The Evolution of Cooperation, Especially in Humans

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dedicated in memory of my sparkly little cousin

Celia Brenner

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Declaration

The literature review in **Chapter 1** is adapted from a forthcoming book chapter: El Mouden, C, West S, Gardner A, Burton-Chellew M. (2012). 'What do humans maximise?', in: *Evolution and Rationality: Decisions, Cooperation and Strategic Behaviour*, Samir Okasha and Ken Binmore (eds) OUP. This is my own work; my coauthors provided comments and advice on draft manuscripts.

Chapter 2 is a published paper: El Mouden, C and Gardner, A. (2008) 'Nice natives and mean migrants: the evolution of dispersal-dependent social behaviour in viscous populations', *Journal of Evolutionary Biology* 21, 1480-1491. The original idea for the paper was AG's, I led the theory and write-up, with significant assistance from AG at all stages; AG wrote the Appendix in response to reviewer comments.

Chapter 3 is a published paper: El Mouden, C., West, S. A. and Gardner, A (2010). 'The enforcement of cooperation by policing', *Evolution*. 64, 2139-2152. The original idea for the paper was developed together with SW and AG. I led the theory and write-up, with assistance from both coauthors. AG wrote the original Mathematica code for the eigenvalue analysis, which I extended.

Chapter 4 is being prepared for publication. I developed the original idea and led the theory and write up. AG and SW provided advice and ideas throughout.

Chapter 5 is in review at Evolution and Human Behaviour as a research paper. I had the original idea for the paper, developed the theory and performed the analysis. AG and SW contributed to the write up.

Chapter 6 is a published paper: Leggett, H., El Mouden, C., Wild, G., and West, S. A. (2011) 'Promiscuity and the evolution of cooperative breeding', *Proceedings of the Royal Society, B* doi:10.1098/rspb.2011.1627. HL and GW conceived the original idea, I helped formulate the inclusive fitness equation, I led the model analysis and wrote the Appendix, GW conducted the numerical investigation, HL led the write-up, to which SW, GW and I contributed.

Chapter 7 is in press: Verbruggen, E., El Mouden, C., Jansa, J. Akkermans, G., Bücking, H., West, S. A. and Kiers, T. (2012) 'Spatial structure and cooperation: Theory and an empirical test using the mycorrhizal mutualism', *American Naturalist*. In collaboration with SW, I developed the theory, analyzed and wrote the theory section of the paper. The experimental work and remaining write-up was undertaken by my coauthors.

The **Appendix** contains my three other publications. **Section 1** is a published book review: El Mouden, C. (2011) Life: social to its core. *Evolution and Human Behaviour* 33(1): 79-80. This is entirely my own work. **Section 2** is a published experimental paper: Harrison, F. H. and El Mouden, C. (2011). 'Money for nothing, cooperation for free: exploring the effects of working for endowments on behaviour in standard economic games', PLoS ONE 6(11): e27623. FH had the original idea and designed the experiments. I collected one-third of the data for the public goods game and all the dictator game data (together with FH). FH performed the statistical analysis and we both wrote the paper. **Section 3** is a published review paper: West, S.A., El Mouden, C. and Gardner, A. (2011). 'Sixteen common misconceptions about the evolution of cooperation in humans', *Evolution and Human Behavior* 32(4): 231-262. SW, AG and I developed the paper's ideas together. SW led the write-up; AG and I wrote some sub-sections and provided extensive comments.

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Abstract

I develop social evolution theory to study the evolution of cooperation as follows: (1) Many organisms undergo a dispersal phase prior to breeding; I demonstrate that knowing ones dispersal status aids the evolution of helping (by non-dispersers) and harming (by dispersers). (2) Policing driven by group-benefits may be selected to enforce cooperation in human and animal societies. I extend existing theory to show that policing may be harder to evolve that previously thought, but that it is maintained more readily than it evolves. (3) Archeological and anthropological evidence suggests that warfare was prevalent during our evolution. I show that, contrary to previous suggestions, between-group competition can favour any social behaviour (pro-social or anti-social) so long as it helps the group compete, and that such traits can be altruistic or mutually beneficial. (4) Reproductive leveling is analogous to policing; in the human literature there is doubt as to whether it can evolve. I extend my previous work to consider the coevolution of culturally and genetically inherited traits for reproductive leveling and selfishness. I find that cooperation can evolve between non-kin if they share the same culture. (5) Monogamy is thought to favour the evolution of cooperative breeding. I show that in the simplest case, because of the cost of competition between non-dispersing siblings, the level of promiscuity has little or no effect on the evolution of cooperation. (6) Spatial structure (limited dispersal) is thought to favour the evolution of inter-specific mutualisms as it aligns the partners' interests. I consider the case of plant-fungi mutualisms and show that spatial structure can disfavour cooperation if it limits the potential fungal partners available to the plant.

Word Count (excluding references and appendices): 46,525 words

Chapter 1. Introduction.

This Chapter provides a background to the field of social evolution and how it is applied to study human behaviour (section 1.1) and an outline of the thesis's structure (section 1.2).

1.1 Background

Research on human evolution and cooperative behaviour is undertaken in biology, psychology, economics, anthropology, sociology and archaeology. Whilst all these disciplines have important and relevant insights for my research, a comprehensive review would be far beyond the scope of this chapter. Therefore, the background section focuses on the question 'what do humans maximise?' as the answer introduces the key concepts of evolutionary theory, gives a flavour of the work being undertaken in other disciplines and neatly illustrates the breath and complexity of this field.

Section 1.1.1 explains the process of natural selection, discusses the purpose of adaptation and explains why cooperation is favoured in nature. Section 1.1.2 considers why organisms are not expected to be perfect or optimal inclusive fitness maximisers. Section 1.1.3 discusses what evolutionary theory predicts humans will maximize and, having established that humans may behave as inclusive fitness maximisers, section 1.1.4 discusses the reasons why people's actions sometimes appear not to maximize inclusive fitness (or anything else). Finally, section 1.1.5 draws on the ideas developed in the earlier sections to discuss whether or when humans are expected to match the predictions of economic rational choice models.

1.1.1 What is the purpose of adaptation?

> *In crossing a heath, suppose I pitched my foot against a stone, and were asked how the stone came to be there, I might possibly answer that, for anything I knew to the contrary, it had laid there for ever...But suppose I had found a watch upon the ground, and it should be enquired how the watch happened to be in that place, I should hardly think of the answer which I had before given, that, for any thing I knew, the watch might have always been there. Yet why should not this answer serve for the watch, as well as for the stone?*
>
> The opening lines of *Natural Theology* (Paley 1802).

What is the difference between a watch and stone? Unlike stones, Paley explains that watches consist of many parts, and that the form of these individual parts may only be understood when we realize they are all contrived for a common purpose (in this case to tell the time). In other words, unlike stones, watches appear designed. Paley argues that organisms are more like watches than stones by demonstrating that, like a watch, the parts of an organism are made up of related parts, contrived for common purposes. For example, consider the eye; the function of an eye's retina, cornea, lens, muscles and nerves cannot be understood unless we appreciate these parts are contrived for the common purpose of seeing. So when Paley asked 'what is the difference between a watch and a stone?', he had identified the question which lies at the heart of evolutionary biology: how is it that the natural world appears designed? (Darwin 1859; Williams 1966; Leigh 1971; Maynard Smith 1982; Gardner 2009). Fifty years later, Darwin provided the answer.

Darwin's (1859) theory of natural selection accounts for both the process and the apparent purpose of adaptation (Gardner 2009). The process is simple: as more individuals are born than survive to reproductive age, any heritable traits associated with increased individual reproductive success will tend to accumulate in

populations. As a consequence of this process, Darwin argued, successive generations of organisms will appear increasingly well designed to maximize their reproductive success. Darwin therefore answered Paley's question in two ways: first, he showed how complex design (adaptation) could result from a natural process; second, he explained what natural design was ultimately for – to maximize Darwinian fitness.

The *Origin of Species* is a masterpiece, but its central argument is informal (non mathematical) and it was written before the mechanism of biological inheritance (genes) was known. This meant Darwin's ideas were not precisely defined. In the 1930s, Fisher formally linked the dynamics of population genetics to the process of natural selection in his seminal work *The Genetical Theory of Natural Selection* (Fisher 1930). He formally defined natural selection in terms of changes in gene frequencies and defined Darwinian fitness as an individual's genetic contribution to future generations. His 'Fundamental Theorem of Natural Selection' (Fisher 1930) shows that genes associated with a greater individual fitness are predicted to accumulate in natural populations. Given this result, Fisher concluded that organisms would appear as if they are striving to maximize their Darwinian fitness (see also Grafen 2002, 2007).

Fisher's theory contained an important caveat; whilst he knew that behaviours may be favoured because of their indirect effect on relatives who share genes, he explicitly chose to ignore this added complexity. It was Hamilton (1964, 1970) who formally incorporated the effect of relatives into fitness, making what is arguably, the most important contribution to evolutionary theory since Darwin. The result was inclusive fitness theory, which provides a better understanding of the process of natural selection, and is the modern view of what natural selection maximizes. An individual's inclusive fitness may be divided into two components: the first

component is direct fitness, which is a measure of an individual's genetic contribution to future generations via its own reproduction and the second component is indirect fitness, which measures the genetic contribution an individual achieves by affecting the reproduction of related individuals (Figure 1.1; Table 1.1). Grafen (2002, 2006a, 2007) has confirmed Hamilton's result more formally, by demonstrating that the phenotypes resulting from the dynamics of natural selection on gene frequencies in a related population, matches the result of an optimization program, where an agent is set to maximize its inclusive fitness within a phenotypic strategy set. Put simply, the fitness that organisms should appear designed to maximize is inclusive fitness.

Table 1.1. An evolutionary classification of social behaviour

Effect on actor (direct fitness, *c*)	**Effect on recipient (indirect fitness, *b*)**	
	+	**-**
+	Mutually beneficial	Selfish
-	Altruistic	Spiteful

A social behaviour refers to any trait that has fitness consequences for another. A Hamiltonian (or evolutionary) classification is determined by the effect the behaviour has on the individual's *average* lifetime fitness, not on short-term fitness consequences or the fitness consequences of single interactions. Both mutually beneficial (+/+) and selfish (+/-) traits may evolve between relatives or non-relatives. Altruistic behaviours (-/+) are favoured when the benefits to related individuals outweigh the cost to self. Spiteful traits (-/-) are very rare, because of the need for negative relatedness (where the actor and recipient on average share less genes than two individuals drawn at random from the population).

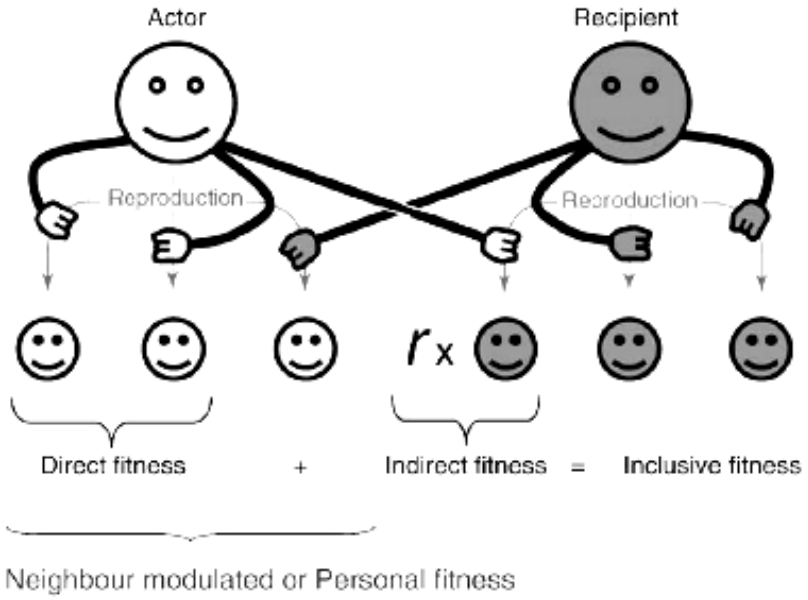


Figure 1.1. Inclusive fitness is the sum of an individual's direct and indirect fitness (Hamilton 1964). Social behaviours affect the reproductive success of self and others. The impact of the actor's behaviour (white hands) on its own reproduction (white offspring) is the direct fitness effect. The impact of the actor's behaviour (white hands) on the reproductive success of social partners (grey offspring), weighted by the relatedness of the actor to the recipient, is the indirect fitness effect. Inclusive fitness does not include all of the reproductive success of relatives (grey offspring), only that which is due to the behaviour of the actor (white hands). Also, inclusive fitness does not include all of the reproductive success of the actor (white offspring), only that which is due to its own behaviour (white hands). A key feature of inclusive fitness is that, as defined, it describes the components of reproductive success which the actor can influence, and therefore what they could be appearing to maximize. Adapted from West *et al*. 2007b.

This expanded view of fitness was instrumental in explaining how the diverse forms of sociality present in the natural world evolved (Hamilton 1996). A behaviour is cooperative if it has been selected for, at least in part, because of the beneficial effect it has on others (West *et al*. 2007b). Explaining the presence of such behaviours appears problematic given that – all else being equal – they reduce the relative fitness of the actor. However, inclusive fitness theory showed that natural selection could favour cooperation or the limitation of conflict under a wide range of conditions. One explanation is that seemingly disadvantageous genes can increase their transmission indirectly by helping other individuals (typically close relatives) that are likely to

share the same gene (Hamilton 1964). Yet cooperation also occurs between unrelated individuals and even between different species (Bourke 2011).

The inherent instability of cooperation between non-relatives is often conceptualized with the aid of the Prisoner's dilemma (Axelrod and Hamilton 1981) or the tragedy of the commons (Hardin 1968), whereby individuals do best by not cooperating (cheating), no matter what their partners do. When there is no scope for repeated interactions or sanctions, this results in an inevitable outcome (hence the 'tragedy') in which all rational actors cheat, even though they would have been better off if they had all cooperated (hence the dilemma). Thus for cooperative behaviour between non-relatives to be evolutionarily stable, it must be favoured by hidden direct-fitness benefits that outweigh any apparent costs and, when confronted with a new instance of sociality, it is the job of evolutionary biologists to elucidate these (Sachs *et al*. 2004; Lehmann and Keller 2006; West *et al*. 2007b).

Over the past 40 years, an extensive literature has developed within evolutionary biology, building upon Hamilton's work, to explain how natural selection may favour the formation and maintenance of cooperation at all levels of biological complexity, including between non-relatives and for different demographic and population structures (Figure 1.2). Broadly speaking, such cooperation may be favoured for one or other of two reasons: because it is also directly beneficial in the long run to the performer, more so than it is costly (as, for example, in group augmentation effects; Kokko *et al*. 2001); or because it is enforced, through policing (Frank 1995, 2003 and see Chapter 3), punishment (Clutton-Brock and Parker 1995; Gardner and West 2004a; Lehmann *et al*. 2007c) or sanctioning of cheaters (Kiers *et al*. 2003), and/or through rewards to cooperators, via mechanisms such as direct or indirect reciprocity (Trivers, 1971). This work illustrates that individuals are not born selfish (contra Dawkins 1976, p.3); they are born as inclusive fitness maximizers and

this may favour them to be selfish, spiteful, mutualistic or altruistic depending on the circumstances in which they find themselves (West *et al*. 2007a).

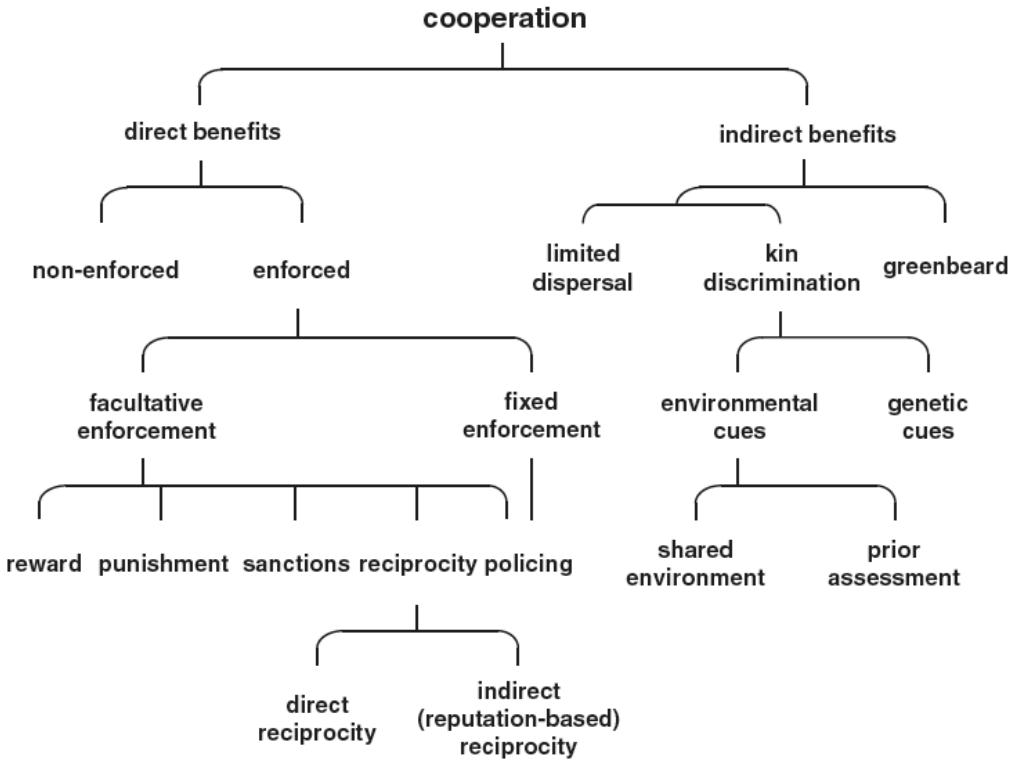


Figure 1.2. A classification of the evolutionary explanations for cooperation. Direct benefits explain mutually beneficial cooperation which may be between non-relatives, whereas indirect benefits explain altruistic cooperation, which is always between relatives (Hamilton 1964). Within these two fundamental categories, different mechanisms can be classified in various ways (Frank 2003; Sachs *et al*. 2004; Lehmann and Keller 2006; Bergmüller *et al*. 2007; West *et al*. 2007b). The classification shown here is for illustrative purposes only. Not all explanations are included (e.g. group competition, group augmentation and the effect of demography is excluded). A single act of cooperation may require multiple explanations, for example it may evolve due to both direct and indirect fitness benefits, or interactions with relatives could be maintained by both limited dispersal and kin discrimination. Furthermore, it does not show how the relative importance of different mechanisms may shift through time. For example, group members may need to be related for a group to form so the benefits of cooperation are sufficient, but once established, cooperation may remain stable even if group members are unrelated due to enforcement mechanisms (Bourke 2011). In most social species, different explanations will apply in different circumstances. For example, in humans, shared environment mechanisms help explain cooperation between kin, whereas reputation and punishment are important for cooperation between non-relatives. Finally, we divide up facultative enforcement strategies here overly simplisticly; a detailed discussion is beyond the

scope of this chapter, and is provided elsewhere (Bergmüller *et al*. 2007). Adapted from West *et al*. 2007b.

1.1.2 Adaptation does not imply perfection or optimality.

Watches vary. A stick in the ground can make a crude sundial, accurate to the nearest hour or so on a sunny day, whilst an atomic clock, cooled to near absolute zero, has a margin of error of about one second in 30 million years. Nevertheless, both sundials and atomic clocks are designed to tell the time. Even a broken watch, that does not work at all, shows evidence of design. According to Paley: 'It is not necessary that a machine be perfect, in order to shew with what design it was made...the only question is, whether it were made with any design at all.' (Paley 1802, p. 8). Applied to nature, Paley's point is that living organisms need not be perfect or optimal fitness maximizers to exhibit adaptations for 'fitness maximizing' design (Gardner 2009).

The closest thing to perfect timekeeping is the atomic clock. If natural selection could produce its version of a 'perfect' watch, it would be a 'Darwinian Demon' (Law 1979). This imagined organism could simultaneously maximize all aspects of its inclusive fitness – it would live forever, in any environment, eat anything, be able to reproduce as soon as it was born and give birth to high quality offspring at an infinite rate whilst also helping its relatives produce their offspring at an infinite rate. Darwinian demons are a fantasy for the same reason that we cannot wear atomic clocks on our wrists: constraints. In nature, phylogenetic, physical, time, developmental, information and resource constraints, mean that organisms are unable to be perfect fitness maximizers. In addition to constraints, organisms must efficiently allocate limited resources to competing concerns. Therefore, from an evolutionary perspective, organisms can never be perfect. However, it is worth asking: when organisms make their complex trade-offs between different aspects of

fitness (e.g. growth vs own reproduction vs relatives' reproduction), can they do so optimally?

Optimality is the achievement of maximum fitness given the constraints an organism faces. Therefore, both atomic clocks and sundials may represent optimal designs in different environments. However, just as a broken watch still shows evidence of design, the presence of fitness-maximizing adaptation does not imply optimality of design. This is because in natural populations, in addition to genes, many other factors influence an organism's chances of survival and reproduction (i.e. its fitness). As the opening lines of Fisher (1930) famously read 'Evolution is not natural selection'. This is because stochastic effects (e.g. unpredictable weather), mutations and population movements all shift gene frequencies, so are evolutionary forces too. If selection pressures are weak (i.e. most individuals survive and reproduce), these non-selective evolutionary forces can be the main drivers of phenotypic change, not natural selection. As these forces do not maximize anything, they divert phenotypes away from natural selection's goal of optimality, degrading any appearance of design. Therefore, even though organisms can be optimal, we expect measurably optimal behaviour to be extremely rare. There is no expectation that organisms will reach the goal of optimality, or even be anywhere near it, just that natural selection will direct genetic variation in that direction.

Although adaptation does not imply optimality, it is important to note that mechanisms under strong selection, such as those concerning life or death decisions or commonly encountered situations, will be more finely tuned toward optimality as here, natural selection will be the dominant evolutionary force. Thus we expect to see organisms performing 'better' in these situations. For example, as sex ratio theory predicts, a fig wasp will vary the sex ratio of her offspring depending upon the number of other females that lay eggs in the same fruit (Herre 1985, 1987). Studies

have demonstrated that the sex ratios the wasps produce are closer to the predictions of evolutionary optimality models in the situations they encounters more frequently (Herre 1987).

1.1.3 What does evolutionary theory predict humans will maximize?

Many animals have evolved the capacity for phenotypic plasticity in order to cope with variable environments. There are various ways phenotypic plasticity can be achieved. Natural selection may favour a range of phenotypes (Smith and Skúlason 1996; Halama *et al*. 2001). For example, many fish species have evolved discrete morphs, which feed in different habitats or adopt different mating strategies (Taborsky 1984; Ehlinger 1989). Alternatively, natural selection may favour the use of proximate cues to help determine optimal behaviour. For example, female great tits use increasing day length, temperature, food availability and social stimulation to time their egg laying with the peak emergence of caterpillars in spring. Studies of a great tit population in Oxford show that since the 1960s, great tits have used these cues to successfully track changes in the temporal availability of caterpillars (Dawson 2008). However, the main way animals achieve behavioural flexibility is through learning.

Learning occurs when an organism changes its behaviour in response to experience (Pearce 1997). By understanding and remembering the consequences of their actions, animals can react better to the behaviour of others and can cope with changes in their local environment (Sullivan 1988; Hollis *et al*. 1997; Mahometa and Domjan 2005). Many animals exhibit surprising abilities at learning. For example, goldfish, chickens, horses, cats and rhesus monkeys, learn equally well to discriminate between two stimuli to gain a reward (Warren 1965). Despite the fact many behaviours are not hard-wired, an animal's actions will still appear designed to maximize inclusive

fitness as its learning is driven by cognitive mechanisms like hunger, libido, pain and fear which are adapted to direct behaviour toward fitness-maximizing goals.

Humans differ from other organisms by degree not by kind; we too derive pleasure from, and direct our efforts toward, fitness-maximizing outcomes (Darwin 1871). We enjoy food, sex, friendship and social recognition and we dislike hunger, pain, fear, failure and ostracism. To illustrate how adaptive preferences can influence our behaviour, we use the literature on sexual preferences as an example. This work shows that men prefer plump women to thin women (81% of people in 58 cultures surveyed; Brown and Konner 1987), and that this preference becomes more extreme when men are hungry (Swami and Tovee 2006), indicating the adaptive explanation may relate to the risk of food shortage (Sugiyama 2004). Men also prefer women with a low waist-to-hip ratio (Singh 1993), which may be adaptive as a low waist-to-hip ratio correlates with offspring with higher cognitive ability (Lasseka and Gaulin 2008). During the few days of the month when women are fertile, they are considered more attractive (Roberts *et al*. 2004) and male partners are more attentive (Haselton and Gagensted 2006) and possessive (Gangestad *et al*. 2002). Whilst fertile, women prefer more masculine males (Penton-Voak, et. al. 1999; Gangestad *et al*. 2005), make more effort to dress attractively (Haselton *et al*. 2007) and are more flirtatious and sociable (Haselton and Gangestad 2006). Finally, ovulating lap dancers earn, on average, $30 an hour more than menstruating lap dancers (Miller *et al*. 2007).

The diverse and growing literature on human behaviour shows our adaptive preferences are many and varied and affect all aspects of our lives. We summarize their effect to be, that overall, humans strive to increase personal 'happiness', and minimize 'unhappiness', although what makes us happy and unhappy changes all the time. We would predict that people are most happy when their conditions are improving, and are less happy when their conditions stagnate, even if they are doing

well. This is because the positive emotions associated with happiness are evolved, proximate mechanisms to guide our behaviour toward adaptive ends.

Humans show an array of adaptations to help us pursue our adaptive preferences. In addition to an unrivalled capacity for problem solving and reasoning, what sets humans apart from all other species, is the fact that we have access to an accumulated cultural knowledge. Culture is defined as information capable of affecting individuals' phenotypes which they acquire from other conspecifics by teaching or imitation (Richerson and Boyd 2004). Many non-human species, including blue tits (Perrins 1979), chimpanzees (McGrew 2004) and dolphins (Simões-Lopes *et al*. 1998), have simple systems of cultural transmission where non-genetic, non-species wide behaviours are passed across the generations. However, only humans have the cognitive capacity to benefit from a cumulative culture where skills are learnt, improved and transmitted to others via teaching or imitation (Boyd and Richerson 1985, 2005a; Henrich 2005, 2008). The positive fitness effect of cumulative culture is evident in even simple human technologies such as a 60,000 year-old stone-tipped spear (Pettit 2005). Its design is the product of multiple innovations to the shaft, hafting and point. No modern human could ever arrive in the Savannah and design such a spear on the spot. We heavily rely upon the accumulated cultural knowledge of past generations for our survival.

Humans show many adaptations for acquiring and using cultural knowledge. We are unusually docile (Simon 1990), highly sensitive to expressions of approval and disapproval by parents and peers (Baum 1994; Henrich and McElreath 2003) and most importantly, we excel at high-fidelity imitation (Tomasello 1999). These imitation skills enable humans to perform social learning via imitation and practice (Whiten and Ham 1992; Heyes and Galef 1996), which ethnographic studies confirm is how we acquire the majority of our skills (as opposed to independently via

individual learning which is what animals typically do; Fiske 1999). Furthermore, our cognition adapts to the local environment as some behaviours learnt during our childhood can become hard-wired in the brain, permanently modifying our minds at the sub-conscious level (Quartz and Sejnowski 1997; Quartz 2002). This is evidenced by between-cultural variation in susceptibility to optical illusions, hand-eye coordination and male stress and aggression levels (Segall *et al*. 1966; Cohen *et al*. 1996; Henrich 2008). It appears that such traits become permanently fixed by about 20 years of age, and remain fixed for life, even if the individual migrates into a new culture (Segall *et al*. 1966).

The cognitive adaptations and reservoirs of cultural knowledge which humans posses mean that, compared to other animals, we display a unique capacity for behavioural flexibility and specialization. People learn from their own life experiences and of those around them and use this experience to decide how best to seek out happiness and avoid pain in the circumstances they find themselves in. Even with identical evolved preferences, as people have different life experiences, we expect them to learn very different behaviours and to possess widely divergent beliefs about how to achieve the same goal. Therefore, behaviours and preferences that appear drastically different on the surface may share a common evolutionary foundation. For example, whilst young men in the West may disrespect their parents, smoke and drive fast cars, young men from other cultures may choose to socialize with elder family members, work long hours and avoid contact with girls; both may be trying to fulfil their desire for social status, but they have learnt to achieve the same goal in very different ways. Similar explanations can sometimes be applied to those that vow to be celibate or to forego material wealth, behaviours often cited as a challenge to evolutionary explanations for human behaviour. Since members of the same family, class, culture or tribal group share many formative experiences in

common, it follows that the variation in beliefs within such groups should be far less than the variation found between them.

Humans, like other group-living vertebrates, show adaptations for sociality. This is evidenced by many elegant behavioural economic experiments, which show that we are often willing to cooperate and to punish those who do not cooperate. Importantly, this includes cooperation or punishment toward strangers in one-shot experimental encounters where there is no way of benefitting from the act (Ledyard 1995; Fehr and Gachter 2002; Fehr and Fischbacher 2003; Gintis *et al*. 2005). Not surprisingly, these social behaviours have a neurological basis: the brain's reward centres (e.g. the striatum; Stanfey 2007) activate and make us feel good when we donate money to charity (Moll *et al*. 2006), observe charitable behaviour (Harbaugh *et al*. 2007), engage in reciprocal cooperation (Rilling *et al*. 2002) and in costly punishment (Quervain *et al*. 2004). There are many possible evolutionary explanations for why we are motivated to perform these social behaviours (Figure 1.2) and a detailed discussion is beyond the scope of this chapter (see Sachs *et al*. 2004; Lehmann and Keller 2006; West *et al*. 2007b, 2011; Bourke 2011). The fact that these behaviours are seen as irrational (with respect to an income-maximizing agent) in economic experiments involving positive externalities has generated the false impression that humans are uniquely social organisms. Such an impression feeds from misconceptions about evolutionary theory, the difference between ultimate and proximate questions, and the ways that cooperation evolves (West *et al*. 2011). Indeed, if the recent history of experimental social sciences had instead tested for income maximizing behaviour in experiments with negative externalities or direct benefits to cooperation, then the same logic may have resulted in the view that humans as irrationally anti-social (Kümmerli *et al*. 2010).

To explain the adaptive value of social behaviours, we must understand the evolutionary trade-offs and constraints the individual experienced in the environment where the behaviours were selected for and were maintained (Herre 1987). As we do not know exactly what our ancestral environment was like, it is not possible to know whether social adaptations we now see were favoured due to direct or indirect benefits. Therefore, we will probably never be able to quantitatively measure the extent to which these adaptations for sociality are (from an evolutionary perspective) mutually beneficial or altruistic. However we can glean insights into the key selective pressures that operated in the past with experiments that utilize implicit cues of key environmental features (Tooby and Cosmides 1990). For example, the use of eye-spots in experiments have proved to be a salient cue of reputational affects, and people condition their cooperation levels on the presence or absence of such cues (Bateson *et al*. 2006; Haley and Fessler 2005; Rigdon *et al*. 2008; Ernest-Jones *et al*. 2011).

1.1.4 Why do humans sometime appear not to maximize inclusive fitness (or anything else)?

Behavioural ecologists commonly use the economic tools of optimization theory and game theory, which assume organisms behave as optimal fitness maximizing agents (Maynard Smith 1982; Davies *et al*. 2011). Constructing such models entails making assumptions about the constraints and trade-offs facing the individual in order to define the available choices (the strategy set) and the potential payoffs (in terms of fitness) they may receive. Crucially, in order to be testable, the model requires feasibly measurable parameters. This means proxies for fitness (e.g. number of offspring, food intake, number of matings or income) and fitness-based payoffs (e.g. cost = time spent on task, calories used or price; benefit = calories gained, matings, change in social rank or profit) must be used (Parker and Maynard Smith 1990). If the

data and model match, the model may correctly capture the adaptive purpose of the behaviour being studied. However, often observations do not fit model predictions and it appears that the individuals are not maximizing their inclusive fitness (or anything else). Indeed, whilst data and models can match well, meta-analyses show that on average, evolutionary or ecological models explain only 2-5% of the variation between natural populations (Møller and Jennions 2002). There are many reasons for this - here we discuss six of them.

First, natural selection acts on average consequences of particular traits. This means that a particular trait may entail some costs, but as long as the cost/benefit analysis is favourable (i.e. on average increases inclusive fitness over the individual's lifetime), the trait can be favoured. For example, in Hedge Sparrows (*Prunella modularis*), females often mate with multiple males, so a male cannot be sure which eggs he sired (Davies *et al*. 1992). As the males invest in parental care, he must estimate his paternity. This potentially difficult task is achieved via a simple approximation; the amount of uninterrupted access he had with the female (Burke *et al*. 1989). This rule is favoured despite the fact that about 15% of males estimate their paternity incorrectly and raise chicks of another male (Burke *et al*. 1989). This fitness cost is carried, as it seems the males are unable to achieve any better with the information they have. As we mentioned above, humans react to a subconscious cue of being watched by behaving more cooperatively. Whilst the cues we respond to usually correlate with being watched, we do make mistakes. For example, if pictures of eyes, or stylised eye-like drawings are displayed, we will contribute more to public goods games, donate more to honesty boxes or litter less (Haley and Fessler 2005; Bateson *et al*. 2006; Burnham and Hare 2007; Ernest-Jones *et al*. 2011). Even seeing three dots configured as a down-pointing triangle (like two eyes and a mouth) instead of an up-

pointing triangle, is enough to make males offer significantly more in a dictator game (Rigdon *et al*. 2008).

The lesson here is that if a few individuals in a population act irrationally, it does not automatically imply that their behaviour is maladaptive or that it needs a specific evolutionary explanation. We expect natural selection to minimize the average cost of errors, but we do not expect it to eliminate them. This may mean an odd individual makes a very costly error, or that everyone who performs the behaviour may incur occasional costs. As these errors in themselves are not adaptive, to seek an explanation for them in isolation is not possible. Psychologists refer to this idea as 'error management theory' (Funder 1987; Haselton and Buss 2000; Haselton *et al*. 2005; McKay and Efferson 2010).

Second, natural selection prefers cheap solutions. In response to the varied constraints organisms face, natural selection tends to favour traits of sundial-like design, by which we mean adaptations that are quick and cheap, but do the job, rather than time consuming, costly and precise. Biologists call these adaptations rules-of-thumb; psychologists and social scientists refer to them as heuristic biases (Kahneman and Tversky 1972; Tversky and Kahneman 1974; Gilovich *et al*. 2002; Gigerenzer and Gaissmaier 2011). For example, the parasitoid wasp *Trichogramma* uses a rule-of-thumb to estimate the volume of a sphere. This sphere is the egg of its host species. Its estimate must be accurate so that it lays the right number of its own eggs inside – too many and the larvae will run out of food, too few and it reduces its reproductive success. The wasp has reduced this complex calculation to a single proxy measure, the angle her head makes with her front leg when balanced on the egg (Wehner 1987). Since the 1970s, psychologists have discovered that humans rely on a diverse array of heuristic biases for many aspects of our decision-making (Gilovich *et al*. 2002; Pohl 2004; Haselton *et al*. 2005). We use heuristics to make

estimates of relatedness, which allows us to avoid incest and direct cooperative behaviours toward those we share genes with. For example, individuals are treated as closer relatives if there was a longer period of association during their childhood (Westermark 1921; Lieberman *et al*. 2003; Corriveau and Harris 2009); if they bear a facial resemblance (De Bruine *et al*. 2008); if they speak the same dialect (Shutts *et al*. 2009) or smell familiar (Russel 1976; Russel *et al*. 1983; Porter and Cernoch 1983; Olsson *et al*. 2006).

