered SES} = \begin{cases} (1, 0) & S + T < 2 \\ (1/2, 1) & S + T > 2 \end{cases} \tag{3.3}$$

Thus, in DOL games individuals that engage exclusively in A-B interactions ($\varphi = 1$) maximise fitness, as illustrated by figure 4.2.

The unstructured SES is the highest fitness state subject to the constraint that $\varphi = \varphi^{(R)}$, whereas the structured SES is the highest fitness state when this constraint is relaxed. One of the central claims of this paper is that this state can only be reached if either (A) individuals are able to control the phenotype of the individual with whom they interact, or (B) they are able to control their phenotype conditionally upon the phenotype of their partner. Neither SES is in general stable, as figure 4.2 illustrates. Increasing assortment so that the equilibrium frequency equals that of the unstructured SES will not 'solve' a DOL game because the population state will also move below the dotted line in figure 4.2 and hence be at a lower fitness than either SES; this is contrary to the claims of Stark (2010).

It is worth considering in more detail the question of whether or not division of labour games can be usefully thought of as cooperative dilemmas. In conventional cooperative dilemmas there

is a distinction between the state of the population that maximises mean fitness, and the state that evolution actually reaches. As it stands this is merely an observation, as there is no reason that evolution should act to maximise the mean fitness of the population, as selection acts upon individuals, not populations. However, many models of the evolution of cooperation have shown that there are certain circumstances in which selection on individuals will cause the population to reach the state that maximises the mean fitness of the population (see for instance Nowak (2006b); Godfrey-Smith (2008)). Conventional models of the evolution of cooperation are not normally phrased in such a manner, preferring instead to talk about conditions that maximise the frequency of cooperation. This quantity is only of interest because (in non-DOL dilemmas) maximising cooperation maximises mean fitness. In DOL games, in contrast we need to be more explicit about the fact that mean fitness is the quantity of interest because the frequencies of each strategy are, by themselves, not sufficient to describe the fitness maximising state.

4 Examples of DOL Games

The defining characteristic of a division of labour game is that the population's mean fitness is maximised by having a polymorphic state, rather than having a pure state composed solely of cooperators. As a brief aside before presenting our central model and to further justify the need for such a formalism, we illustrate three examples of DOL games from three disparate areas of biology. Other authors have looked at the evolution of the division of labour, most notably in the context of bi-parental care of offspring (Harrison et al., 2009) and in foraging duties in eusocial insects (Pollock et al., 2012), here I consider a number of other examples.

Firstly, slime moulds: slime moulds come in two phenotypic varieties, spores and stalks. The current hypothesis being that the presence of stalks increases the fecundity of spore cells by increasing their dispersal radius (Strassmann et al., 2011, 2000). We thus have two strategies: spore (Sp) and stalk (St). Stalk cells receive no benefit from either other stalk cells or from spore cells, they also have a base line fitness of 0. Spore cells receive a benefit $b > 0$ from a stalk cell and have a base line fitness of 1 (for the purposes of this example). The resulting payoff matrix is given by table 4.1. This is a division of labour game only if $b > 1$. Thus whilst stalk cells may be thought of as altruists in the sense of Hamilton (Hamilton, 1964a; West et al., 2007), they are certainly not cooperators in a prisoner's dilemma, as a population composed entirely of stalk cells would in fact minimise mean fitness, not maximise it.

	Sp	St
Sp	1	$1+b$
St	0	0

TABLE 4.1: Payoff matrix for slime moulds playing either spore (Sp) or stalk (St).

Our second example is anisogamy. The hypothesis that we examine is that male sex cells are adapted to eject mitochondria in order to minimise intra-organelle conflict (Hurst and Jiggins, 2005). Crucially we conduct the analysis from the view point of the nuclear DNA, as opposed to the mitochondrial DNA. A zygote formed from two female sex cells is less fit than one formed from a male and a female sex cell because of conflict between bi-parentally inherited organelles. A zygote formed from two male sex cells is unviable. Let the fitness of the zygote formed from a male and female cell be 1, and let the cost incurred by conflict be k. The payoff matrix for the two strategies male (M) and female (F) is given by table 4.2. As $k > 0$ this is a DOL game. Some conventional models of the evolution of anisogamy (Bell, 1978) portrayed the male as a form of parasite on the altruism of the female (see also Roughgarden et al. (2006) for a criticism of this line of thinking). However, by our view neither the male nor the female may be construed as the cooperator or the defector. The species benefits from having a reduction in organelle conflict, and for this to be achieved two different, yet complementary, roles must be performed.

	F	M
F	$1-k$	1
M	1	0

TABLE 4.2: Payoff matrix for the nuclear genes of male (M) and female (F) in which there is a cost to organelle conflict.

Our final example comes from genetics and the phenomenon of over-dominance. It has been shown (Traulsen and Reed, 2012) that the interaction of two alleles at a single locus in a diploid organism has an exact correspondence to two-player evolutionary game theory if one considers the allele (and not the typical biological individual) as the agent in the game. Each diploid individual is one round of a 'game' between two alleles. Consider two alleles A and B. In addition assume that the heterozygote has a higher fitness than either homozygote (i.e. over-dominance or heterozygotic-superiority). The corresponding fitnesses are ω_{AA}, ω_{AB} and ω_{BB}. The fitness of an allele is the fitness of the zygote in which it resides multiplied by the probability that it is transmitted. The transmission probability is one half under normal Mendelian genetics, but may differ from one half if one considers the action of meiotic drive (Burt and Trivers, 2009). The resulting payoff matrix is given by table 4.3. The condition for overdominance is identical to the condition of a division of labour game. Population mean fitness is maximised by a polymorphic state of both alleles, this is a special case of a DOL game as here $S = T$, (i.e. the off diagonal elements are equal). Because of this, evolution does tend towards a frequency of alleles that maximises the mean fitness of the population. In our language this is the unstructured SES. Note also that fitness could be further increased if *all* individuals were heterozygotic, this would be the structured SES.

	A	B
A	$\frac{1}{2}\omega_{AA}$	$\frac{1}{2}\omega_{AB}$
B	$\frac{1}{2}\omega_{AB}$	$\frac{1}{2}\omega_{BB}$

TABLE 4.3: The payoff matrix for a allele A against allele B in a diploid individual.

As a general point, we do not claim that the above examples have not been sufficiently studied in their own right. Instead, we are making the claim that the phenomenon of division of labour games is common across a broad spectrum of biological examples. Our contribution is to recognise that these seemingly disparate examples all share deep commonalities. Table 4.4 considers whether or not a number of examples can be considered as (A) DOL games and (B) cooperative dilemmas. We include an additional example here, not discussed in above, of public good production in a biofilm. We argue that this is not a DOL game and is in fact a standard cooperative dilemma, as there is only one task to be performed, and mean fitness is a monotonic function of the amount of this public good being produced.

Phenomenon	Division of Labour Game?	Cooperative Dilemma?
Public good production in biofilms (Crespi, 2001)	No	Yes
Diploid Genetics	Yes, in the case of overdominance	Only when one considers meiotic drive
Anisogamy	Yes	No for nuclear DNA, yes for mitochodrial DNA
Slime moulds	Yes, if $b > 1$	Yes

TABLE 4.4: Division of labour games in nature. Columns indicate whether the situation can be thought of as (A) a division of labour game and (B) a cooperative dilemma.

5 Models and Results

We present here an extension of simple *one-gene one-phene* game theoretic models in order to show that a conditional expression of phenotype is necessary for a population to reach the structured SES. In our model the phenotype of an individual is expressed conditionally upon the phenotype of the parent and may thus be construed as a manner of parental effect (Mousseau and Fox, 1998; Marshall and Uller, 2007). Individuals are characterised by two genes that determine their strategy. The first gene, the social strategy, $x \in [0, 1]$, is the probability of playing strategy A. The second gene, the replication strategy, $\beta \in [-1, +1]$, determines the probability that an offspring is of the same ($\beta > 0$) or opposite ($\beta < 0$) phenotype to its parent. This can be

interpreted as either the influence the parent has on the offspring, or the reaction of the offspring to the parent's phenotype.

When a parent-offspring pair interact the parent's phenotype is determined by its social strategy. The phenotype of the offspring is determined by the replication strategy of the parent. If $\beta > 0$ it copies the phenotype of the parent with probability β, and if $\beta < 0$ it adopts the opposite phenotype to its parent with probability $|\beta|$, otherwise it reverts to its own social strategy. When two random members of the population meet both adopt a strategy determined solely by their social strategy.

We allow for an arbitrary level of genetic assortment, or relatedness, $r \in [0, 1]$. This can be modelled as follows: with probability r an offspring remains with its parent and the two interact, and with probability $1 - r$ the offspring interacts with a random member of the population. Note that r is a measure of assortment in exactly the same manner as φ (see section 3). However, we use a distinct symbol here as r is a measure of *genetic* assortment, whereas φ measures *phenotypic* assortment. In conventional models of game theory these two parameters are exactly equivalent; the distinction only becomes apparent in the presence of a separation of phenotype and genotype.