Heuristics are cheap, but they are only adapted to maximize an aspect of fitness in a particular situation. They allow fast decision-making and are accurate in commonly encountered situations, but they can cause us to behave in irrational ways, particularly when faced with novel situations. For this reason, the approximation used by the *Tricogramma* wasp, is only accurate over the natural range of egg sizes she encounters; if presented with an abnormally small or large host egg, her rule of thumb fails and she would lay too many or too few eggs (Wehner 1987). In humans, the approximations for relatedness we use for incest avoidance cause problems in China and Taiwan where occasionally parents adopt a female infant and rear her alongside their son to whom she will eventually marry. Compared to marriages where spouses were raised apart, such marriages result in lower fertility and higher divorce rates (Wolf 1995). There is a vast and growing literature detailing how heuristic biases cause humans to make irrational decisions, particularly when faced with complex calculations or unfamiliar environments (Gilovich *et al*. 2002; Pohl 2004; Haselton *et al*. 2005).

Third, the solution to fitness trade-offs can change across time and circumstances. Behaviours may not appear to maximize anything because different fitness components are traded off against each other and, as our circumstances and life-stages change, these different components will change in importance (Stearns 1992).

For example, we may be risk-prone as adolescents, when our desire for social status outweighs our desire to avoid danger and become risk-averse once we have children when our desire to keep our children and ourselves from harm outweighs any desire to be 'cool' (Steinberg 2008). Similarly, an old lung or skin-cancer patient will probably wish they had not smoked or used sun beds when young. To an economist, such remorsefulness may appear irrational due to the inconsistency of preferences, but we are not designed to behave consistently, but to make the best of the situations we find ourselves in, continually altering our preferences throughout our lives in response to what makes us happy.

Fourth, novel environments produce non-optimal behaviour. Even behaviorally flexible organisms can only be expected to respond optimally within the range of natural variation they or their species have encountered before. Therefore in unfamiliar environments (such as a laboratory) organisms will make mistakes. In humans, this source of error is variously known as the 'mismatch hypothesis' (Hagen and Hammerstein 2006), artifact biases (Haselton *et al*. 2005) or, in cooperation research, the 'big mistake hypothesis' (Boyd and Richerson 2005b). As the physical and cultural environment humans live in is radically different from the one we evolved in we may exhibit traits that are not adapted for our current environment. For example, in our ancestral environment, individuals with strong preferences for sugary, fatty, and salty foods were favoured, as they sought out higher quality diets. Today in developed countries, these evolved preferences are contributing to obesity and heart disease (Birch 1999). Similarly, strong sexual desires drove males to find the best mates and more willing mates. Now they also maintain a multi-billion dollar-a-year porn industry. It is not immediately clear whether suffering from heart disease or spending income on pornography is maladaptive or not, but this is not the point, and attempts to find out are often misguided. The point is that if these

behaviours are not offset by other benefits then they will be selected against provided the environment remains stable over an evolutionary timescale. For example, it is possible that the factors contributing to heart disease may have inclusive fitness benefits in early life, and that an attraction towards pornography may be a relatively harmless side-effect of a beneficial sex-drive. As we discussed earlier, this idea is particularly relevant when studying cooperation because of the importance that demography and population structure play in the origin and maintenance of sociality.

Fifth, it is hard to measure fitness. Even when a behaviour is adaptive, the parameters being measured may be unable to detect the relationship. For example, behavioural ecologists use proxies of fitness, such as number of offspring, number of mates, longevity, food intake, body size, antler length etc. Better proxies (i.e. those more closely correlated with inclusive fitness) are expected to result in a better fit between the model prediction and the observed behaviours. Models that assume people are designed to maximize their income or social status are like models that assume a stag is designed to maximize its antler length or a bee its nectar intake; they will be accurate only insofar as income, social status, antler length or nectar intake are accurate proxies for inclusive fitness. Furthermore (due to the reasons discussed above) behaviours are more likely to appear mistaken (i.e. the adaptive relationship will be missed), if observed for only a short time, in isolation, in a single individual, or when the individual is in an unfamiliar environment.

Sixth, and finally, not all traits are the target of selection. This means that the existence of such traits, which may be genetically or culturally inherited, cannot be understood with economic tools. For example, a genetic trait of interest may be a by-product of selection for another gene; this applies to fleece colour of Soay Sheep (*Ovis aries*; Gratten *et al*. 2007) and red-ginger hair colour in humans (Valverde *et al*. 1995).

In Soay Sheep, the gene for dark fleeces is favoured as it is tightly linked (i.e. physically close on the chromosome) to a gene that is essential for cell protein function (Gratten *et al*. 2007). Similarly in humans, ginger hair has no adaptive value however the gene for ginger hair also causes pale skin, which is selected for when there is little sunshine as it increases vitamin D synthesis from UVB radiation (Jablonski and Chaplin 2000). Adaptive explanations may help explain the fundamental desires that drive our culturally inherited behaviours. However, they say little about the cultural activities such as music, literature, sport, fashion and art that we undertake to fulfill those desires. In other words, much of what we value does not have an adaptive explanation so we do not believe that the minutiae of human behaviour can be studied using economic tools.

1.1.5 Is measurably optimal behavior ever expected in humans?

As optimal behaviour is not expected in the natural world, behavioural ecologists do not use economic tools to test whether animals behave optimally. Instead, they use them to make testable predictions that can help them understand why animals show the adaptations they do. Behavioural ecology is often a *qualitative* science, where evolutionary explanations are established by comparative studies across different conditions or between species (Frank 1998; Davies *et al*. 2011). Having said that, in the natural world, in vary rare cases optimality models fare much better, and can accurately predict *quantitative* differences. The best-known case of such 'as-if' optimization is that of sex ratio where very simple models can make accurate quantitative predictions (Charnov 1982; West 2009). For example, there is a tight fit between observed sex-ratios in the fig-pollinating wasps and those predicted by theory, which says that as the number of females laying eggs in a fruit increases, the sex ratios in the broods should become less female-biased (West *et al*. 2000).

Why do the models and data match so well in the case of sex-ratios? The first reason is that sex allocation represents a very basic tradeoff (male vs female), so it's easy to construct models that accurately reflect the constraints and choices available to the organism. Second, the traits can be measured precisely as it is relatively easy to sex offspring and count the number of surviving grandchildren. Third, the sex ratio is very strongly tied to fitness so it is strongly selected for, meaning that natural selection will be the dominant evolutionary force, allowing the trait to get close to the goal of optimality. Finally, the sex ratio is a neat quantitative trait, with the expectation that mutations of small effect can readily arise that will nudge the sex ratio by a small amount, allowing a great precision of adaptation even in a very simple organism like the fig wasp. Cases like the sex ratio indicate when as-if optimality is most likely to be observed: when there is a simple set of choices, where decisions can be precisely quantified, when there are high-stakes and where the individual can control their choice. However it should be noted that even in the case of sex-ratios, deviations from predictions are observed, but these deviations are not random. In fact, as predicted, they negatively correlate with the strength of selection and with the reliability of environmental cues (West *et al*. 2002c). Other instances of 'as-if' optimization in animals are found in clutch sizes and various aspects of foraging behaviour (Davies *et al*. 2011).

The human capacity for learning, reasoning and behavioural flexibility means that, despite the mistakes we sometimes make, in a wide range of cases we do behave in ways that match the predictions of optimality models. Indeed, we are so good at fine-tuning our real time behaviour toward achieving specific goals that it is typically studied using rational choice models. The maximand for a rational choice model is an individual's utility. Utility is maximized over short timescales and correlates to desires or wants (Marshall 1920) so can be related to inclusive fitness (Grafen 1998). It

is a numerical representation of a preference ranking over a set of alternatives. Given its properties as a universal exchange rate, it is no surprise that in today's world so many people have decided that making money is an effective way to be happy. This justifies the oft-made simplifying assumption that utility represents a single preference for income maximization.

So long as we model the constraints and choices humans face carefully, rational choice models can accurately describe human behavior, i.e. we do achieve 'as-if' optimization of utility in real time in many situations (Tversky and Kahneman 1986). Like 'as-if' optimality in nature, we predict that human behaviour is more likely to quantitatively match the predictions of rational choice models when: the decision can be precisely measured (as is the case for market behaviour); the choices are simple or routine (so there is a clear optimal decision and we do not need to rely on heuristics); the stakes are high (so the individual is motivated to 'care' about the outcome); and when the individual it in total control of their choice. It is often hard to give economic explanations for our actions, particularly when they affect others or have other far-reaching consequences. When attempting to model such behaviour, care is needed to avoid unrealistic assumptions, for example by only specifying cognitively feasible concerns. Furthermore, parsimonious explanations are often possible with simpler models that only focus on personal costs and benefits. For example, an experimental participant that acts in a way that benefits or harms others may not be motivated beyond personal concerns; such externalities may be a by-product of experimental design (Kümmerli *et al.* 2010).

Humans are so good at achieving 'as-if' optimization of utility in the way rational choice models predict, it is no surprise that most economic theories assume we behave in this way. However, we do not match the predictions of rational choice models all the time (Sen 1977). This is especially true for non-market behaviour. In

the previous section, we discussed why our behaviour may not appear to maximize anything; economists will be familiar with many of these reasons. In addition to these however, the predictions about human behaviour arising from the expectation that individuals will strive to maximize their inclusive fitness will not always match the predictions of models which assume we strive for real-time utility maximization. Like body weight and antler length, income and happiness are proxies for inclusive fitness and this is the only maximand that the average lifetime consequences of a person's actions might be consistent with. Furthermore humans are not expected to have consistent preferences during their lives (as rational choice theory assumes). Instead, preferences vary in importance and will shift over time as an individual's personal circumstances and knowledge changes. These preference changes may seem irrational from an economic perspective, but they can make sense from an inclusive fitness point of view. Most importantly, whether we appear to solve a particular problem in an economically rational way will depend on the time frame of the problem. Over short time frames, we do maximize our pleasure. However, we are not designed to maximize pleasure in the same way over our lifetime, as what gives us pleasure will continually change. Finally, optimization models, no matter how sophisticated or complex, cannot describe all our behaviour as we are a product of evolution, not just natural selection. In other words, we may be good, but we are not optimally designed.

1.1.6 What do Humans Maximise?

How would an alien biologist that was capable of observing our behaviours and reading our minds sum up what humans maximize? They would probably say that, intentionally, humans do not aim to maximize any one thing over their life times, but do generally try to maximize their pleasure and minimize their pain over short time

frames. The alien may note that we receive pleasure and pain from adaptively sensible sources, and that we are very good at learning or inventing new ways to increase our pleasure. They may also note that the happiest people are those whose lives have improved constantly, rather than those who started at the top and just had to stay there. Finally, whilst realizing that humans engage in many instances of cooperation, their energies appear to be mostly directed towards benefitting themselves, their reproductive opportunities, and the well being of their relatives. Whilst fascinating for many reasons, the alien would conclude that humans, along with all other organisms, are best described as striving to maximize their inclusive fitness over their lifetimes, yet are imperfect and non-optimal, but often in predictable ways.

1.2 Thesis outline

In the first two chapters of this thesis, I develop general theory which provides theoretical overviews of mechanisms that favour the evolution of cooperation: chapter 2 explores how knowing whether one has dispersed or not could effect the evolution of social behaviours and chapter 3 considers the evolution of cooperation in groups via the repression of within-group competition. Given their generality, these results may be relevant to diverse species. In chapters 4 and 5, I focus specifically on extending and developing ideas originally presented in the human literature: chapter 4 examines how competition between groups can select for social behaviours, and chapter 5 extends the model presented in chapter 3 to consider the coevolution of reproductive levelling and competitivness. In the final two chapters, I present the result of collaborative work on specific non-human models. Chapter 6 considers the effect of promiscuity on the evolution of cooperative breeding and chapter 7 combines theoretical and experimental work to consider how partner

choice may help plants maintain cooperation in the plant-fungal mutualism. A more detailed summary of each chapter is given below.

Chapter 2: When might social behaviours, which depend on the dispersal status of the actor, evolve? There has been much interest in the evolution of social behaviour in viscous populations (Hamilton 1964). Whilst low dispersal increases the relatedness of neighbours, which tends to promote the evolution of indiscriminate helping behaviour, it can also increase competition between neighbours, which tends to inhibit the evolution of helping and may even favour harming behaviour (Queller 1992; West *et al*. 2002a). In the simplest scenario, these two effects exactly cancel, so that dispersal rate has no impact on the evolution of helping or harming (Taylor 1992*a*). In this chapter I show that the dispersal rate does matter when individuals can adjust their social behaviour conditional on whether they have dispersed or whether they have remained close to their place of origin. I find that non-dispersing individuals are weakly favoured to indiscriminately help their neighbours, whilst dispersing individuals are more readily favoured to indiscriminately harm their neighbours.

Chapter 3: Policing is regarded as an important mechanism for maintaining cooperation in human and animal social groups. A simple model providing a theoretical overview of the coevolution of policing and cooperation has been analysed by Frank (1995, 1996b, 2003, 2009), and this suggests that policing will evolve to fully suppress cheating within social groups when relatedness is low. Here, I relax some of the assumptions made by Frank, and investigate the consequences for policing and cooperation. First, I address the assumption that the individual cost of investment into policing is reduced when selfishness dominates. I find that relaxing this assumption leads to policing being favoured only at intermediate relatedness. Second, I address the assumption that policing fully recovers the loss of fitness

incurred by the group owing to selfishness. I find that relaxing this assumption prohibits the evolution of full policing. Finally, I consider the impact of demography on the coevolution of policing and cooperation, in particular the role for kin competition to disfavour the evolution of policing, using both a heuristic 'open' model and a 'closed' island model. I find that large groups and increased kin competition disfavour policing, and that policing is maintained more readily than it invades. My results suggest that policing may be harder to evolve than previously thought.

Chapter 4: Conflict, or warfare between groups has been suggested as a potentially important mechanism favouring the evolution of altruism in humans as the cost of being an altruist may be outweighed by the benefits of succeeding in between-group competition (Choi and Bowles 2007; Bowles 2006; 2009). Here, I build upon models by Bowles (2006; 2009) to examine in more detail what forms of sociality are favoured when groups compete. I show that: (1) group competition can favour the evolution of pro-social or anti-social behaviours, what matters is that they improve the group's ability to compete; (2) even when they are anti-social and reduce the fitness of group mates, the behaviour is still mutually beneficial or altruistic because for group members, the fitness costs of the anti-social behaviour are outweighed by the benefits of increased group survival. In contrast to earlier models, my results suggest that competition between groups alone cannot explain the evolution of pro-social behaviour in humans.

Chapter 5: Reproductive levelling refers to mechanisms where conflict between group members is limited such that resources are equally distributed. Anthropological evidence suggests that egalitarianism is common in small-scale societies (Fried 1967; Boehm 1982; 1997; 2001, Knauft 1991), which suggests that mechanisms such as reproductive levelling may have been important during human

evolution. Previous models of reproductive levelling have assumed its existence, and considered its impact upon the evolution of cooperation, without considering the evolution of reproductive levelling itself (Bowles 2006). Here, I extend the model presented in chapter 3 to examine the co-evolution of traits for costly reproductive levelling and competitiveness. I consider both the genetical and cultural evolution of reproductive levelling and competitiveness and find that: (1) costly culturally-inherited reproductive levelling can be selectively favoured, in such a way that it selects for genetically-inherited self-restraint; (2) reproductive levelling is most strongly favoured when group members are genetically unrelated but share the same culture. Assuming that cultures can evolve in the way my model assumes, my results suggest that reproductive levelling could be a potentially important mechanism to explain how gene-culture coevolution may lead to the evolution of pro-sociality in large, genetically-heterogeneous groups.

Chapter 6: Empirical data suggest that monogamy or low levels of promiscuity have played a key role in the evolution of cooperative breeding and eusociality (Boomsma 2007, 2009; Hughes *et al.* 2008; Cornwallis *et al.* 2010). However, from a theoretical perspective, low levels of promiscuity, can favour dispersal away from the natal patch, and have been argued to select against cooperation, in a way that cannot be explained by inclusive fitness theory (Nonacs 2011). Here, I use an inclusive fitness approach to model selection for juveniles to stay and help parents breed in a simple patch-structured population. The model predicts that the level of promiscuity has either no, or a slightly negative influence on selection for helping. This prediction is driven by the fact that staying to help leads to increased competition between relatives for the breeding position - when promiscuity is low (and relatedness is high), the best way to aid relatives is by dispersing to avoid competing with them. Furthermore, my coauthors found the same results with an individual based

simulation, showing that this is not an area where inclusive fitness theory 'gets it wrong' (contra Nonacs 2011). My results suggest that whether promiscuity has a negative or positive effect on selection for staying to help will depend upon a large number of biological assumptions. The aim of this chapter is not to argue that low promiscuity disfavours cooperation – there is plenty of empirical evidence that it does. Rather, this model highlights that in nature the benefits of helping must be decoupled from the costs of competition.

Chapter 7: Explaining mutualistic cooperation between species remains a major challenge for evolutionary biology (Herre *et al.* 1999; Sachs *et al.* 2004; West *et al.* 2007b). Why cooperate if defection potentially reaps greater benefits? It is commonly assumed that spatial structure (limited dispersal) aligns the interests of mutualistic partners (Frank 1994; Doebeli and Knowlton 1998; Bever and Simms 2000; Hoeksema and Kummel 2003; Lion and van Baalen 2008; Platt and Bever 2009; Hodge *et al.* 2010). But is spatial structure consistently positive for cooperation? Here, I examine the role of spatial structure in the maintenance of mutualisms and show that when hosts interact with multiple partners and enforce cooperation at a fine scale, then spatial structure (limited dispersal) can actually disfavour cooperation by limiting the suite of potential partners. I find that the effect of spatial structuring depends on the scale (fine or coarse level) at which hosts reward their partners. My coauthors tested these model predictions using novel molecular methods to track the abundance of competing, closely related, cooperative and less-cooperative arbuscular mycorrhizal (AM) fungal symbionts on host roots over multiple generations. The results concur with my model predictions as when the soil is mixed to reduce spatial structure, the relative success of the more cooperative AM fungal species increases. This challenges previous suggestions that high spatial structuring is critical in stabilizing cooperation in the mycorrhizal mutualism (Bever *et al.* 2009).

Chapter 2.

Nice natives and mean migrants: the evolution of dispersal-dependent social behaviour in viscous populations.

This chapter appears as the following publication: El Mouden, C and Gardner, A. 2008. 'Nice natives and mean migrants: the evolution of dispersal-dependent social behaviour in viscous populations', *Journal of Evolutionary Biology* 21, 1480-1491.

2.1 Introduction

Cooperation abounds in nature, and poses one of the greatest problems for evolutionary biology. If natural selection is the survival of the fittest, then how can individuals be favoured to promote the reproductive success of others? (Hamilton 1996; West *et al*. 2007a). Despite this apparent problem, cooperation occurs at all levels of biological organisation from within eukaryotic cells, to multi-cellular organisms to complex human and animal societies (Leigh 1977; Buss 1987; Maynard Smith and Szathmáry 1995).

One explanation for cooperation is provided by kin selection (Hamilton 1963, 1964, 1970; Maynard Smith 1964). Because relatives share genes in common, an individual who improves the reproductive success of her relatives may increase her overall genetic contribution to future generations, and genes that promote cooperation may increase in frequency. Hamilton (1964) suggested two major mechanisms for ensuring that cooperative social behaviours be directed primarily towards high relatedness social partners. Firstly, he suggested that individuals might be able to

recognise and act preferentially towards their genealogical kin (kin discrimination). However, this mechanism may require complicated cognitive faculties, and may be inherently unstable (Crozier 1986; Rousset and Roze 2007), so it appears to be rather limited in its application. Secondly, he proposed that if individuals do not disperse far from their place of origin (population viscosity), then neighbours would tend to be genealogical relatives, and hence even indiscriminate cooperation may be favoured (see also Wright 1945). This requires no cognitive function, nor indeed any perception of the external world, and hence viscosity appears to provide a very general explanation for cooperation at all levels of biological organisation (West, *et al*. 2007b).

However, later work showed that cooperation is not so readily favoured in viscous populations. Although reduced dispersal increases the relatedness of social partners, which acts to favour the evolution of cooperation, it also increases competition between social partners for space and resources, which acts to disfavour the evolution of cooperation (reviewed by Queller 1992 and West *et al*. 2002a). In the simplest scenario these two effects exactly cancel, so that there is no net impact of the dispersal rate on the evolution of cooperation (Taylor 1992a, b; see also Wilson *et al*. 1992). Experimental and observational data support these predictions showing that local competition can reduce or completely negate any effect of limited dispersal on selection for cooperation (West *et al*. 2001; Griffin *et al*. 2004; Kümmerli *et al*. 2009). Indiscriminate cooperation is favoured in a purely viscous population only as readily as it is favoured in a fully dispersing population.

In addition to disfavouring cooperation, localised competition may also favour harming behaviour (reviewed by Gardner and West 2004b). However, Taylor's (1992a) model, which implicitly describes the evolution of indiscriminate harming as

well as helping behaviour in viscous populations, again predicts that there is no net impact of dispersal rate. Whilst limited dispersal increases competition between neighbours, promoting the evolution of harming, it also increases the relatedness of neighbours, which inhibits the evolution of harming, and these two effects exactly cancel. Neither indiscriminate helping nor indiscriminate harming are affected by the dispersal rate in this simplest scenario.

Taylor's (1992a) simple model provides a useful benchmark upon which to rest and conceptualise more elaborate (and potentially more realistic) models in which the relationships between dispersal, relatedness and competition are altered so that limited dispersal can make an impact on helping and harming behaviour. Analytical extensions of the basic model that have been shown to promote the evolution of indiscriminate helping behaviour include the inclusion of overlapping generations (Taylor and Irwin 2000; Irwin and Taylor 2001; Grafen 2007; Lehmann *et al*. 2007a; Taylor, *et al*. 2007), trans-generational social behaviour and niche construction (Lehmann 2007), elastic population structure (Taylor 1992b; van Baalen and Rand 1998; Mitteldorf and Wilson 2000), group (or 'budding') dispersal (Gardner and West 2006; Lehmann *et al*. 2006), sex-biased dispersal (Johnstone and Cant 2008), and kin discrimination (Perrin and Lehmann 2001). The evolution of harming has received much less attention (e.g. Gardner *et al*. 2004a, 2007b; Lehmann *et al*. 2007a; Johnstone and Cant 2008). A major challenge is to find minimal additions to the basic model that allow social behaviour to be promoted by population viscosity, without invoking unrealistic cognitive and other faculties that will reduce the generality of the mechanism.

In this article, we consider that individuals can assess their dispersal status, i.e. whether they have dispersed or whether they have remained close to their place of origin, and we ask whether they will be favoured to adjust their social behaviour

conditional upon this information. Whilst dispersers and non-dispersers do not differ in the resource competition they experience with neighbours, they can differ with respect to their relatedness to their neighbours. Specifically, non-dispersers are generally predicted to be more highly related to their neighbours and dispersers will be less related to their neighbours, relative to Taylor's (1992a) model for the same degree of local competition. Thus, we predict that helping will more readily be favoured among non-dispersers and harming more readily favoured among dispersers, relative to the case where social behaviour is not adjusted according to dispersal status. This mechanism does not require that individuals assess any aspect of their social partners directly, nor indeed necessarily any aspect of their external environment. Thus, it appears to be rather general in its application.

2.2 Models and Analyses

2.2.1 Basic model

Our analysis is based on the infinite island model analysed by Taylor (1992a; see also Wright 1931). A summary of the model notation used in this article is provided in Table 2.1. We consider an infinite population of asexual haploid individuals organised into patches of n individuals, and initially we assume that no social interaction takes place. Individuals produce a large number $K >> 1$ of offspring, then die, and each offspring disperses to another, randomly chosen, patch with probability m or else, with probability 1-m, remains in its patch of origin. After dispersal, n offspring are chosen on each patch to mature to adulthood, while the rest die, and this returns the population to its original size for the beginning of the next lifecycle.

Table 2.1. A summary of symbols used in the analysis

Symbol	*Definition*	*Symbol*	*Definition*
a	Fecundity cost of harming for harmer	M	Mutant
A	Personal-cost function for harming	n	Number of breeding spaces per patch
b	Fecundity benefit of helping for recipients	N	non-disperser
B	Recipient-benefit function for helping	q_X	Abundance of class X (Appendix 2.4 only)
$\tilde{b}$	Fitness effect of social behaviour on recipient	r	Coefficient of relatedness between patch mates (i.e. not including self)
c	Fecundity cost of helping for helper	R	Coefficient of relatedness within a patch (i.e. including self)
C	Personal-cost function for helping	t	Generation number
$\tilde{c}$	Fitness effect of social behaviour on actor	u	Generic non-disperser class (Appendix 2.4 only)
c_X	Reproductive value of class X (Appendix 2.4 only)	U	Set of all non-disperser classes (Appendix 2.4 only)
d	Fecundity cost of harming for victims	v	Generic disperser class (Appendix 2.4 only)
D	Victim-cost function for harming	V	Set of all disperser classes (Appendix 2.4 only)
D	disperser	w	Darwinian fitness
F	Fecundity of a focal individual	x	Helping strategy
$\overline{F}$	Average fecundity of focal patch	$\hat{x}$	Mutant helping strategy
g	Genetic 'breeding' value for social behaviour (Appendix 2.4 only)	$\overline{x}$	Average helping strategy
i	Number of mutants among a focal individual's patch mates	y	Harming strategy
K	Average number of offspring per individual in the population	$\hat{y}$	Mutant harming strategy
m	Dispersal rate	$\overline{y}$	Average harming strategy

Assuming that all genetic variation in the population is neutral with respect to reproductive success, we can use the recursion approach of Taylor (1992a) to

determine the relatedness structure of the population at equilibrium. We note that the expected relatedness of any individual in generation t to a randomly chosen member of her own patch (including herself) is $R_t = 1/n + ((n\text{-}1)/n)r_t$, where $1/n$ is the probability of choosing herself, in which case the relatedness is 1, and $(n\text{-}1)/n$ is the probability of choosing a different individual, in which case the relatedness is r_t, the relatedness of two different individuals in the same patch. The only way for two different individuals from the same patch to be related is if neither dispersed, which occurs with probability $(1\text{-}m)^2$. In this case, the two individuals will have an expected relatedness equal to the relatedness of two individuals chosen at random from that same patch in the previous generation, $R_{t\text{-}1}$. This allows us to write down a recursion:

$$R_t = \frac{1}{n} + \frac{n-1}{n}(1-m)^2 R_{t-1}, \qquad (1)$$

which can be solved for equilibrium ($R_{t\text{-}1} = R_t = R$) to yield an equilibrium relatedness of an individual to her group (including herself) of:

$$R = \frac{1}{n-(n-1)(1-m)^2}, \qquad (2)$$

as determined by Taylor (1992a). Substituting this into the expression $R = 1/n + ((n\text{-}1)/n)r$ and solving for r obtains the equilibrium relatedness between different individuals within the same patch:

$$r = \frac{(1-m)^2}{n-(n-1)(1-m)^2}, \qquad (3)$$

i.e. the F_{ST} of Wright (1943). Note that both these 'whole-patch' and 'others-only' relatedness coefficients (R and r; Pepper 2000) are expressed as averages over all individuals, with dispersers and non-dispersers taken together. However, dispersing

individuals always find themselves in a patch containing no relatives other than themselves, and so restricting attention to dispersers only, the equilibrium relatedness is $R_D = 1/n$ to the whole patch and $r_D = 0$ to the other individuals in the patch. Noting that $R = m\,R_D + (1\text{-}m)R_N$ and $r = m\,r_D + (1\text{-}m)r_N$, where R_N and r_N are the whole-patch and others-only relatedness for non-dispersing individuals, we can solve to find that:

$$R_N = \frac{n + (n-1)m(1-m)}{n\left(n - (n-1)(1-m)^2\right)}, \tag{4}$$

and:

$$r_N = \frac{1-m}{n - (n-1)(1-m)^2}, \tag{5}$$

for non-dispersers. Note that for $0 < m < 1$, we have $R_D < R < R_N$ and $r_D < r < r_N$, i.e. dispersers experience a lower relatedness to their social partners, and non-dispersers a higher relatedness to their social partners, than average (Figure 2.1). Taylor (1992a) found that unconditional helping behaviour cannot be favoured if it incurs a net fecundity cost for the actor; the result also extends to unconditional harming behaviour, revealing that this is also disfavoured if the actor incurs a net fecundity cost. In the next two sections, we explore whether and when the increased relatedness experienced by non-dispersers favours helping behaviour that is expressed only by non-dispersers, and whether and when the decreased relatedness experienced by dispersers favours harming behaviour that is expressed only by dispersers.

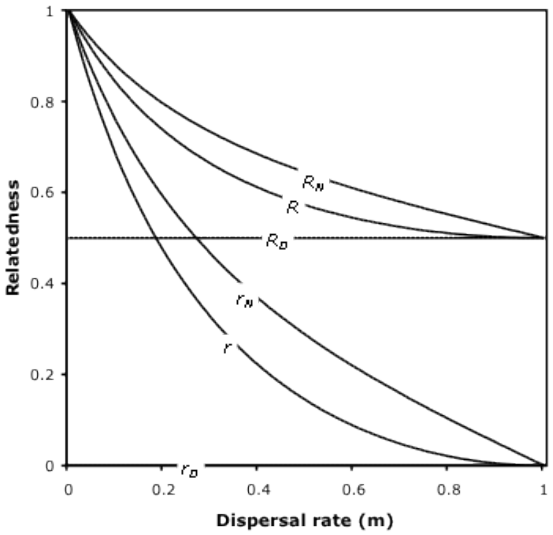


Figure 2.1. Relatedness coefficients in viscous populations. Increased dispersal (m) separates kin and hence reduces the relatedness between patchmates (r) and the relatedness of an individual to the patch as a whole (i.e. including itself; R). These relatedness coefficients correspond to Taylor's (1992a) model of unconditional social behaviour, in which neither costly helping nor costly harming are favoured by selection. Our model considers that nondispersing individuals will experience higher than average relatedness to their patch ($rN > r$ and $RN > R$) whilst dispersing individuals will experience lower than average relatedness to their patch ($rD < r$ and $RD < R$), and thus nondispersers may be favoured to exhibit indiscriminate helping behaviour, and dispersers may be favoured to exhibit indiscriminate harming behaviour. Numerical solutions are calculated assuming a patch size of two individuals ($n = 2$).

2.2.2 Evolution of helping in non-dispersers

We now consider a vanishingly rare genetic mutation that causes the bearer to express indiscriminate, others-only helping behaviour that incurs a relative personal fecundity cost c and gives a relative fecundity benefit b that is shared equally by her n-1 patch mates; these fecundity effects are expressed relative to the baseline fecundity, which is set equal to 1, and we assume b, $c << 1$. Crucially, we assume that this helping behaviour is expressed conditionally with respect to the individual's own dispersal status; in particular, we assume that only non-dispersers, who have remained in their natal patch, exhibit helping behaviour.

In order for the mutation to be favoured by selection it must, on average, increase the expected fitness of its bearers, i.e. the number of surviving offspring that the individual produces over her lifetime (Price 1970). There are two routes by which a gene may impact on the fitness of its bearer: firstly, by having a direct impact on that individual's personal reproductive success, and secondly, by having an indirect impact on the individual's personal reproductive success due to its presence among relatives who socially interact with her (neighbour-modulated fitness; Hamilton 1963, 1964). There are neither direct nor indirect effects of the gene on carriers that have dispersed away from their patch of origin, since they do not express the helping behaviour and they do not encounter any other individuals carrying the mutant gene. So, the condition for the gene to be favoured is if it leads, through direct and/or indirect effects, to an increase in the expected number of surviving offspring (i.e. Darwinian fitness) of non-dispersers (see Appendix 2.4 for mathematical details). The average fitness in the population is 1, because the population size remains constant from generation to generation, and dispersers and non-dispersers have the same expected fitness, so a condition for the mutant gene to be favoured by natural selection is that the expected fitness of a disperser carrying the gene exceeds 1.

We now proceed to calculate the expected fitness of a non-disperser who is carrying the mutant gene, and this is a function of her fecundity and the intensity of competition experienced by her offspring for one of the n breeding spaces within whichever patch they find themselves in after the dispersal event. The relative (to the population average) fecundity of a mutant non-disperser that shares her patch with i other mutant non-dispersers is:

$$F_{\mathrm{MN},i} = 1 - c + \frac{i}{n-1} b . \qquad (6)$$

Thus, the focal individual produces a total of $KF_{MN,i}$ offspring, of which $mKF_{MN,i}$ disperse while $(1\text{-}m)KF_{MN,i}$ remain in the natal patch.

We consider first the fate of the $mKF_{MN,i}$ dispersing offspring of the focal mutant individual. Each arrives in a separate patch, where they compete with $(1\text{-}m)nK$ locals and mnK other dispersers. As there are n breeding spaces within the patch, the probability that a particular dispersing offspring of the focal mutant survives competition to find a breeding space is $n/(1+(1\text{-}m)nK + mnK) \approx 1/K$. Thus, the expected number of survivors among the dispersing offspring of the focal mutant is $mKF_{MN,i}/K = mF_{MN,i,}$.

We now consider the fate of the $(1\text{-}m)KF_{MN,i}$ non-dispersing offspring of the focal mutant individual, who compete a breeding space on their natal patch. The competitors include $(1-m)K\overline{F}_{MN,i}$ non-dispersing locals plus $mKnF$ incoming migrants, where $\overline{F}_{MN,i}$ is the average relative fecundity of parents in the focal patch, which is:

$$\overline{F}_{\mathrm{MN},i} = 1 + \frac{i+1}{n}(b-c). \qquad (7)$$

Again, there are n breeding spaces available, and so the expected number of successful non-dispersing offspring due to the focal mutant individual is $(1-m)KF_{MN,i}/\left((1-m)K\overline{F}_{MN,i} + mK\right)$ which, to first order in b and c, is:

$$(1-m)\left(1-c+\frac{i}{n-1}b-(b-c)\left(\frac{i+1}{n}\right)(1-m)\right). \qquad (8)$$

By adding the expected number of surviving dispersing and non-dispersing offspring we can find the expected fitness of the focal mutant non-disperser, which is:

$$w_{\mathrm{MN},i} = 1 - c + \frac{i}{n-1} b - (1-m)^2 (b-c) \frac{i+1}{n}. \qquad (9)$$

This expected fitness is conditional on there being i other (non-dispersing) mutants in the focal individual's patch. As outlined above, the condition for invasion of the mutant gene is $w_{\mathrm{MN}} > 1$, where w_{MN} is the average fitness of all non-dispersing mutant individuals, i.e. $w_{\mathrm{MN}} = E_{\mathrm{MN}}(w_{\mathrm{MN},i})$. This average fitness obtains:

$$w_{\mathrm{MN}} = 1 - c + bE_{\mathrm{MN}}\left(\frac{i}{n-1}\right) - (1-m)^2 (b-c) E_{\mathrm{MN}}\left(\frac{i+1}{n}\right). \qquad (10)$$

Note that $i/(n\text{-}1)$ is the proportion of (non-disperser) mutants among the social partners of the focal individual, and so the average of this quantity, taken over all non-dispersing mutants in the population, is equal to r_{N}, the others-only relatedness experienced by non-dispersers. This makes use of the assumption that the mutant is vanishingly rare in the population. Similarly, $(i\text{+}1)/n$ is the proportion of (non-disperser) mutants among all the individuals in the focal individual's patch, including herself; so, averaging this quantity over all non-disperser mutants in the population obtains R_{N}, the whole-group relatedness experienced by non-dispersers. Making this substitution, we obtain a condition, in the form of Hamilton's rule, for when the helping mutant will invade from rarity:

$$-c + br_{\mathrm{N}} - (1-m)^2 (b-c) R_{\mathrm{N}} > 0. \qquad (11)$$

Because we have assumed weak selection, this condition for increase is frequency independent (Rousset 2004, p80), and therefore it also describes the progress of the mutant gene when it is no longer rare. Although condition (11) has been derived using a neighbour-modulated (or 'direct') fitness approach to kin selection, it easily yields an inclusive fitness interpretation. Helping by non-dispersers is favoured

when the sum of three components, equal to the inclusive fitness effect of their helping, is positive. Firstly, their help leads to a personal loss of c offspring. Secondly, their help leads to b extra offspring for the other individuals on the patch, and these are valued at r_N, the relatedness to patch mates. Thirdly, the net increase of b-c offspring on the patch leads to enhanced competition for locals, to the extent that these extra offspring do not disperse and nor do the other locals (i.e. $(1\text{-}m)^2$), and this leads to the competitive exclusion of local offspring (including own offspring) which are valued by R_N, the whole-group relatedness appropriate for non-dispersers.

Condition (11) is expressed in terms of fecundity effects, but can be re-written as $-\tilde{c} + \tilde{b} r_N > 0$, in terms of the direct ($-\tilde{c}$) and indirect ($\tilde{b}$) fitness effects of helping. The direct cost to the actor is $\tilde{c} = c + (1-m)^2 (b-c)/n$ and the benefit to the recipient is $\tilde{b} = b(1-m)^2 (b-c)(n-1)/n$; both fitness effects are positive under the assumption $b > c$, and so the helping behaviour is altruistic ($\tilde{c}$ & $\tilde{b} > 0$; Hamilton 1964).

Note that the relatedness coefficients can be written in the form of equations (4) and (5); making this substitution into the inequality (11) obtains an invasion condition that is expressed wholly in terms of model parameters (n, m, b and c):

$$b/c > \frac{n^2(2-m) - (n-1)(1-m)^3}{1 + m\left(2n - (n-1)(3-m)m - 3\right)}. \qquad (12)$$

The above analysis has determined when a mutation that incrementally increases (or decreases, if we change the sign of b and c) the level of helping indiscriminate exhibited by non-dispersers will invade from rarity under the action of natural selection. The RHS of inequality (12), which we may denote $f(n,m)$, presents a quantity that must be exceeded by the benefit/cost ratio of helping in order for non-dispersing individuals to be favoured to indiscriminately help their patch mates. We

see that as n increases, the RHS of the inequality becomes larger ($\partial f/\partial n > 0$) i.e. helping is less favoured as the number of individuals per patch increases (Figure 2.2a). We see that helping is most readily favoured at intermediate migration rates ($\partial f/\partial m < 0$ for $m < m^*$ there is some threshold $0<m^*<1$ such that $\partial f/\partial m > 0$ for $m > m^*$; Figure 2.2a). At the extreme of a very viscous population, $m \to 0$, the condition for helping to be favoured in non-dispersers is $b/c > 1+(2n-1)n$. However, the selection pressure here is vanishingly small (LHS (11) $\to 0$ as $m \to 0$), and so helping is a nearly-neutral character in extremely viscous populations and is not expected to be elaborated by natural selection. At the other extreme of a fully mixing population, costly helping is never favoured ($f \to \infty$ as $m \to 1$). Numerical investigation reveals that the optimum migration rate for helping (i.e. $m = m^*$) is less than 0.5, but converges upon 0.5 as $n \to \infty$, and the condition for helping to evolve at this optimum is approximately $b/c > 4n-2$.

We have determined when a small increase or decrease in helping by non-dispersers will be favoured by natural selection, and we can use this to determine the absolute level of helping that is expected to evolve, i.e. the evolutionarily stable strategy (ESS; Maynard Smith and Price 1973). If we assume that expressing a level x of helping incurs a personal fecundity cost $C(x)$ and an others-only fecundity benefit $B(x)$ (where $B, C << 1$; see Appendix 2.4), then the incremental increases in cost and benefit that result from adopting a mutant strategy $\hat{x}$, rather than the resident strategy $\bar{x}$ are $C(\hat{x}) - C(\bar{x}) = C'(\bar{x})(\hat{x} - \bar{x}) = c$ and respectively. Substituting these into the invasion condition (12), we find that an incremental increase in the helping strategy of non-dispersers ($\hat{x} > \bar{x}$) is favoured when the condition is satisfied, an incremental decrease in helping ($\hat{x} < \bar{x}$) is favoured when the reversed condition is satisfied, and a candidate (non-zero) evolutionarily stable strategy (x^*; i.e. a resident strategy that is

uninvadable by any mutant strategy $\hat{x}$) is implicitly defined by changing the invasion condition to an equation:

$$\frac{B'(x^*)}{C'(x^*)} = \frac{n^2(2-m)-(n-1)(1-m)^3}{1+m(2n-(n-1)(3-m)m-3)}. \qquad (13)$$

Strictly, this condition applies only if the ESS takes a positive value; and $x^* = 0$ if $B'(0)/C'(0) < 0$). An explicit solution for the ESS level of helping x^* requires explicit functional forms for $C(x)$ and $B(x)$. Numerical solutions for an illustrative example are given in Figure 2.2*b*.

2.2*a* 2.2*b*

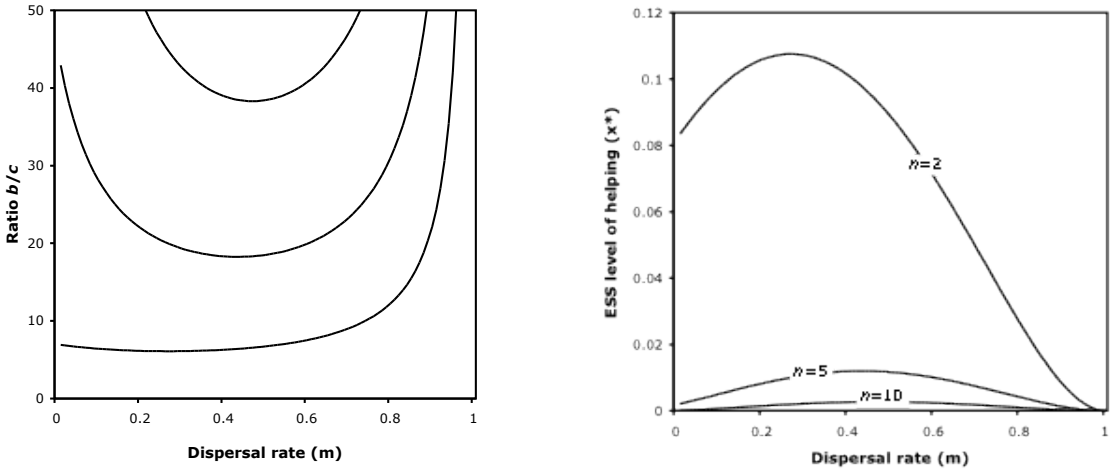


Figure 2.2. Indiscriminate helping by non-dispersers. (a) The benefit/cost ratio (b/c) below which a mutant gene increasing the level of helping is favoured by selection, for n = 2, 5 and 10 individuals per patch, and a range of dispersal rates $0 < m < 1$. (b) The ESS level of helping, assuming $C(x) = kx$ and $B(x) = k\alpha x^{\beta}$ and $\alpha = 4$, $\beta = 0.5$, $k \to 0$, n = 2, 5 and 10, and $0 < m < 1$.

2.2.3 The evolution of harming in dispersers

We now investigate the evolutionary success of a vanishingly rare mutant gene that causes bearers to express harming behaviour upon dispersal to a new patch, and which confers a relative personal fecundity cost a and a further relative fecundity cost d that is shared equally among the n-1 other individuals on the patch. Again we

express a and d relative to baseline fecundity, and we assume that $a, d << 1$. This mutant gene will spread if, on average, it increases fitness of its bearers (whether they are dispersers or non-dispersers), either due to direct or indirect fitness effects. There are no direct or indirect effects of the mutant gene on non-dispersing mutants as these do not express the gene nor do they encounter dispersing mutants who do express the harming behaviour. Therefore, the mutant gene can only invade from rarity if it increases the average fitness of mutant dispersers (see Appendix 2.4 for mathematical details). The relative fecundity of a mutant disperser is simply:

$$F_{\mathrm{MD}} = 1 - a \,. \tag{14}$$

This focal individual produces a total of KF_{MD} offspring, of which mKF_{MD} disperse to a new patch and $(1\text{-}m)KF_{\mathrm{MD}}$ remain in the natal patch. Using the same approach as in the previous section, the probability that any one of the dispersing offspring of the focal mutant will survive the competition and find a breeding space is $1/K$. This means that the expected number of dispersing offspring due to the focal individual is $mF_{\mathrm{MD}} = m(1\text{-}a)$.

We now consider the fate of the $(1\text{-}m)KF_{\mathrm{MD}}$ non-dispersing offspring of the focal mutant disperser, which remain on the natal patch to compete for breeding spaces. The competitors consist of $(1-m)K\overline{F}_{\mathrm{MD}}$ non-dispersing locals, plus mKn incoming migrants, where $\overline{F}_{\mathrm{MD}}$ is the average relative fecundity of parents in the focal patch and is given by:

$$\overline{F}_{\mathrm{MD}} = 1 - \frac{1}{n}(a + d). \tag{15}$$

Again, there are n breeding spaces available, and so the expected number of successful non-dispersing offspring due to the focal mutant individual is $n(1-m)KF_{\mathrm{MD}} / \left((1-m)K\overline{F}_{\mathrm{MD}}n + mKn\right)$, which is, to first order in a and d, equal to:

$$(1-m)(1-a) - (1-m)^2(a+d)\frac{1}{n}. \tag{16}$$

By adding the expected numbers of dispersing and non-dispersing offspring together this we can find the average fitness of a mutant disperser, and this is:

$$w_{\mathrm{MD}} = 1 - a + (a+d)(1-m)^2\frac{1}{n}. \tag{17}$$

Note that $1/n$ is equal to R_{D}, the relatedness coefficient for the whole patch the mutant is found in (for dispersing mutants, r_{D} is 0). In order for the mutant gene to invade from rarity it must provide a fitness advantage i.e. $w_{\mathrm{MD}} > 1$. This gives an inequality in the form of Hamilton's rule:

$$-a + (a+d)(1-m)^2\frac{1}{n} > 0 \tag{18}$$

Again, we can derive an inclusive fitness interpretation. Harming by dispersers is favoured when the sum of three components, equal to the inclusive fitness effect of harming, is positive. Firstly, the harming act leads to a personal loss of a offspring. Secondly, the act will inflict a cost d on fellow patch mates; but, since these are related by $r_{\mathrm{D}} = 0$, this component of inclusive fitness is zero. Thirdly, the net decrease of $a+d$ offspring on the patch leads to decreased competition for locals and hence an effective increase in local offspring (including the mutants own offspring) that would otherwise have been competitively excluded. These are valued by $R_{\mathrm{D}} = 1/n$ the whole-group relatedness for dispersing mutants.

Note that condition (18) can be recovered from condition (11) by substituting $a = c$ and $d = -b$ and replacing the relatedness coefficients for non-dispersers (r_N and R_N) with those for dispersers (r_D and R_D). As we did for condition (11), condition (18) can be re-expressed in terms of fitness effects, i.e. $-\tilde{c} + \tilde{b}r_D$, where $\tilde{c}$ and $\tilde{b}$ are the direct and indirect fitness consequences of harming. We find that $\tilde{c} = a - (1-m)^2(a+d)/n$ and $\tilde{b} = -d + (1-m)^2(a+d)(n-1)/n$; both fitness effects are negative when condition (18) is satisfied so harming, whenever it is favoured by selection, is selfish ($\tilde{c}$ & $\tilde{b} < 0$; Hamilton 1964).

We now rearrange inequality (18) to give a condition:

$$d/a > \frac{n}{(1-m)^2} - 1, \qquad (19)$$

which can be used to determine when a mutation that incrementally increases (or decreases) the level of indiscriminate harming exhibited by dispersers will invade from rarity under the action of natural selection. It is helpful to denote the RHS of condition (19) by $g(n,m)$. We find that, as with the helping analysis considered in the previous section, harming by dispersers is progressively more difficult to evolve as the number of individuals per patch increases (i.e. $\partial g/\partial n > 0$; Fig. 2.3a). Also, harming is less easily favoured with increasing dispersal rates (i.e. $\partial g/\partial m > 0$; Fig. 2.3a): at the extreme of a very viscous population ($m \to 0$) harming is favoured whenever the ratio of the harm inflicted on patch mates to harm inflicted upon self is greater than the number of individuals the disperser shares a patch with (i.e. $d/a > n-1$; Hamilton 1971), and at the opposite extreme of a fully mixing population harming will not evolve under any circumstances ($g \to \infty$ as $m \to 1$). We note that, if the dispersal rate is tuned so as to be most favourable to harming (i.e. $m \to 0$) then

harming by dispersers evolves more easily than does helping by nondispersers in the corresponding best-case scenario for the latter to evolve (i.e. $m = m^*$; and $n\text{-}1 < 4n\text{-}2$).

Again, we can locate a candidate ESS to examine the absolute level of harming that we expect to evolve among dispersing individuals. We assume that expressing a level y of harming incurs a personal fecundity cost $A(y)$ and an others-only fecundity cost of $D(y)$ (where $A, D << 1$; see Appendix 2.4), hence the incremental increases in personal cost and damage to victims that results from adopting the mutant strategy $\hat{y}$ rather than the resident strategy $\bar{y}$ are given by $D(\hat{y}) - D(\bar{y}) = D'(\bar{y})(\hat{y} - \bar{y}) = d$ and $A(\hat{y}) - A(\bar{y}) = A'(\bar{y})(\hat{y} - \bar{y}) = a$ respectively. Substituting these into invasion condition (18), we find that an incremental increase in the harming strategy of non-dispersers ($\hat{y} > \bar{y}$) is favoured when the condition is satisfied, an incremental decrease in harming ($\hat{y} < y$) is favoured when the condition is reversed, and a candidate ESS (y^*; i.e. a resident strategy that is uninvadable by any mutant strategy y) is implicitly defined by changing the invasion condition to an equation:

$$\frac{D'(y^*)}{A'(y^*)} = \frac{n}{(1-m)^2} - 1. \qquad (20)$$

Again, this applies strictly to ESSs that take intermediate values, and $y^* = 0$ if $D'(0)/A'(0) < 0$. An explicit solution for the ESS level of helping y^* requires explicit functional forms for $D(y)$ and $A(y)$. A numerical solution for an illustrative example is given in Figure 2.3b. Note that, whilst we have modelled helping and harming behaviours as separate traits, an alternative (but equivalent) analysis could treat this social behaviour as a single trait varying from extreme conflict to extreme cooperation.

2.3*a* 2.3*b*

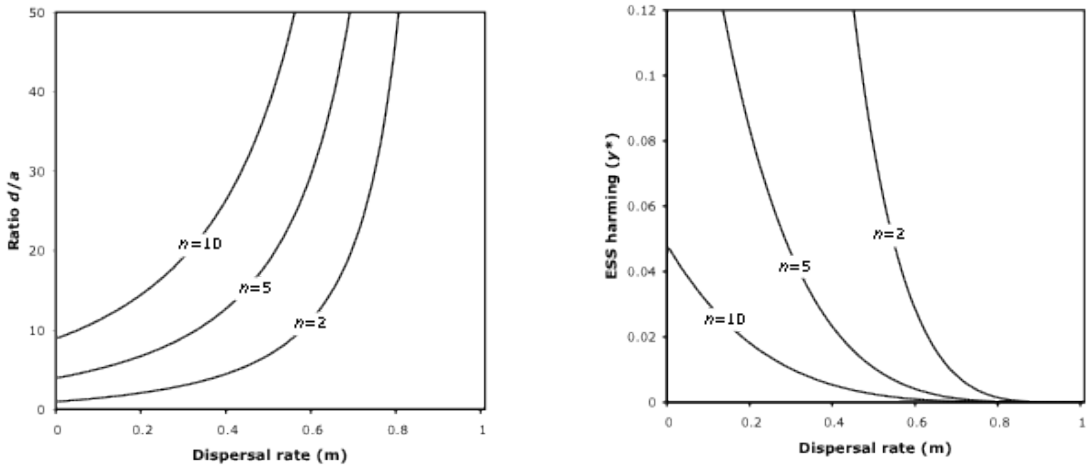


Figure 2.3. Indiscriminate harming by dispersers. (a) The harm / cost ratio (d/a) below which a mutant gene increasing the level of harming is favoured by selection, for n = 2, 5 and 10 individuals per patch, and a range of dispersal rates $0 < m < 1$. (b) The ESS level of harming, assuming $A(y) = ky$ and $D(y) = k\alpha y^{\beta}$ and $\alpha = 4$, $\beta = 0.5$, $k \to 0$, $n = 2$, 5 and 10, and $0 < m < 1$.

2.3 Discussion

Hamilton (1964, 1971) suggested that indiscriminate helping behaviour should be commonest in viscous populations, because these were associated with greater relatedness between neighbouring individuals. However, more recent work has emphasised the potential for kin competition to inhibit helping behaviour in viscous populations (reviewed by Queller 1992 and West *et al*. 2002a), and in the simplest scenario of an inelastic island model there is no net impact of dispersal rate upon the evolution of helping (Taylor 1992a). There is much interest in determining which minimal modifications to the basic model can be made in order to allow social behaviour to flourish. We have examined the possibility that social behaviour is adjusted conditionally upon the dispersal status of the actor, and have shown that this allows both helping and harming behaviour to evolve in viscous populations. Because no external information need be assessed by the individual – for example, its

dispersal status may be inferred according to whether it is a winged or wingless morph or whether it experienced a period of cooling or desiccation associated with aerial dispersion – and hence there is no reliance on complex cognitive function, this appears to be a rather general mechanism for the evolution of social behaviours in viscous populations.

In the simplest case of an inelastic island model with dispersal-independent social behaviour (Taylor 1992a) the effect of increased relatedness associated with population viscosity is exactly cancelled by the effect of increased competition between social partners. Thus, both helping and harming behaviours evolve no more readily in viscous populations than they do in fully mixing populations. In contrast, dispersal-dependent social behaviour decouples relatedness and competition so that, while dispersers and non-dispersers experience the same level of competition with their social partners, they differ in their genetic relatedness. In particular, non-dispersers are more related than average to their social partners and dispersers are less related than average to their social partners, and this can allow for the evolution of indiscriminate helping by non-dispersers and indiscriminate harming by dispersers. In the special cases of a completely viscous or completely mixing population, individuals are either all non-dispersers or all dispersers, and so at these extremes our analysis recovers Taylor's (1992a) results and here we predict that neither costly helping nor costly harming will evolve (for example, whilst dispersers are expected to invest more into harming as the population becomes more viscous, the frequency of dispersers is decreasing towards zero). Thus, we predict that costly helping and harming behaviours are most abundant at intermediate population viscosity.

We have found a basic asymmetry between helping and harming evolution such that, over all, helping is relatively weakly favoured and harming is relatively strongly

favoured in viscous populations. This is because zero relatedness (on average) is guaranteed by one's own dispersal, irrespective of the population level of dispersal, whereas the relatedness achieved by not dispersing is nonzero only insofar as one's social partners are also non-dispersers. In a highly viscous population conditions are favourable to the evolution of harming by dispersers, because they experience strong local competition and zero relatedness to their social partners. Conversely, while the low competition experienced in highly dispersive populations is conducive to helping, even those individuals not dispersing will have low relatedness to their social partners, who will often be immigrants. So whilst the level of harming exhibited by a disperser is predicted to increase with increasing population viscosity, the level of helping by a non-disperser is predicted to maximise at intermediate population viscosity. Thus, if a sample of species were collected that had relatively low viscosity then we would support Hamilton's (1964) prediction that, within this sample, there will be a positive correlation between viscosity and the level of helping. However, a sample of species with relatively high population viscosities might show the reverse trend.

The implications of this simple mechanism for the evolution of indiscriminate helping by non-dispersers were also considered by Perrin and Lehmann (2001). However, in that previous study, the authors evaluated fitness by considering the fecundity of actors averaged over the disperser and non-disperser classes (their equation 5a), instead of averaging the fitness component obtained by disperser and non-disperser, with the result that the fitness consequences for disperser and non-disperser where not properly separated. This led to an underestimation of the intensity of kin competition (Lehmann 2003, pp14-16; L. Lehmann, *pers. comm*.). Thus, the results of the present analysis on the evolution of helping are quantitatively and also qualitatively different from those obtained previously. For example, Perrin and

Lehmann's (2001) analysis predicted that the evolutionarily stable level of helping should increase monotonically with population viscosity, whereas we have described a dome-shaped relationship, with maximal helping at intermediate viscosity. In addition, Perrin and Lehmann (2001) did not consider the evolution of indiscriminate harming by dispersing individuals.

Our model of dispersal-dependent social behaviour may provide insight into the evolution of social parasitism in aphids, whereby individuals disperse to new colonies and, upon arrival, downregulate their usual altruistic colony-defence function and upregulate selfish, personal reproduction (Abbot *et al*. 2001; Foster 2002). Although social parasitism is known in several taxa, this might usually be attributed to direct discrimination of own versus alien colonies. However, aphids are presently understood to lack the faculty for kin recognition that would permit them to directly assess genetic relatedness of their neighbours, and so it may be the simple act of dispersal that provides the cue for this facultative change in their social behaviour (Foster 2002). The present analysis is also conceptually related to a study of dispersal-dependent sex allocation by Taylor and Crespi (1994), which predicted (and empirically tested and confirmed in a species of thrips) that dispersing and non-dispersing individuals would allocate differentially to sons versus daughters due to the correlation between dispersal status and relatedness to neighbours. In particular, Taylor and Crespi's (1994) diploidy model predicted that non-dispersers should exhibit a slight bias towards daughters, and dispersers a strong bias towards sons. This is analogous to our result that non-dispersers are weakly favoured to help and dispersers are strongly favoured to harm, and indeed in both cases the pattern is due to the different relatedness experienced by dispersers and non-dispersers.

A correlation between dispersal status and social behaviour can have important consequences for population demography, suggesting avenues for future research

directions. By modifying the fitness differences between dispersers and non-dispersers, this association can drive selection for changes in the rate and pattern of dispersal. Perrin and Lehmann (2001) considered the role for indiscriminate helping by non-dispersers to mediate the evolution of dispersal, and the subsequent feedback on the helping trait itself. As with our analysis, Perrin and Lehmann (2001) found that helping was only weakly favoured in viscous populations, and hence the co-evolution of helping and dispersal did not have a dramatic impact on either trait. However, the role for harming by dispersers to mediate the evolution of population viscosity remains to be considered, and our analysis suggests potential for rather stronger effects. An association between dispersal status and social behaviour might also impact upon inter-specific interactions, influencing the distribution and abundance of competing species. For example, Duckworth (2007) has described a positive correlation between dispersal and aggression in blue birds that appears to mediate species range expansions.

More generally, we have highlighted that the kin selection coefficient of relatedness describes the conditional information that an individual has with regard to the genetic similarity of its social partners, rather than their actual genetic similarity (e.g. Frank 1998, ch 6). Typical extensions of Taylor's (1992a) simple island model that promote the evolution of social behaviour in viscous populations do so by decoupling the genetic structure of the population from the intensity of local competition; for example, by introducing 'elasticity' so that cooperative patches can grow in size, localised competition is reduced while genetic structuring remains high. In contrast, the degree of genetic structure and the intensity of localised competition described in our model is exactly as occurs in Taylor's (1992a) model, and the decoupling of relatedness and localised competition occurs through use of conditional information: whether or not the individual dispersed.

2.4 Appendix

2.4.1 Natural selection and social behaviour

We consider a locus G controlling social behaviour of one type of individual (i.e. non-disperser or disperser), and we denote an individual's genetic 'breeding' value (Price 1970; Falconer 1981; Grafen 1985) for the behaviour as g. The change in the population average breeding value is given by:

$$\Delta\overline{g} = \sum_{u\in U} c_u \operatorname{cov}_u\left(w/\overline{w}_u, g\right) + \sum_{v\in V} c_v \operatorname{cov}_v\left(w/\overline{w}_v, g\right), \tag{A1}$$

where: U is the set of all non-disperser classes (defined according to the number of non-dispersers among their social partners); V is the set of all disperser classes (also defined according to the number of non-dispersers among their social partners); c_u and c_v are the class reproductive values of non-disperser class u and disperser class v respectively; $\overline{w}_u$ and $\overline{w}_v$ are the average fitnesses for individuals of class u and v respectively; and cov is the statistical covariance taken over individuals of the indicated set (Price 1970; Taylor 1996b).

2.4.2 Natural selection and social behaviour in non-dispersers

Consider first that the gene affects social behaviour in non-dispersers only. We can expand the selection component due to dispersers as:

$$\sum_{v\in V} c_v \operatorname{cov}_v\left(w/\overline{w}_v, g\right) = \sum_{v\in V} c_v \beta_v\left(w/\overline{w}_v, g\right) \operatorname{var}_v\left(g\right), \tag{A2}$$

where β is a least-squares linear regression, and var is the statistical variance, both taken over the indicated class of individuals. Expanding the regression into partial regressions, we have:

$$\begin{aligned}\beta_v\left(w/\overline{w}_v, g\right) = {} & \beta_v\left(w/\overline{w}_v, g \mid g'_{\mathrm{N}} \,\&\, g'_{\mathrm{D}}\right) + \beta_v\left(w/\overline{w}_v, g'_{\mathrm{N}} \mid g \,\&\, g'_{\mathrm{D}}\right)\beta_v\left(g'_{\mathrm{N}}, g\right) \\ & + \beta_v\left(w/\overline{w}_v, g'_{\mathrm{D}} \mid g'_{\mathrm{N}} \,\&\, g\right)\beta_v\left(g'_{\mathrm{D}}, g\right),\end{aligned} \tag{A3}$$

where g'_{N} and g'_{D} are the average breeding values of the individual's non-disperser and disperser social partners, respectively. Note that, because the gene is not expressed in dispersers, $\beta_v(w/\ \overline{w}_v, g \mid g'_{\mathrm{N}}$ and $g'_{\mathrm{D}}) = \beta_v(w/\ \overline{w}_v, g'_{\mathrm{D}} \mid g$ and $g'_{\mathrm{N}}) = 0$. Also, because the focal individual is a disperser it is unrelated to the other individuals on its patch, and so $\beta_v(g'_{\mathrm{N}}, g) = \beta_v(g'_{\mathrm{D}}, g) = 0$. Hence, $\beta_{\mathrm{v}}(w/\ \overline{w}_v, g) = 0$ and so, from equations (A1) and (A2), selection on the gene is given by:

$$\Delta\overline{g} = \sum_{u \in U} c_u \operatorname{cov}_u\left(w/\overline{w}_u, g\right). \tag{A4}$$

Thus, if the gene is expressed only in non-dispersers, selection for or against the gene is determined solely by the fitness consequences for non-dispersers.

2.4.3 Natural selection and social behaviour in dispersers

Alternatively, consider that the gene affects social behaviour in dispersers only. We can expand the selection component due to non-dispersers as:

$$\sum_{u \in U} c_u \operatorname{cov}_u\left(w/\overline{w}_u, g\right) = \sum_{u \in U} c_u \beta_u\left(w/\overline{w}_u, g\right) \operatorname{var}_u\left(g\right), \tag{A5}$$

and, expanding the regression into partial regressions, we have:

$$\begin{aligned}\beta_u\left(w/\overline{w}_u, g\right) = {} & \beta_u\left(w/\overline{w}_u, g \mid g'_{\mathrm{N}} \,\&\, g'_{\mathrm{D}}\right) + \beta_u\left(w/\overline{w}_u, g'_{\mathrm{N}} \mid g \,\&\, g'_{\mathrm{D}}\right)\beta_u\left(g'_{\mathrm{N}}, g\right) \\ & + \beta_u\left(w/\overline{w}_u, g'_{\mathrm{D}} \mid g'_{\mathrm{N}} \,\&\, g\right)\beta_u\left(g'_{\mathrm{D}}, g\right),\end{aligned} \tag{A6}$$

where g'_N and g'_D are once again the average breeding values of the individual's non-disperser and disperser social partners, respectively. Note that, because the gene is not expressed in non-dispersers, $\beta_u(w/\ \bar{w}_u, g \mid g'_N \text{ and } g'_D) = \beta_u(w/\ \bar{w}_u, g'_N \mid g \text{ and } g'_D) = 0$. Also, because the focal individual is unrelated to its disperser social partners, $\beta_u(g'_D, g) = 0$. Hence, $\beta_u(w/\ \bar{w}_u, g) = 0$ and so, from equations (A1) and (A5), selection on the gene is given by:

$$\Delta\bar{g} = \sum_{v \in V} c_v \operatorname{cov}_v\left(w/\bar{w}_v, g\right). \tag{A7}$$

Thus, if the gene is expressed only by dispersers, selection for or against the gene is determined solely by the fitness consequences for dispersers.

2.4.4 Invasion condition for non-disperser social behaviour

We now determine the invasion condition for a gene affecting social behaviour of non-dispersers, and we do so under the assumptions that: (1) the gene has vanishingly small impact on the phenotype; and (2) social behaviours in general have vanishingly small impact upon fecundity. Because the population is not growing in size, we can write the fitness of a focal non-disperser individual $j \in J_u$ in class u as $w_{uj} = 1 + \delta w_{uj}$, the average fitness of individuals of this class as $w_u = 1 + \delta\ \bar{w}_u$, and the reproductive value of this class as $c_u = q_u + \delta c_u$, where q_u is the proportion of all individuals in the population that belong to class u. These three deviation terms are all vanishingly small quantities (δw_{uj}, $\delta\ \bar{w}_u$, $\delta c_u << 1$).

Taking equation (A4), and making the individual notation more explicit, the action of selection is given by:

$$\begin{aligned}\Delta\bar{g} &= \sum_{u\in U}\left(q_u + \delta c_u\right)\mathrm{cov}_u\left(w_{uj}/\bar{w}_u, g_{uj}\right) \\ &= \sum_{u\in U} q_u \,\mathrm{cov}_u\left(w_{uj}/\bar{w}_u, g_{uj}\right) + \sum_{u\in U}\delta c_u \,\mathrm{cov}_u\left(w_{uj}/\bar{w}_u, g_{uj}\right).\end{aligned} \tag{A8}$$

Due to the weak-selection assumption, $\mathrm{cov}_u(w_{uj}/\ \bar{w}_u, g_{uj}) << 1$, and so the second term of equation (A8) is negligible. Using $w_{uj} = 1 + \delta w_{uj}$ and $w_u = 1 + \delta\ \bar{w}_u$, and discarding higher order terms of δ, we can rewrite equation (A8) as:

$$\begin{aligned}\Delta\bar{g} &= \sum_{u\in U} q_u \,\mathrm{cov}_u\left(w_{uj} - \delta\bar{w}_u, g_{uj}\right) \\ &= \sum_{u\in U} q_u \,\mathrm{cov}_u\left(w_{uj}, g_{uj}\right) \\ &= q_{\mathrm{N}} \sum_{u\in U}\frac{q_u}{q_N}\left(\sum_{j\in J_u}\frac{q_{uj}}{q_u} w_{uj} g_{uj} - w_u g_u\right),\end{aligned} \tag{A9}$$

where $q_{\mathrm{N}} = \Sigma_U\, q_u = 1\text{-}m$ is the total fraction of individuals in the population that are non-dispersers. Making the standard assumption that gene frequencies are equal in all classes, i.e. $g_u = \ \bar{g}$ for all $u \in U$, this obtains:

$$\begin{aligned}\Delta\bar{g} &= (1-m)\left(\sum_{u\in U}\sum_{j\in J_u}\frac{q_{uj}}{q_N} w_{uj} g_{uj} - \bar{w}_{\mathrm{N}}\bar{g}\right) \\ &= (1-m)\,\mathrm{cov}_{\mathrm{N}}\left(w_{uj}, g_{uj}\right),\end{aligned} \tag{A10}$$

where $\mathrm{cov}_{\mathrm{N}}$ is a covariance taken over the set of all non-disperser individuals in the population. Natural selection acts to increase the average genetic breeding value of the population when $\Delta\ \bar{g} > 0$. In order to track the invasion success of a rare variant gene, we may assign it a value of $g = 1$ and all its alleles a value of $g = 0$, in which case we can write $\Delta\ \bar{g} = (1\text{-}m)\ \bar{g}(w_{\mathrm{MN}}\text{-}\ \bar{w}_{\mathrm{N}})$, where w_{MN} is the average fitness of all the non-disperser carriers of the variant gene. Hence, the variant gene invades if, on average,

it increases the fitness of its non-disperser carriers above that of the average non-disperser individual ($w_{MN} > \bar{w}_N$).

2.4.5 Invasion condition for disperser social behaviour

The action of selection upon genes for disperser social behaviour (equation (A7)) has the same form as that for non-disperser social behaviour (equation (A4)), and an invasion condition can be determined by following the procedure of the last section and simply switching the non-disperser notation to the corresponding disperser notation (i.e. u to v, U to V, and N to D). This reveals that a rare genetic variant that alters the social behaviour of disperser individuals is favoured if the average fitness of its disperser carriers is greater than the average fitness of all dispersers in the population ($w_{MD} > \bar{w}_D$).

Chapter 3.
The enforcement of cooperation by policing.

This chapter appears as the following publication: El Mouden, C., West, S. A. and Gardner, A 2010. 'The enforcement of cooperation by policing', *Evolution*. 64, 2139-2152.

3.1 Introduction

Explaining cooperation is a major challenge for evolutionary biology. Natural selection favours those individuals who have the greatest relative fitness (Darwin 1859; Fisher 1930; Price 1970). However, all else being equal, cooperation reduces the relative fitness of the actor, as it involves providing a benefit to other individuals. The challenge is therefore to explain how cooperation can evolve by natural selection (Hamilton 1966; Sachs *et al*. 2004; Lehmann and Keller 2006; Lehmann *et al*. 2007c; West *et al*. 2007a). In the social sciences, the problem of cooperation is famously encapsulated by the "tragedy of the commons" (Hardin 1968) which considers a group of herdsmen deciding how many animals to graze on a shared pasture. The benefit of adding an extra animal accrues directly to the individual herdsman whilst the cost of overgrazing is shared by all. Hence, each herdsman is favoured to add further animals to the commons, resulting in severe overgrazing and the destruction of the shared resource. Finding solutions to avert this tragedy is of interest to evolutionary biologists, economists and moral and political philosophers (Ostrom 1990; Binmore 1994, 1998; Frank 2003, 2009).

Two major ways for the tragedy of the commons to be averted are through kin-selected self-restraint or repression of competition (Frank 2003). The former mechanism works when relatives interact, as individuals that improve the reproductive success of those with which they share genes are more successful in transmitting copies of these genes to future generations (Hamilton 1963, 1964a; Maynard Smith 1964; Hamilton 1970). However, theory predicts that even groups of close relatives can foster significant internal conflict, and so there is still often scope for very costly competition within groups (Frank 1995, 1996b, 2003). Repression of competition is when there is a mechanism that reduces competition among the members of a group (Frank 2003). In contrast to kin-selected self-restraint, complete repression of competition is predicted to lead to harmonious groups because the individual can pursue the maximization of its inclusive fitness only by promoting the success of its group (Leigh 1971, 1977; Alexander 1987; Frank 2003; Gardner and Grafen 2009).