Although Hamilton phrased his formulation of relatedness on the notion of co-ancestry (Hamilton, 1964a) it was soon realised that the most general formulation of relatedness is in terms of a regression coefficient on genetic value (Orlove, 1975; Grafen, 1985) (see Gardner et al. (2011) for a recent review). r is defined by: $r = {}^{\mathrm{Cov}(g',g)}/_{\mathrm{Var}(g)}$, where g is the value of the gene at the focal locus and g' the value of the gene of the partner. Note that it is possible for the same value of relatedness to be embodied in a number of different ways. It may be that individuals meet only clonal individuals a certain fraction of the time, or that they meet individuals who are only somewhat similar to themselves *all* of the time, or anything else in-between. The claim of inclusive fitness theory is that the action of selection is the same in either case. We therefore model r in the simplest possible way; namely, that individuals meet clonal individuals with probability r (and random individuals with probability $1 - r$).

Frequently an individual's phenotype is determined not solely by the genotype or the abiotic environment of the individual, but through manipulation of the phenotype by the parent; this is referred to as a parental (or maternal) effect (of phenotype) (Mousseau and Fox, 1998; Marshall and Uller, 2007). Parental effects are common across many taxa, as can be seen in eusocial insects (Hölldobler and Wilson, 1990), in plants (Bernardo, 1996), and even in bacteria such as rhizobium (Bever and Simms, 2000). In addition, asymmetric cell division (Roegiers and Jan, 2004) can be thought of as parental control of phenotype at the cellular level**.** What is required for the solution to a DOL game that is also a cooperative dilemma is some manner of context sensitive determination of phenotype. However, this is not sufficient, as there remains a potential conflict between strategies. Therefore, in addition to context sensitive development of phenotype, genetic assortment, or relatedness, is necessary for the resolution of a DOL cooperative dilemma. We therefore complement the model presented in this paper with an alternative

formulation of our model to further illustrate the generality of our claims, see appendix B. In the alternative model we construe the value β to be a reaction to the phenotype of another individual who is not necessarily the parent of the focal individual. In this model one individual, chosen at random, reacts to the phenotype of the other individual. The results of this model are *qualitatively* the same as the one presented here, and all the key points we make here apply equally well to this alternative model.

We proceed by calculating the expected fitness of each individual as a function of its genotype and of the state of the population. When an individual with social strategy p meets an individual with social strategy q it receives a payoff given by:

$$F[p,q] = pq + p(1-q)S + (1-p)qT \tag{5.1}$$

which is the expected payoff to mixed strategy p on meeting q. On meeting a random individual the expected payoff to an individual with genotype $[x,\beta]$ is given by $\pi_{\text{rand}} = F[x, E[x]]$, where $E[x]$ is the average value of x. On being paired with a clonal individual the payoff is defined as:

$$\pi_{\text{self}}[x,\beta] = \begin{cases} (1-\beta)\,F[x,x] + \beta x & \beta \geq 0 \\ (1+\beta)\,F[x,x] - \beta\left(\frac{S+T}{2}\right) & \beta < 0 \end{cases} \tag{5.2}$$

The fitness of the individual is given by:

$$\pi = (1-r)\,\pi_{\text{rand}} + r\pi_{\text{self}} \tag{5.3}$$

We calculate ESSs for all points on the TS-plane (see appendix A). We find that they are always unique and that all ESSs lie at the extreme values for x and β. We also model selection via numerical integration of the replicator equation (Taylor and Jonker, 1978). We do this by selecting an initial population, but fixing r in each case. In the limiting case of an infinitely large population the initial state is fully specified by stating the density of individuals that exist in the population with a given genotype (x,β), subject to the constraint that total density equals one. In order to integrate this numerically we break up the space of possible genotypes into a discrete grid of 100 by 100 possibly genotypes. We investigate the dynamics by starting from both a uniform initial condition and from a random one. Here random means simply selecting a value for density at each point independently from the uniform distribution $[0,1]$, and normalising so that this distibution sums to one. In the latter case we repeat the numerical experiment multiple times for different random initial conditions. We find that in every case the evolutionary dynamics lead to the population settling in the unique ESS.

We also analyse two special cases of the full model for means of comparison: the pure strategy model, in which $\beta = 0$ for all individuals and x is either one or zero, and the mixed strategy model in which $\beta = 0$ for all individuals, but x may vary continuously. These two cases are used as bench marks to illustrate when more sophisticated strategies will be at a selective advantage.

The state of the population is characterised by the frequency of phenotype A, x_A and by the extent to which interactions are phenotypically correlated: cov (p, p') (covariance of phenotype for all interacting pairs). Figure 4.3 shows the equilibrium values of these two variables for one generic non-DOL and one DOL prisoner's dilemma, illustrating that, as relatedness increases, selection favours those strategies that increase the amount of negative phenotypic assortment, and move x_a towards one half. This illustrates that such a manner of conditional expression of phenotype is sufficient for a population to move towards the structured SES. We also show the fitness attained by the full model, as compared with the mixed and pure strategy models. These models reach the structured SES, the unstructured SES and all-A state respectively, illustrating the fitness advantage to conditional expression of phenotype in DOL games for sufficiently high relatedness.

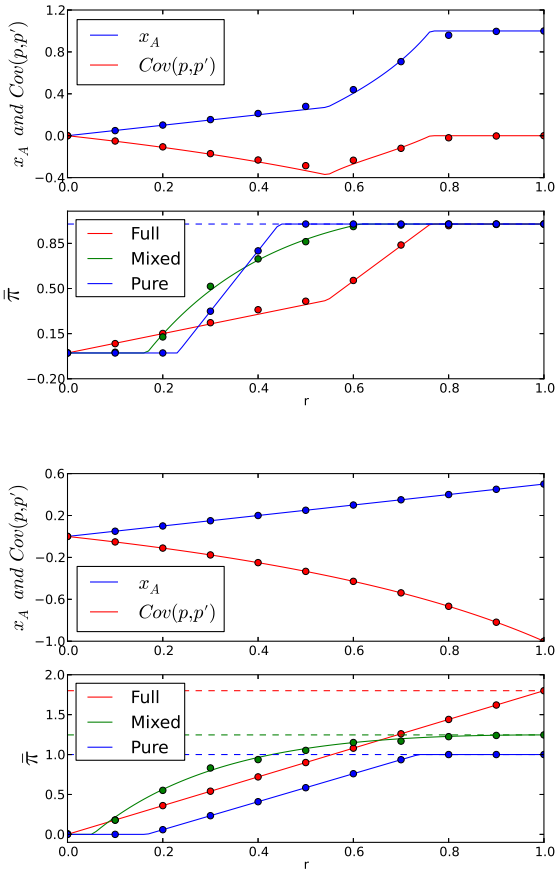


FIGURE 4.3: Equilibrium states for one non-DOL game (top: $(S, T) = (-0.3, 1.8)$) and one DOL game (bottom: $(S, T) = (-0.2, 3.8)$) as a function of relatedness. Top figures: x_A and cov (p, p') plotted against r. Bottom figures: solid lines represent the actual achieved fitness of the three models ($\bar{\pi}$) for increasing r. The three dashed lines represent the theoretical fitness that the population would obtain at (from top to bottom) (A) the structured SES, (B) the unstructured SES and (C) a population composed entirely of strategy A (note that in the non-DOL game these three states coincide, and thus only one line is shown). The points are the result of numerical integration of the replicator equation, and the lines the exact ESS of the model (see appendix A).

Figure 4.4 categorises the nature of all ESSs on the TS-plane for varying levels of r (see appendix A for a derivation). When $r = 0$ the ESSs are the same as in the conventional pure strategy models of evolutionary game theory (see Nowak (2006a); Santos and Pacheco (2005)). For $0 < r < 1$ we find seven qualitatively different types of ESSs. These ESSs may be understood as strategies that maximise individual fitness, as given by equation (5.3). The first part of equation (5.3), π_{rand}, is maximised by the social strategy corresponding to the ESS in the pure strategy case. The second part is maximised by $[0, -1]$ and $[1, -1]$ if $S + T > 2$, or by $[1, +1]$ otherwise. The resulting ESS is a compromise between maximising both terms, the relative importance of each changes with r. For small r the optimal strategy is to choose a social strategy to maximise π_{rand}, and then to choose a replication strategy to maximise π_{self}, given the constraint of what has been chosen for the social strategy. This latter choice depends upon which of three qualitatively different regions the game lies in:

1. $S + T < 2P$: the unlike pairing is the worst possible outcome, and thus it pays to meet A with A and B with B.
2. $2P < S + T < 2R$ all strategies do best when paired with an A, favouring the strategies $[1, +1]$ and $[0, -1]$.
3. $S + T > 2$ unlike pairings are the best possible outcome, so it pays to meet A with B and B with A.