A potentially important means of repressing competition is by policing. The theory of policing has developed in two directions: (1) general theory intended to capture how mechanisms that enforce an equitable distribution of shared group resources may evolve (e.g. Frank 1995, 1996b, 2003, 2009); and (2) theory developed specifically for eusocial insects (Ratnieks *et al*. 2006; Ratnieks and Helanträ, 2009) such as honeybees where egg-laying workers are policed (their eggs are eaten) (Ratnieks 1988; Ratnieks and Visscher 1989). This work considers how mechanisms of kin-discrimination can lead to policing in haplodiploid societies. Here, we are concerned with the former approach, which aims for an illustrative overview of how the shared benefits of cooperation can drive the evolution of policing mechanisms to limit within-group conflict. Such policing is found at all scales of biological complexity: for example, intragenomic conflict is limited by fair meiosis (Leigh 1977); uncooperative symbionts

can face sanctions from their host, such as in the legume-rhizobia mutualism (West *et al*. 2002b; Kiers *et al*. 2003; 2006b) and 'reproductive leveling' in human tribal groups is enforced via strict food sharing norms (Bowles, 2006). Such general models highlight how comparable evolutionary mechanisms may result in cooperation evolving in very different contexts.

We develop the general theory of 'group-benefits' driven policing (Frank 1995, 1996b), in three ways. First, we relax an implicit assumption in the original model that the individual cost of investment into policing is lower in more selfish groups. Second, we relax the assumption that the group cost of selfishness is totally recovered by policing. Third, we consider policing in a demographic context: the population processes that allow relatedness to build up (e.g. low dispersal) can often lead to intensified competition between relatives (Taylor, 1992a; Queller 1992; West *et al*. 2002a), but this has been neglected in previous models of policing. We consider the general impact of increased kin competition on the evolution of group-benefits driven policing, and provide an illustration using Wright's (1931) infinite island model.

3.2 Models and Analyses

3.2.1 Frank's model of selfishness and policing

In this section, we introduce and re-derive the results of the policing model developed by Frank (1995, 1996b, 2003, 2009), which forms the basis of our subsequent analyses. Frank (1995, 1996b) considered the evolution of a policing behaviour that represses competition and limits the detrimental impact of within-group competition on the level of group resources, assuming that the fitness w of an individual is given by:

$$w \propto \left(a' + (1-a')\frac{z}{z'} - ca \right)\left(1-(1-a')z'\right), \tag{1}$$

where: a is the individual's investment into group policing; a' is the average level of policing by members of the group; z is the individual's investment into selfishness; z' is the group average level of selfishness; and c scales the individual cost of investment into policing (a summary of all notation is given in Table 3.1). Fitness is given by the individual's relative share (first multiplicative term) of total group resources (second multiplicative term). The individual's relative share of group resources is given by 1 in all policed encounters, and by her relative selfishness z/z' in all unpoliced encounters. The total group resources are reduced as unpoliced encounters and selfishness increase within the group. (A variant of the model considered by Frank (1996b), implements a personal cost of investment into policing alternatively as a third multiplicative fitness component, which does not qualitatively affect results.) Importantly, Frank's (1995) model assumes that investment into policing incurs a reduction in the individual's *share* of the group resources, and hence the absolute cost of policing is lower when the group has fewer resources (i.e. it is cheaper to police a more selfish group). We relax this assumption in the next section.

In the absence of policing ($a=a'=0$), the model describes a basic tragedy of the commons scenario (Frank 1996a, 2009, 2010), where fitness is given by:

$$w \propto \frac{z}{z'}(1-z'), \tag{2}$$

where an individual's share of group resources increases with its relative investment in selfishness (z/z'), which favours the evolution of increased selfishness, and the tragedy occurs because a more selfish group has reduced resources ($1-z'$). The model shows that one way the tragedy can be averted is when there is within-group

relatedness, which favours some degree of self-restraint. If within-group relatedness is r, then the evolutionarily stable (Maynard Smith and Price 1973) level of selfishness is $z^*=1-r$ (Frank 1995, 1996b).

Table 3.1. A summary of symbols used in the analysis

Symbol	*Definition*
a	policing behaviour
z	selfishness / competitiveness
c	cost of policing
b	efficiency of policing at restoring group resources
r	coefficient of relatedness ("whole-group" relatedness)
w	Darwinian fitness
R	"others-only" relatedness
B	fitness benefit to the recipient of a social behaviour

Symbol	*Definition*
C	fitness cost to actor of a social behaviour
n	number of breeding spaces per group (group size)
F	fecundity of an individual
F'	mean fecundity of group
$\overline{F}$	mean fecundity of population
s	scale of competition
m	dispersal rate
K	number of offspring

Frank (1995) found that relatively high relatedness disfavours policing ($a^* = 0$ when $r>1-c$; Figure 3.1a). This is because kin-selected self-restraint can maintain relatively high cooperation in high-relatedness groups, so that the benefits of policing do not outweigh its costs. Note, however, that even in groups with considerable relatedness, there is scope for significant selfishness. For example, in a large group of full siblings, relatedness is approximately one half, so the ESS level of selfishness is also equal to one half, and hence the group's fitness is only half as great as it could be if selfishness were completely abolished. Conversely, a relatively low within-group relatedness favours full investment into policing ($a^* = 1$ when $r<1-c$; Figure 3.1a). A further result, not emphasized by Frank (1995), is that policing results in increased selfishness $z^* > 1-r$; Figure 3.1b). Hence, Frank's model of repression of competition

by policing works by recovering the group cost of selfishness and not by reducing selfishness itself.

Frank (1995, 1996b) considered the evolutionary dynamics and statics of policing and selfishness, but did not show how policing fits into the standard classification of social behaviours provided by Hamilton (1964b; Table 1.1). The classification depends on the others-only relatedness of group mates (R; the expected relatedness of two individuals sampled at random from the same group without replacement; Pepper 2000). Details of how the classification is determined are given in Appendix 3.4. We find that:

Result 1 – In Frank's (1995) model, policing may be altruistic or mutually beneficial. Full policing is altruistic when others-only relatedness is high ($R > [n(1-c)-1]/[(n-1)(1+cn)]$) and mutually beneficial when others-only relatedness is low ($R < [n(1-c)-1]/[(n-1)(1+cn)]$).

3.1*a* 3.1*b*

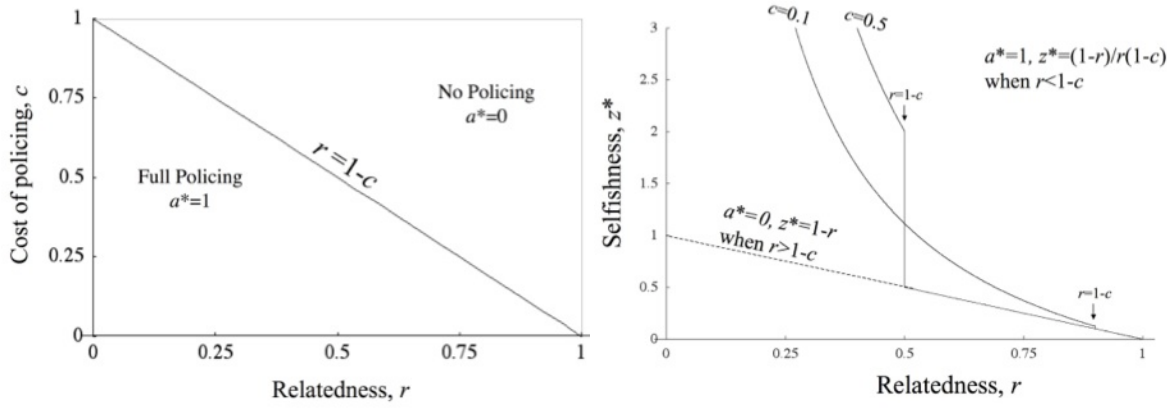


Figure 3.1 Frank's model of policing and selfishness In Frank's policing model (Frank 1995; Figure 3.1*a*) full policing (a^*=1), which results in a complete repression of within-group competition and a fair distribution of group resources, is predicted to invade whenever relatedness is low (r<1-c). When relatedness is high (r>1-c), no policing occurs (a^*= 0) and the model reduces to the tragedy of the commons and selfishness (Figure 3.1*b*) is limited only by kin-selected self-restraint (z^*=1-r). Policing always favours increased selfishness (dz^*/da>0, $z^*=(1-r)/r(1-c)$).

3.2.2 The individual cost of policing

Owing to multiplicativity of terms, Frank's (1995, 1996b) model of policing implicitly assumes that the magnitude of the individual cost of investment into policing increases with group resources (i.e. $\partial w/\partial a = -c(1-(1-a')z')$, where $1-(1-a')z'$ is the total group resources). In other words, policing gets cheaper to maintain as groups become more selfish. However, it is difficult to think of a biological scenario where this would be the case. In this section, we assume instead that policing requires a fixed investment of time or energy. The relationship between the cost of policing and selfishness will depend on the biological details; our aim here is to illustrate the importance of such details and provide a base upon which more complex models, that consider specific biological cases, may be developed.

We make the marginal cost of policing a fixed proportion of group fitness ($\partial w/\partial a = -c$), which we implement using a modified fitness function:

$$w \propto \left(a' + (1-a')\frac{z}{z'}\right)\left(1-(1-a')z'\right) - ca. \qquad (3)$$

In Appendix 3.4, we show that this leads to the following predictions:

Result 2 – Full policing ($a^*=1$) is only favoured at intermediate relatedness (specifically, when $r(1-r)>c$; Figure 3.2a). At the extremes of relatedness ($r(1-r)<c$), policing cannot evolve and the model recovers the tragedy of the commons result ($a^*=0$ and $z^*=1-r$). This is in contrast to Frank's (1995, 1996b) model, in which policing is favoured at arbitrarily low relatedness.

Result 3 – When there is full policing, selfishness increases ($z^*>1-r$), but tends to a lower level than in Frank's (1995, 1996b) model ($z^* = (1-r)/r$; Figure 3.1b).

3.2.3 The group cost of selfishness

Frank's (1995, 1996b, 2003) models assumed that policing fully recovers the loss of group fitness due to selfishness. This seems appropriate if policing removes the opportunity for individuals to behave selfishly (as in fair meiosis; (Leigh 1977). However, if policing simply works to remove the fitness benefits of selfishness, without directly suppressing selfishness itself (as in the eating of worker-laid eggs in honeybees, which does not directly prevent worker egg-laying but removes the benefits of doing this; Ratnieks *et al.* 2006), then we would expect selfishness to continue to incur some group-level cost even in the presence of full policing. We now develop the model described in the previous section to allow for a less than perfect efficiency of policing, with only a proportion b of the group fitness lost to selfishness being recovered by policing. In particular, we write:

$$w \propto \left(a' + (1-a')\frac{z}{z'}\right)\left(1-(1-ba')z'\right) - ca \,. \qquad (4)$$

We find that this adjustment impacts upon the model behaviour in several ways (see Appendix 3.4 for details):

Result 4 – Full policing is never stable ($a^* < 1$; a full expression for a^* is given in Appendix 3.4). This is in contrast to Frank's (1995, 1996b) model, in which full policing may be stable ($a^* = 1$), and an intermediate level of policing is never stable.

Result 5 – Reduced efficiency of policing (lower b) reduces the scope for policing to invade ($a^* > 0$ only if $r(1\text{-}r) > c/b$, see Appendix 3.4 for full expression of a^*) and leads to a lower ESS level of policing when it does invade ($\mathrm{d}a^*/\mathrm{d}b > 0$; Figure 3.2a).

Result 6 – Policing is more easily maintained by selection than it initially evolves. The condition for invasion of policing (instability of $a = 0$) is more stringent that the condition for an internal stable equilibrium to exist ($0 < a^* < 1$; Figure 3.4).

Result 7 – Policing promotes self-restraint ($z^*<1-r$; Figure 3.2b). When there is policing, selfishness is $z^* = c/rb$ which, in the region where policing can invade from rarity ($r(1-r) > c/b$, is lower than that which is obtained in the absence of policing ($z^* = 1-r$)). This is in contrast to Frank's (1995, 1996b) model, where policing always favours increased selfishness ($z^*>1-r$; Figure 3.1b)

Result 8 – Policing is an altruistic behaviour. This is in contrast to Frank's (1995, 1996b) model, in which policing could be either altruistic or mutually beneficial (see Result 1).

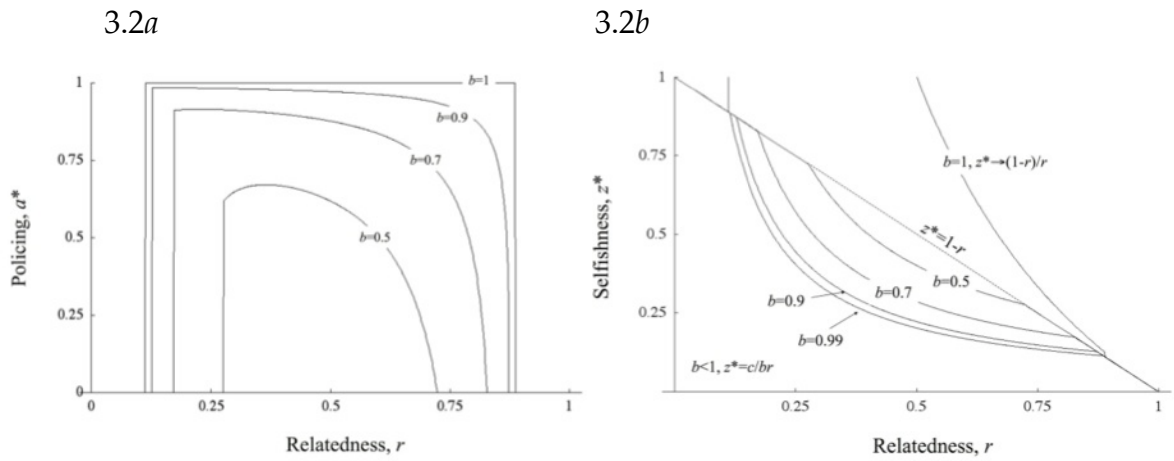


Figure 3.2 Modified individual and group cost models. In Frank's model, the cost of policing was proportional to the value of group resources so high levels of selfishness were favoured as they allowed vanishingly cheap policing. When the cost is adjusted to be proportional only to the level of policing an individual performs (i.e. $\partial w/\partial a = -c$, we assume here c=0.1; Figure 3.2a) policing is favoured to invade only at intermediate relatedness (when $r(1-r)>c/b$). If the efficiency with which a policing behaviour restores group resources is imperfect ($b<1$) intermediate levels of policing are favoured (Figure 3.2a) and when it invades, it always promotes self-restraint ($dz^*/da<0$, $z^*=c/br$; Figure 3.2b). This is in contrast to perfect policing (b=1, a=1) which favours increased selfishness ($z^*\rightarrow(1-r)/r$; Figure 3.2b). When policing is not stable (a^*=0 is stable when $r(1-r)<c/b$), the model reduces to the tragedy of the commons (z^*=1-

r). As the efficiency of a policing behaviour at restoring group resources b is reduced, the ESS level of policing falls ($da^*/db<0$; see Appendix 3.4).

3.2.4 Demography and policing – illustrative overview

Frank's (1995, 1996b) models consider the impact of whole-group relatedness (r) upon the coevolution of policing and cooperation, and our developments of this basic model reveal that an intermediate level of relatedness is crucial for policing to evolve. However, the ecological and demographic processes that lead to relatedness within groups can often also lead to intensified competition between relatives (reviewed by Queller 1992; West *et al*. 2002a), which has not been explicitly considered in Frank's models. For example if individuals do not disperse far from where they were born (population viscosity, Hamilton 1964b), then relatives must compete more intensely for space and resources.

We extend the model developed in the previous section, by making explicit the impact of policing and selfishness upon fecundity, and by making explicit the impact of personal fecundity and the fecundity of competitors upon Darwinian fitness. We employ a "scale of competition parameter" (s; Frank, 1998), which measures the extent to which an increase in an individual's fecundity leads to intensified competition within its social group. This approach has been useful for both theoretical (West *et al*. 2002a; West and Buckling 2003; Gardner and West 2004b, 2006; Gardner *et al*. 2009; Lively 2009; Gardner, 2010) and empirical (West *et al*. 2001; Griffin *et al*. 2004; West *et al*. 2006; Kümmerli *et al*. 2009) studies of social evolution. Following the approach taken by those earlier studies, we define the fitness of an individual as:

$$w = \frac{F}{sF' + (1-s)\overline{F}}, \tag{5}$$

where: F is the relative fecundity of the individual, and is given by

$$F = \left(a' + (1-a')\frac{z}{z'}\right)\left(1-(1-ba')z'\right) - ca\,; \tag{6}$$

F' is the average fecundity of the individuals in its social group, and is given by

$$F' = 1-(1-ba')z' - ca'\,; \tag{7}$$

and $\bar{F}$ is the average fecundity of individuals over the whole population, and is given by

$$\bar{F} = 1-(1-b\bar{a})\bar{z} - c\bar{a}\,. \tag{8}$$

Thus the fitness of an individual is determined by their fecundity (F) relative to the fecundity of their competitors, a proportion s of which are locals (group mates, who have average fecundity F') and a proportion 1-s of which are random individuals drawn from the population as a whole (having average fecundity $\bar{F}$). In Appendix 3.4, we derive the following results:

Result 9 – Local competition reduces the scope for policing to invade ($a^*>0$ when $r(1-r)[(1-s)/(1-rs)^2]>c/b$, which is increasingly stringent as s increases; for a^*, see Appendix 3.4) . Increased local competition both narrows the range of relatedness values that favour the invasion of policing ($d[r_{max}-r_{min}]/ds<0$ where r_{max} and r_{min} are the maximum and minimum relatedness permitting policing to invade from rarity) and shifts this range to higher levels of relatedness ($dr_{max}/ds>0$, $dr_{min}/ds>0$; Figure 3.3a). When competition is entirely local (s=1) policing is never favoured. Policing is still maintained more readily than it invades (Figure 3.4).

Result 10 – Increased local competition leads to higher selfishness ($dz^*/ds > 0$). In the absence of policing $z^*=(1-r)/(1-rs)$ which rises from $z^*=1-r$ when s=0 to z^*=1 when

s=1. As before, policing promotes self-restraint ($z^*=c(1-rs)/br(1-s)$) in the region where it can invade from rarity (when $r(1-r)[(1-s)/(1-rs)^2]>c/b$).

Queller (1994) suggested that the effects of local competition can be easily accounted for by redefining relatedness so that it measures genetic similarity of two individuals with respect to their "economic neighbourhood" (the arena in which they compete) rather than the with respect to the population as a whole. The condition for policing $r(1-r)[(1-s)/(1-rs)^2]>c/b$ may be re-written as $\rho(1-\rho) > c/b$ by substituting whole-group relatedness, r with Queller's relatedness, ρ where $\rho=(r-sr)/(1-sr)$ (see also Gardner and West 2006). Queller's relatedness captures both the genetic structure of a population (relatedness, r) and the effect of changing the scale of competition (s), with Queller's relatedness being an increasing function of the former ($\partial\rho/\partial r > 0$) and a decreasing function of the latter ($\partial\rho/\partial s < 0$). Results 9 and 10 may be derived from the basic model results (Results 2, 4-7) by substituting "r" with "ρ". If competition is entirely local, regardless of the genetic relatedness, there is no incentive to perform cooperative behaviours as all group members are valued equally (i.e. when s=1, ρ=0). This is why the effect of kin-selected self-restraint on selfishness disappears as competition becomes localised which then confines policing to higher levels of relatedness.

3.3*a* 3.3*b*

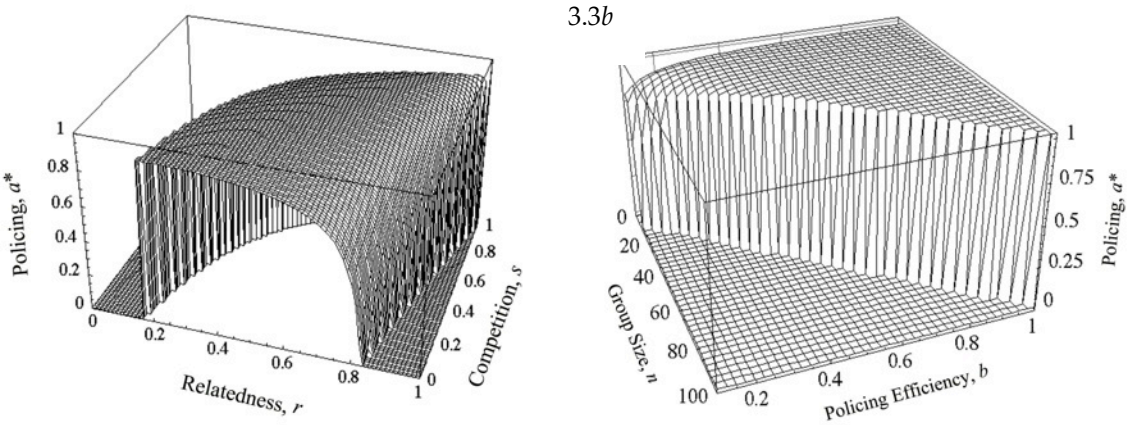


Figure 3.3 Policing and Demography. Increased local competition reduces the scope for policing ($a^*>0$ when $r(1-r)[(1-s)/(1-rs)^2]>c/b$). In the open model (Figure 3.3*a*, where we assume c=0.1, b=0.7), as the scale of competition (s) increases, policing is restricted to higher, narrower levels of relatedness. When s=0, policing is equal to the line for b=0.7 on Figure 3.2*a*. In the Island model (Figure 3.3*b*, where we assume c=0.1), which considers a population subdivided into groups of size n with migration occurring between them, we find that policing invades less readily in larger groups ($a^*>0$ when $(n-1)/n^2>c/b$).

3.2.5 Demography and policing - island model

In the previous section we provided an illustrative overview of how intensified kin competition impacts upon the coevolution of policing and cooperation. This revealed that, all else being equal, increased competition reduces the scope for policing and self-restraint to evolve. In particular, we examined the impact of increased kin competition (s) under the assumption of fixed relatedness (r). However, the same demographic processes may determine the degree of relatedness and kin competition, so it can be misleading to consider the effects of changing the value of either of these quantities in isolation. Here, we explicitly consider the co-dependency of relatedness and kin competition in a specific infinite island model setting (Wright 1931). The model assumes that an infinite population of asexual haploid individuals compete for resources in groups of size n. The relative fecundity of an individual is determined by its success in social interactions within the group (as in Equations 6-8). Individuals then have a very large number of offspring (K) proportional to their

fecundity, of which a proportion m migrate to randomly chosen groups and a proportion 1-m remain in the natal group. We assume that parents die at each generation and that dispersing individuals will not find relatives in their new group. In the next generation, native and immigrant individuals compete in every group for the n breeding spots. The fitness of an individual is determined by its success relative to others in its group. In Appendix 3.4, we show that:

Result 11 –The migration rate m has no net effect on the evolution of policing ($da^*/dm = dz^*/dm = 0$). Decreasing the migration rate both increases relatedness ($r=1/(n-(n-1)(1-m)^2$, so $dr/dm<0$) and also increases local competition ($s=(1-m)^2$, $ds/dm<0$). However these effects have no net impact on Queller's relatedness ρ, which is the relatedness of an individual to its social partners relative to its competitors, (substituting the island model values for r and s into ρ gives an effective relatedness of $\rho=1/n$ which is not a function of m).

Result 12 – Increasing group size disfavours the invasion of policing (policing can increase from rarity when $(n-1)/n^2>c/b$, in large groups this is approximately when $b/c>n$, for a^* see Appendix 3.4; Figure 3.3b). The larger the group, the lower the whole-group relatedness (which falls from $r=1$ when the group size $n=1$ to $r\rightarrow 0$ as $n\rightarrow\infty$) and the higher the selfishness. Policing is maintained in larger groups more readily than it invades in them (Figure 3.4). In the absence of policing, selfishness is stable at $z^*=(n-1)/n$ and as before, in the region where policing can invade, it always promotes self-restraint, given by $z^*=cn/b$.

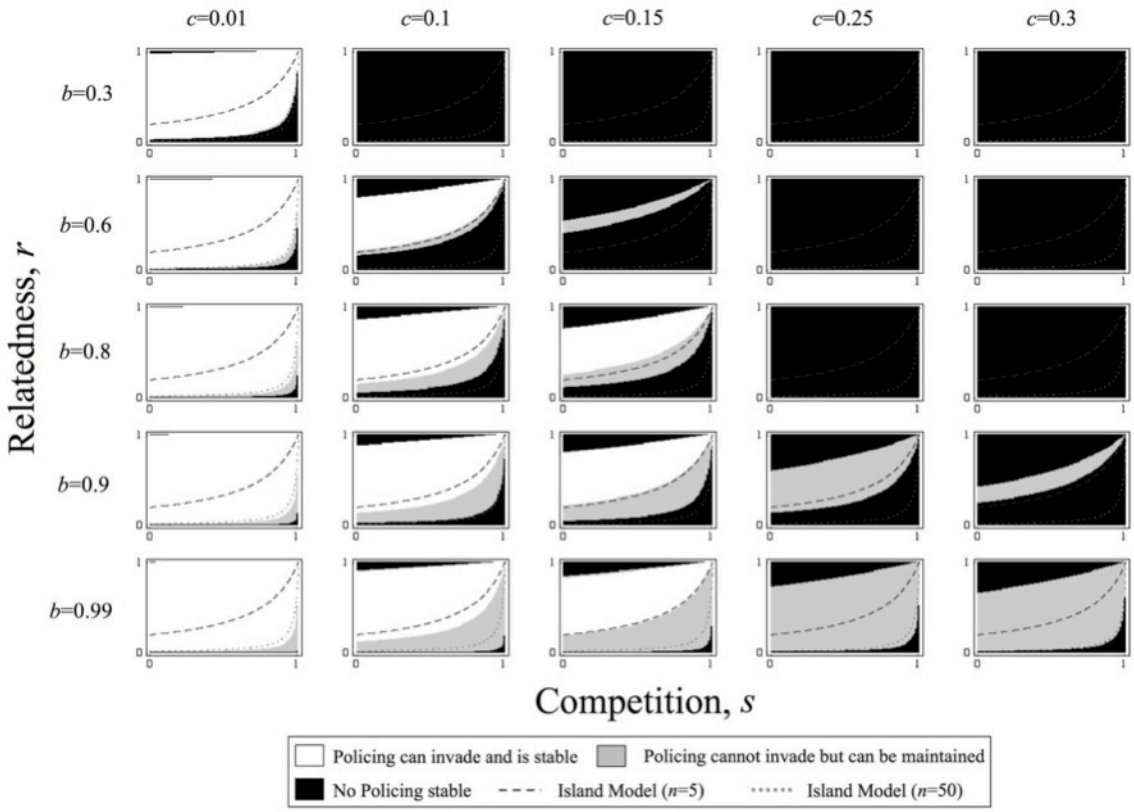


Figure 3.4 Policing and Demography - Stability Analysis. A numerical investigation (following the methodology of Otto and Day (2007, pp 237-242)) shows policing is always stable when it invades and that policing is maintained outside the region defined by the invasion condition. If the ESS for policing is stable, (the real part of its associated eigenvalue is negative), we plot a white or grey point; white denotes when only policing is stable (no policing is unstable if the marginal fitness for policing, dw/da is positive when $a^*=0$, which is when $r(1-r)[(1-s)/(1-rs)^2]>c/b$) and grey denotes when both policing and no policing are stable i.e. policing cannot invade but it would be stable if already present. If only no policing is stable (the real part of the ESS's associated eigenvalue is positive and $r(1-r)[(1-s)/(1-rs)^2]<c/b$), we plot a black point. The Island model stability results can be calculated from the open model results as $s=(1-m)^2$ and $r=1/[n-(n-1)s]$. They highlight that once present, policing could be maintained in much larger groups (plotted for $n=5$ and $n=50$).

3.3 Discussion

Group-benefits driven policing – enforcement of fairness in the distribution of resources among group mates – has been regarded as a potentially important mechanism for the evolution of cooperation and the major transitions in evolution

(Leigh 1977; Alexander 1987; Frank 2003; Gardner and Grafen 2009). In this paper we have examined Frank's (1995, 1996b) model of policing, which was developed to provide a general overview of group benefits driven policing theory. The basic predictions of his model was that policing evolves most readily when within-group relatedness is low, that this leads to an increase in selfishness and, when it does evolve, it will completely abolish competition among group mates (Figure 3.1). However, his model assumes that the individual cost of investment into policing vanishes as group selfishness increases, and also that policing totally recovers any loss of group fitness due to selfishness – even though individuals continue to invest in selfishness. We have examined the consequences of relaxing these assumptions and find these basic results are changed substantially. Specifically we find that policing does not evolve when relatedness is low (Figure 3.2a), favours a reduction in individual selfishness (Figure 3.2b), cannot repress all within-group conflict (i.e. $a^*<1$; Figure 3.2a), is easier to maintain at lower relatedness than to initially evolve (Figure 3.4), and is altruistic.

We also examined the consequences of placing group-benefits policing in its proper demographic context. We have shown that within-group relatedness is crucial for policing to be favoured, and yet the demographic processes that can generate within-group relatedness may also lead to intensified kin competition, which has not been considered explicitly in previous models. We found that, in general, an increased competition for resources within groups leads to a reduction in policing. Further, in the context of Wright's (1931) infinite island model, we found that the relatedness-inflating effects of low migration exactly cancel the associated increase in local competition, so the evolution of policing is solely dependent upon group size, with larger groups disfavouring policing (Figure 3.3b). In short, all of our model developments reduce the scope for policing to invade from rarity. However our

results also show that policing is easier to maintain than it is to evolve, specifically that it is stable at lower relatedness (Figure 3.4). This result mirrors previous work on the evolution of punishment and cooperation which showed that punishment was maintained more readily than it evolved (Gardner and West 2004a).

We found that group-benefits policing requires intermediate relatedness within groups in order to be favoured by natural selection. We emphasize that this refers to the 'whole-group' relatedness, which includes the relatedness of the individual to itself, and not the 'others-only' relatedness, which is the relatedness between social partners (Pepper 2000). Thus, the model could be applied to study policing in interspecific mutualisms, where a host individual is not genetically related to its symbiont(s), but nevertheless could reap the benefits of policing its social partners (mediated through non-zero whole-group relatedness). Our model predicts policing evolves most readily when whole-group relatedness is one half, which is obtained when unrelated partners are in a 1:1 relationship or when one individual forms a symbiosis with a clonal group. However, our model is not intended to fully capture the biology of mutualisms. Importantly, it treats all individuals as equivalent, incurring the same costs and benefits for the same investments into policing and selfishness, whereas real-world mutualisms bring non-equivalent individuals from different species together in a complementary relationship. Development of this basic policing model to explicitly incorporate the biology particular to mutualisms represents an important direction for future research (Murray 1985; West *et al* 2002a,b; Yu *et al* 2004; Foster and Wenseleers 2006).

Group-benefits driven policing may have been important in human evolution (Bowles 2006, 2009). As policing is maintained more readily than it invades, it could have evolved at a time when humans lived in small tightly-knit groups and then been retained as much larger societies developed, particularly if social norms

allowed the cost of policing to be reduced. To investigate the evolution of social behaviours in humans, the effect of both population structure and cultural transmission (if they are culturally inherited) on their evolution must be understood. Whilst the importance of cultural transmission in enhancing/limiting selection for cooperation has received considerable attention (Boyd and Richerson 1985; Henrich and Boyd 1998; Henrich and Gil-White 2001; Boyd and Richerson 2005a; Lehmann and Feldman 2008a; Lehmann *et al*. 2008), the importance of population structure has not. For example, budding dispersal, where groups migrate to new territories (the 'tribe-splitting' model of Haldane 1932) may be a better description of human population movements than the basic island model used here (which assumes individuals disperse independently). Both theoretical and empirical work (Gardner and West 2006; Lehmann *et al*. 2006; Kümmerli *et al*. 2009) show that budding dispersal favours the evolution of cooperation. This illustrates the importance of considering whether models capture the details of human population structure when investigating the evolution of behaviours such as policing. Extending the island model (Wright 1931; Taylor 1992a) to study policing in a demographic context which more accurately reflects the structure of human Palaeolithic society is an important task for the future.

In this paper, we have focused upon models of policing that work owing to group benefits (following Leigh 1977; Alexander 1987; Frank 2003). However, group benefits are not necessary for policing to be favoured and for policing to favour cooperation. Perhaps the best understood policing system, from a theoretical and empirical standpoint, is that of honeybees and other hymenoptera. Here, workers prevent the rearing of eggs laid by other workers by eating them upon discovery, and this has been shown to reduce the extent to which workers do lay eggs (Wenseleers *et al*. 2004a, b). The selective force favouring policing in such cases is the nepotistic

interests of the egg-eating workers, who value queen-laid unfertilized eggs above worker-laid unfertilized eggs, because they are more genetically related to the former than to the latter (Ratnieks 1988; Ratnieks and Visscher 1989; Wenseleers *et al* 2005; Wenseleers and Ratnieks 2006; Ratnieks and Helanterä 2009). Consequently, this egg-eating behaviour can evolve even when it does not provide a colony-level benefit (Wenseleers *et al*. 2004a; Wenseleers *et al*. 2004b). However, such policing can also provide colony level benefits, which can maintain policing even in cases where there are not relatedness asymmetries (Martin *et al* 2002).

How do our results relate to our understanding of the major evolutionary transitions? A major transition can occur when individuality shifts from a lower to higher level (Buss 1987; Maynard Smith and Szathmary 1995; Queller 2000). For such transitions to be possible, internal conflict must be resolved (Frank 2003). This can occur owing to clonality of individuals within social groups, or if there is a total repression of within-group competition (Buss 1987; Maynard Smith and Szathmary 1995; Gardner and Grafen 2009). Policing has been suggested as a potentially important factor in driving major transitions (Frank 1995, 1996b, 2003; Ratnieks and Helanterä 2009). We suggest caution, as this may depend upon whether policing evolves via group-benefits or kin discrimination, and as the extent to which individuals can discover other ways to pursue their selfish interests. This suggestion is consistent with recent work on social insects which suggests lifetime monogamy (which leads to high within-colony relatedness) was an essential step in the evolution of eusociality (Boomsma 2007, 2009; Hughes *et al*. 2008). It appears that worker policing was only important after eusociality became irreversible as a means to limit within-colony reproductive conflicts that emerged as queens began multiply mating (Ratnieks *et al*. 2006; Ratnieks and Wenseleers 2008). More generally, this emphasises

how the origin versus the maintenance evolution of cooperation behaviours can involve different selective forces (West *et al*. 2007a).

To conclude, we have shown that policing mechanisms that enforce the fair sharing of group resources are harder to initially evolve than previously thought. We show that contrary to previous suggestions, policing is unlikely to be favoured at low relatedness and that, when it evolves, it often favours self-restraint. Furthermore policing is unlikely to result in a complete repression of within-group competition. This suggests group-benefits driven policing will be of limited importance in major transitions. Major tasks for the future include the study of policing in different demographies, particularly with reference to humans and understanding the role of policing in intra-specific mutualisms.

3.4 Appendix

3.4.1 Frank's model of selfishness and policing

Here, we analyse the dynamics and statics of Frank's (1995) model of the coevolution of selfishness and policing, deriving the results stated in section (a) of the main text. In the context of this model, the average fitness of an individual is given by equation (1) of the main text. The direction of selection acting upon any trait is given by the marginal fitness with respect to that trait, i.e. $\mathrm{d}w/\mathrm{d}z$ and $\mathrm{d}w/\mathrm{d}a$ for selfishness and policing, respectively. These derivatives may be separated into partial derivatives, describing the effect of an individual's trait value, and the effect of the average trait value of its group, on this individual's fitness, using the chain rule methodology of Taylor and Frank (1996; see also Frank 1995, 1996b, 2003; Taylor 1996a; Taylor *et al*. 2007). This gives $\mathrm{d}w/\mathrm{d}z = \partial w/\partial z + r\,\partial w/\partial z'$ and $\mathrm{d}w/\mathrm{d}a = \partial w/\partial a + r\,\partial w/\partial a'$, where r

$= dz'/dz = da'/da$ is the kin-selection coefficient of (whole-group) relatedness (Taylor and Frank 1996; Pepper 2000), or:

$$\frac{dw}{dz} = (1-a)\left(\frac{(1-r)(1-(1-a)z)}{z} - r(1-ca)\right), \quad \text{(A1)}$$

and:

$$\frac{dw}{da} = rz(1-ca) - c(1-(1-a)z). \quad \text{(A2)}$$

First, we consider the evolution of selfishness in the absence of policing ($a = 0$). Evaluating the RHS of equation (A1) at $a = 0$ and $z \to +0$ gives $+\infty$, which is positive and hence selfishness is always favoured in the absence of policing. The ESS level of selfishness z^* is determined by setting the RHS of equation (A1) to zero and solving for z; in the absence of policing ($a = 0$), this gives $z^* = 1-r$. More generally, holding the level of policing fixed at a, the ESS level of selfishness is given by $z^*=(1-r)/[1-a(1-r(1-c))]$. When there is full policing ($a^*=1$) selfishness is neutral ($dw/dz=0$), but in the limit of full policing ($a \to 1$), the ESS level of selfishness is $z^* \to (1-r)/(1-c)r$. This is stable as any reduction in selfishness ($z=[(1-r)/(1-c)r]-\delta$, where δ is a vanishingly small quantity), favours an increase in policing ($dw/da>0$).

Next, we determine when policing can evolve, by evaluating the RHS of equation (A2) at $a = 0$ and $z = 1-r$. This gives $dw/da=r(1-r)-cr$, and hence the condition for policing to invade is $r<1-c$. Assuming that policing does evolve, we determine the evolutionary stability of full policing ($a = 1$) by evaluating the RHS of equation (A2) at $a = 1$ and $z =(1-r)/(1-c)r$, which obtains $dw/da=1-c-r$. Hence, full policing is evolutionarily stable ($a^* = 1$) if $r < 1-c$. Setting the RHS of both equations (A1) and (A2) to zero, and simultaneously solving for a and z, yields no biologically feasible

solutions; hence, an intermediate level of policing ($0 < a^* < 1$) is never evolutionarily stable. This means that when $r>1-c$, policing cannot evolve ($a^* = 0$), whereas when $r<1-c$, then policing can evolve, and the level of policing increases until full policing is established ($a^* = 1$). Policing favours an increase in selfishness: differentiating the ESS level of selfishness $z^*=(1-r)/[1-a(1-r(1-c))]$ with respect to a yields a positive slope ($dz^*/da=[(1-r)(1-(1-c)r)]/[1-a(1-(1-c)r)]^2>0$) when $r<1/(1-c)$ which is always met as $1/(1-c)>1$.

To investigate whether policing evolves due to direct or indirect fitness benefits, we must rearrange the marginal fitness for policing to be in the form of Hamilton's rule. Above, we expressed the marginal fitness for policing as $dw/da= dw/da+r(dw/da')$, where r is the whole-group relatedness (relatedness to average group member, including onself; Pepper 2000). We now re-write in terms of others-only relatedness R (relatedness to average group member, not including self; Pepper 2000), which is the relatedness term that appears in a formal statement of Hamilton's rule. Substituting in the expression $r=1/n + [(n-1)/n]R$, we may rearrange equation (A2) into the form $RB-C$, where $-C$ is the direct fitness effect and RB is the indirect fitness effect and their sum is the inclusive fitness effect (Hamilton 1963, 1964b). Hence, we have $B=[(n-1)/n](dw/da')$ and $-C=dw/da+[(1/n)(dw/da')]$. For Frank's model, this obtains $B=[(n-1)/n](z(1-ac))$ and $C=c(1-(1-a)z)-[(1/n)(z(1-ac))]$. We evaluate B and C at the ESS $a=a^*=1$ and $z=z^*=(1-r)/(1-c)r$, which gives $B = [(n-1)(n(1-c)-1)(1-R)]/[(1-c)n(1+R(n-1))]$ and $C =c+1/n-1/(1+R(n-1))$. Social behaviours are classified according to the sign of B and C (Hamilton 1964; Table 1.1): we find that policing is altruistic ($B > 0$ and $C > 0$) if $R > [n(1-c)-1]/[(n-1)(1+cn)]$, and otherwise it is mutually beneficial ($B > 0$ and $C < 0$) – and this is Result 1 of the main text.

3.4.2 The individual and group cost of selfishness

Here, we determine the evolutionarily stable levels of policing and selfishness for our modified model of policing (sections (b) and (c) of the main text). Note that the model presented in section (c) of the main text is a generalization of the model presented in section (b); thus, the results of both sections can be derived from the model of section (c), with those pertaining to section (b) being given in the special case of $b = 1$. The average fitness of an individual is given by equation (4) of the main text. Marginal fitness for selfishness (z) and policing (a) are given by:

$$\frac{\mathrm{d}w}{\mathrm{d}z} = \frac{(1-r)(1-a)}{z} - (1-ab)\big(1-a(1-r)\big), \tag{A3}$$

and:

$$\frac{\mathrm{d}w}{\mathrm{d}a} = brz - c. \tag{A4}$$

We first consider selfishness. Holding the level of policing fixed at a, and setting the RHS of equation (A3) equal to zero and solving for z, we obtain an ESS level of selfishness of $z^*=[(1-a)(1-r)]/[(1-ab)(1-a(1-r))]$. If we assume there is no policing (set $a=0$), as in Frank's model, this gives selfishness as $z^*=1-r$. The marginal fitness for policing at this point is given by evaluating equation (A4) at $z = 1-r$, obtaining $\mathrm{d}w/\mathrm{d}a = br(1-r)-c$, and hence policing can invade only when $r(1-r)>c/b$ (Results 2 and 6 of the main text). For full policing ($a=1$) when $b=1$ we find that selfishness is neutral ($\mathrm{d}w/\mathrm{d}z=0$) at $a=1$. In the limit of full policing ($a \to 1$) selfishness tends to $z^* \to (1-r)/r$ (Result 2 and 3, main text). This ESS is stable as any reduction in selfishness ($z=(1-r)/r-\delta$ where δ is a vanishingly small quantity) favours an increase in policing ($\mathrm{d}w/\mathrm{d}a>0$). For full policing ($a=1$) when $b<1$ we find that $\mathrm{d}w/\mathrm{d}z=-(1-b)r$, so selfishness is disfavoured and will decline to zero and this obtains $\mathrm{d}w/\mathrm{d}a=-c$ which

is always negative. Hence for $b<1$, full policing is never stable (Result 4 of the main text). This means that when policing can invade from rarity ($r(1-r)>c/b$), the ESS level of policing must be intermediate ($0 < a^* < 1$). To find this explicitly, we set the RHS of equations (A3) and (A4) to zero and simultaneously solve $a=a^*$ and $z=z^*$. This yields two results, only one of which is a biologically feasible ESS at $z^*=c/rb$ and

$$a^* = \frac{bc + (c - br)(1 - r) + \sqrt{4bc(1 - r)(br(1 - r) - c) + (bc + (c - br)(1 - r))^2}}{2bc(1 - r)} \qquad \text{(A5)},$$

(Result 4 and 8 of the main text). The maintenance and stability of this ESS (Results 6 of the main text) is discussed in the next section. Using the approach outlined in the previous section, we may rearrange the RHS of equation (A4) into the form $RB-C$, where $B=bz(n-1)/n$ and $C=c-bz/n$. Evaluating this at the above ESS values of z^* and a^*, we find that $B > 0$ and $C > 0$, and hence policing is always altruistic in this variant model (Result 8 of the main text). Finally, we examine the impact of b – the efficiency with which a policing behaviour restores the group resources – upon the ESS level of policing.

Differentiating the RHS of equation (A5) with respect to b obtains:

$$\frac{da^*}{db} = \frac{bc - (c - br)(1 - r) - \sqrt{4bc(1 - r)(br(1 - r) - c) + (bc + (c - br)(1 - r))^2}}{2b^2\sqrt{4bc(1 - r)(br(1 - r) - c) + (bc + (c - br)(1 - r))^2}} \qquad \text{(A6)},$$

which is always positive, hence the ESS level of policing is stable at higher values as the efficiency of policing improves (see Figure 3.2b).

3.4.3 Demography and policing - Illustrative overview

Here, we consider the evolution of policing and selfishness in a demographic context, using the illustrative overview presented in section (d) of the main text where the

average fitness of an individual is given by equation (5). The marginal fitness for selfishness (z) and policing (a) are given by:

$$\frac{\mathrm{d}w}{\mathrm{d}z} = \frac{(1-a)(1-r)-(1-ab)\big(1-a(1-r)-rs\big)z}{z\big(1-ac-z(1-ab)\big)}, \tag{A7}$$

and:

$$\frac{\mathrm{d}w}{\mathrm{d}a} = \frac{(1-s)brz-c(1-rs)}{1+a(bz-c)-z}. \tag{A8}$$

First we consider the evolution of selfishness. The ESS level of selfishness (z^*) is found by setting the RHS of equation (A7) to zero and solving for z; in the absence of policing ($a = 0$), this gives $z^* = (1\text{-}r)/(1\text{-}rs)$. More generally, if the level of policing is held at a, the ESS level of selfishness is given by:

$$z^* = \frac{(1-r)(1-a)}{(1-ab)\big((1-a)(1-r)+(1-s)r\big)} \tag{A9}$$

We determine the evolutionary stability of policing by evaluating the RHS of equation (A8) at $a = 0$ and $z = (1\text{-}r)/(1\text{-}rs)$, which obtains an invasion condition for policing of $r(1\text{-}r)[(1\text{-}s)/(1\text{-}rs)^2]>c/b$. For full policing ($a$=1) we find that $\mathrm{d}w/\mathrm{d}z$=-[(1-b)r(1-s)]/[1-c-(1-b)z] which is zero for b=1 and negative for all b<1, so selfishness is neutral in the special case of fully efficient policing and is otherwise disfavoured and is expected to decline to zero. Evaluating equation (A8) at $z = 0$ and $a = 1$ yields –[c(1-rs)]/(1-c), which is always negative and hence full policing (a=1) is never stable. This means that when policing can invade from rarity ($r(1\text{-}r)[(1\text{-}s)/(1\text{-}rs)^2]>c/b$), the ESS level of policing must be intermediate ($0 < a^* < 1$). To find this ESS, we set both equations (A7) and (A8) equal to zero, and solve simultaneously for a and z at a=a^* and z= z^*. This yields a single biologically feasible result of z^*=c(1-rs)/br(1-s)) and a

rather complicated a^* (which can be recovered by replacing Queller's relatedness (r-sr)/(1-sr) for r in equation (A5)).

To show that policing is maintained more readily that it invades we evaluate the marginal fitness for policing (A8) at the ESS level of selfishness (A9). This yields:

$$\frac{\mathrm{d}w}{\mathrm{d}a} = \frac{br(1-a)(1-r)(1-s) - c(1-ab)(1-a(1-r)-rs)(1-rs)}{(1-ab)(ca^2(1-r)+r(1-s)-a(c(1-rs)))}. \quad \text{(A10)}$$

Evaluating equation (A10) at $a = 0$ and c=br(1-r)[(1-s)/(1-rs)^2] (the maximum cost of policing at which policing can invade), we find some policing is favoured (dw/da>0) when b>ρ (where ρ is Queller's relatedness (r-rs)/(1-rs)). As full policing $a = 1$ is never favoured (dw/da<0), this means an ESS must exist between 0<a<1 where dw/da=0 outside the region where policing can invade (Result 6 of the main text). We conduct a numerical investigation of the stability of this ESS using the methodology of Otto and Day (2007, pp 237-242; results in Figure 3.4) which confirms that when policing can invade, the ESS is always stable and that policing is maintained in regions where it cannot invade (particularly at lower relatedness and higher c/b ratios). When the ESS is stable, the eigenvalues are negative complex numbers indicating that policing and selfishness spiral in towards their ESS values.

To examine the effect of local competition s on the coevolution of selfishness and policing, the invasion condition r(1-r)[(1-s)/(1-rs)^2]>c/b is solved for r, to yield the maximum and minimum relatedness values that permit policing to invade from rarity. These bounds take the form r_{max}=X+Y and r_{min}=X-Y where:

$$X = \frac{1-s(1-2c/b)}{2(1-s(1-sc/b))}, \quad \text{(A11)}$$

and:

$$Y = \frac{(1-s)\sqrt{1-4c/b}}{2\left(1-s\left(1-sc/b\right)\right)}. \qquad \text{(A12)}$$

To know whether local competition favours or disfavours the evolution of policing, we first examine how an increase in local competition affects the range of relatedness values over which policing may invade. This range evolve is given by r_{max}-$r_{min} = 2Y$. Differentiating Y with respect to s yields

$$\frac{dY}{ds} = -\frac{s(c/b)(1-\tfrac{1}{2}s)(1-s)\sqrt{1-4c/b}}{(1-s)\left(1-s\left(1-s(c/b)\right)\right)^2} \qquad \text{(A13)},$$

which is negative when $c/b < ¼$, which is a necessary (but not sufficient) condition for the evolution of policing, hence the range of relatedness values over which policing invades decreases with increasingly local competition ($d(r_{max}\text{-}r_{min})/ds < 0$; Result 9 of the main text).

If increasing local competition leads to policing invading at higher levels of relatedness, then dr_{max}/ds and dr_{min}/ds are positive. For these to be positive, both dX/ds must be positive and dX/ds must be greater than dY/ds. Differentiating X with respect to s yields:

$$\frac{dX}{ds} = \frac{c/b\left(1-s+s^2\left(\tfrac{1}{2}-c/b\right)\right)}{\left(1-s\left(1-sc/b\right)\right)^2} \qquad \text{(A14)},$$

which is positive when $c/b>0$. Setting the RHS of equation (A13) equal to the RHS of (A14) gives the condition that $dX/ds > dY/ds$ when $c/b>0$. Hence as local competition increases, policing is favoured to invade at higher levels of relatedness (Result 8 of the main text).

To investigate the effect of increased local competition on selfishness we differentiate z^* (A9) with respect to s, which yields

$$\frac{\mathrm{d}z^*}{\mathrm{d}s} = \frac{r(1-r)(1-a)}{(1-ab)(1-a(1-r)-rs)^2} \qquad \text{(A15).}$$

This is positive for all $0 < r < 1$, which is a necessary (but not sufficient) condition for the evolution of policing which means selfishness increases as competition becomes more localised (Result 10 of the main text).

3.4.4 Demography and policing - Island Model

We now consider the evolution of policing in an island model setting (Wright 1931; Taylor 1992a), as described in the main text of section (e). We assume that the average fecundity F of a focal individual is given by equation (6), the average relative fecundity of all individuals in a group F' is given by equation (7) and the average relative fecundity of all individuals in the population $\bar{F}$ is given by equation (8) in the main text. The focal individual has KF offspring of which mKF disperse and (1-m)KF remain in the natal group. Each dispersing offspring arrives in a new group with (1-m) $\bar{F}nK$ natives and m $\bar{F}nK$ other immigrants. Therefore the chance that a particular dispersing offspring succeeds in obtaining one of the n breeding spaces is $n/[1+ (1-m)\ \bar{F}nK + m\ \bar{F}nK] \approx 1/\ \bar{F}K$ assuming K is very large. Therefore, the expected fitness of the focal individual that is obtained through its dispersing offspring is $mFK/\ \bar{F}K = mF/\ \bar{F}$. Turning now to the (1-$m$)$FK$ non-dispersing offspring of the focal individual, these find themselves competing for the n breeding spaces in the natal patch with (1-m)$F'K$ natives (including each other) and m $\bar{F}nK$ immigrants, therefore the total number of offspring is (1-m)$F'nK$+m $\bar{F}nK$. Hence the expected fitness of the focal individual that is obtained through its non-dispersing offspring is

$[(1\text{-}m)FnK]/[(1\text{-}m)F'nK+m\ \bar{F}nK] = [(1\text{-}m)F]/[(1\text{-}m)F'+m\ \bar{F}]$. Thus the total number of dispersing and non-dispersing offspring of a focal individual (which is equal to an individual's fitness) is given by:

$$w = m\frac{F}{\bar{F}} + \frac{F(1-m)}{(1-m)F' + m\bar{F}} \tag{A16}$$

Using equation (A16), we substitute the fecundity values given in section (d) to calculate the marginal fitnesses for selfishness (z) and policing (a) where:

$$\frac{\mathrm{d}w}{\mathrm{d}a} = \frac{brz\left(1-(1-m)^2\right) - c\left(1-r(1-m)^2\right)}{1+a(bz-c)-z} \tag{A17},$$

and

$$\frac{\mathrm{d}w}{\mathrm{d}z} = \frac{(1-a)(1-r) - (1-ab)\left(1-a(1-r)-r(1-m)^2\right)z}{z\left(1+a(bz-c)-z\right)} \tag{A18}.$$

Note that equations (A7) and (A8) recover equations (A17) and (A18), respectively, if we make the substitution $s = (1\text{-}m)^2$, i.e. in the context of the inelastic island model the scale of competition is equal to the probability that two randomly chosen group mates both fail to disperse (Gardner and West 2006). Relatedness r may also be expressed as a function of model parameters, by deriving a recursion for relatedness over successive generations and calculating its equilibrium value (Taylor 1992a). In generation t, the expected relatedness of an individual to a randomly chosen member of its group (including itself), is $r_t = 1/n + ((n\text{-}1)/n)R_t$ where $1/n$ is the probability of choosing itself (in which case relatedness is 1) and $(n\text{-}1)/n$ is the probability of choosing another individual (in which case relatedness is R_t, the "others-only" relatedness). The only way for two different individuals from the same group to be related is if neither of them dispersed (which occurs with a probability of $(1\text{-}m)^2$), in

which case their relatedness is equal to the whole-group relatedness of the previous generation (r_{t-1}). Hence, we may write the following recursion:

$$r_t = \frac{1}{n} + \frac{n-1}{n}(1-m)^2 r_{t-1} \qquad \text{(A19)},$$

which can be solved at equilibrium ($r_{t-1}=r_t=r$) to give an equilibrium relatedness of:

$$r = \frac{1}{n-(n-1)(1-m)^2} \qquad \text{(A20)},$$

as determined by Taylor (1992a). Substituting equation (A20) into equations (A17) and (A18) obtains:

$$\frac{\mathrm{d}w}{\mathrm{d}z} = \frac{\left(1-(1-m)^2\right)\left((1-a)(n-1)+(1-ab)\left(a(n-1)-n\right)z\right)}{z\left(1+a(bz-c)-z\right)\left(1+(n-1)\left(1-(1-m)^2\right)\right)} \qquad \text{(A21)},$$

and:

$$\frac{\mathrm{d}w}{\mathrm{d}a} = \frac{\left(1-(1-m)^2\right)(bz-cn)}{\left(1+a(bz-c)-z\right)\left(1+(n-1)\left(1-(1-m)^2\right)\right)} \qquad \text{(A22)}.$$

We now determine the evolutionarily stable levels of policing and selfishness. Considering selfishness first, we find the ESS level of selfishness (z^*) by setting the RHS of equation (A21) to zero and solving for z whilst holding the level of policing at a; this gives:

$$z^* = \frac{(1-a)(n-1)}{(1-ab)\left(a+n(1-a)\right)} \qquad \text{(A23)}.$$

In the absence of policing ($a = 0$), this is $z^* = (n\text{-}1)/n$. Evaluating equation (A22) at $z^* = (n\text{-}1)/n$, gives an invasion condition for the evolution of policing of $(n\text{-}1)/n^2 > c/b$

(Result 11 of the main text). To find the stable level of policing, we set both equations (A21) and (A22) equal to zero, and solve simultaneously for a and z at $a=a^*$ and $z=z^*$. This yields one biologically feasible solution of $z^*=(c/b)n$ and a complicated a^*, which can be recovered by substituting $\rho=1/n=r$ in equation (A5) (Result 12 of the main text). To investigate whether policing may be maintained in larger groups than it can invade, we evaluate the marginal fitness for policing (A22) at the ESS level of selfishness (A23), which yields:

$$\frac{\mathrm{d}w}{\mathrm{d}a}=\frac{m(m-2)\left(cn\left(a+n(1-a)\right)+b\left(1-n+cna^2(n-1)+a\left(n-1-cn^2\right)\right)\right)}{(1-ab)\left(1+2m(n-1)-m^2(n-1)\right)\left(1+ca^2(n-1)-acn\right)} \qquad \text{(A24)}$$

Evaluating equation (A24) at $a=0$ and $c=(b(n\text{-}1))/n^2$ (the maximum cost of policing at which policing can invade), we find policing is still favoured ($\mathrm{d}w/\mathrm{d}a>0$) when $b>1/n$ (which is equivalent to the open model result of $b>\rho$ as for the Island model, Queller's relatedness $\rho=1/n$). Full policing $a=1$ is not stable ($\mathrm{d}w/\mathrm{d}a<0$), so an ESS must occur between $0<a<1$ at the point where $\mathrm{d}w/\mathrm{d}a=0$ (Result 12 of the main text). We conduct a stability analysis for the Island model ESS using the methodology of Otto and Day (2007, pp 237-242). The results can be obtained from Figure 3.4 by substituting s for $(1\text{-}m)^2$ and r for $1/[n\text{-}(n\text{-}1)s]$.

To investigate how changing migration rates affect both relatedness and the intensity of local competition, we first differentiate relatedness ($r=1/(n\text{-}(n\text{-}1)(1\text{-}m)^2)$) with respect to m, which yields $\mathrm{d}r/\mathrm{d}m=-[2(1\text{-}m)(n\text{-}1)]/[1+(1\text{-}(1\text{-}m)^2)(n\text{-}1)]^2$. This is always negative, so increasing the migration rate decreases relatedness. Differentiating the scale of competition term, $s=(1\text{-}m)^2$, in terms of m gives $\mathrm{d}s/\mathrm{d}m=-2(1\text{-}m)$ which is also always negative meaning that increasing migration decreases local competition. Both these effects must cancel resulting in the migration rate m having no net effect on the

evolution of policing as a^* and z^* and ρ are not functions of m, so $da^*/dm = dz^*/dm = d\rho/dm = 0$ (Result 11, main text).

Chapter 4. Group competition and the evolution of cooperation in humans.

4.1 Introduction

Darwin (1871) was the first to suggest that hostility between groups could have played a key role in the evolution of cooperative social behaviours in humans. The presence of cooperation in human society poses a problem from an evolutionary perspective as, all else being equal, helping another individual reduces the relative fitness of the helper so natural selection should favour non-helpers over helpers (Hamilton 1964). Theoretical and empirical work suggests that conflict or war between groups may favour the evolution of cooperative behaviours, because the benefit of being in a winning cooperative group may outweigh the cost of cooperation (Choi and Bowles 2007, Bowles 2006; 2009; Lehmann and Feldman 2008). Theoretical model of group conflict have been parameterized with archaeological and ethnographic data, to estimate empirically the influence of conflict (Bowles 2006; 2009). However, it is not always clear exactly why cooperation is favoured in these models.

In this paper we ask whether conflict only favours pro-social traits (e.g. teamwork) or whether it can also favour anti-social traits (e.g. uncontrolled aggression). This latter case, that conflict between-groups may favour harmful traits, has not been examined. Furthermore, we examine to what extent predictions from existing models are applicable to all forms of social behaviour, as currently it is unclear whether conflict

favours any sort of social trait (e.g. food sharing), or just those directly related to conflict (e.g. helping to make spears for the group) (Lehmann and Feldmann 2008). Our investigation into the influence of group competition on the evolution of social behaviours uses Bowles's (2006; 2009) theoretical framework as a starting point to illuminate the selective forces involved.

4.2 Model and Analysis

The model we develop is only intended to provide a heuristic overview and for that reason, we consider the simplest possible case of a social behaviour p, which influences both the relative reproductive success of the individual within the group, and the ability of her group to compete with other groups. We assume the social behaviour is directly costly to the individual performing it so that if she invests in this trait, she incurs a fitness cost (scaled by c, such that for an individual i, in group j, her relative fitness within the group is reduced by cp_{ij}). We further assume that the social behaviour has a fitness effect on the p_j group members (we do not specify the way in which this fitness effect is conferred). This effect is scaled by b, such that the individual's relative fitness within her group is changed by bp_j. For generality, we allow b to be positive or negative, and so consider social behaviours that either help or harm the other group members. For example, a helping trait may be collective childcare and a harming trait might be fighting other group members.

In addition to affecting within-group relative fitness via helping or harming, we assume that investing in the social behaviour, p helps the group succeed if it comes into conflict with other groups. Every generation we assume, that with probability κ, the group will compete against another randomly chosen group in the population and that the group will win that contest with probability λ. Bowles (2006, 2009) used

different functions for λ and we examine both here. In Bowles (2006), λ was related to the average (absolute) level of the social behaviour expressed by members of group j (so $\lambda=f(p_j)$) and in Bowles (2009), λ was related to the average level of the social behaviour expressed by the group p_j, relative to the population average, p (so $\lambda=f(p_j/p)$). Following Bowles (2006; 2009), we assume competitive encounters are 'all or nothing' events, so if the group loses, it goes extinct and if it wins it momentarily doubles in size and then splits into two daughter groups (of equal size). We assume that only between-group competition determines the group's fitness, so if groups do not compete (which occurs with probability $1-\kappa$), group fitness is 1. Summing these probabilities gives the average fitness of a group j as $w_j = 0[\kappa(1-\lambda)]+2[\kappa\lambda]+1[(1-\kappa)] = 1-\kappa+2\kappa\lambda$.

Table 4.1. A summary of symbols used in the analysis

Symbol	*Definition*
p	social behaviour
c	personal cost
b	helping or harming
κ	frequency of group contest
λ	probability of winning a contest

Symbol	*Definition*
r	within-group relatedness
w	Darwinian fitness
n	group size
B	fitness benefit to the recipient of a social behaviour
C	fitness cost to actor of a social behaviour

The overall fitness of an individual i in group j, w_{ij}, is found by multiplying her success within the group by her group's resources, so the more resources she has, the higher her fitness. More formally, if the group's resources are given by F_G, and her

share is determined by her within-group success F_I, relative to her group mates' success $\bar{F}_I$, then her fitness $w_{ij} = F_G(F_I / \bar{F}_I)$. Here, we have

$$w_{ij} = \left(1 - \kappa + 2\kappa\lambda\right)\frac{1 + bp_j - cp_{ij}}{1 + bp_j - cp_j}, \quad (1)$$

where p_{ij} is the individual's investment into the social behaviour and p_j is the average level of the social behaviour performed by members of the group. Model notation is summarised in Table 4.1. We use the neighbour modulated fitness modelling approach (Taylor and Frank, 1996) to assess the evolutionary stability of the social behaviour p.

4.2.1. Probability of winning a group contest, λ is related to the average (absolute) level of the social behaviour in the group.

Here, we follow Bowles (2006) and assume that the probability of winning a contest λ, is related by a scaling factor z to the average level of the social behaviour p_j, so that the probability of winning a conflict is given by: $\lambda = z + (1-2z)p_j$, where z may vary between 0 and 0.5. When z=0, the average level of the social behaviour, p_j is equal to the probability of winning and as z increases, the social behaviour becomes less important in conflict such that when z=0.5, the probability of winning a conflict is independent of p_j. In Appendix 4.4, we show three main results.

First, group conflict favours the evolution of both helping (+b) and harming (-b) behaviours (Figure 4.1A). All that matters is that the within-group social behaviour (b) is correlated with winning contests (λ), and that the inclusive fitness benefit the individual gains from winning contests outweighs the costs (-c and –b) incurred by performing the behaviour. The within-group effect (b) of the social behaviour (p) does affect the evolutionary stable level of the social behaviour (ESS; Maynard Smith and

Price, 1973), but it does not affect when the social behaviour can invade from rarity (Figure 4.1A). The reason why helping and harming are both favoured may be because we followed Bowles (2006, 2009) and assumed that within-group social behaviour (b) does not affect the group's fitness (w_j=1-κ+2$\kappa\lambda$). Here group fitness is solely determined by the effect between-group competition has on the group's resources (F_G). For this reason, in the absence of group contest, the social behaviour is never favoured (p^*=0; dw/dp<0 if κ=0) as if an individual incurred the cost c of performing the social behaviour, it would gain less of the group's resources relative to any non-social group mates ($F_I < \bar{F}_I$), resulting in a lower relative fitness.

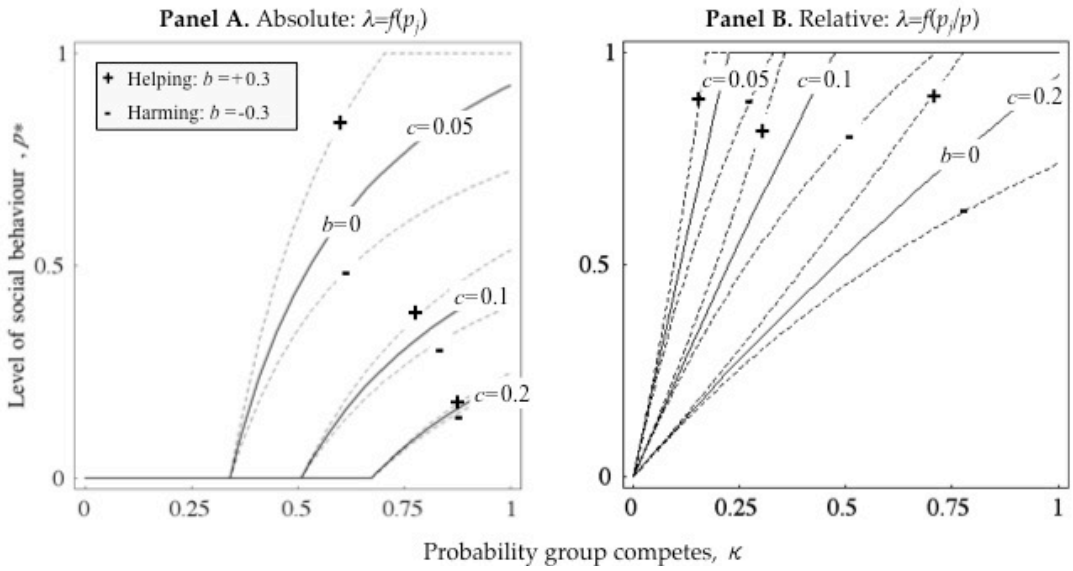


Figure 4.1 Helping and harming behaviours may be favoured when groups compete. The within-group social behaviour, b may be helpful (+b) or harmful (-b), however this has no effect on the invasion condition for the social behaviour (p>0; solid lines are b=0, right/lower dotted lines are b=-0.3, left/higher dotted lines are b=0.3). Whether the social behaviour is favoured or not depends on its ability to improve the group's chance at surviving contests, λ. If group contests are more frequent (higher κ), this favours higher levels of the social behaviour because the benefits from increased group survival will outweigh the costs of performing it (determined by b and c). If we follow Bowles (2006) and assume the group's chance of surviving is related to the average level of the social behaviour in the group (λ=f(p_j); Panel A), we find when contests are infrequent (low κ), the social behaviour is not favoured. By contrast, if we follow Bowles (2009) and assume the group's chance of surviving is related to the average level of social behaviour in the group relative to the population average level of the social

behaviour ($\lambda=f(p_j/p)$; Panel B), then, if groups compete, the social behaviour is always favoured whatever the cost ($p^*>0$ when $\kappa>0$). Plotted for $n=32$, $R=0.076$ (where $r=(1/n)+((n-1)/n)R$), Panel A: $z=0$; Panel B: $\mu=2$.

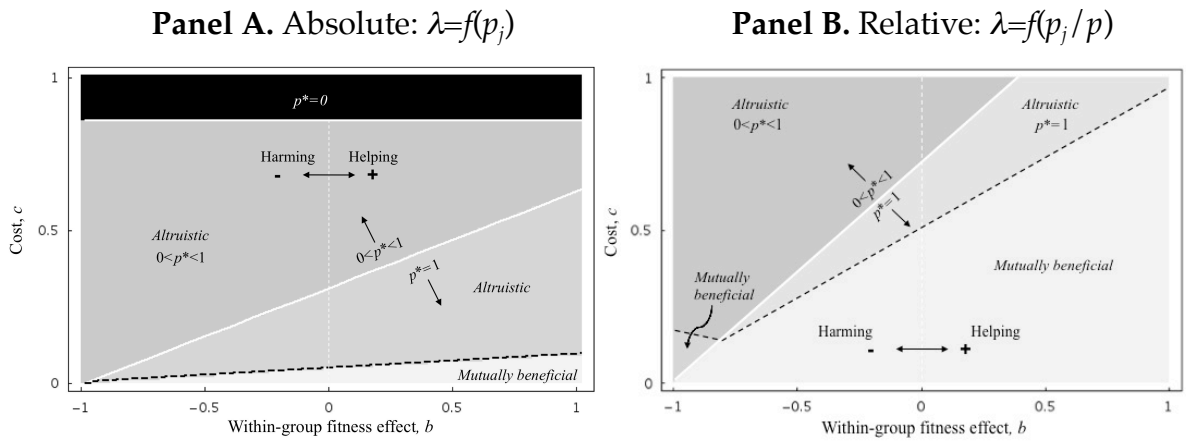


Figure 4.2 The social behaviour is either altruistic or mutually beneficial. When compared to the fitness of a rare defector who does not perform the social behaviour, the social behaviour may be altruistic or mutually beneficial. It may sound nonsensical to say a harming behaviour is also be altruistic or mutually beneficial, but this is because harming represents only a partial fitness effect, whilst the '*B*' and '*C*' terms in Hamilton's rule captures the overall fitness effect, which includes the group-level benefit the social behaviour yields due to increased group survival. Hamilton's rule states that a behaviour or trait will be favoured by selection, when $RB-C>0$; where $-C$ is the fitness cost to the actor, B is the fitness benefit to the recipient weighted by R, their others-only relatedness. A behaviour is termed altruistic if $B>0$ and $C>0$, mutually beneficial if $B > 0$ and $C < 0$ (Hamilton, 1964; West *et al.*, 2007b). Panel A plotted for $n=10$, $r=0.5$, $\kappa=0.3$ and $z=0$. Panel B plotted for $n=10$, $r=0.3$, $\kappa=0.6$ and $\mu=10$.