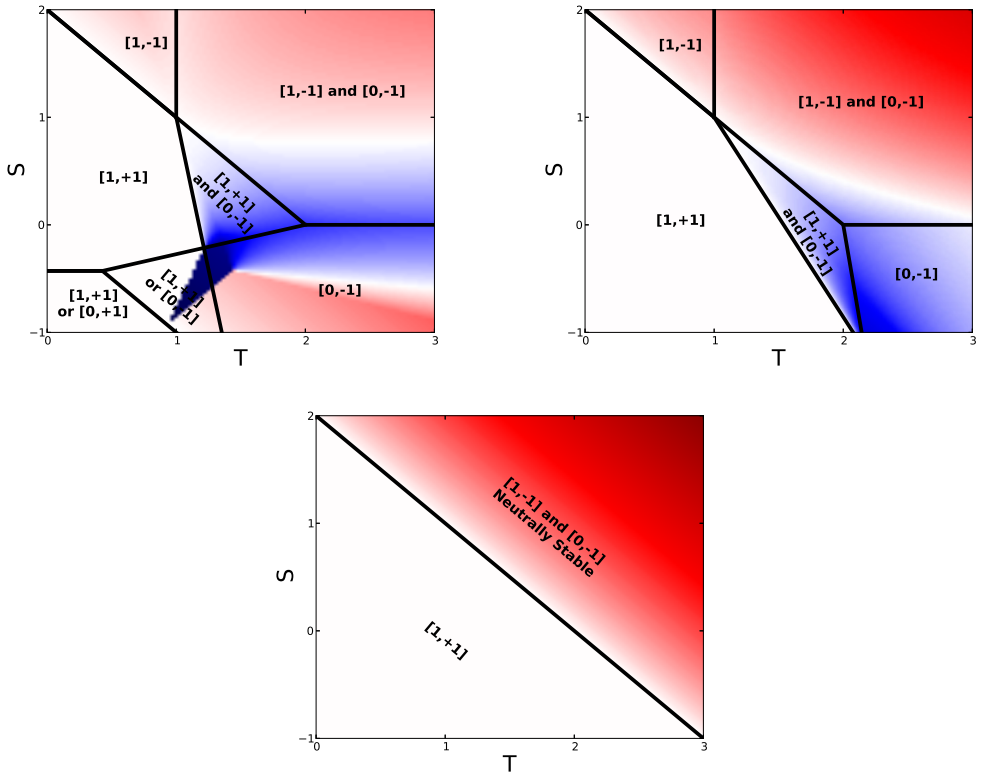


FIGURE 4.4: ESSs in the TS-plane. Solid black lines denote qualitatively different regions of ESSs. Colour represents the fitness of the full model minus the fitness of the mixed strategy model. Blue/red regions indicate where the mixed/full model have higher fitness. From left to right: $r = 0.3$, 0.7, and 1.

As r increases no qualitatively new ESSs emerge, instead the regions in which they are stable smoothly deform. Take for example the region $[1, +1]$ and $[0, -1]$. For small r this region lies mostly in the non-DOL snowdrift game. Selection favours a ratio of A and B given by the fixed point of the pure strategy game. Upon meeting a relative the As do better from meeting another A, so the replication strategy is to make one's offspring like oneself (i.e. $\beta = +1$). Bs also do better upon meeting As and thus make their offspring unlike themselves ($\beta = -1$). As r increases this region encroaches on the prisoner's dilemma. The additional gains that an $[1, +1]$ makes on meeting a relative begin to outweigh the losses it makes upon being paired with a random individual (as AA scores more highly than AB in this region). As r approaches one the ESSs begin to merge with the strategies that create the structured SES ($[1, +1]$ for $S + T < 2R$ and one of $[1, -1]$ or $[0, -1]$ for $S + T > 2R$). When $r = 1$ the strategies $[1, -1]$ and $[0, -1]$ have exactly equal fitness in the DOL region and are thus neutrally stable.

The colour of figure 4.4 summarises the key results of this paper by plotting the fitness of the full model, minus the fitness of the mixed strategy model for three incremental values of r. The reason for comparing the full model to the mixed model is that the mixed model obtains the next highest fitness to the full model and we specifically wish to illustrate the additional fitness increment that is obtained from the addition of context sensitive strategies, over mixed

strategies. In the absence of relatedness the two models are equivalent. At intermediate levels of r, in some regions of game space, the mixed strategy model actually achieves a higher fitness than the full model. At higher levels of r the full model reaches a state with significantly higher fitness. Furthermore, this effect becomes more pronounced the further into the DOL region the game lies. Mixed strategies allow for the maintenance of heterogeneity despite being genetically homogeneous, which allows them to reach the unstructured SES. They are not able, however, to control phenotypic assortment and are thus unable to reach the structured SES. This is achieved through individuals creating full negative assortment on phenotype. The key feature of the full model is the separation of genotype from phenotype. This allows for a complete decoupling of phenotypic assortment from genotypic assortment, thus allowing individuals to fully explore the population state (as described by figure 4.2). This is a feature of collective living organisms such as cellular slime moulds, the individual cells of which are able to develop into one of two phenotypes, and to do so in a manner that is sensitive to their social environment (Strassmann et al., 2011; Nanjundiah and Sathe, 2013). These key features mean that the colonies begin to exhibit adaptation in their own right by having high levels of specialisation and functional integration and are thus candidates for individuality (Queller, 1997; Clarke, 2011; Herron et al., 2013), rather than being merely groups of cooperators.

6 Discussion

In recent years there has been a call to move evolutionary theory beyond the modern synthesis, possibly via some form of extended synthesis (Pigliucci, 2007; Pigliucci and Müller, 2010; Laland et al., 2015). One particular phenomenon that may not be adequately taken into account is phenotypic plasticity (West-Eberhard, 2003) and how this affects, and is affected by, genetic evolution. Evolutionary game theory, despite a large amount of success in explaining interesting biological phenomena (see for example Bulmer (1994)), is very much rooted in the *one-gene one-phene* paradigm of genetics. In order to understand phenotypic plasticity we must begin to extend these models.

This paper has presented a simplest case scenario that extends the conventional models of evolutionary game theory in order to incorporate the possibility of context sensitive expression of phenotype. This was done by allowing for a general genetic encoding of a context sensitive strategy via a two locus genome. Rather than considering the abiotic environment of the individual as the key driving force for phenotypic plasticity, we look instead at how social interaction (and in particular the social interactions between parent and offspring) can be its main driver (Uller, 2008; Badyaev and Uller, 2009; Marshall and Uller, 2007). This is crucial if one wants to gain an understanding of the early stages of the evolution of multicellularity and other analogous fraternal transitions in evolution (Maynard Smith, 1964; Queller, 2000). In particular, a crucial feature of such transitions is the presence of epigenetic expression of phenotype (Jablonka and Lamb, 2006). We have also shown that the evolution of conditional epigenetic strategy can also come about through a different route by investigating an alternative formulation of the model,

whereby individuals react to the strategy of other individuals in the population, see appendix B. In the first instance the epigenetic determination of strategy comes about through parental manipulation of the child's phenotype, and in the second instance it comes about through the conditional expression of phenotype as a reaction to the phenotype of the individual with whom it interacts. However, particularly when the individuals are clonally related, this distinction is only a matter of perception. If two individuals share identical genes then one individual telling the other what to do and one individual reacting to another individual are indistinguishable cases. The second model differs only in the fact that individuals can also react to the phenotype of individuals who they are not related to.

One of the key features for selection to favour conditional expression of phenotype is that there exists some benefit to the simultaneously performing of two different tasks. This is a feature that the conventional Dawesian cooperative dilemmas do not possess. We have shown, however, that by extending the notion of cooperative dilemmas to include the DOL games (those games where polymorphic states are socially efficient), one can find scenarios where conditional expression of phenotype will be at a selective advantage, i.e. when relatedness is high. A key advantage of our model is that it can be used to analytically explore all possible two-player interactions. We find that conditional strategies become increasingly favoured the further into the DOL region the game lies. The reason for the success of the conditional strategies is that there exists, in these games, a fundamental problem; as these games are cooperative dilemmas they require positive assortment for their resolution (Eshel and Cavalli-Sforza, 1983). However, as they benefit from heterogeneous interactions they are "solved" by negative assortment. This tension is only resolved with phenotypic plasticity, which allows for a simultaneous positive assortment on genotype and a negative assortment on phenotype.

A Appendix: Determining ESSs on the TS-plane

In this appendix we illustrate how to calculate the properties and location of the qualitatively different regions of ESSs on the TS-plane. We do this firstly for the special case of mixed strategies and then for the full model involving strategies that are able to act conditionally upon the phenotype of their parents.

A.1 Mixed Strategies

A mixed strategy individual is characterised by a single number, $x \in [0, 1]$, which represents the probability of playing strategy A in any given encounter. The *expected* payoff an individual with strategy p receives on meeting an individual with strategy q is given by:

$$F(p, q) = pq + p(1 - q)S + (1 - p)qT \tag{A.1}$$

The population is genetically assorted, and thus individuals play a clonally related individual with probability r and a random one with probability $1 - r$. Thus, the expected payoff to an individual with strategy x is given by:

$$\pi(x) = rF(x, x) + (1 - r)F(x, E[x]) \tag{A.2}$$

where $E[x]$ is the mean value of x.