Second, when compared to the fitness of a rare defector who does not perform the social behaviour (p), the social behaviour may be altruistic or mutually beneficial (Figure 4.2A; Table 1.1). According to Hamilton's rule, social behaviours are favoured when $RB-C>0$, where R is the average relatedness between actors and recipients and B and C are the fitness effects on the recipient and actor respectively (Hamilton, 1964). The social behaviour always yields indirect fitness benefits, even if the within-group term b is negative, because the benefit group mates receive from increased group success always outweighs any fitness effect of the within-group trait, b. Note, the '*B*' and '*C*' terms in Hamilton's rule differ from '*b*' and '*c*' model terms because

they capture the overall fitness effect, not the within-group component of fitness. Altruism occurs when the actor incurs a direct fitness cost ($-C>0$) whilst group mates enjoy a fitness benefit ($B>0$) and mutually beneficial behaviours occur when both the actor and recipients benefit ($-C<0$, $B>0$). In this model intermediate levels of the social behaviour, p^*, are always altruistic. When $p^*=1$, the behaviour is mutually beneficial when the cost, c is very low (the value is given in equation A11 of Appendix 4.4), otherwise the behaviour is altruistic (Figure 4.2A).

Third, the optimal level of the social behaviour (p^*) is correlated with the likelihood of group contests (κ), within-group relatedness (r) and the cost of performing the social behaviour (c). The optimal level of the social behaviour is positively correlated with the likelihood of group contests, κ ($dp^*/d\kappa>0$), as if conflicts becomes more likely, the benefit gained by winning more of them becomes greater relative to any within group costs. It is positively correlated with within group relatedness ($dp^*/dr>0$, where r, is Wright's (1922) coefficient of relatedness, which measures how much of the genetic variance in the social behaviour occurs between-groups) as higher within-group relatedness increases the indirect fitness benefits the individual receives. Finally, the optimal level of the social behaviour is negatively correlated with the individual cost of performing the behaviour, c as higher costs reduce the overall fitness benefit an individual receives. If the social behaviour is very costly, it cannot evolve ($p^*=0$ when $c>[2r\ (1-2z)\kappa]/[(1-r)(1-(1-2z)\kappa)]$) and conversely, when the social behaviour is very cheap, maximum expression occurs ($p^*=1$ when $c<[2(1+b)r(1-2z)\kappa]/[1-r+(1-r)(1-2z)\kappa]$). For costs between these values, intermediate levels of the social behaviour are favoured ($p^*=[2\kappa r(1-2z)- c(1-r)(1-(1-2z)\kappa]/[2\kappa(1-2z)(c-br)]$).

4.2.2 Probability of winning a contest is related to the relative level of the social behaviour expressed by the group (p_j/p).

Bowles (2006) assumes that the likelihood of the winning a between-group contest, λ is determined by the average (absolute) investment in social behaviour by group members. However, the likelihood of winning is more likely to depend upon how much the group invests in social behaviour relative to the groups it competes against (i.e. whoever invests more wins; Bowles 2009). To allow for this, we follow Bowles (2009) and define the conflict success term, λ, in (1) as:

$$\lambda = \frac{1}{1 + e^{2\mu\left(1 - p_j/p\right)}}, \tag{2}$$

where e is the natural logarithm and μ is a scaling function. With this adjustment, if the average investment by group members in the social behaviour (p_j) is the same as the population average (p), then on average it is evenly matched against other groups so will win contests with a probability ½ (i.e. when $p_j=p$, $\lambda=0.5$). If the group invests below the population average in the social behaviour ($p_j<p$), then it will win less than half the time ($\lambda<0.5$), and likewise, if it invests more than the population average ($p_j>p$), it will win more than half the time ($\lambda>0.5$). The scaling function, μ determines the degree to which having higher/lower levels of social behaviour than the population average alters the probability of winning (becoming more pronounced as μ increases). In Appendix 4.4 we show that this modification qualitatively changes the previous results in two ways.

First, if there is any competition between groups ($\kappa>0$), then the social behaviour is always favoured ($p^*>0$), irrespective of how costly it is (Figure 4.1B). This is because if groups did not perform the social behaviour, any group investing in a non-zero level

of the social behaviour would win every contest, and all other groups would go extinct. Consequently, no matter how costly, a non-zero optimal level of the social behaviour (p^*) is always favoured ($p^*=1$ when $c< [r\kappa\mu(1+b)]/[1- r(1-\kappa\mu)]$ and when the cost is higher than this $p^*=(\kappa\mu r)/[c(1-r(1-\kappa\mu))-br\kappa\mu)]$). With the previous assumption, a fractional increase in the level of the social behaviour led to only a fractional increase in a group's success so the cost of investing in the trait could be greater than the benefit. Second, whilst the Hamiltonian classification still shows that the behaviour is always mutually beneficial or altruistic, now intermediate levels of the social behaviour, p^*, may be mutually beneficial or altruistic (Figure 4.2B). As before, the social behaviour is mutually beneficial when the cost is low, and altruistic when the cost is high (the condition is given in A13 of Appendix 4.4).

4.3 Discussion

We investigated the effect of competition between groups on the evolution of social behaviours. For completeness, we calculated the likelihood a group will succeed in conflict success in two different ways: linked to (1) the average expression of the social behaviour in the group (absolute; figures 4.1A and 4.2A) and (2) this average expression relative to the population average expression (relative; figures 4.1B and 4.2B). Our results show that whichever way we calculate the probability of success, between-group competition can favour any social behaviour that helps the group win contests, be it pro-social (associated with helping group-mates) or anti-social (associated with harming group mates). Furthermore, we show that when it evolves, this behaviour is always altruistic or mutually beneficial because, even if it causes harm to group mates, the costs are more than compensated by the benefits of greater success in conflicts. As we show that helpful or harmful social behaviours are

favoured so long as they are associated with improved competitiveness, our results complicate the widely held view that the presence of competition between groups may be a mechanism driving the evolution of general pro-social preferences in humans.

Our results suggest that group competition only favours within-group social behaviours that are directly associated with the ability to win conflicts. We follow earlier models (Bowles 2006; 2009) and assume that the evolution of any within-group helping or harming behaviour, b, is perfectly linked to the group's ability to win contests, λ (they are both properties of a single trait p). This assumption means that the within-group social behaviour, b in our model is not an adaptation i.e. it is not selected due to its fitness-maximising properties; it occurs because we assume it is inseparable from a behaviour that is adaptive (the ability to win conflicts). Whilst natural selection will always minimize the deleterious consequences of an adaptive behaviour, the adaptive benefits of some behaviours (e.g. aggression) may be inseparable from their harmful side effects.

Our model assume that the average fitness of a group member (w_j) is entirely determined by competition between groups. This is unrealistic; if group members harm each other we would expect their group to do worse than a group where individuals help each other. However we favour this assumption for two reasons. First, we are interested in the simplest case, and if we added the complication that within-group social behaviours could affect group fitness, it would only make a quantitative difference to our results. There would still be parameter values where helping and harming are possible. Second, relaxing this assumption would not aid our understanding of how group competition influences the evolution of social behaviours. It would merely capture the well-known fact that helping behaviours

may be favoured if they increase group fitness and harming behaviours may be disfavoured if they decrease group fitness. In reality, competition between groups will not be the only factor affecting the average fitness of a group member. For example fitness will also be affected by how group members share food, care for children and sanction bad behaviour. Given that many factors contribute to an individual's inclusive fitness, there are always evolutionary trade-offs and so even if harming behaviour helped groups succeed in conflicts, it may not be favoured by natural selection if, for example, people who harmed made bad parents.

Our results do not support the view that between-group competition alone can favour generalized individually costly pro- or anti-social tendencies. Instead, we find it only favours behaviours directly associated with increased competitiveness. However, our model considers group competition in isolation; a fruitful avenue for future research will be to re-examine the established evolutionary explanations for cooperation in the presence of group competition (West *et al*. 2007b). For example, between-group competition may favour cooperation between non-kin when combined with the mechanism of group augmentation (Kokko *et al*. 2001). Models of group augmentation assume that group size correlates with individual survival, and show that costly cooperation between non-relatives can evolve if it helps groups to grow in size (e.g. by helping to rear unrelated offspring; Kokko *et al*. 2001). As group size is probably a good predictor of success in contests, the presence of between-group competition may favour the evolution of cooperation in large non-kin groups. It will be interesting to see whether group competition also increases selection for punishment mechanisms (Clutton Brock 1995).

Our results show the social behaviour always provides indirect fitness benefits, no matter how harmful it is (Figure 4.2). This result highlights the important difference between specific fitness effects (such helping or harming other group members) and

the overall fitness consequences of a behaviour (in this case altruistic or mutually beneficial). Hamilton's rule measures the overall lifetime fitness consequences of a behaviour, not partial fitness effects such as the fitness effects within a group or effects measured over a short timescale (Hamilton 1964). It is a complete and general description of the action of natural selection (Gardner *et al*. 2011). Behaviours are often altruistic, even if they have short-term costs. For example, if bees die stinging a honey badger, colony productivity will drop which is costly to the remaining bees, however stinging remains altruistic because the benefit of having the hive saved far outweighs any costs the remaining bees suffer from reduced productivity. Similarly, in this model, the harmful social behaviour is indirectly beneficial as the costs group mates incur from being harmed are outweighed by the benefits they gain if their group wins contests. It is worth noting that models sometimes adopt other definitions for altruism by defining the relatedness, benefits and costs differently from the very specific definitions used in Hamilton's rule (Table 1.1; West *et al*. 2011). For example, previous models of group competition (Bowles 2006, 2009) defined altruism as individually costly but group beneficial by likening the model's '*b*' and '*c*' terms to the indirect and direct fitness terms in Hamilton's rule. If we used that definition here, we would have classified the harming behaviour as spiteful (West *et al*. 2011), allowing us to conclude that between-group competition favours the evolution of spite in humans. This potential for confusion, highlights why it is important to use the Hamiltonian definitions when discussing the evolution of social behaviours.

How could our models be tested with empirical data? One possibility, used by Bowles (2006, 2009) is to attempt to parameterise our model with empirical data, and examine if that leads to conditions where certain forms of social behaviour would be favoured. However, Frank (1998) has argued that this is not how such theory should

be used. The problem is that countless other models could be developed which also predict the occurrence of social behaviours, in response to a number of other factors, and so it would be impossible to distinguish a role of between group competition *per se*. Put another way, if alternative hypotheses are allowed for, it is hard to distinguish between them. This is the case even with much simpler traits, where the relevant traits can be measured much more easily, such as population sex ratios (West 2009), l*et al*one with the archaeological data used by Bowles (2006, 2009), which will be subject to huge confidence intervals. The alternative and much more powerful approach is to use our model to make comparative predictions for how the levels of social behaviours should vary across groups, populations, cultures or species (Frank 1998; West 2009). For example, one could test our model predictions by seeing whether social behaviours are correlated with the frequency of between group conflicts or success rates in conflicts.

To conclude, our aim here was not to argue that group competition could not be a factor in the evolution of helping. One can readily imagine why traits for coalitional, cohesive groups may be favoured. However, one can also imagine why traits that reduce cooperation within the group, such as aggression and violence, could also be favoured when groups compete. We suggest that the presence of group competition alone is insufficient to explain the existence of pro-social, cooperative groups and that more theoretical and empirical work is needed to understand the role of group competition on the evolution of cooperation.

4.4 Appendix

Here, we determine the evolutionarily stable levels of the social behaviour for our group competition model. We calculate the direction of selection acting upon the

social behaviour p from the individual fitness in equation (1) of the main text, by calculating the marginal fitness with respect to p, i.e. $\mathrm{d}w/\mathrm{d}p$. To do this we use the neighbourhood modulated fitness (or kin selection) approach. This uses the chain rule methodology of Taylor and Frank (1996) to give $\mathrm{d}w/\mathrm{d}p = \partial w_{ij}/\partial p_{ij} + r\,\partial w_j/\partial p_j$ where $r = \partial p_j/\partial p_{ij}$ which is the kin-selection coefficient of (whole-group) relatedness (Taylor and Frank, 1996; Pepper 2000). This derivative may be separated into partial derivatives, which describes the effect of an individual's trait value ($\partial w_{ij}/\partial p_{ij}$), and the effect of the average trait value of its group ($\partial w_j/\partial p_j$), on a focal individual's fitness.

4.4.1 Contest success is determined by absolute (group average) level of the social behaviour.

We now derive the results presented in section 4.2.1 of the main text by conducting an invasion and stability analysis of (A1). Assuming that $\lambda = z + (1-2z)p_j$, in equation (1) in the main text, at the population average ($p_{ij} = p_j = p$), this obtains:

$$\frac{\mathrm{d}w}{\mathrm{d}p} = r\big(2\kappa(1-2z)\big) - (1-r)\frac{c}{1+p(b-c)}\Big(1-\kappa+2\kappa\big(z+(1-2z)p\big)\Big) \qquad \text{(A1)}$$

First, we evaluate equation (A1) assuming there is no social behaviour (i.e. set $p=0$), this obtains $\mathrm{d}w/\mathrm{d}p = 2\kappa r(1-2z) + c(1-r)\kappa(1-2z)$, and hence the social behaviour cannot invade ($\mathrm{d}w/\mathrm{d}p < 0$) when:

$$c > [2\kappa r(1-2z)]/[(1-r)(1-(1-2z)\kappa)], \qquad \text{(A2)}$$

note this is not a function of the within-group social behaviour term b. When the cost is below this, some level of the social behaviour is favoured ($\mathrm{d}w/\mathrm{d}p < 0$). For the maximum expression of the social behaviour ($p^* = 1$) we find that $\mathrm{d}w/\mathrm{d}p = 2\kappa r(1-2z)$-

$[c(1-r)(1+\kappa(1-2z)]/[1+b-c]$ and hence the social behaviour will reach its maximum expression when:

$$c<[2(1+b)r(1-2z)\kappa]/[1-r+(1-r)(1-2z)\kappa]. \quad \text{(A3)}$$

For intermediate costs, an intermediate ESS level of the social behaviour is favoured. To find this, we set the RHS of equation (A1) equal to zero and solve for p, which obtains:

$$p^* = \frac{2\kappa r(1-2z) - c(1-r)\left(1-(1-2z)\kappa\right)}{2\kappa(1-2z)(c-br)} \quad \text{(A4)}$$

We now assess how different model parameters affect levels of the social behaviour. In the absence of group contest (κ=0), equation (A1) becomes dw/dp=-(1-r)[c/(1+p(b-c))] which is always negative, so selection on the social behaviour, p is neutral when groups do not compete. When groups do compete, to examine how increasing the frequency of group contests (κ) affects the ESS level of the social behaviour p we differentiate the RHS of (A4) with respect to κ. This obtains:

$$\frac{dp^*}{d\kappa} = \frac{c(1-r)}{2(c-br)(1-2z)\kappa^2} \quad \text{(A5)}$$

which is positive when $0<p^*<1$ (it is negative when $c<br$, but from (A3), we see when the cost is this low, we have p^*=1). To consider how increasing the average relatedness between group members, r affects the ESS level of the social behaviour p, we differentiate the RHS of (A4) with respect to r, which obtains:

$$\frac{dp^*}{dr} = \frac{c\left(c+(2+b-c)(1-2z)\kappa-b\right)}{2(c-br)^2(1-2z)\kappa}. \quad \text{(A6)}$$

This is positive when $c>[b-(2+b)(1-2z)\kappa]/(1-(1-2z)\kappa)$. If we compare this inequality to the boundary conditions for $0<p^*<1$, we can see this always holds when intermediate levels of p^* are favoured. To examine how increasing the cost, $-c$ of performing the social behaviour affects its evolution, we differentiate the RHS of (A4) with respect to $-c$, which obtains:

$$\frac{\mathrm{d}p^*}{\mathrm{d}(-c)} = r\frac{2(1-2z)\kappa - b(1-r)\left(1-(1-2z)\kappa\right)}{2(c-br)^2(1-2z)\kappa} \qquad \text{(A7)}$$

which is negative when $b>2(1-2z)\kappa/[(1-r)(1-(1-2z)\kappa)]$. For intermediate levels of the social behaviour, p^*, this inequality is always satisfied so increasing the cost will decreases the stable level of the social behaviour. If the within-group helping/harming behaviour b, is below this value, intermediate levels of the social behaviour are not favoured. This can be shown as the condition for $p^*>0$ (A2) is higher than the condition for $p^*=1$ (A3) when $b>2(1-2z)\kappa/[(1-r)(1-(1-2z)\kappa)]$. Finally, to investigate how changing levels of within-group helping/harming affect levels of the social behaviour, we differentiate the RHS of (A4) with respect to b, which obtains:

$$\frac{\mathrm{d}p^*}{\mathrm{d}b} = r\frac{c(1-r+2r)(1-2z)\kappa - c(1-r)}{2(c-br)^2(1-2z)\kappa} \qquad \text{(A8)}$$

which is positive when $c<[2\kappa r(1-2z)]/[(1-r)(1-(1-2z)\kappa)]$, which is always satisfied as this is the condition for the social behaviour to be favoured (see A2).

We investigate whether the social behaviour is maintained by direct or indirect fitness benefits by calculating the fitness components of Hamilton's rule (Hamilton 1964) and establishing when they are positive or negative. The change in relative fitness is $\Delta W=W_{ij}-\bar{W}$, where $\bar{W}=1$ and $W_{ij}=w_{ij}/w$. If fitness, w_{ij} is given by equation (1) in the main text, then assuming $\lambda=z+(1-2z)p_j$, W_{ij} is:

$$W_{ij} = \frac{\left(1-\kappa+2\kappa\left(z+(1-2z)p_j\right)\right)\left(\frac{1+bp_j-cp_{ij}}{1+bp_j-cp_j}\right)}{1-\kappa+2\kappa\left(z+(1-2z)p\right)}; \qquad \text{(A9)}$$

We consider the change in relative fitness, ΔW, of a rare defector who does not perform the social behaviour. Assuming weak penetration (the 'tracer allele' approach; Grafen 2006b), we have $p_j = p(n\text{-}1)/n$ to account for the group member (representing a fraction $1/n$ of group j) who defects. To find the direct fitness term in Hamilton's rule $-C$, we assume the focal individual is a defector, so we set $p_{ij}=0$ and $p_j=(n\text{-}1)p$ in (A9) which yields:

$$-C = -\frac{2\kappa p(1-2z)\left(n+b(n-1)p\right)-cpn(n-1)\left(1-\kappa+2\kappa\left(z+(1-2z)p\right)\right)}{n\left(n+p(b-c)(n-1)\right)\left(1-\kappa+2\kappa\left(z+(1-2z)p\right)\right)} \qquad \text{(A10)}$$

and this is always positive in the region where $0<p^*<1$ is favoured (we found this by substituting p^* in A4 for p in A10 and comparing when this value of $-C>0$ with the boundary conditions for p^*). To examine the region where $p^*=1$ is favoured, we substitute $p=1$ for p in A10 and examine when it is negative ($-C<0$), which is when:

$$c < \frac{2\left(b(n-1)+n\right)(1-2z)\kappa}{(n-1)n\left(1+(1-2z)\kappa\right)} \qquad \text{(A11)}$$

thus $-C>0$ for costs, c above this. If not performing the social behaviour is directly costly, this implies performing it directly beneficial. Therefore, at intermediate levels and for costs above the value given in the RHS of equation (A11), we have a directly beneficial behaviour, otherwise, the social behaviour is directly costly.

To calculate the indirect benefits term B, we assume the focal individual performs the social behaviour, but that one member of her group is a defector, so we set $p_{ij}=p$ and $p_j=(n\text{-}1)p$ in (A9). This yields:

$$B = -\frac{cpn\left(1-\kappa+2\kappa(1-2z)\right)+2p\kappa(1-2z)\left(n+bp(n-1)\right)}{n\left(n+p(b-c)(n-1)\right)\left(1-\kappa+2\kappa\left(z+(1-2z)p\right)\right)}. \quad \text{(A12)}$$

which is always negative in the region where $p^*>0$ is favoured. If not performing the social behaviour always has indirect costs, this implies performing it indirectly beneficial, hence our result that the social behaviour is altruistic except for at low costs when p=1, where it is mutually beneficial (A11; Figure 4.2A).

4.4.2 Contest success is determined by the average level of the social behaviour in the group relative to the population average.

We now derive the results presented in section 4.2.2 of the main text by assuming that $\lambda=1/[1+e^{2\mu(1-(pj/p))}]$ in equation (1) in the main text. At the population average ($p_{ij}=p_j=p$), this obtains:

$$\frac{\mathrm{d}w}{\mathrm{d}p} = \frac{\kappa\mu r(1+bp)-cp\left(2(1-r)+\kappa\mu r\right)}{2p(1+bp-cp)} \quad \text{(A13)}$$

In the absence of group competition ($\kappa = 0$), the social behaviour cannot spread (as $\mathrm{d}w/\mathrm{d}p<0$ when $\kappa = 0$). If $\kappa>0$, some positive level of the social behaviour ($p>0$) is always favoured as $p^*=0$ is never stable (when $p=0$, $\mathrm{d}w/\mathrm{d}p\rightarrow+\infty$). Maximum expression of the social behaviour ($p^*=1$) occurs when $\mathrm{d}w/\mathrm{d}p > 0$ at $p=1$ which is when:

$$c < \frac{\kappa\mu r(1+b)}{\kappa\mu r+2(1-r)} \quad \text{(A14)}$$

When the cost of performing the social behaviour is above this threshold, an intermediate level of the social behaviour is stable. To find the ESS level of the social behaviour, we set the RHS of equation (A13) to zero and solve for p which gives:

$$p^* = \frac{\kappa\mu r}{c\left(2\left(1-r\right)+\kappa\mu r\right)-b\kappa\mu r}. \tag{A15}$$

Following the methodology described in section 4.4.1, we now calculate the fitness components of Hamilton's rule and determine when they are positive or negative. If fitness, w_{ij} is given by equation (1) in the main text, then as w=1, W_{ij} =w_{ij}. The direct fitness term is:

$$-C = -\frac{\left(n+bp\left(n-1\right)\right)\kappa\tanh\left[\mu/n\right]-cp\left(n-1\right)}{n+p\left(b-c\right)\left(n-1\right)} \tag{A12}$$

and this is positive (-C>0), when:

$$c > \frac{\left(n+bp\left(n-1\right)\right)\kappa\tanh\left[\mu/n\right]}{p\left(n-1\right)} \tag{A13}$$

and negative (-C<0) for values of c below this. Both intermediate (0<p^*<1) and full (p^*=1) expressions of p are favoured for costs above and below the value given on the RHS of A13, therefore the social behaviour may be directly costly (-C>0) or directly beneficial (-C<0) for $p \leq 1$.

Following the methodology described in section 4.4.1, the indirect benefits term B is found to be:

$$B = -\frac{\left(n\left(1+p\left(b-c\right)-bp\right)\right)\kappa\tanh\left[\mu/n\right]+cp}{n+p\left(b-c\right)\left(n-1\right)} \tag{A14}$$

which is always negative (B<0) for all the model parameter values. Thus, if not performing the social behaviour always incurs an indirect cost, this implies that performing it is always indirectly beneficial, hence our result that the social

behaviour is either altruistic or mutually beneficial, depending on whether the cost, *c* lies above or below the cost threshold given in A13 (Figure 4.2B).

Chapter 5.
Reproductive levelling and the evolution of cooperation between non-kin.

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5.1 Introduction

Cooperation can be found at all levels of biological complexity, between genes within a genome, between cells within multicellular organisms, and between individuals within societies (Maynard Smith and Szathmáry, 1995; Bourke 2011). However, cooperative behaviours which benefit others whilst reducing the actor's relative fitness pose a challenge for the biological and social sciences. This is because natural selection favours individuals with a higher relative fitness so, all else being equal, cooperation should be selected against (Darwin 1859; Fisher 1930; Price 1970). A large theoretical literature has arisen to address this problem, showing that cooperation can be favoured between genetic relatives (kin selection) or when cooperation leads to a direct benefit for the actor (reviewed by Sachs *et al*. 2004; Lehman and Keller 2006; West *et al*. 2007a).

One potentially important way cooperation in humans may have evolved is via norms or social institutions which promote reproductive levelling (Bowles 2006). Reproductive levelling is when fitness differences between group members are reduced via the equal distribution of resources, or the limitation of competition

between group members, and hence leads to more egalitarian groups. A possible role of reproductive levelling in our evolution is supported by: archaeological and anthropological evidence that ancestral humans lived in broadly egalitarian societies (Knauft 1991; Fried 1967; Boehm 1982, 1993, 1997, 2001); economic experiments showing that human possess preferences for equality (Dawes *et al*. 2007); and empirical studies suggesting that these preferences limit inequalities in wealth, status or power (Woodburn 1982; Boehm 1982, 1993, 1997, 2001).

However, from a theoretical perspective, it is not clear how and why reproductive levelling itself should be favoured (Bowles 2006; Boyd 2006; Bell 2009). Previous theoretical work assumes that cultural norms for reproductive levelling are present, and consider its impact upon the evolution of cooperation, without considering the evolution of reproductive levelling itself (Bowles 2006). However, because reproductive levelling can be a form of cooperation, this approach examines the consequences of cooperation (reproductive levelling) for the evolution of cooperation (other cooperative traits), and hence avoids the ultimate problem of whether reproductive levelling itself would be favoured (West *et al*. 2011). It has been suggested that natural selection on genes alone cannot explain the extent and complexity of social behaviours found in humans (Bell *et al*., 2009, Henrich and Henrich 2007; Richerson and Boyd 2004) These authors suggest that culturally inherited cooperation created a novel selective environment which led to uniquely human forms of genetically inherited cooperation evolving. The results of earlier work on reproductive levelling (Bowles 2006), in particular, have been cited in support of the idea that pre-existing culturally inherited cooperation leads to natural selection favouring genes for sociality that would otherwise be maladaptive (Bell *et al*. 2009).

Our aim here is to examine if and how reproductive levelling can be favoured and whether it can lead to more cooperative groups. The idea of reproductive levelling is captured by models in the biological literature which consider how cooperation can be favoured by what is sometimes termed policing or repression of competition (Leigh 1971, 1977; Ratnieks 1989; Frank 1995, 2003). In such models, a policing trait is costly to the individual performing it, but leads to a more equitable sharing of resources within groups. We allow such a trait to coevolve with a competitiveness trait that determines the extent to which individuals exploit resources cooperatively or selfishly. Although our interest is the scenario when reproductive levelling is a culturally inherited trait and competitiveness is a genetic trait, we consider, for completeness, all possible combinations of cultural or genetic inheritance.

5.2 Model and Analyses

In the absence of reproductive levelling, fitness is determined by the share of group resources that an individual obtains, which we capture with Frank's (1995, 1996a,b, 2003, 2009, 2010) tragedy of the commons model. We assume an individual i in group j displays a level of competitiveness s_{ij} and that her share of the group resources is determined by how competitive she is, relative to her group mates (s_{ij}/s_j). If an individual invests in an above-average level of competitiveness ($s_{ij}/s_j>1$), she outcompetes her group mates and receives a greater share of the group's resources. We also assume that this competition between group members reduces the group's resources, such that the total resources (we scale resources such that a fully cooperative group has one unit) available for group members is given by $1-s_j$. Consequently, in the absence of reproductive levelling, an individual's fitness is given by $w_{ij}=(s_{ij}/s_j)(1-s_j)$. This leads to the classic 'tragedy of the commons' scenario,

where the evolution of increased competitiveness is favoured, even though this leads to a reduction in the available resources (Hardin 1968; Frank 1995, 2010).

We assume that an individual can invest in a reproductive levelling behaviour at level τ, ($0<\tau<1$), which has three fitness consequences. First, it reduces fitness differences between group members by ensuring resources are shared more evenly; a proportion τ_j of resources are distributed equally between group members (where τ_j is the average group investment in reproductive levelling). The individual's share of the remaining portion $1-\tau_j$ of group resources is still given by her relative competitiveness, s_{ij}/s_j. Second, reproductive levelling stops the group's resources being lost due to selfish exploitation; the result is to increase group fitness by $b\tau_j$ (so group fitness is $w_j=1-(1-b\tau_j)s_j$), where b ($0<b<1$) is the efficiency of reproductive levelling, representing the proportion of the group's resources that are prevented from being lost; we assume reproductive levelling cannot be perfectly efficient ($b\neq1$). Third, an individual pay a fitness cost, $-c$ to perform her personal level of the behaviour τ_{ij}. Model notation is summarized in Table 5.1. If w_{ij} denotes the relative fitness of an individual i in group j, then:

$$w_{ij} = \left(\tau_j + \left(1-\tau_j\right)\frac{s_{ij}}{s_j}\right)\left(1-\left(1-b\tau_j\right)s_j\right) - c\tau_{ij}. \quad (1)$$

This is the same fitness function used in chapter 3, which builds upon previous policing models by Frank (1995, 1996b, 2003). The individual faces a trade off between the cost of reproductive levelling, which she pays for personally, and the benefit she receives, which is determined by the average investment in reproductive levelling by all group members. The individual benefits from increased group

resources, however in addition to the personal cost, increased reproductive levelling also limits the individual's ability to compete and selfishly exploit the group's resources.

Table 5.1. A summary of symbols used in the analysis

Symbol	*Definition*
τ	reproductive levelling
s	Competitiveness / selfishness
c	cost of reproductive levelling
b	efficiency reproductive levelling restores group resources
r_τ	coefficient of (whole-group) relatedness for the reproductive levelling trait

Symbol	*Definition*
r_s	coefficient of (whole-group) relatedness for the selfishness trait
w	Darwinian fitness
R	others-only relatedness (the 'R' term in Hamilton's rule)
B	fitness benefit to the recipient of a social behaviour
C	fitness cost to actor of a social behaviour

5.2.1 The tragedy of the commons and reproductive levelling

We are primarily concerned with how culturally inherited reproductive levelling coevolves with genetically inherited cooperation. However, before considering that scenario, it is useful to consider two other special cases of our model. In Appendix 5.4 we show that:

Result 1. In the absence of reproductive levelling, ($\tau_{ij}=\tau_j=0$), we recover the classic 'tragedy of the commons' model of Frank (1995, 1996a,b, 1998, 2003, 2009, 2010). In this case, if within-group relatedness for the competitiveness trait is r_s, then the evolutionarily stable (ESS; Maynard Smith and Price 1973) level of selfish competition is $s^*=1-r_s$ (Frank 1995, 1996a; Taylor and Frank 1996; Figure 5.1*b*). Competitiveness is negatively correlated with relatedness because higher relatedness favours increased kin-selected self-restraint.

Result 2. If both traits have the same mode of inheritance (either both genetic, or both cultural), such that both traits have the same within-group relatedness, r, then high levels of costly reproductive levelling can be favoured at intermediate relatedness (when $r(1-r)>c/b$) and this leads to a reduction in competitiveness (Figure 5.1). The results of this special case are identical to that presented in an earlier paper on the genetic evolution of policing (see chapter 3).

5.2.2 Gene-culture coevolution

We now consider the scenario where the relatedness terms may differ. We follow Bowles (2006) to examine the case when the competitiveness trait is genetically inherited (r_s measures average genetic similarity) and the reproductive levelling trait is culturally inherited (r_τ measures average cultural similarity). These relatedness terms, r_s and r_τ, are statistical measures of variation, describing how closely the individual's trait values (s_{ij} and τ_{ij}) match her group's average trait value (s_j and τ_j); the tighter the correlation, the higher the relatedness. Previous analyses of this model assumed the selfishness and reproductive levelling were inherited in the same way i.e. that $r=r_s=r_\tau$ (see Chapter 3). However, the two relatedness terms could differ if the traits have different mechanisms of inheritance (i.e. one is cultural, one is genetic). We make no assumptions about the transmission or inheritance structure of the trait such as whether it is horizontal vs vertical (Lehmann *et al*. 2008) or blended vs particulate (Gardner 2011). Consequently, these relatedness terms could measure the cultural or genetic similarity between the focal individual and their group; we return to this topic in the discussion.

In Appendix 5.4, we show that this leads to the following predictions:

Result 3. Costly reproductive levelling can evolve, and it is most strongly favoured when genetic relatedness is very low ($r_s \to 0$) and cultural relatedness very high ($r_\tau \to 1$; $\tau^* > 0$ when $r_\tau(1-r_s) > c/b$; Figure 5.2). This is because low genetic relatedness leads to high competitiveness, which increases the benefits of a mechanism such as reproductive levelling that can prevent the tragedy of the commons. High genetic relatedness disfavours reproductive levelling ($\tau^* = 0$ when $r_\tau(1-r_s) < c/b$) as kin-selection alone can reduce competitiveness thus the added benefits of reproductive levelling no longer outweigh the costs. High cultural relatedness makes it easier for reproductive levelling to evolve, as this strengthens the feedback between the cost an individual pays to perform reproductive levelling and the benefits she receives which is determined by the group-average investment in reproductive levelling.

Fig. 5.1a

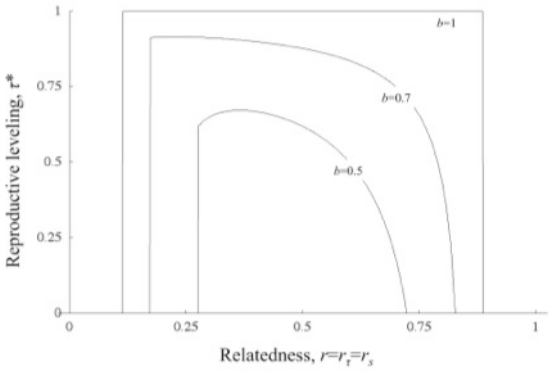


Fig. 5.1b

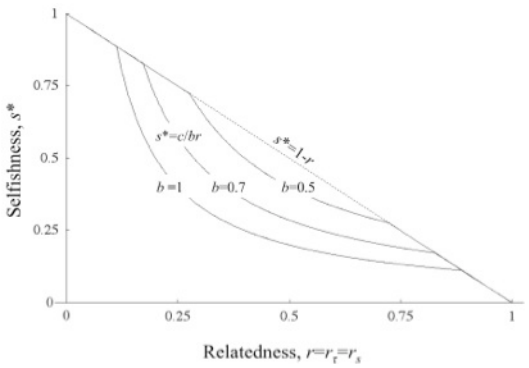


Figure 5.1. The coevolution of reproductive levelling and competitiveness when both traits are inherited in the same way (i.e. both genetically or both culturally). Reproductive levelling is favoured only at intermediate relatedness (when $r(1-r) > c/b$; Figure 5.1*a*) and when it invades, it always promotes self-restraint ($ds^*/d\tau < 0$, $s^* = c/br$; Figure 5.1*b*). If reproductive levelling is not perfectly efficient at restoring group resources ($b < 1$), intermediate levels of reproductive levelling, and smaller reductions in competitiveness are favoured. When reproductive levelling is not favoured ($\tau^* = 0$ is stable when $r(1-r) < c/b$), the model reduces to the tragedy of the commons ($s^* = 1-r$) and competitiveness is only limited by kin-selected self-restraint (Figure 5.1*b*). We assume the cost of reproductive levelling, $c = 0.1$. These predictions are identical to those made by the policing model presented in Chapter 3.

Result 5. When it evolves, intermediate levels of reproductive levelling ($0<\tau<1$) are favoured. Full reproductive levelling is not favoured ($\tau^*<1$) so the ESS level must be an intermediate value (τ^* is given in Appendix 5.4). For the model parameter values, τ^* is bounded between $0<\tau^*<1$, and so when it evolves, reproductive levelling enforces the fair distribution of some, but not all of the group's resources ($\tau^*<1$ so $w_j<1$). In other words, competitiveness is reduced, but some within group conflict remains, and so individuals are not acting so as to maximise group fitness.