For a strategy x to be at equilibrium it must maximise equation (A.2), with the additional condition that $E[x] = x$. That is:

$$\frac{d\pi}{dx}|_{E[x]=x} = 0 \tag{A.3}$$

This occurs at the point:

$$x^* = \frac{S + rT}{(1 + r)(S + T - 1)} \tag{A.4}$$

Note: that this is equal to the unstructured ESS when $r = 1$. Furthermore, the equilibrium is stable only if $\frac{d^2\pi}{dx^2} < 0$, which occurs for $S + T > 1$.

From $x^* = 1$ it follows that: $S = -rT$ and $x^* = 0$: $S = \frac{1+r-T}{r}$. These are the lines at which the mixed strategy equilibrium leave the valid region for x and thus delimit the regions in which the ESS is a mixed strategy and the regions in which it is a pure strategy (see figure 4.5).

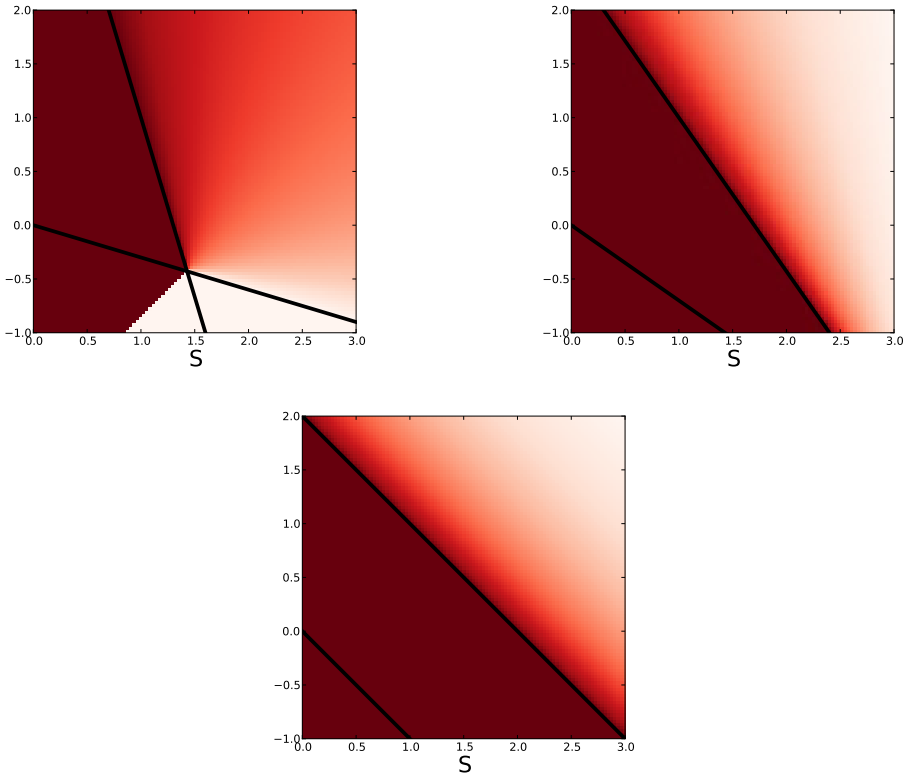


FIGURE 4.5: The mean value of phenotype A for the mixed strategy case with $r = 0.3$, 0.7 and 1.0. Also shown the lines: $S = -rT$ and $S = \frac{1+r-T}{r}$.

A.2 Conditional Strategies

Individual strategy is characterised by a two locus genome, the social strategy, x, and the replication strategy, β. With probability $1-r$ an individual is paired with another random individual, in which case the expected payoff is given by:

$$\pi_{\text{rand}}(x) \quad = \quad F(x, E[x]) \tag{A.5}$$

as is the case for the mixed strategy model. With probability r an individual is paired with a clonal relative, in which case individual one determines the phenotype of individual two (which role the individual takes is decided at random). If $\beta < 0$ then individual two *opposes* the phenotype (i.e. does the opposite) of individual one with probability $-\beta$, otherwise individual two chooses a phenotype based on its own social strategy. Thus, if no opposition occurs the expected payoff is given by $F(x, x)$. If, however, opposition occurs then each player gets either S or T with probability one half. The expected payoff is then given by:

$$\pi_{\text{self}}(x, \beta < 0) = (1+\beta)F(x,x) - \beta\left(\frac{S+T}{2}\right) \tag{A.6}$$

If $\beta > 0$ then the second individual *copies* the phenotype of the first with probability β, and gets $R = 1$ with probability x or $P = 0$ with probability $1 - x$. Otherwise, payoff is again determined by $F(x, x)$. Thus:

$$\begin{aligned} \pi_{\text{self}}(x, \beta > 0) &= (1-\beta)F(x,x) + \beta x R + \beta(1-x)P & \text{(A.7)} \\ &= (1-\beta)F(x,x) + \beta x & \text{(A.8)} \end{aligned}$$

and thus:

$$\pi_{\text{self}}[x, \beta] = \begin{cases} (1-\beta)\, F(x,x) + \beta x & \beta \geq 0 \\ (1+\beta)\, F(x,x) - \beta\left(\frac{S+T}{2}\right) & \beta < 0 \end{cases} \tag{A.9}$$

The overall payoff is given by:

$$\pi(x, \beta) = (1-r)\, \pi_{\text{rand}}(x) + r\pi_{\text{self}}(x, \beta) \tag{A.10}$$

Mixed strategy equilibria must satisfy the condition that they are at a maximum of fitness. However, we find that the fitness function has no maxima within $x \in [0, 1]$ and $\beta \in [-1, +1]$. This is found from verifying that the equation:

$$\begin{pmatrix} \frac{\partial \pi}{\partial \beta}|_{E[x]=x} \\ \frac{\partial \pi}{\partial x}|_{E[x]=x} \end{pmatrix} = 0 \tag{A.11}$$

has no solutions.

Combinations of pure strategies may form ESSs. These are found simply by requiring that fitness of all strategies at equilibrium must be equal. For example, for $[1, +1]$ and $[1, -1]$ to form

an ESS it is necessary that $\pi(1,1) = \pi(1,-1)$. There are no solutions for all four strategies having equal fitness, nor are there any solutions for the four possible combinations of three strategies. However, the following pairwise equalities have solutions:

$$\pi(1,1) = \pi(0,-1)$$

at:

$$x = \frac{r(3S+T-2)-2S}{2(1-r)(1-S-T)} \tag{A.12}$$

and:

$$\pi(1,-1) = \pi(0,-1) \tag{A.13}$$

at:

$$\frac{S}{S+T-1} \tag{A.14}$$

and:

$$\pi(1,1) = \pi(0,1) \tag{A.15}$$

at:

$$\frac{r\,(S-1)-S}{(1-r)(1-S-T)} \tag{A.16}$$

no other mixtures of pure strategies have solutions.

These equilibria may be either stable or unstable. Stability occurs only if:

$$\frac{\partial}{\partial E[x]}\left(\pi(1,\beta)-\pi(0,\beta)\right) < 0 \tag{A.17}$$

Each of the three found ESSs are unstable if $S+T>1$.

The ESSs comprised of single pure strategies are found simply by checking which of the pure strategies has maximum fitness. Figure 4.6 shows how the TS-plane is split into different qualitative regions of ESSs. The regions in which certain ESSs exist is found by asserting that a mixed ESS lies within the interval of validity: $x \in [0,1]$. Line A is found from setting equation (A.12) equal to one, and line C is found from setting equation (A.14) equal to one. Lines D, E and F are found by setting equations (A.14), (A.12) and (A.16) equal to zero. Line B is the point where the mixed ESS composed of $[1,-1]$ and $[0,-1]$ is equal in fitness to the mixed ESS composed of $[1,1]$ and $[0,-1]$, and finally, line G is when the pure state of $[0,+1]$ is equal in fitness to the pure state of $[0,-1]$. Table 4.5 summarises all of this information.