Result 6. When reproductive levelling does evolve, we find a lower level of competitiveness is favoured ($s^*=c/b\,r_\tau$) than when it is absent ($s^*=1-r_s$). This repression of competition occurs because reproductive levelling reduces the relative benefit of competing over resources.

Fig. 5.2a Fig. 5.2b

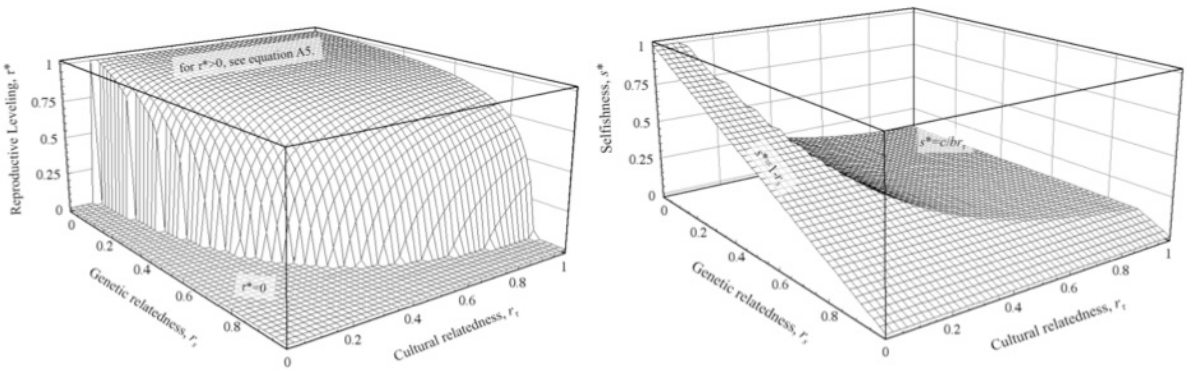


Figure 5.2. The coevolution reproductive levelling and competitiveness when reproductive levelling is inherited culturally, and competitiveness is inherited genetically. Reproductive levelling is favoured and enforces the fair distribution of the group's resources when genetic relatedness (r_s) is low and cultural relatedness (r_τ) is high (Figure 5.2a; $\tau^*>0$ when $r_\tau(1-r_s)>c/b$). Here, relatedness is a statistical measure of the average cultural or genetic similarity between group members compared to the population average; it makes no assumptions about the nature of inheritance. The greatest reduction in genetically inherited competitiveness ($s^*=c/b\,r_\tau$; Figure 5.2b) occurs when everyone shares the same cultural norms for reproductive levelling ($r_\tau\rightarrow1$). When reproductive levelling is not favoured ($\tau^{**}=0$ is stable when $r_\tau(1-r_s)<c/b$), the

model reduces to the tragedy of the commons ($s^*=1-r_s$) and competitiveness is only limited by kin-selected self-restraint (Figure 5.1*b*, 5.2*b*). Plotted where the individual cost of performing reproductive levelling, $c=0.05$, and the efficiency of reproductive levelling, $b=0.5$ (i.e. at best, reproductive levelling can restore the group's resources to half of what could be possible if competitiveness were absent).

To investigate whether reproductive levelling is maintained by direct or indirect fitness benefits, in the Appendix, we calculate the fitness components of Hamilton's rule (Hamilton 1964). We examine the change in relative fitness ΔW, of a rare defector who performs no reproductive levelling. We find that:

Result 7. When it is at its ESS level, reproductive levelling is a mutually beneficial trait, by which we mean that it provides a benefit to both the actor and the other individuals of the group (West *et al* 2007b; Table 1.1). By performing the trait, an individual gains both direct and (if $R>0$) indirect fitness benefits, when compared to the fitness of a rare defector who does not perform the trait.

5.3 Discussion

We have examined the evolution of a reproductive levelling trait, which reduces fitness differences between group members by enforcing the fair distribution of the group's resources. We have shown that reproductive levelling: (1) can be favoured by either genetic and/or cultural selection (Figure 5.1*a*, 5.2*a*); (2) is mutually beneficial; (3) favours a reduction in competitiveness (Figure 5.1*b*, 5.2*b*); (4) is most strongly favoured when group members share the same culture but are genetically unrelated (Figure 5.2). We chose to model a relatively simple scenario, in order to elucidate the underlying selective forces, and also to clarify links with the biological literature on how policing or repression of competition can favour cooperation. These results confirm that reproductive levelling mechanisms could play a role in the evolution of

cooperation, and counters the suggestion that such mechanisms cannot evolve genetically (Bell *et al*. 2009; Svensson 2009; Bowles 2006; Boyd 2006).

Why is reproductive levelling favoured? Reproductive levelling is a mutually beneficial trait that is favoured because both the actor and any related group-mates benefit from improved group resources (Hamilton 1964). If we assume that both the competitiveness and reproductive levelling traits share the same mode of inheritance (i.e. both genetical or both cultural), reproductive levelling is most strongly favoured at intermediate relatedness. This is because of a trade off: relatedness must be high enough for the indirect benefits of reproductive levelling to be significant, but not too high, otherwise kin-selected self-restraint reduces competitiveness to a point where reproductive levelling is no longer cost-effective (Figure 5.1). The potential benefit of gene-culture coevolution is that if the traits are inherited differently, the relatedness coefficicents are different, so these opposing selective constraints are decoupled. Reproductive levelling is most strongly favoured when genetic relatedness is low, as this increases the benefits of a mechanism to limit the harmful effects of competitiveness, and when cultural relatedness is very high as this maximizes the indirect benefits the actor gains from performing the trait. Estimates of genetic and cultural relatedness among human groups indicate this situation, of low genetic and high cultural relatedness, was common during human evolution (Bell *et al*. 2009).

If we assume that both reproductive levelling and competitiveness traits are inherited genetically, our results are the same as those predicted by policing models (see chapter 3 and Frank 1995, 1996b, 2003, 2009; Leigh, 1971, 1977). Policing is a well-known and important mechanism by which within-group competition is repressed, and evidence for it is found at all scales of biological complexity (Leigh 1977; West *et al*. 2002b; Kiers *et al*. 2003; Ratneiks 1988; Ratnieks and Visscher 1989; Ratneiks and Wenseleers 2008). It is possible that genetically controlled policing mechanisms are

important in humans too as it can enforce cooperation in large groups where relatedness is low (see chapter 3). Assuming genetic inheritance does not imply behaviours are hard-wired or that culture does not matter. For example, natural selection may favour a genetically-inherited preference for egalitarianism between group members, however, as discussed in chapter 1, the way in which an individual can fulfil this preference would vary greatly, depending upon the learning opportunities, social structure, environment and culture they experience. Given the experimental and ethnographic evidence that suggests a preference for egalitarianism may be universal (Dawes *et al*. 2007; Boehm 1982, 1997, 2001), this seems a fruitful avenue for further research and it highlights the benefits that increased interaction with the biological literature may bring.

To conclude, our results present a possible mechanism whereby gene-culture co-evolution could favour cooperation in large groups of genetically unrelated individuals. However, there are two caveats here, which could apply to many models of gene-culture coevolution. First, we examine the coevolution between genetically inherited competiveness and culturally inherited reproductive levelling, which implicitly assumes the cultural environment is stable enough to alter selection on genes in a consistent way. Given that almost all the complex traits, like competitiveness, that have been studied involve many genes with small effects, any selective change would take hundreds of generations (Lynch and Walsh 1998). Thus for our model predictions to apply, it would require long periods of cultural stability (generating a high cultural relatedness long enough to favour genes for reduced competitiveness). Assuming both traits are inherited genetically avoids this problem as the population dynamics for both traits operate on the same timescale.

Second, we assume that natural selection can lead to fitness maximizing adaptations via culturally inherited traits. A special feature of Mendelian inheritance is that

genetic relatedness will be approximately equal across most of the genome, in such a way that all genes will tend to be striving towards the same fitness maximization (Grafen 1985, 2006, 2007). It is precisely this which allows the construction of complex adaptations which are based upon multiple genes, and provides justification for the maximization or optimizing approach (Grafen 2007; Gardner 2009). In contrast, the same unity of interest cannot be assumed with cultural selection, because the pattern of transmission can vary across space, time or traits. Therefore it is unclear whether a trait such as reproductive levelling could evolve via cultural selection. It would be extremely useful to apply Grafen's (1999, 2007) formal Darwinism methods to cultural selection, and determine what, if any, maximizing principle would emerge from the cultural dynamics (Gardner 2009).

5.4 Appendix

Here we perform an invasion analysis and assess equilibrium conditions, deriving the results stated in the main text. To understand when the traits of competitiveness and reproductive levelling are favoured by selection, we examine how changing the levels of these traits affect an individual's fitness. The direction of selection is found using the neighbour-modulated fitness methodology of Taylor and Frank (1996; see also Frank 1995, 1996b, 2003; Taylor 1996a; Taylor *et al*. 2007). Assuming vanishing variation in the trait about the population average, the marginal fitness for competitiveness is:

$$\mathrm{d}w_{ij}/\mathrm{d}s_{ij} = \partial w_{ij}/\partial s_{ij} + r_s\, \partial w_{ij}/\partial s_j, \qquad \text{(A1)}$$

and for reproductive levelling is:

$$\mathrm{d}w_{ij}/\mathrm{d}\tau_{ij} = \partial w_{ij}/\partial \tau_{ij} + r_\tau\, \partial w_{ij}/\partial \tau_j, \qquad \text{(A2)}$$

where $r_s = \mathrm{d}s_j/\mathrm{d}s_{ij}$ and $r_\tau = \mathrm{d}\tau_j/\mathrm{d}\tau_{ij}$ represent the kin-selection coefficients of whole-group relatedness for the competitiveness and reproductive levelling traits respectively (relatedness to average group member, including oneself; Taylor and Frank 1996; Pepper 2000). The average fitness of an individual is give by equation (1) in the main text. For the population average, ($s_{ij}=s_j=s$ and $\tau_{ij}=\tau_j=\tau$), this gives the marginal fitnesses for competitiveness (s) and reproductive levelling (τ) as:

$$\frac{\mathrm{d}w}{\mathrm{d}s} = \frac{(1-r_s)(1-\tau)}{s} - (1-\tau b)\left(1-\tau(1-r_s)\right), \qquad \text{(A3)}$$

and:

$$\frac{\mathrm{d}w}{\mathrm{d}\tau} = br_\tau s - c\,. \qquad \text{(A4)}$$

We first consider the evolution of competitiveness in the absence of reproductive levelling. To do this we set $\tau=0$ in equation (A3), set the RHS to zero, and solve for s. This gives competitiveness as $s^*=1-r_s$, (result 1 of the main text). The marginal fitness for reproductive levelling at this point is found by evaluating equation (A4) at $s^*=1-r_s$, which obtains $\mathrm{d}w/\mathrm{d}\tau = b\,r_\tau(1-r_s)-c$; hence for reproductive levelling to invade (which occurs when $\mathrm{d}w/\mathrm{d}\tau>0$), we need $r_\tau(1-r_s)>c/b$ (result 3 of the main text). If both traits are inherited in the same way ($r_s=r_\tau$), then this becomes $r(1-r)>c/b$ (result 2 of the main text). If $\tau=1$, we have $\mathrm{d}w/\mathrm{d}s=-(1-b)r_s$, and, as we assume the efficiency of reproductive levelling $b<1$, competitiveness, s, is disfavoured and will decline to zero and this obtains $\mathrm{d}w/\mathrm{d}\tau=-c$ which is always negative. This means reproductive levelling cannot reach fixation (result 5 of the main text). To find the ESS levels of reproductive levelling and competitiveness, we set the RHS of equations (A3) and

(A4) to zero and simultaneously solve for $\tau=\tau^*$ and $s=s^*$. This yields $s^*=c/b\,r_\tau$ (result 6 of the main text) and two results for τ^*, only one of which is a biologically feasible:

$$\tau^* = \frac{bc + \left(c - br_\tau\right)\left(1 - r_s\right) + \sqrt{4bc\left(1 - r_s\right)\left(br_\tau\left(1 - r_s\right) - c\right) + \left(bc + \left(c - br_\tau\right)\left(1 - r_s\right)\right)^2}}{2bc\left(1 - r_s\right)}, \quad \text{(A5)}$$

which is result 5 of the main text.

To investigate whether reproductive levelling is maintained by direct or indirect fitness benefits, we must calculate the fitness components of Hamilton's rule (Hamilton, 1964). The change in relative fitness is $\Delta W=W_{ij}-\bar{W}$, where $\bar{W}=1$ and $W_{ij}=w_{ij}/w$. If fitness, w_{ij} is given by equation (1) in the main text, W_{ij} is:

$$W_{ij} = \frac{\left(\tau_j + \left(1 - \tau_j\right)\left(s_{ij}/s_j\right)\right)\left(1 - \left(1 - b\tau_j\right)s_j\right) - c\tau_{ij}}{1 - s\left(1 - b\tau\right) - c\tau} \quad \text{(A6)}$$

We consider the change in relative fitness, ΔW, of a rare defector who performs no reproductive levelling. Assuming weak penetration (the 'tracer allele' approach; Grafen, 2006b), we have $\tau_j=\tau(n\text{-}1)/n$ as one group member (representing a fraction $1/n$ of group j) performs no reproductive levelling. To find the direct fitness term in Hamilton's rule $-C$, we assume the focal individual is a defector, so we set $\tau_{ij}=0$ and $\tau_j=(n\text{-}1)\tau$. This yields:

$$-C = -s\left(1 - b\tau\left(1 - \frac{1}{n}\right)\right) \quad \text{(A7)}$$

which is always negative ($-C<0$) for our model parameters. If not performing reproductive levelling is directly costly, this implies performing it is directly beneficial. To find the indirect benefits term B, we assume the focal individual

performs reproductive levelling, but that one member of their group is a defector, so we set $\tau_{ij}=\tau$ and $\tau_j=(n\text{-}1)\tau$. This yields:

$$B = -s\left(1 - b\tau\left(1 - \frac{1}{n}\right)\right) - \frac{c\tau}{1 - c\tau - s(1 - b\tau)} \qquad \text{(A8)}$$

this is always negative ($B<0$) for our model parameters. If not performing reproductive levelling has indirect fitness costs, this implies performing it yields indirect fitness benefits, thus, when compared to the fitness of the defector, reproductive levelling is mutually beneficial (result 7 of the main text).

Chapter 6.
Promiscuity and the evolution of cooperative breeding.

This chapter has been submitted for publication in Proceedings of the Royal Society, B: Leggett, H., El Mouden, C., Wild, G., and West, S. A. (2011) Promiscuity and the evolution of cooperative breeding.

6.1 Introduction

It has been argued that monogamy or low levels of promiscuity have played a key role in favouring the evolution of cooperative breeding and eusociality. Hamilton's inclusive fitness theory (Hamilton 1964, 1970; Grafen 2006a) explains how cooperation can be favoured when it is directed towards relatives who also carry the gene for cooperation. Higher levels of promiscuity would decrease relatedness within family groups (Figure 6.1a), which could therefore reduce selection for staying and helping in the natal family group (Charnov 1981). Empirical support for this idea has come from the observations that: (a) eusociality has only evolved in species with strict lifetime monogamy, and (b) both the occurrence and amount of cooperative breeding in birds is negatively correlated with the level of promiscuity (Boomsma 2007; Hughes *et al*. 2008; Boomsma 2009; Cornwallis *et al*. 2010).

However, it is unclear whether greater promiscuity should always select against staying to help in natal groups. From an empirical perspective, some of the highest promiscuity rates ever recorded are cooperative breeders, such as the superb fairy wren, and the Australian magpie (Cornwallis *et al*. 2010). From a theoretical

perspective, previous predictions have been based on verbal or heuristic models. More explicit models, which allow the action of selection to emerge as a consequence of demographic assumptions, have shown that an increased relatedness will not always favour cooperation because it can be negated by increased local competition between relatives (reviewed by West *et al*. 2002a and Lehmann and Rousset 2010). Furthermore, a class of theoretical models make the opposite prediction, showing that because higher relatedness within groups leads to increased competition between relatives, selection can favour dispersal away from the natal group to reduce competition between relatives (Figure 6.1b; e.g. Hamilton and May 1977; Taylor 1988; Frank 1998; Gandon and Rousset 1999; Ronce *et al*. 2000; Rousset and Gandon 2002). Finally, a recent attempt to explicitly model the effect of promiscuity on cooperation by Nonacs (2011), with population genetic simulations, found that the level of promiscuity had little, or even a positive effect on selection for cooperation, and that this might be an area where inclusive fitness theory "gets it wrong".

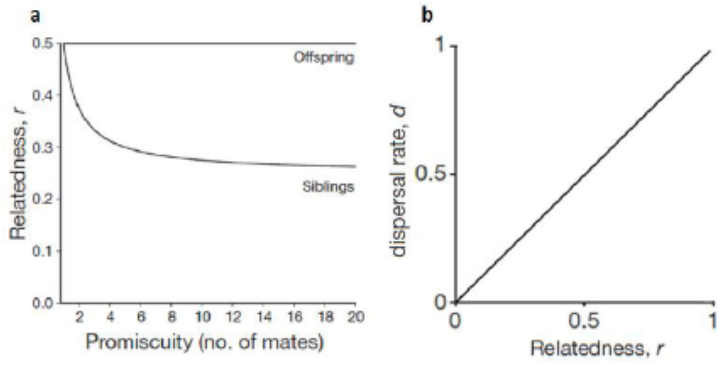


Figure 6.1. Promiscuity, relatedness and dispersal rate within cooperatively breeding groups. **(A)** Promiscuity and relatedness. Promiscuity (number of mates) plotted against the mean genetic relatedness between potential helpers and both their siblings and offspring. An individual is always related to its offspring with $r = 0.5$. However, as promiscuity increases, the relatedness to siblings decreases from $r=0.5$ to $r=0.25$ (full-siblings to half-siblings) (Charnov 1981); redrawn from (Cornwallis *et al*. 2010). (**B)** Higher relatedness within groups can lead to increased competition between relatives, which selects for dispersal away from natal groups to reduce competition between relatives (Hamilton and May 1977).

Here, we use an inclusive fitness approach to model how promiscuity influences selection to stay and help in natal groups. Our aim is to consider the simplest possible scenario to explicitly examine how the level of promiscuity, through its effect on within group relatedness, has multiple consequences. Our approach is therefore analogous to that taken by Taylor (1992a), when considering how limited dispersal influences cooperation. In particular, we examine how lower levels of promiscuity lead to higher within-group relatedness, which can, in turn, increase the indirect benefits of both staying to help relatives and dispersing to reduce competition among relatives (Wild 2006). In addition, we check the validity of our inclusive fitness model by comparing its predictions with that made by an individual based model.

6.2 The Model

We consider a population of cooperatively breeding, diploid individuals. To address the effect of promiscuity on cooperative breeding behaviour, we must assume the individuals are sexual. In order to avoid complications of modelling different sexes, we assume individuals are sexual hermaphrodites.

We assume the population is subdivided into a very large, but fixed, number of breeding patches (say, P patches). Each patch is of equal quality and supports one adult breeder at a time, plus a large number of juvenile offspring. In nature, the cooperative behavior of juvenile helpers can increase the breeder's survival and/or fecundity (Heinsohn and Legge 1999; Griffin and West 2003; Rousset and Ronce 2004; Russell *et al*. 2007). We focus on the former case, where the survival of the breeder is boosted by the presence of helpers. We avoid the latter case in order to avoid complications associated with the cascading effects that fecundity benefits exert on

breeder fitness (more helpers result in more offspring, producing greater numbers of helpers, which in turn increases fecundity further, etc).

Since juvenile helpers often direct their help toward the individual(s) occupying the same patch on which helpers, themselves, were born (e.g. helpers help their own parents (Clutton-Brock 2009; Hatchwell 2009), we assume that an offspring's tendency to help is linked to its decision to disperse. In other words, an offspring that remains on its natal patch does so in order to help the incumbent breeder, while an offspring that disperses from its natal patch will compete (without helping) on a new patch for the opportunity to breed, which occurs upon the (possible) death of a breeder. We explain our assumptions about the basis for an individual's "tendency" to help (or "decision" to not disperse) in greater detail below. For now, simply note that all helpers exert the same level of effort, and it is just this "tendency" to help that varies among individuals in our model population.

Now suppose we observe the population described above at discrete, evenly spaced points in time. We assume that, between successive observations, the following series of five events occurs:

1. *Mating.* Each breeder chooses exactly M mates from the global population of breeders. Mate choice occurs uniformly at random, and with replacement. Note that larger (resp. smaller) M indicates that breeders are more (resp. less) promiscuous.

2. *Birth.* Each breeder produces a very large number (K) of offspring, and each offspring carries one chromosome pair. An offspring's "maternally" inherited chromosome is one of the two chromosomes (chosen uniformly, at random) belonging to the local breeder, while the offspring's "paternally" inherited

chromosome is one of two chromosomes (chosen uniformly, at random) belonging to one of the breeder's M mates (chosen uniformly, at random with replacement). The local breeder, then, will be considered "mother" to offspring produced on her patch, while the local breeder's mates will be considered potential "fathers". Of course, the local breeder will also be potential "father" to offspring produced off-site, while the local breeder's mates will also be "mother" to offspring produced on the patches they, themselves, occupy.

3. *Dispersal.* Genes found on the chromosomes carried by an individual offspring determine d, the probability with which that individual disperses from its natal patch. We assume that maternally inherited and paternally inherited genetic information contribute equally and additively to the offspring's dispersal phenotype. Thus, we treat d as the mean of two phenotypes: one that would be produced by two identical copies of the maternally inherited gene, and a second that would be produced by two identical copies of the paternally inherited gene. We assume that offspring that disperse successfully find their new patch by choosing from among the P patches in the population uniformly at random. Note there is a possibility that an offspring actually disperses to its natal patch, and in this case becomes a non-helping, individual-in-wait. Since offspring that do not disperse stay to help the incumbent breeder, the probability $1 - d$ quantifies an individual's aforementioned "tendency" toward helping.

4. *Helping.* Once the decision to help/disperse has been made, the direct benefit to the breeder can accrue. We assume that breeder mortality decreases at a rate proportional to the total number of helpers found on its patch immediately following dispersal. We assume that the constant of proportionality is small (say, of order $1/K$), and model the probability of breeder mortality with the function,

$$\mu(\bar{d}) = \exp\{-k(1 - \bar{d})\}, \quad (1)$$

where k is a positive constant that controls how quickly breeder mortality decays with increasing numbers of helpers (larger k means helping is more effective at reducing mortality), and where $\bar{d}$ is the mean d among a breeder's current brood of offspring. Given this model for breeder mortality, we model the probability of breeder survival as

$$S_b(\bar{d}) = 1 - \mu(\bar{d}). \quad (2)$$

5. *Competition for vacant patches.* If a breeder survives, then we assume it retains its patch, and breeds there again in the next time step. However, if a breeder does not survive, then we assume that competition for the vacant patch occurs among helpers (those native offspring that did not disperse) and non-helping individuals-in-wait (those non-native offspring that dispersed to the contested patch). Because a helper may compete for a breeding site more/less successfully than a non-helper due to, for example, different territory inheritance patterns (Griffin and West 2003), costs of dispersal (Ronce 2007), costs of helping itself (Heinsohn and Legge 1999), we introduce a positive constant c that allows the competitive ability of a non-helper to vary relative to that of a helper. When $c > 1$ a non-helper has a competitive advantage over a helper, when $c < 1$ a non-helper has a competitive disadvantage, and when $c = 1$ helpers and non-helpers are competitively equivalent. Mathematically, c acts as a competitive weight given to non-helpers, so that the terms $\frac{1}{\#helpers + c \cdot \#non\text{-}helpers}$ and $\frac{c}{\#helpers + c \cdot \#non\text{-}helpers}$ express the probability that a given helper and given non-helper, respectively, secure the breeding site on which they compete.

When the competition phase (event 5, above) is complete, all unsuccessful competing offspring die (this includes offspring competing on patches where the incumbent breeder survived), and the cycle repeats.

6.3 Methods of Analysis and Results

We analyzed the model using the neighbour-modulated (direct) fitness approach of Taylor and Frank (1996; see also Frank 1998; Taylor, *et al*. 2007). For convenience, our inclusive fitness treatment assumes that the number of patches (P) and brood size (K) are both very large (ideally infinite). We make the standard suite of genetic assumptions (weak selection, additive gene action, etc.), discussed in detail elsewhere (Taylor 1989; Taylor and Frank 1996; Rousset and Billiard 2000; Rousset 2004; Gardner *et al*. 2011).

We show in Appendix 6.5 that, given the life history assumptions described above, the marginal inclusive fitness of a focal juvenile with a reduced tendency to help (i.e. increased tendency to disperse) can be described by:

$$\begin{aligned} \Delta W \quad = \quad & (1 - S_b)\,[(c-1)/(1-d+cd)] && \text{I} \\ & + \ \bar{R}\, S_b' && \text{II} \\ & + r\,(1 - S_b)\,[1/(1-d+cd)] && \text{III} \\ & - r\, S_b' && \text{IV} \end{aligned} \tag{3}$$

where S_b' is the marginal survival of the breeder (this is negative, since increased dispersal leads to reduced breeder survival), $\bar{R} = 0.5$ is the average relatedness

between a juvenile and its parent, and r is the average relatedness between a focal juvenile and the average juvenile that competes on the focal juvenile's natal patch. The coefficient r can be written as $r = [(1- d)/(1 - d + cd)]\ [(1/M)+1]/4$, where M is the number of different mates a breeder has (i.e. M is the degree of promiscuity).

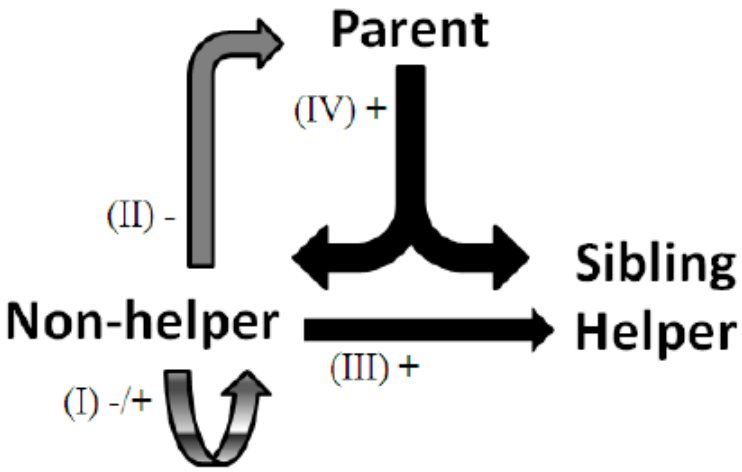


Figure 6.2 The costs and benefits of helping at the natal patch. Numerals relate to the lines of equation 3. – is a cost, + is a benefit: (I)-/+ is the direct cost/benefit associated with a decreased/increased chance of becoming a breeder; (II)+ is the benefit of increasing parent survival; (III)- is the cost of increasing parent survival, leading to decreased chances of the helper or siblings becoming a breeder; (IV)- is the cost of not dispersing, leading to increasing competition among siblings.

Equation 3 shows that the inclusive fitness effects of reduced helping (i.e. increased dispersal) are given by four components (Figure 6.2).

I. The direct fitness effect of reduced helping. That is, the difference between the focal individual's own probability of securing a breeding site in the case that it disperses (i.e. does not help), and the focal individual's own probability of securing a breeding site in the case that it does not disperse (i.e. does help). When $c > 1$ this term counts as a benefit, and when $c < 1$ this term counts as a cost.

II. The change in adult breeder survival that is associated with reduced help. This term counts as a cost, since $S_b' = -k\mu < 0$.

III. Reduced local competition due to the focal individual's increased tendency to disperse (i.e. decreased tendency to help). This is a benefit given to the average juvenile that competes on the focal juvenile's natal patch.

IV. The benefit of increased opportunity to succeed the current breeder. When there is less help available to the breeder, adult survivorship is decreased which, in turn, increases the probability that the local breeding site will become available to a juvenile competing on the patch. Again, this benefit is given to the average juvenile that competes on the focal juvenile's natal patch.

The sign of ΔW determines when reduced helping confers a selective advantage. Reduced helping is favoured when $\Delta W > 0$ and is disfavoured when $\Delta W < 0$. When $\Delta W = 0$, the population-wide level of helping is at an equilibrium. Simple algebra shows that the sign of ΔW is determined by a quadratic function of d, so it is possible to provide an exact expression for the equilibrium value of d, when it exists. That said, the expression for such a d is complicated, and its existence requires that a second complicated expression (in mathematical terms, the second expression is called the discriminant) be positive. To avoid complicated mathematical expressions that convey no biological insight, and to explore the possibility that long-term action of selection leads to non-equilibrium levels of behaviour we turned to numerical simulation.

Our numerical procedure began by assuming a set of initial conditions that covered the entire range of phenotype space evenly. In this way, the numerical procedure simulated several evolutionary trajectories simultaneously. Any given trajectory was constructed by using the most recently observed population average d to calculate ΔW. When ΔW was positive (resp. negative) the population average d was increased (resp. decreased) by an amount that was proportional to ΔW itself. These two basic steps were repeated until all trajectories of a given simulation run were sufficiently close to one another. A subset of the numerical results was confirmed with individual-based simulation of the model system (Figure 6.4; details of the assumptions and a version of the script used to generate simulation results are available on request).

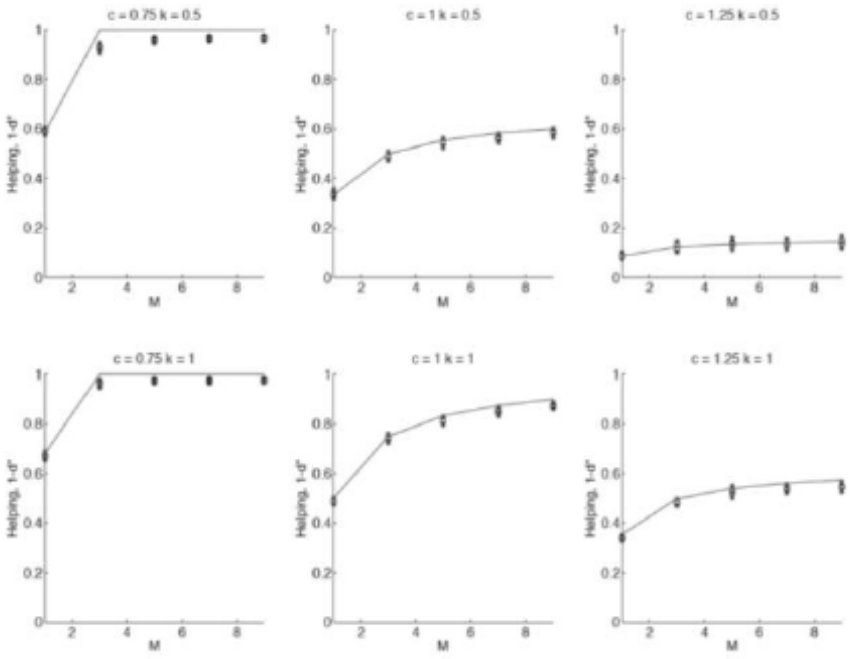


Figure 6.3 Helping and promiscuity. The predicted rate at which individuals stay and help (1-d) is plotted against the level of promiscuity (M), for different rates of the competitive ability of non-helpers (c) and the survival benefit conferred upon breeders (k). The proportion of individuals selected to stay and help decreases with increased competitive abilities of non-helpers, c, and with decreased survival benefit conferred upon breeders, k.

We found that the predicted level of helping increased with increasing promiscuity (Figure 6.3). Hence, the tendency to help juveniles born on the same patch increased with decreasing relatedness (from increasing promiscuity). The reason for this is that the rate of promiscuity only has consequences for lines III and IV in equation 3. Promiscuity does not influence either direct fitness consequences (Line I) or relatedness to parent (Line II). In contrast, lower levels of promiscuity lead to a higher relatedness to other helpers on a patch, which increases the indirect benefit of dispersing to reduce competition for relatives.

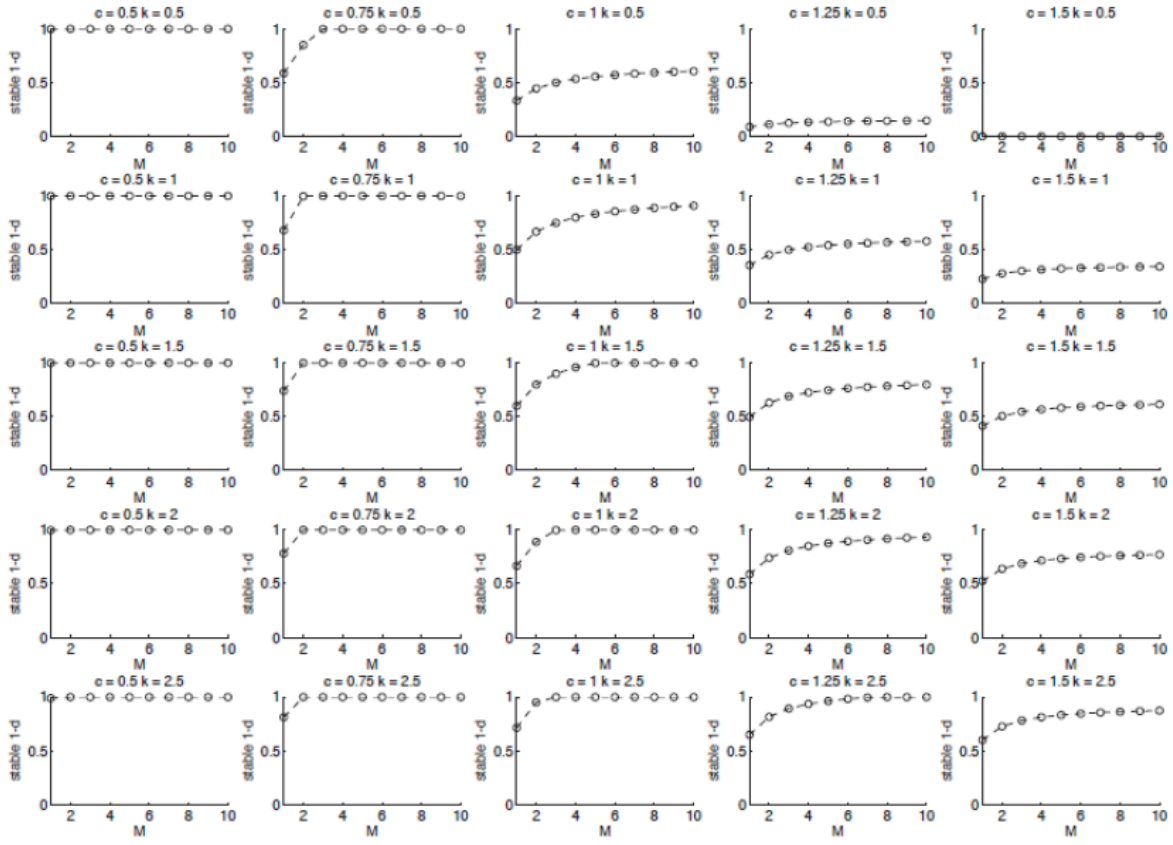


Figure 6.4 Inclusive fitness versus simulations. The predicted rate at which individuals stay and help (1-*d*) is plotted against the level of promiscuity (*M*), for different rates of the competitive ability of non-helpers (c) and the survival benefit conferred upon breeders (*k*). The results obtained using inclusive fitness theory (solid lines) and individual based simulations (dots) are in close agreement.