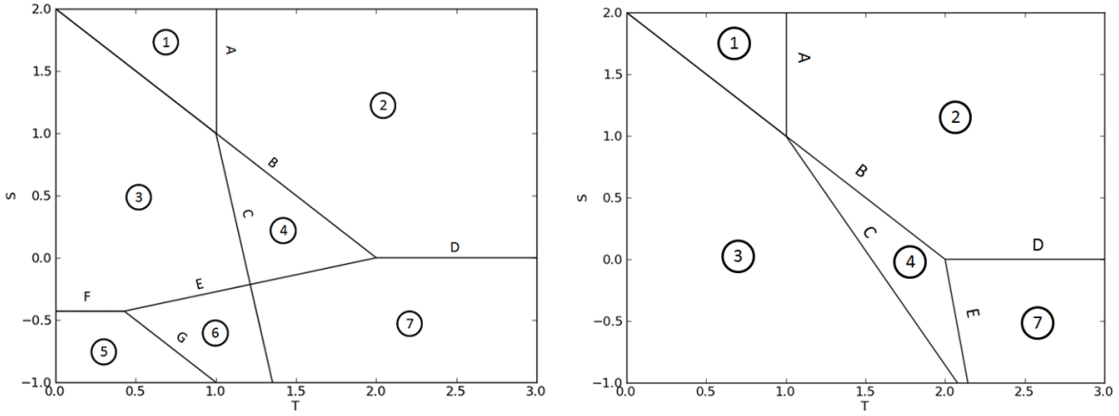


FIGURE 4.6: Schematic for different ESS in the TS-plane. Left: $r = 0.3$ and right: $r = 0.7$.

Label	Equation
A	$T = 1$
B	$S = 2 - T$
C	$S = \frac{2-2T+rT}{r}$
D	$S = 0$
E	$S = \frac{r(2-T)}{3r-2}$
F	$S = \frac{r}{r-1}$
G	$S = -T$

Region	ESS	Frequency of 1st strategy or tipping point
1	Pure $[A, -1]$	NA
2	$[A, -1]$ and $[B, -1]$ coexist	$\frac{S}{S+T-1}$
3	Pure $[A, +1]$	NA
4	$[A, +1]$ and $[B, -1]$ coexist	$\frac{r(3S+T-2)-2S}{2(1-r)(1-S-T)}$
5	$[A, +1]$ and $[B, +1]$ are bistable	$\frac{r(S-1)-S}{(1-r)(1-S-T)}$
6	$[A, +1]$ and $[B, -1]$ are bistable	$\frac{r(3S+T-2)-2S}{2(1-r)(1-S-T)}$
7	Pure $[B, -1]$	NA

TABLE 4.5: Equations of lines separating ESS regions and equilibria values for different ESSs (see figure 4.6).

B Appendix: Alternative Formulation of the Model Using "Reactive" Strategies

B.1 Introduction

In the main paper we presented a model in which the parent controlled the phenotype of the offspring based on a genetic "replication" strategy. The conclusions that we drew were that, in DOL games, the maximum fitness state could only be reached if (A) there exists genetic assortment (i.e. relatedness) and (B) individuals are able to determine the phenotype of the individual with whom they interact. With this appendix we show that the conclusions of the main paper are more general than the particular modelling choices that we made. Specifically, a *qualitatively* similar outcome pertains if individuals react to the phenotype of their opponent based on a genetic copying strategy. As before we envisage individuals paired with genetically similar individuals, but in this case they are not necessarily parent-offspring pairs. The phenotype of one of the individuals, chosen at random, reacts to the phenotype of the other individual. This is in contrast to the previous model where the phenotype of an individual was determined by another individual (in this case the parent). We go on to show that such a model is sufficient for the population to reach the maximally fit state.

The difference between the models is not a profound one, as we shall show. If two individuals are clonally related then there is no difference between being told what to do by a relative, and reacting conditionally to a relative, as in each case the responsible genes are in common between the two individuals. The real difference in this model is that here individuals are able to react to the phenotype of other individuals with whom they are not related.

B.2 The Reactive Strategy Model

As before, an individual's genotype has two loci. Genotype G_i is thus specified via $G_i = [x_i, \beta_i]$. Individuals develop into one of the two possible phenotypes (A or B) in a manner dependent upon their genotype. Individuals are paired and engage in fitness altering interactions according to the standard RSTP payoff matrix. Upon being paired one of the two individuals is arbitrarily, and at random, chosen to be player one, and the other player two. Player one develops into one of the two possible phenotypes stochastically. With probability x_i it becomes an A and with probability $1-x_i$ it becomes a B. Player two then develops into one of the possible phenotypes. If β is positive it copies the phenotype of player one with probability β, and with probability of $1-\beta$ it develops stochastically according to its value of x in the same manner as before. If β is negative, then with probability $-\beta$ it develops into the opposite phenotype of player one, and with probability $1+\beta$ it develops stochastically according to its value of x. β thus plays the role of "desired phenotypic assortment" of the individual in question. It plays a role analogous to β in the previous model, but here it is interpreted as a "reaction" strategy rather than a replication strategy.

We model this situation via the replicator equation, rather than following an agent based approach. The replicator equation is (Taylor and Jonker, 1978):

$$\dot{x}_i = x_i(\pi_i - \bar{\pi}) \tag{B.1}$$

where x_i represents the fractional density of a strategy type i, $\dot{x}_i$ the rate of change of the density and π_i the payoff to strategy type i. $\bar{\pi}$ represents the mean fitness of all strategies, so that strategies increase in frequency in proportion to their current densities and the relative fitness with respect to the average. The state of the population is specified by the density of individuals playing strategy $[x_i, \beta_i]$, for all $x_i \in [0, 1]$ and $\beta_i \in [-1, 1]$. Evolution is modelled simply by integration of the replicator equation through time. We record the mean fitness ($\bar{\pi}$) as well as the mean values for each locus ($\bar{x}$ and $\bar{\beta}$). The replicator equation is constructed by calculating the expected payoff for an individual playing strategy (x_1, β_1) meeting an individual playing strategy (x_2, β_2) (given by $F[(x_1, \beta_1), (x_2, \beta_2)]$). From this we can construct payoffs and integrate the replicator equation until equilibrium is reached. As each player has equal chance of being player one we break the fitness function into the payoff received upon being player one ($F^{(1)}$) and that received upon being player two ($F^{(2)}$) and note that:

$$F[(x_1, \beta_1), (x_2, \beta_2)] = \frac{1}{2}F^{(1)}[(x_1, \beta_1), (x_2, \beta_2)] + \frac{1}{2}F^{(2)}[(x_1, \beta_1), (x_2, \beta_2)] \tag{B.2}$$

The two fitness functions are given by:

$$\begin{aligned} F^{(1)}[(x_1, \beta_1), (x_2, \beta_2)] = {} & (1 - |\beta_2|)\left(x_1\left(x_2 R + (1 - x_2)\, S\right) + (1 - x_1)\left(x_2 T + (1 - x_2)\, P\right)\right) \\ & + \begin{cases} \beta_2\left(x_1 R + (1 - x_1)\, P\right) & \beta_2 > 0 \\ -\beta_2\left(x_1 S + (1 - x_1)\, T\right) & \beta_2 \leq 0 \end{cases} \end{aligned} \tag{B.3}$$

and:

$$\begin{aligned} F^{(2)}[(x_1, \beta_1), (x_2, \beta_2)] = {} & (1 - |\beta_1|)(x_2(x_1 R + (1 - x_1)T) + (1 - x_2)(x_1 S + (1 - x_1)P)) \\ & + \begin{cases} \beta_1(x_2 R + (1 - x_2)P) & \beta_1 > 0 \\ -\beta_1(x_2 T + (1 - x_2)S) & \beta_1 \leq 0 \end{cases} \end{aligned} \tag{B.4}$$

As before, two special cases of the model are also considered by restricting the permissible set of genotypes. Firstly, the mixed strategy model in which individuals are restricted to have $\beta = 0$. Thus, individuals cannot affect phenotypic assortment. One can consider this as the mixed strategy model, as a genotype specifies a probability of playing a certain strategy, although all individuals only ever express one phenotype for the duration of their lifetime. Secondly, we consider a pure strategy model, in which individuals may only play with one of the two pure strategies: $G \in \{[1, 0], [0, 0]\}$.

We also assume there is a level of relatedness parametrised via r. We assume the presence of some form of assorted interactions, but do not specify the detailed mechanism underlying them, see the main text. We simply state that the fitness an individual receives is r times the fitness it receives on playing itself, plus $1 - r$ times the fitness received on playing a random individual, that is individual I receives payoff:

$$F(r) = rF[I, I] + (1 - r)\, F[I, \bar{I}] \tag{B.5}$$

where $\bar{I}$ is the average individual.

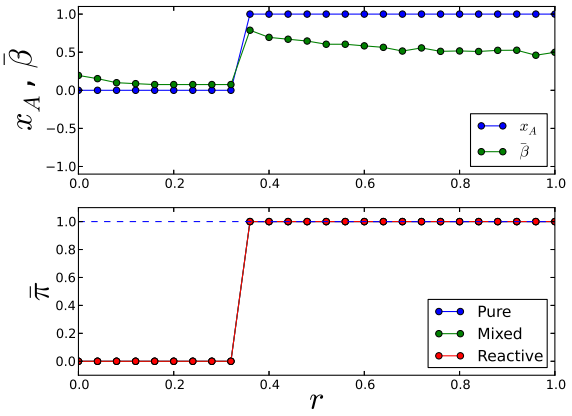
As before we create an initial population of individuals by randomly populating the space of genetic individuals, and normalising so that this distribution sums to one. Again we break up the space of potential genotypes into a discrete 100×100 grid in order to perform numerical integration. We also investigate the model starting from uniform, rather than random, initial conditions, and find the results to be the same in each case. We integrate via the replicator equations until equilibrium is reached and record the average value of x and β as well as the final mean fitness ($\bar{\pi}$). We investigate how x and β depend on the game and the value of r. We also compare final fitness with the two special cases of the model.

B.3 Results

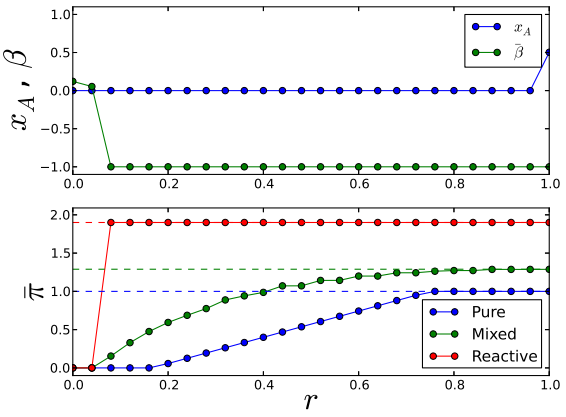
Figure 4.7 shows a figure analogous to figure 4.3. The fitness obtained at equilibrium for the three versions of the model are plotted against increasing relatedness. In the non-DOL game the three versions of the model exhibit identical behaviour. In every model, for low r, all individuals play always defect, and evolve to have a β value equal to 0 (in other words they don't react to the phenotype of their opponent). This is unsurprising as the game is a prisoner's dilemma. Note that the fact that individuals may react to the phenotype of their opponents makes no difference to this game as it is always rational to defect against ones opponent regardless of their strategy. As r increases all three models exhibit an abrupt transition from the all-B state to the all-A state. In the case of the reactive strategies they do this simply by always playing A, in which case any positive value of β will amount to the same behaviour, and thus β evolves through random drift to approximately $1/2$. Notice that close to the transition β is markedly higher than one half. This is because there is selective pressure on β whilst x is evolving towards one. Close to the transition the evolution of x is much slower, creating an initial selective pressure on β. Note that in this non-DOL prisoner's dilemma the addition of more complicated reactive strategies has absolutely no effect, as the "rational" behaviour is to always perform a certain action regardless of the action of ones opponent.

The second example game is a DOL prisoner's dilemma. It thus has a structured SES whereby all pairs of individuals are of unlike types (A-B). Note that the mere presence of reactive strategies is not sufficient for the structured SES to be reached as, although individuals are theoretically capable of creating this structure, it is not in their interests to do so. Only when relatedness

increases will the reactive agents reach the structured SES. Note also that the fixed point of the evolutionary dynamics for the mixed strategy agents transforms smoothly towards the unstructured SES as r increases. Likewise, the fixed point for the pure strategy agents transforms smoothly towards the all-A state for increasing r. The key features of this graph are directly comparable to that of the figure 4.3. Firstly, the three models are identical in the non-DOL games. Secondly, for the DOL game, but in the absence of relatedness, the three models give identical results. Thirdly, for DOL games the conditional strategy version of the model is the only one that is able to reach the structured SES. We can conclude that this manner of complementary strategies can only come about if (A) the game is a DOL game, and there is thus some overall benefit to complementary tasks, (B) the individuals have the *ability* to develop conditionally upon the phenotype of the individual with whom they interact with and (C) there is the motivation to collaborate, i.e. that there is a high level of relatedness. Note that, ordinarily, if $\beta = -1$ it always pays to play B when player one, as other individuals will always play A. This is not true however if $r = 1$, in which case individuals should consider the welfare of their opponent as equally important to their own. Hence for $r = 1$, x_A is equal to one half (see top right panel of figure 4.7).



(a) Non-DOL game $S, T = (-0.5, 1.5)$.

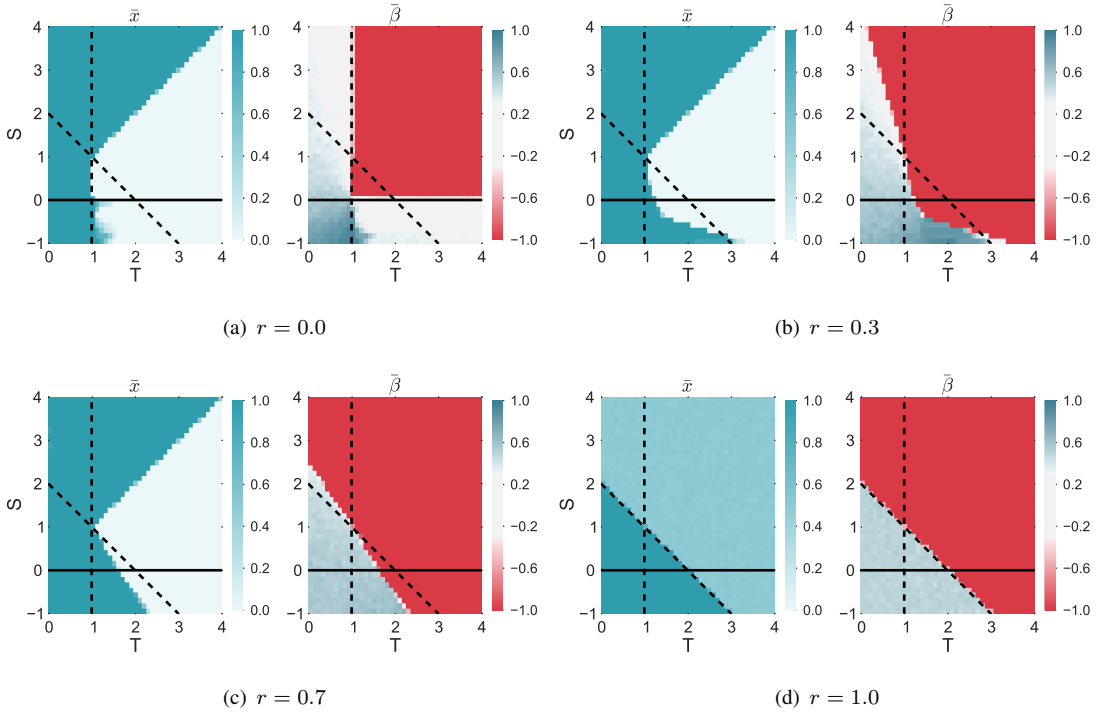


(b) DOL game $S, T = (-0.2, 4.0)$.

FIGURE 4.7: The final fitness obtained for each of the three models for one non-DOL prisoner's dilemma and one DOL prisoner's dilemma for increasing values of r. Also shown: the value of x and β for the full model only. Dashed lines represent the theoretical maximum obtainable fitness in each case. Each point is the mean of 12 runs of the model. Note that in the non-DOL game the three models are indistinguishable, and hence only one set of points is visible.

Figure 4.8 shows the fixed point in terms of both x and β for the space of all possible two-player symmetric games represented by the TS-plane. Note that the diagonal dotted line divides the space into non-DOL (below) and DOL (above) games. The structured SES above the line is to have $\beta = -1$ and $x = 1/2$. Below the line it is simply to have a population composed entirely of strategy A. For low r it is clear that the outcome of evolution is not to reach this optimal state over the entire space. However, the situation is more subtle than in the previous version of the model. Even in the absence of relatedness in the snowdrift game it is rational for an individual to oppose the strategy of the opponent, i.e. to meet A with B and B with A. Under normal assumptions of evolutionary game theory this is achieved in a probabilistic manner, however, if individuals can react to the strategy of their opponent deterministic strategies perform better. Thus, in some regions of the game space individual freely-mixed selection will reach the SES.

However, this is not a surprise since individuals will evolve to this state even in those games where it does not maximise mean fitness. Note also that in this model, unlike the conventional snowdrift game, a mixture of strategies does not evolve. Given that individuals will always oppose you it pays to always play B if $S < T$ or to always play A if $S > T$. Figure 4.9 summarises the key points by showing a schematic overview of whether genetic assortment or phenotypic plasticity (or both) are necessary to "solve" the dilemma for every qualitative type of game.



(a) $r = 0.0$ (b) $r = 0.3$

(c) $r = 0.7$ (d) $r = 1.0$

FIGURE 4.8: Intensity of colour indicates the averaged equilibrium value of the two genes x and β over the space of possible games represented by S and T. The TS-plane is broken into 41×41 discrete games, and each point is the mean of 12 repeats of the simulation. The four panels correspond to 4 increasing values of r.

For $r = 1$ the system will always evolve to the structured SES, this is identical to the main version of the model. For intermediate values of r the situation transitions between the outcome in the well-mixed game, and the outcome in the case where $r = 1$. There is thus a gradual transition between selection being driven towards maximising the mean fitness of the population, and selection maximising the one off fitness of the individual. Note that we are not claiming that selection actually selects groups of individuals to maximise their mean fitness, merely that when relatedness is high the action of selection on individuals is indistinguishable from such a scenario.

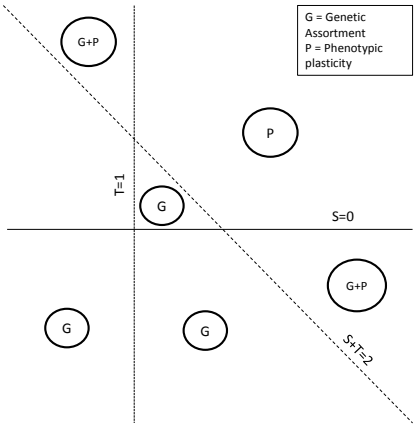


FIGURE 4.9: Schematic showing which of genetic assortment (G) and phenotypic plasticity (P) are necessary for the resolution of each type of game. See figure 4.1 for comparison.

Overall, this second model demonstrates that in DOL games higher mean fitness states can be achieved provided that (i) strategies allow for some degree of phenotypic plasticity, and (ii) there exists a degree of genetic assortment. It should be noted that in detail the model described here exhibits different behaviour from both the pure strategy model and the version of the model described in the main paper. However, the central point of our paper is well supported by this alternative version of the model, showing that our claims are more general than the specific assumptions we must make in order to construct a model. What is necessary for the evolution of function specialisation is some manner by which individuals can react to, or be manipulated by, the social environment in which they find themselves. This was achieved in the main paper by assuming that parents could determine the phenotype of their offspring based on their own phenotype. In this appendix this was achieved by assuming that individuals could react conditionally to the phenotype of the individuals with whom they interact. Whilst some details of the two model were different, particularly in the well-mixed case, the final outcome for high relatedness were the same. For this diversification of roles to evolve there must be both the mechanism, i.e. some form of conditional expression of phenotype, and the "motivation", i.e. high levels of relatedness.

Chapter 5

Discussion

1 Overview

Cooperation, the act of forgoing reproductive potential in order to increase the reproductive potential of others, is a widespread feature in the biological world; the presence of which is puzzling. A naive reading of Darwinism suggests that selection should prune out any tendency of an individual not to maximise its own number of offspring. One explanation for this 'paradox' of cooperation concerns positive assortment (Eshel and Cavalli-Sforza, 1983; Michod and Sanderson, 1985; Godfrey-Smith, 2008). Positive assortment, a form of population structure, means that individuals meet other individuals who are correlated, either genetically or phenotypically, with themselves to a degree that is statistically greater than would have been expected from random encounters alone. In the presence of positive assortment cooperation may be evolutionarily stable.

In the vast majority of models of the evolution of cooperation population structure is taken for granted. Increasingly, however, there are a growing number of studies that ask what would happen if positive assortment were itself subject to evolution (Powers, 2010; Powers et al., 2011; Ryan and Watson, 2015; Santos et al., 2006a). A number of authors have looked at the evolution of assortment from the perspective of limited dispersal (Johnson and Gaines, 1990; Le Galliard et al., 2005; Hochberg et al., 2008), or through the evolution of kin recognition (Tang-Martinez, 2001; Waldman et al., 1988; Giron and Strand, 2004; Rousset and Roze, 2007; Schausberger and Croft, 2001). However, there lacks an overall theory of how and why assortment can evolve in a general manner, which is independent of the actual mechanism that brings assortment about. These studies come from the realisation that many aspects of the biological world that lead to positive assortment are: (A) subject to genetic variation and (B) show many of the hallmarks of adaptations; see chapter 3. Thus, it is of vital importance to theoretical evolutionary biology to understand how cooperation and population structure will co-evolve. This concurrent evolution of cooperation and population structure has previously been referred to as *social niche construction* (SNC).

Previous models of SNC provide a powerful framework for understanding the coevolution of cooperation and population structure. However, further work is required for a more complete understanding of SNC, in particular:

1. Models of SNC lack some key real world features that may affect their results: specifically the presence of crossover.
2. Powers et al. (2011) include a cost to assortment by introducing an intrinsic cost to individuals in smaller groups. However, the implications of this cost are not systematically investigated. Models of social evolution would benefit from a more general and systematic parametrisation of cost.
3. Whilst models of SNC show that the evolution of positive assortment may be stable, it often cannot get started. That is, there is no gradualist adaptive walk from a population of freely-mixed individuals to a population composed of positively-assorted altruistic individuals. Thus, we cannot give a conventional Darwinian account of the concurrent evolution of assortment and altruism.
4. Models of SNC do not present formal mathematical results, and it would therefore be beneficial to systematically describe all possible scenarios (within some reasonably constrained set) for how cooperation and assortment will evolve in each others presence.
5. These models have not been applied to many specific biological systems; only in doing so will we fully test these ideas and realise their shortcomings.
6. Positive assortment on strategy leads to a decrease in the diversity of types and of heterogeneous interactions. Typical models of the evolution of cooperation assume that cooperation consists of a single action, but in reality cooperation in nature often comes about through the interaction of differing complementary types.

This thesis has addressed these issues in an attempt to work towards a more formal theory for the evolution of assortment, and of social niche construction in general.

2 Key Findings

Firstly, in chapter 3 I outlined a formal justification for an issue with our current understanding of social niche construction. Namely, that there is no gradualist adaptive walk from a well-mixed population of defectors to an assorted population of cooperators. I presented a solution to this issue by investigating a game theoretic model in which individuals interact in multiple strategic interactions in parallel. If individuals interact in many social dilemmas simultaneously, then there may exist a feedback process whereby the weaker dilemmas transform the stronger dilemmas into weaker ones and eventually all dilemmas are resolved.

I have applied the idea of the evolution of assortment to a biological scenario: the evolution of assortative mating in the presence of meiotic drive. This involved extending the known isomorphism between evolutionary game theory and the population genetics of diploid organisms (Traulsen and Reed, 2012). The application of the ideas of SNC here lead to the hypothesis that assortative mating, which is prevalent in nature, may be an adaptation that overcomes meiotic drive. This is an hypothesis that could be tested experimentally, and serves as a test case for the ideas of SNC. The paper also involved extending the models of the evolution of assortment to include both cost and crossover. The main conclusions here were that assortment can never reach fixation if it comes at a cost to the individual. Furthermore, crossover does not negate the effect of the evolution of assortment, even though the evolution of assortment depends critically upon the build-up of linkage disequilibrium between the cooperative allele and the positive assortment allele. Note that this model does not contradict the findings of chapter 3, as there I considered the primitive state to be freely-mixed defectors, whereas this paper was concerned with the *maintenance* of cooperation through assortment. In addition, I also presented an analytical solution for the case of zero crossover and a more general numeric solution for non-zero crossover. In these cases I was able to describe the equilibria over the space of all two-player, two-strategy, symmetric games. This lends a more systematic approach to the models of the evolution of assortment.

The final contribution of this thesis involves a formalisation of the idea of cooperative division of labour. Many instances of cooperation in nature do not conform to the conventional Dawesian idea of a cooperative dilemma because such dilemmas assume that there is a single cooperative strategy for which the population would be best off if everyone performed this action. This is simply not the case in many real biological situations, such as cellular slime moulds (Strassmann et al., 2000, 2011), siphonophorae (Dunn and Wagner, 2006) and volvocine algae (Michod and Herron, 2006). In such situations the benefits of cooperation come about through heterogeneous interactions between complementary strategies. This is of particularly importance when one considers positive assortment. Positive assortment, by its very nature, reduces the frequency of heterogeneous interactions. However, positive assortment is necessary to overcome the inherent cooperative dilemma; this is apparently a *catch-22* situation. However, it may be resolved if one realises that phenotypic plasticity allows for simultaneous positive assortment on genotype and negative assortment on phenotype, thereby resolving the apparent paradox. This realisation is the key point of the third and final paper. The results presented in this chapter are in agreement with papers addressing the so called volunteers dilemma (Archetti, 2009; Boza and Számadó, 2010), however, these studies look at games where groups benefit if only a certain fraction of individuals cooperate. We instead construe division of labour games to be games in which individuals perform any number of arbitrary actions, and show that groups can only maximise mean fitness if individuals are able to react conditionally to their social environment. The link to the evolution of assortment is less immediately apparent however, but the resolution of the division of labour problem is important to a full understanding of when and why assortment may evolve and the details of the outcomes when it does so. Understanding cooperative division of

labour is of particular importance when one considers the evolution of multicellularity and other fraternal transitions in individuality (Queller, 1997, 2000).

A further understanding of the evolution of assortment, and of social niche construction in general, is vitally important to the field of evolutionary biology; and in particular to our understanding of the major transitions in evolution (Maynard Smith and Szathmary, 1997; Bourke, 2011; Queller, 2000; Godfrey-Smith, 2009) and the notion of the biological individual (Godfrey-Smith, 2009; Okasha, 2009; Clarke, 2011). A major transition is said to have occurred whenever a collection of particles that were previously able to reproduce independently become sufficiently integrated that they are no longer able to reproduce, except as part of a larger whole. In addition, when the constituent parts are related to one another, the transition is said to be fraternal. Although it has not always been made explicit, much of this work has been motivated by gaining a further understanding of the major transitions in evolution; in particular the fraternal ones. Much of the work in the field has been in categorising and understanding those features of the biological world that make cooperation within an organism possible; such as the unicellular bottleneck or the fairness of meiosis. However, merely stating that a biological trait is conducive to cooperation does not, in itself, explain the presence of the trait. A full explanation of the transitions from within the field of Darwinism must explain the adaptive walk from free living individuals to fully fledged individuals at the higher level. In further expanding the explanation of the evolution of cooperation I have shed light on the evolution of communal living by, for instance, offering an explanation of how such a process may get started. Furthermore, the final paper illustrates the path to functional specialisation, and does so under the same theoretical framework as the models of the evolution of cooperation. This is an important and often neglected step in the explanation of the evolution of individuality, as often models are only concerned with explaining why cooperation is stable, rather than where the benefits of cooperation actually come from.

A full account of the evolution of fraternal transitions from free-living individual particles to fully integrated specialised collectives would require an evolutionary account of how individual particles form highly assorted collectives, with differentiated, specialised parts. My analyses have illustrated how assortment can evolve from a freely-mixed primitive state (chapter 3). Furthermore, I have illustrated how the constituent parts of assorted collectives can specialise into differing roles through phenotypic plasticity (chapter 4).

3 Further work

The ideas in this thesis have set down important foundational work in developing a better understanding of the evolution of assortment and social niche construction. However, much work remains to be done in order to gain a more complete understanding of the field. Specifically, for the understanding of the evolution of assortment it would be beneficial to develop our knowledge

of the effects of the separation of time scales between the evolution of cooperation and assortment. In chapter 3, the concurrent evolution of cooperation and assortment occurred in such a way that the social strategy was always close to equilibrium. Assortment evolves only gradually through the introduction of small mutations. The ratio of the time scales of the evolution of the two parameters is, implicitly, dictated by the mutation rates and the initial conditions; however, this could be formalised and investigated more systematically. It may be that assortment evolves only slowly when compared to the evolution of social strategy. These two elements, cooperation and assortment, may have completely different genetic bases, and may therefore evolve at different speeds. A fuller understanding of this is important for our general understanding of SNC. In particular, this separation of time scales is of importance to the "getting started problem". As chapter 3 has shown, the evolution of assortment in the prisoner's dilemma has difficulty getting started. This is because a selective pressure only exists if there is a polymorphism in the cooperative trait. A polymorphism could be maintained if significant selection could occur at the assortment modifying allele whilst the social strategy is out of equilibrium. However, if the social strategy is effectively always at equilibrium, and the pace of selection is dictated by the assortment gene, then the getting started problem is exacerbated.

Furthermore, my discussion of the getting started problem in the evolution of assortment rests strongly on notions of equilibria. One of my claims is that assortment will not evolve in a prisoner's dilemma because there is no cooperation at equilibrium. The analytical demonstration of this claim assumes an infinite population at equilibrium. However, many features of real populations can cause a deviation from equilibrium; most notably mutation and migration. Some interesting work has been done in applying the concepts of statistical physics to population biology, and in particular the replicator equation (McKane and Newman, 2004, 2005). A fruitful line of further inquiry might be to apply these techniques to the evolution of assortment.

It is the nature of theoretical modelling that the researcher must make a number of arbitrary modelling decisions when building a model of the phenomenon of interest; this work is no exception. Whenever such a decision is made, then one must either make sure that this decision does not affect the results in any meaningful way, or otherwise begin to enumerate the consequences of all possible modelling choices. Whenever a parameter choice or modelling decisions was made I investigated many alternatives before making an eventual decision. However, one can never enumerate all possible choices. This is particularly true of the assortment mechanisms that were employed when studying the evolution of assortment. There are many ways in which we could conceivably pair individuals assortatively. Whilst many mechanisms were investigated, there are many other ways in which this could have been done. The SNC research program can be enriched by describing and categorising the different manners in which the evolution of assortment may come about.

In order to connect my work on the evolution of assortment to more biological bodies of work it would be beneficial to frame these models in a more biologically plausible manner. This would make the work more accessible to population geneticists. In particular the model of the evolution of assortment could be framed in terms of island models of limited dispersal (seeWright (1943);

Slatkin and Takahata (1985); Barton and Slatkin (1986); Hartl and Clark (1998)). Island models are mathematical models from population genetics in which a population is divided into smaller sub-populations, or islands; between which some limited migration can occur. Thus, migration rate acts in effect to parameterise the level of assortment that the population is subject to. The evolution of assortment could be realised through the evolution of the migration rate.

All such work must make a trade-off between being general and pertaining to any real case. Further progress could be made by investigating more specific biological examples of SNC. These would inspire further generality of the models and often suggest further routes of investigation that the modeller would not have otherwise thought of. Progress in this area should be made via a dialogue between the general and the specific. Ultimately, of course, the success or otherwise of this research program must be judged on experimental results.

The last paper, concerning the evolution of functional specialisation, stands somewhat apart from the first two papers. In the model here, assortment was an imposed parameter rather than being itself subject to evolution. This was intentional, as the main point of the paper concerned the evolution of phenotypic plasticity as a response to a division of labour game. However, now that we understand the evolution of phenotypic plasticity and assortment at a deeper level, in isolation from each other, it may be beneficial to combine these two models. This could result in a more general framework for understanding the onset of multicellularity and the nature of the fraternal major transitions (Queller, 1997, 2000). Again there are potential questions about the separation of time scales between the evolution of phenotypic plasticity, assortment and social strategy. Godfrey-Smith (2009) defines biological individuality as a three dimensional concept. Collections of organisms become individuals when they have a high degree of functional integrity, go through a unicellular bottleneck (which facilitates genetic homogeneity) and have a reproductive division of labour. It may be beneficial to further explore, both mathematically and philosophically, how these ideas relate to a more general version of the model presented in paper three. Godfrey-Smith's notion of the individual has some correspondence with the parameters of my model. These include: phenotypic plasticity, which leads to specialisation and hence functional integration; the evolution of relatedness, which may come about through a unitary bottleneck, which itself leads to high relatedness; and the reproductive division of labour, which is itself a type of role diversification, albeit a special one. The model that I present in chapter 4, suitably extended, may shed some light on this process. We may be able to answer questions about whether the evolution of individuality is constrained to happen in a certain order and what the effects of the interplay of the different elements are. For instance, are there theoretical reasons that a proto-individual would have to evolve functional specialisation before, after or concurrently with high levels of relatedness, or are these parameters free to evolve in any order?

In all cases the research program should be to (A) extend the generality of the ideas of the evolution of assortment, (B) apply the ideas of social niche construction to more specific biological contexts and (C) to make further efforts to link these ideas to experimental work. The latter may be difficult, as many of these processes are inherently slow, hard to measure, happened a long time ago and do not leave clear fossil evidence. However, laboratory bacterial populations

may be a good place to start, particularly bacterial biofilms (Powers et al., 2012; Shapiro, 1998; Crespi, 2001; Steenackers et al., 2016).

A number of side projects also present themselves. In particular, I think extending the parallel between genetic conflict and game theory would be a fruitful line of further research; the complete power of this isomorphism is not fully appreciated. Fully taking into account all the possibilities of genetic systems and mapping them to existing ideas in evolutionary game theory may be a PhD's worth of work in itself. For instance the phenomenon of imprinting could be understood through the formalism of asymmetric, or bi-matrix, games. Imprinting (Feil and Berger, 2007; Martienssen and Colot, 2001; Reik and Walter, 2001) is the phenomenon whereby gene expression is dependent upon the parent of origin of the gene. Asymmetric games represent those situations in which the competing strategies play differing, non-genetically determined roles. Thus, there may be a further isomorphism between these two fields. Polyploidy (Sattler et al., 2016), in which individuals have more than two copies of each gene, could be understood through the theory of multi-player games (van Veelen, 2011; Gokhale and Traulsen, 2010). The study of genes at different loci could be modelled through the multi-species replicator equation (Weibull, 1997). Genes at different loci can be thought of as individuals in differing species, as they can never out-compete each other (Gardner et al., 2007), epistasis is however a form of social interaction and linkage disequilibrium is a form of population structure. Coalitions of genes, such as those between the nuclear and mitochondrial DNA could be understood with cooperative game theory (see any comprehensive text on game theory, such as Binmore (1992)), an idea that has itself not been fully incorporated into the standard arsenal of evolutionary game theory.

This thesis has taken a step towards a more general understanding of cooperation and social niche construction; and hence the nature of biological individuality. Further theoretical and empirical work should be done in these areas. If we begin to think in this manner, then some of the arbitrary features of the biological world should begin to form a more coherent pattern. Furthermore, the ideas of social niche construction begin to shed light on evolutionary theory itself.

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