6.4 Discussion

Our model predicts that selection for staying to help: (1) either shows no relationship or increases with higher levels of promiscuity; (2) is decreased when non-helpers are more likely to inherit a territory (higher *c*); (3) increases when helping provides a greater survival benefit to the breeder (Figure 6.3). In addition, we found close agreement between the predictions of our inclusive fitness analyses and an individual based simulation (Figure 6.4).

Our model shows that, even in a very simple scenario, the consequences of promiscuity for the level of cooperation can be far more complicated than expected from heuristic and verbal arguments (see equation 3 and Figure 6.2). Previous theoretical arguments have focused on how promiscuity reduces relatedness to siblings, and hence reduces the indirect benefit of staying to help, leading to the prediction that promiscuity selects against helping (Figure 6.1). However, when all the population level consequences of staying to help are explicitly modelled, there can also be indirect costs of staying to help. Specifically, staying to help increases the chance that both the breeder survives, and that the helper inherits the patch if the breeder dies (Lines IV and III of equation 3 respectively). Consequently, staying to help decreases the chance that another sibling who has stayed to help will inherit the patch.

Why does our model predict that promiscuity has either no or a positive effect on selection to stay and help (Figure 6.3)? Equation 3 shows that in our model, staying to help has four fitness consequences: (I) direct cost of not dispersing to breed elsewhere; (II) indirect benefit of increased survival of parent; (III and IV) indirect cost of creating greater competition for non-dispersing siblings by reducing the likelihood that the breeder will die, and the helper staying to compete for the

breeding spot if the breeder dies. The indirect benefit of staying to help (II) is not altered by promiscuity, because the helper is always related to their parent by R=0.5. Consequently, the effect of promiscuity is driven by its indirect cost of making greater competition for siblings (III and IV). Lower levels of promiscuity lead to a higher relatedness between siblings, which favours dispersal to reduce the competition that these siblings face. Another way of looking at this is that low promiscuity does favour helping, but that the way to help siblings is to disperse away (to reduce competition), rather than to stay and help. This illustrates the general point that verbal arguments can be misleading because they focus on conspicuous traits and ignore less conspicuous consequences.

From a theoretical perspective, one of the most important points that arises from our model is that, even in a simple situation, things can be much more complicated than expected. Specifically, competition between relatives can either negate or reverse predictions that follow from simple intuition. In this respect, our paper is analogous to when Taylor (1992a) showed that limited dispersal didn't necessarily favour cooperation, because it could also lead to increased competition between relatives. A major theme since Taylor's pivotal paper has been examining exactly what kind of demographic factors lead to limited dispersal favouring cooperation (reviewed by West *et al*. 2002a; Lehmann and Rousset 2010). Analogously, we are not saying that the idea that monogamy and low levels of promiscuity will favour cooperation is wrong, but rather saying that this is only likely to hold given certain biological assumptions.

A problem with our model is that by concentrating on survival benefits, our model neglected one of the main aspects of helping that we had hoped to capture - the benefit to siblings from staying to help. Extending our model to include survival benefits to siblings did not change things (results not shown), because the strict

density dependence means that this is cancelled out by the increased competition that it also creates. We suggest that the best avenue for future research is to take an entirely different approach, and examine fecundity benefits in a population where there is not strict density dependence (Rousset and Ronce 2004; Grafen 2007; Alizon and Taylor 2008; Wild *et al*. 2009; Taylor and Grafen 2010; Wild 2011). This task will be non-trivial, as even the simple scenario we considered here, with only survival effects, and strict density dependence, led to a model that we had to solve numerically, rather than analytically. We suspect that this alternative approach would lead to a negative correlation between selection to stay to help and promiscuity, as has been found in the empirical literature (Boomsma 2007, 2009; Hughes *et al*. 2008; Cornwallis *et al*. 2010). Noteworthy here is that the species where the benefits of helping are primarily on the survival of breeders, the superb fairy-wren, also has one of the highest levels of promiscuity (Russell *et al*. 2007).

Finally, our results contradict the possibility that the influence of promiscuity on the evolution of cooperation could be an area where inclusive fitness theory makes "spurious predictions" (Nonacs 2011). Nonacs (2011) found that a population genetic simulation predicted a flat or slightly positive relationship between promiscuity and helping, and contrasted this with arguments based on kin selection theory that promiscuity should select against cooperation (e.g. Charnov 1981; Boomsma 2007, 2009; Cornwallis *et al*. 2010). However, he did not also construct an inclusive fitness model, and so he was comparing scenarios with very different assumptions, rather than comparing different theoretical approaches (for a related example on the evolution of dispersal, see pp. 117-120 of Frank 1998). We have found the same pattern as Nonacs with an inclusive fitness analysis, and also shown that when population genetic simulations are produced for the same scenario, they give the same results. This is not surprising, given the large literature showing the

equivalence of different approaches (Queller 1992; Taylor 1996b; Rousset 2004; Gardner *et al*. 2007a; Gardner *et al*. 2011). More generally, the comparison of our simulation and inclusive fitness analyses support the previous suggestion that the inclusive fitness approach will often be computationally simpler, and hence facilitate both the deriving of predictions and our conceptual understandings of why those predictions arise (p. 671 of Taylor 1996b; p. 120 of Frank 1998; p. 119 of Pen and Weissing 2000).

6.5 Appendix

In this Appendix, we develop a class-structured model (Taylor and Frank, 1996) to calculate the marginal inclusive fitness of a focal juvenile that is given in equation (3) of the main text.

If d is the probability of dispersal for a focal individual, $\bar{d}$ is the average level of dispersal from the natal patch, and d' is the population average level of dispersal. To calculate the probability that a juvenile becomes a breeder, we note that a helper will share a patch with $(1-\bar{d})K$ other helpers and $d'cK$ immigrants, thus, all else being equal, if the parent dies, a focal helper will become a breeder with probability $[(1-d)]/[K((1-\bar{d})+d'c)]$. A dispersing juvenile will be one of $d'cK$ dispersers and will share the patch with $(1-d')K$ helpers. Thus if the breeder dies, all else being equal, it will inherit the breeding site with probability $dc/[K(1-d')+Kd'c]$. Therefore, the probability a focal juvenile will inherit a breeding site is given by:

$$w_{AJ} = \left(1 - S_b\left(\bar{d}\right)\right)\frac{1-d}{K\left(\left(1-\bar{d}\right)+cd'\right)} + \left(1 - S_b\left(d'\right)\right)\frac{cd}{K\left(\left(1-d'\right)+cd'\right)} \qquad \text{(A1)}$$

In this model, we consider the fitness effect the focal individual (actor) has on its parent breeder and juvenile helper siblings (the recipients). Both these two classes of recipients have adult and juvenile fitness components. For the adult class, the adult breeder (w_{AA}) gains fitness by surviving, which occurs with probability $w_{AA}=S_b(\bar{d})$ and gains fitness by producing juveniles (w_{JA}), where $w_{JA}=KS_b(\bar{d})$. For the juvenile class, a juvenile gains fitness by inheriting a breeding site (w_{AJ}), which is given by equation (A1) and gains fitness by producing offspring if the juvenile becomes an adult breeder (w_{JJ}), where $w_{JJ}=K\,w_{AJ}$. These fitness components are combined using the following reproductive values:

$$W=\begin{pmatrix} w_{JJ} & w_{JA} \\ w_{AJ} & w_{AA} \end{pmatrix}_{d=\bar{d}=d'}=\begin{pmatrix} 1-S_b(d') & KS_b(d') \\ S_b(d')/K & S_b(d') \end{pmatrix} \tag{A2}$$

To calculate the juvenile and adult components of fitness we weight the class fitnesses by the reproductive values. The fitness of a focal juvenile, W_J, is given by

$$W_J=\frac{1-S_b(d')}{KS_b S_b(d')}w_{JJ}+w_{JA}=\frac{1}{KS_b(d')}\left(\left(1-S_b(\bar{d})\right)\frac{1-d}{(1-\bar{d})+cd'}+\left(1-S_b(d')\right)\frac{cd}{(1-d')+cd'}\right), \tag{A3}$$

and the fitness of a focal adult, W_A, is given by

$$W_A=\frac{1-S_b(d')}{KS_b(d')}w_{JA}+w_{AA}=\frac{S_b(\bar{d})}{S_b(d')}. \tag{A4}$$

The direction of selection acting upon the dispersal trait, d is given by the marginal fitness with respect to that trait weighted by the class reproductive values. This is given by:

$$\frac{\mathrm{d}W}{\mathrm{d}d'}=K\frac{\mathrm{d}W_J}{\mathrm{d}d'}+\frac{\mathrm{d}W_A}{\mathrm{d}d'} \tag{A5}$$

These derivatives may be separated into partial derivatives, describing the effect of an individual's trait value, and the effect of the average trait value of its group, on the juvenile and adult class fitnesses, using the chain rule methodology of Taylor and Frank (1996; see also Taylor 1996; Taylor *et al*. 2007). This gives $\mathrm{d}W_J/\mathrm{d}d' = \partial W_J/\partial d + \bar{r}\,\partial W_J/\partial \bar{d}$ and $\mathrm{d}W_A/\mathrm{d}d' = \partial W_A/\partial d + \bar{R}\,\partial W_A/\partial \bar{d}$, where $\bar{r} = \mathrm{d}\bar{d}/\mathrm{d}d$ is the kin-selection coefficient of (whole-group) relatedness between a focal individual and other members of the juvenile class, and $\bar{R} = \mathrm{d}\bar{d}/\mathrm{d}d$ is the kin-selection coefficient of (whole-group) relatedness between a focal individual and members of the adult class (Taylor and Frank, 1996; Pepper 2000). This yields the marginal fitness given in the main text, which uses the notation $d'=d$ and gives the relatedness between two juveniles as $r=\bar{r}[(1-d)/(1-d(1-c)]$.

Chapter 7.

Spatial structure and inter-specific cooperation: Theory and an empirical test using the mycorrhizal mutualism

7.1 Introduction

Despite their ubiquity, explaining the evolutionary persistence of cooperative mutualisms between species remains a major challenge (Sachs et al. 2004; West et al. 2007).The problem is that mutualisms are vulnerable to 'free-riders' or 'cheaters', partners who do not cooperate, but may gain the benefit of others cooperating (Axelrod and Hamilton 1981). These exploiters are common in mutualistic systems, ranging from rhizobia that fail to provide fixed N_2 to their legume hosts (Denison and Kiers 2011) to pollinators that lay eggs in hosts but defect from pollinating duties (Goto et al. 2010). Theory suggests that, without mechanisms to prevent exploitation, natural selection should favour these 'free-riders' because they pay reduced costs but still reap benefits of the partnership.

The spatial structuring or partitioning of symbiotic partners is often invoked as a mechanism for maintaining mutualisms (Frank 1994; Doebeli and Knowlton 1998; Bever and Simms 2000; Hoeksema and Kummel 2003; Foster and Wenseleers 2006; Gardner et al. 2007; Lion and van Baalen 2008; Platt and Bever 2009; Hodge et al. 2010). In particular, spatial structuring has been proposed to be important in stabilizing mutualisms in which hosts differentially allocate resources to partners varying in benefit. It has been argued that spatial structure can facilitate the evolution of cooperation by separating patches of high-quality mutualists from their

antagonistic counterparts on a single host (Bever et al. 2009). When mutualists of similar quality are clustered, a host may preferentially reward patches of cooperators and/or punish patches of cheaters. However, spatial structure could also limit the local diversity of partners, reducing the range of partners among which hosts might discriminate.

Here, we model the role of spatial structure in maintaining mutualism (interspecific cooperation), focusing on the symbiosis between the majority of land plants and arbuscular mycorrhizal (AM) fungi. This symbiosis, which primarily involves the exchange of carbohydrates from plants for mineral nutrients from the fungal partners, offers ideal experimental opportunities to study spatial structuring: a single host root can be colonized by several AM fungal species that range in benefit from mutualistic to antagonistic (Hoeksema et al. 2010). Although spatial structure has been suggested to play an important role in the maintenance of the mycorrhizal mutualism (Chanway et al. 1991; Wilkinson 1998; Bever et al. 2009; Hodge et al. 2010), observations from the field and lab experiments do not consistently reveal strong structuring of different fungal communities (van Tuinen et al. 1998; Jansa et al. 2003; Alkan et al. 2006) but rather distantly related AM fungi are found to intermingle on a small spatial scale in host roots (Jansa et al. 2003). Whether hosts discriminate among intermingled fungal partners, allocating resources preferentially to the best ones, has been the subject of an on-going debate (Fitter 2006; Kiers and van der Heijden 2006; Bever et al. 2009; Helgason and Fitter 2009; Smith et al. 2009). Recent manipulative work supports the hypothesis that hosts reward fungal symbionts at a fine scale, preferentially allocating C to small patches of hyphae when they deliver more P resources to their host (Kiers et al. 2011). The reciprocal was also found to be true, with fungi preferentially allocating P to small patches of roots that delivered more C. This bi-directional control allows the mutualism to function like a

biological market (Noë and Hammerstein 1995), in which partners offering the best rate of exchange are rewarded. However, it is not yet known how fungal spatial structuring influences this partner control.

To generate predictions, we first determined theoretically how the relative fitness of 'high-quality' and 'low-quality' mutualists should depend on the extent to which they are spatially structured. If plants can only preferentially allocate resources to fungi at a relatively coarse level, such as sections of the root system, we predict that spatial structuring will increase the relative fitness and proliferation of the high-quality mutualist. In contrast, if plants preferentially allocate resources at a much finer level, such as groups of cortical cells or at the level of the arbuscule (the site where nutrient transfer occurs; Parniske 2008), then we predict that spatial structuring will decrease the relative fitness and proliferation of the high-quality mutualist. These opposite predictions arise because spatial structure can limit the local availability of partners, reducing the extent to which hosts can reward more cooperative partners on a fine scale. We then test these competing predictions by using a multi-generational selection experiment, manipulating spatial structuring to determine how this influences the relative fitness of the low-quality AM fungal species. In the past, it has been difficult to track AM fungal fitness in mixed cultures over multi-generations. We resolved those constraints by developing specific quantitative molecular markers for our focal AM fungal species, allowing us to follow their relative fitness - when in direct competition - over multiple generations. We manipulated spatial structure by either mixing the soil or leaving it non-mixed after every successive generation.

7.2 Predicting the consequences of spatial structure

Our aim is to provide a general model, that makes qualitative predictions, and which could also be applied to other mutualisms, rather than a more specific model that makes quantitative predictions. We examine the situation where there is a host interacting with two mutualists (in our case mycorrhizal fungal species) that differ in mutualistic quality. Our model defines the high-quality fungal species as a mutualist that transfers sufficient resources to the plant to be considered beneficial, and the lower-quality species as one that transfers fewer resources to the plant, (less beneficial/less cooperative). We assume that both fungal mutualists gain some baseline fitness benefit (w_0) by interacting with the plant, regardless of the degree of spatial structuring, but as the high-quality fungus transfers more resources, it experiences a higher energetic cost c. The fitness benefit experienced by the high-quality fungi depends upon the plant's ability to differentiate between high- and low-quality fungi.

We assume, as has recently been shown empirically, that plants preferentially allocate resources to higher-quality mutualists (Kiers et al. 2011). We consider two evolutionary scenarios, which differ in the scale at which plants can preferentially allocate resources (coarse or fine-scale rewarding). The fungi occur in patches, where a patch is a section of the root network on a single plant. If plants can only adjust their level of resource allocation between different sections of the root network, then differential allocation is at a "coarse" level, between patches. In contrast, if plants are able adjust their level of resource allocation at a finer scale, for example towards specific groups of cortical cells or at the level of the arbuscule or interface with one fungus (Kiers et al. 2011), then differential allocation is at a "fine level", within patches.

We examine the influence of population structuring, by considering the extent to which fungal strains are mixed between and within root patches. At one extreme, in highly structured populations, we would expect to see each patch (associated with a single host plant) dominated by one or a small number of fungal strains, but to find different strains in different patches. At the other extreme, in highly unstructured (panmictic) populations, we would expect to see a mixture of strains in each patch, and a similar mix in all patches. This corresponds to Wright's (1935) F_{st}, a measure of population structuring, going from high (1.0) to low (0). Mixing the soil would reduce the fungal population structure, resulting in a lower F_{st}. We denote the fitness effect of population structuring by $f(F_{st})$ where f increases monotonically with F_{st} and the sign of the function f differs for high and low quality fungal strains and is determined by the level of control the plant has over resource allocation. This function need not be the same for the two types of fungal species or for the different levels of plant control. To highlight this we use $f_{Hc}(F_{st})$, $f_{Hf}(F_{st})$, $f_{Lc}(F_{st})$ and $f_{Lf}(F_{st})$ for the high ($_H$) and low ($_L$) quality fungal strains under coarse ($_c$) and fine ($_f$) control respectively.

When there is coarse control, preferential allocation by hosts is to different parts of the root network. Under this scenario, plants will be best able to discriminate between high- and low-quality fungi, when each part of the root network (patch) tends to contain only one type of fungi, but different types occur in different patches. Higher population structuring (higher F_{st}) will allow greater discrimination and hence favours the high-quality fungal strain. Conversely, low quality fungi do better if patches are more evenly mixed so the plants will be less able to discriminate against them. Therefore fitness of a high-quality fungus (w_{Hc}) is given by $w_{Hc} = w_0 - c + f_{Hc}[F_{st}]$ and the fitness of a low quality fungus (w_{Lc}) is given by $w_{Lc} = w_0 - f_{Lc}[F_{st}]$. This leads to the prediction that, under coarse control, the relative fitness of low-quality

mutualists ($v_{Lc} = w_{Lc} / w_{Hc}$) decreases as populations becomes more structured (fig. 7.1). Conversely, we predict that spatial structuring (high F_{st}, low mixing) under coarse control favors high-quality mutualists and hence selects for cooperation.

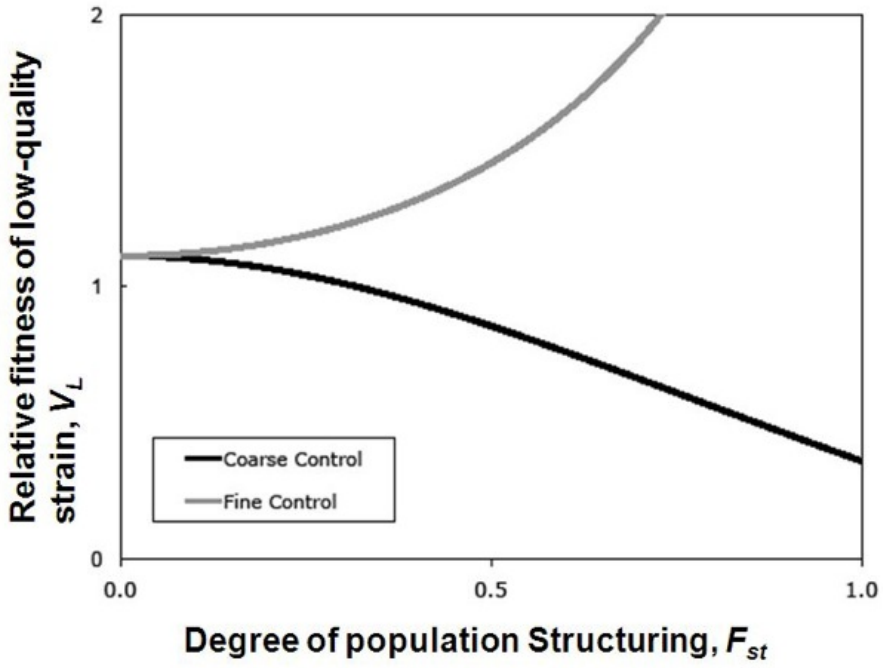


Figure 7.1 The effect of spatial structure (F_{st}) on the relative fitness of low-quality (less cooperative) fungal species (v_L). F_{st}, is a measure of population structuring; high structuring ($F_{st} \rightarrow 1$) implies no mixing, low structuring ($F_{st} \rightarrow 0$) implies mixing. The black line represents coarse level control (v_{Lc}, which assumes a single plant interacts with patches of fungi on its root system) and the grey line represents fine level control (v_{Lf}, which assumes plant interacts with individual strains of fungi at the level of the arbuscule). Fine control predicts that high spatial structuring (no mixing, $F_{st} \rightarrow 1$) favours low-quality strains whereas course control predicts high spatial structuring (no mixing, $F_{st} \rightarrow 1$) favours high-quality strains. Here, we illustrate the simplest case, where the function f is the same for high and low quality fungi and for coarse and fine control, but this need not be the case. We assume the fitness function, f of F_{st} takes the form $f = x(\pm F_{st})^y + z$. Fig. 7.1 is plotted for w_0=1, x = 0.5, y = 2, z = 0 and the cost to the high-quality fungus, c = 0.1. Qualitatively, the result that the low-quality mutualist is favoured at high Fst under fine control and disfavoured under coarse control, holds for all values of x, y, z and $c > 0$.

Under fine control, we assume that the plant can allocate resources at a finer scale, towards specific fungal strains within patches of a single root-system. Consequently, plants will be best able to discriminate between low- and high-quality fungi when

both types are evenly mixed (low F_{st}). Because the fitness of high-quality fungi will be positively correlated with the extent to which plants can discriminate, the fitness of a high-quality fungus when there is fine control (w_{Hf}) is given by $w_{Hf} = w_0 - c - f_{Hf}[F_{st}]$. Conversely, if patches are composed solely of low-quality partners, the plant is less able to preferentially choose the high-quality fungi, hence the fitness of a low quality fungus (w_{Lf}) is given by $w_{Lf} = w_0 + f_{Lf}[F_{st}]$ as lower-quality mutualists will be more successful when there is low mutualist diversity (high F_{st}). These fitness equations lead to the opposite prediction from the coarse control scenario. When there is fine control, they predict that the relative fitness of a low-quality mutualist ($v_{Lf} = w_{Lf} / w_{Hf}$) increases as populations become more structured (fig. 7.1). Here, spatial structuring favours low-quality fungi and selects against cooperation. Although the extent to which this is the case, would be reduced if plants can also move resources between patches (i.e. a combination of fine and coarse control).

7.3 Material and methods

7.3.1 Culture of plants and fungi

For our experimental test, we used two closely related AM fungi of the highly cosmopolitan sub-genus Glomus Ab (Schwarzott et al. 2001), *Glomus intraradices* Schenck & Smith (but see Stockinger et al. 2009 for discussion of *G. intraradices* re-classification) and *Glomus custos* Cano & Dalpé. The use of closely-related AMF allowed us to focus only on fungal cooperative strategy while excluding differences associated with radically contrasting life history traits (Denison and Kiers 2011). Both strains were isolated from Southwest Spain but represented two ends of the mutualistic continuum. At comparable colonization levels, inoculation with the "high-quality mutualist" *G. intraradices* results in significantly higher host biomass

production and phosphorus (P) content compared to the "low-quality mutualist" *G. custos* in both monocot and dicot plant species (Kiers et al. 2011). We chose *Plantago lanceolata* L. as a host because it is a model plant species in AM fungal research and is readily colonized and highly responsive to a broad range of AM fungal taxa (Maherali and Klironomos 2007).

7.3.2 Experimental setup

Our experiment consisted of three fungal treatments: *G. intraradices* alone, *G. custos* alone, and an equal mixture of both ("combined"). We planted seedlings in 2.6 L pots, grown from seeds that were surface-sterilized with 10% sodium hypochlorite. We inoculated roots of seedlings with ~1500 spores and 1.0 g of *in vitro* root material of either *G. intraradices* or *G. custos* (provided by Mycovitro S.L. Biotechnología ecológica, Granada, Spain). These cultures were originally started from single spores, and thus considered to exhibit high genetic uniformity (but see Angelard et al. 2010). In the "combined" treatment, we halved inoculum amounts so that seedlings received equal amounts of both AM fungal species. Plants grew in autoclaved, nutrient-poor dune sand (as in: Scheublin et al. 2007), with the following characteristics: pH: 7.2; OM: 0.2 %; P: 0.3 mg/kg ($CaCl_2$-extracted); N-total 190 mg/kg. We maintained soil humidity at 70% water holding capacity and biweekly, we added nutrients, 8 ml of 1/2 P Hoagland solution (Arnon and Hoagland 1940) per pot. We randomized pots biweekly on benches, and they grew for 12 weeks per generation under semi-controlled light intensity with a 16 h light and 8 h dark rhythm.

We grew plants for three consecutive seasons. After the first generation, we randomly assigned the soils to either a "mixed soil" treatment (reduced spatial structuring i.e. low population structure) or "non-mixed soil" treatment (control

spatial structuring i.e. high population structure). In both treatments, we removed the central root with a core (2 x14 cm), and replaced it with an equal core of sterile soil (± 50 g). For the mixed treatment, we removed all soil and roots from the pots and cut the roots into ~1 cm pieces. Contents were then mixed and transferred back into their respective pots separately. In the non-mixed treatment, we left the soil and roots outside the core intact. We treated all pots with an excess of demineralized water to standardize for soil compactness, and then planted a new seedling. We replicated each fungal treatment x soil treatment 12 times for a total of 72 pots. In the beginning of the third generation, one plant in the high-quality/non-mixed treatment died resulting in only 11 biological replicates.

7.3.3 Plant and fungal measurements

After each generation, we harvested the aboveground plant material, which was oven dried it at 70°C for 72 h, and weighed. After the first generation, we determined P content by the molybdate blue ascorbic acid method following acid digestion (Watanabe and Olsen 1965) and N and C contents by dry combustion on an Elemental Analyzer (NC2500, ThermoQuest Italia, Rodana, Italy). For each pot, we removed four evenly spaced soil cores (same as used for removing central root system) and which were replaced by sterilized sand. Half the cores were freeze-dried overnight and the other half used to measure the mycorrhizal colonization using trypan blue staining and the magnified intersection method (McGonigle et al. 1990; 100 intersections per replicate).

To compare the relative abundance of competing fungal species in the "combined" fungal treatments, we performed DNA analyses after each generation and measured the fungal abundances in the single species treatments after generation three. We pooled roots from two cores per replicate, weighed and ground them in a

microcentrifuge tube using a 4 mm glass bead in a FP120 bead beater (ThermoSavant, Holbrook, USA; 4.0 m s^{-1}, 2 x 10 s). We spiked each sample with 2 x 10^{10} copies of a plasmid containing the cassava mosaic virus sequence motif. This served as an internal standard in order to exclude variations in isolation efficiency or pre-analysis DNA degradation (see also Appendix 7.67.). We extracted DNA from the resulting mixture using the DNeasy Plant Mini Kit (Qiagen, Hilden, Germany) following the standard protocol and eluted in 100 µl of the provided elution solution. We analyzed the abundance of each species by quantitative PCR primers targeting the mitochondrial large subunit (mtLSU) rRNA gene (see: Kiers et al. 2011). These primers were specifically designed to quantify the mtLSU copy numbers of each species, and have been found to be highly specific and accurate (for design, optimization and validation see Appendix 7.6).

7.3.4 Statistical analyses

We performed all statistical analyses using SPSS version 17.0. To establish the effects of the factor "species" (with levels: "high-quality", "low-quality", "combined") on plant biomass, we tested N content, P content, and hyphal colonization of roots by MANOVA (Hyphal colonization of roots was arcsin-transformed to meet assumptions). Then, in order to assess which of these response variables contributes most to differences between inoculation treatments, we performed a Discriminant Analysis. To test whether response variables were significantly different between plants inoculated with different species we performed a one-way ANOVA for each response variable separately, followed by Tukey's b post-hoc test and pair-wise ANOVAs for each of three species treatments.

We performed separate ANOVAs for shoot biomass differences in mixed and non-mixed pots between first and final generation for the "combined" treatment. We

performed an ANOVA on colonization responses (i.e. percentage root colonized by hyphae, arbuscules or vesicles as indication of differences in the abundance of fungal nutrient transfer structures (e.g. arbuscules) versus storage structures (e.g. vesicles, see Johnson et al. 2010) in the final generation. For the single species treatments, we also compared relative abundance of arbuscules compared to vesicles for both mixed and non-mixed treatments, which were ln transformed to meet assumptions for ANOVA. We analyzed qPCR-derived abundance data of each species for the "high-quality", "low-quality", and "combined" fungal treatment between mixed and non - mixed soil treatments using the non-parametric Mann-Whitney U test because the requirement for a normal data distribution of ANOVA was not met. Ranked abundance of each strain did not significantly deviate in their homogeneity of variance - which is a requirement of the Mann-Whitney U test (Ruxton and Beauchamp 2008). In case of "no detection", we replaced species abundance by the detection level, after confirming that these cases did not appear to represent statistical outliers.

7.4 Results

7.4.1 Benefits conferred by fungal species to host plants

Plant growth and nutritional status after the first generation confirmed that the two fungal species differed in mutualistic benefits conferred to their hosts. This is consistent with previous plant growth results that utilized these fungal species (Kiers et al. 2011). MANOVA analysis indicated a significant treatment effect on plant responses (Pillai's trace, $V = .98$, $F_{8,134} = 4.64$, $P < .001$), and Discriminant Analysis suggested an especially high difference between the low-quality mutualist versus the high-quality mutualist and combined treatment (Appendix 7.6). Above ground

biomass contributed most strongly to this separation, followed by shoot P content, hyphal colonization and lastly shoot N content (Appendix 7.6).

Inoculation by the high-quality mutualist (*G. intraradices*) resulted in significantly higher aboveground biomass ($F_{1,46}$ = 18.15, P =.001) and shoot P content ($F_{1,46}$ = 10.92, P = .002) than the low-quality mutualist (*G. custos*; fig. 7.2). The plants that were inoculated with both species ("combined treatment") did not differ from plants that were only inoculated with the "high-quality" species *G. intraradices* (Biomass: $F_{1,46}$ = 0.40, P = .531; P content: $F_{1,46}$ = 0.20, P = .661; fig. 7.2). We found no significant differences in plant N content ($F_{2,69}$ = 0.34, P = .710) suggesting that biomass differences are predominantly the result of an improved P nutritional status and/or lower C costs. The hyphal colonization levels of the high-quality and low-quality species in the single-inoculation treatments were high (80 ± 1.8% SEM versus 71 ± 3.3%, respectively; all error estimates onwards represent 1 SEM unless stated otherwise), confirming that the host was able to form effective associations with both partners. Although these colonization levels differed significantly ($F_{1,46}$ = 6.67, P = .013), the high colonization (above 70%) in each treatment and its low contribution to treatment separation in Discriminant Analysis (see Appendix 7.6) suggest that differences in mycorrhizal colonization were not the primarily cause for differences in host benefit.

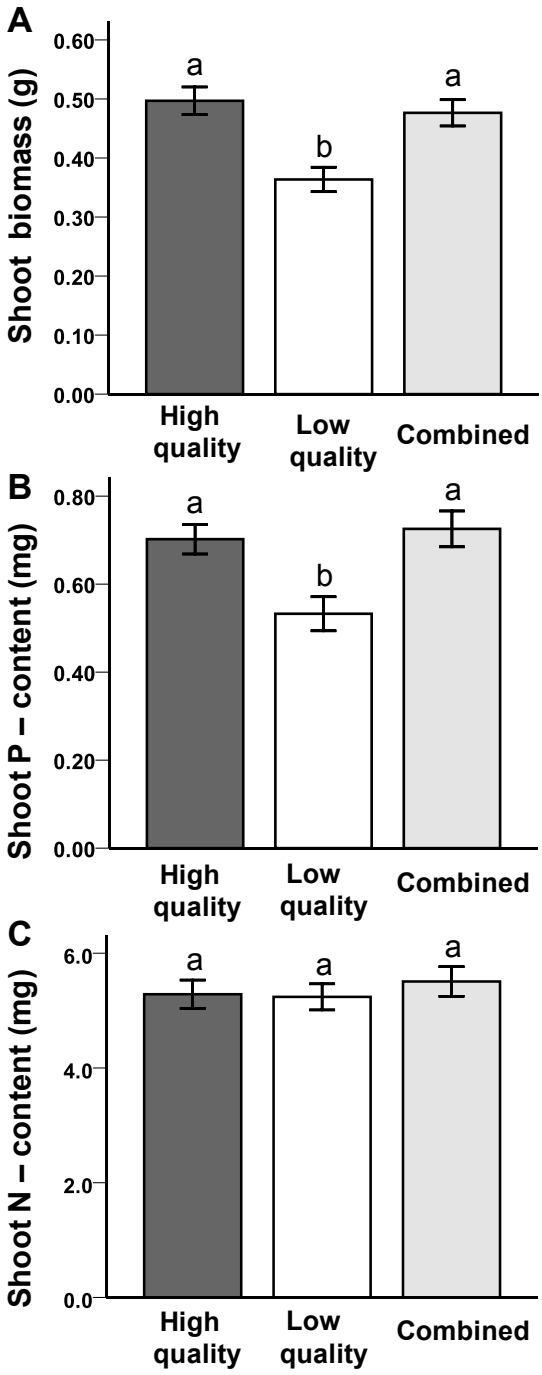


Figure 7.2. Effect of fungal treatments on shoot biomass and P content. Shoot biomass (*A*) and P content (*B*) of plants from generation one were significantly higher for plants inoculated with the high-quality species than with the low-quality species. For N content (*C*) there were no significant differences between treatments. When inoculated with both AM fungal species ("combined treatment"), the host plant biomass did not differ significantly from plants that were only inoculated with the high-quality species. Bars represent mean ± 1 standard error. Different letters represent significant differences according to Tukey' b post-hoc test.

7.4.2 Mixed versus non-mixed soil, effects on AMF mutualists

We found that soil mixing significantly affected the success of competing high- and low-quality mutualists. In the combined treatment, the abundance of the low-quality mutualists was significantly reduced after two consecutive generations of soil mixing ($Z_{1,22}$ = -2.83, P = .004, fig. 7.3). This effect was not due to a higher tolerance to soil mixing by the high-quality species. In the single species treatments, mixing reduced the abundance of the high-quality species (from 8.2 (± 1.4) $\cdot 10^7$ to 4.4 (± 0.8) $\cdot 10^7$ marker copies g^{-1} root; $Z_{1,21}$ = -2.28, P = .023), whereas there was no significant negative effect of mixing on the abundance of the low-quality species in the single species treatment. There was even a marginal positive effect of soil mixing on the low-quality species when grown alone (from 6.2 (± 0.4) $\cdot 10^6$ to 10.5 (± 2.3) $\cdot$ 10^6 marker copies g^{-1} root; $Z_{1,22}$ = -1.67, P = .083).

7.4.3 Plant benefit under mixed and non - mixed conditions

When both high- and low-quality fungal mutualists were present, soil mixing had a marginally significant positive effect on host shoot biomass compared to non-mixed conditions ($F_{1,22}$ = 3.40, P = .079; fig. 7.4*A*). A positive effect of soil mixing on the symbiosis is further supported by the finding that in host roots, under mixed conditions, more arbuscules and fewer vesicles were formed than under non-mixed conditions, whereas there was no difference in hyphal colonization (arbuscules: $F_{1,22}$ = 8.77, P = .007; vesicles: $F_{1,22}$ = 7.92, P = .010; hyphae: $F_{1,22}$ = 0.03, P = .860; fig. 7.4*B*). A high relative abundance of arbuscules (structures important for nutrient transfer) compared to vesicles (structures for storage) has been shown to be a useful indicator for mutualistic quality of AM fungi (Johnson et al. 2003; Johnson et al. 2010). This higher arbusculae abundance could indicate increased colonization by the high-quality species because we found that high arbuscule abundance (compared to

vesicle abundance) is a consistent identifiable trait of the high-quality species under both mixed ($F_{1,22} = 42.28$, $P = .001$) and non-mixed conditions ($F_{1,21}$ 40.38, $P = .001$).

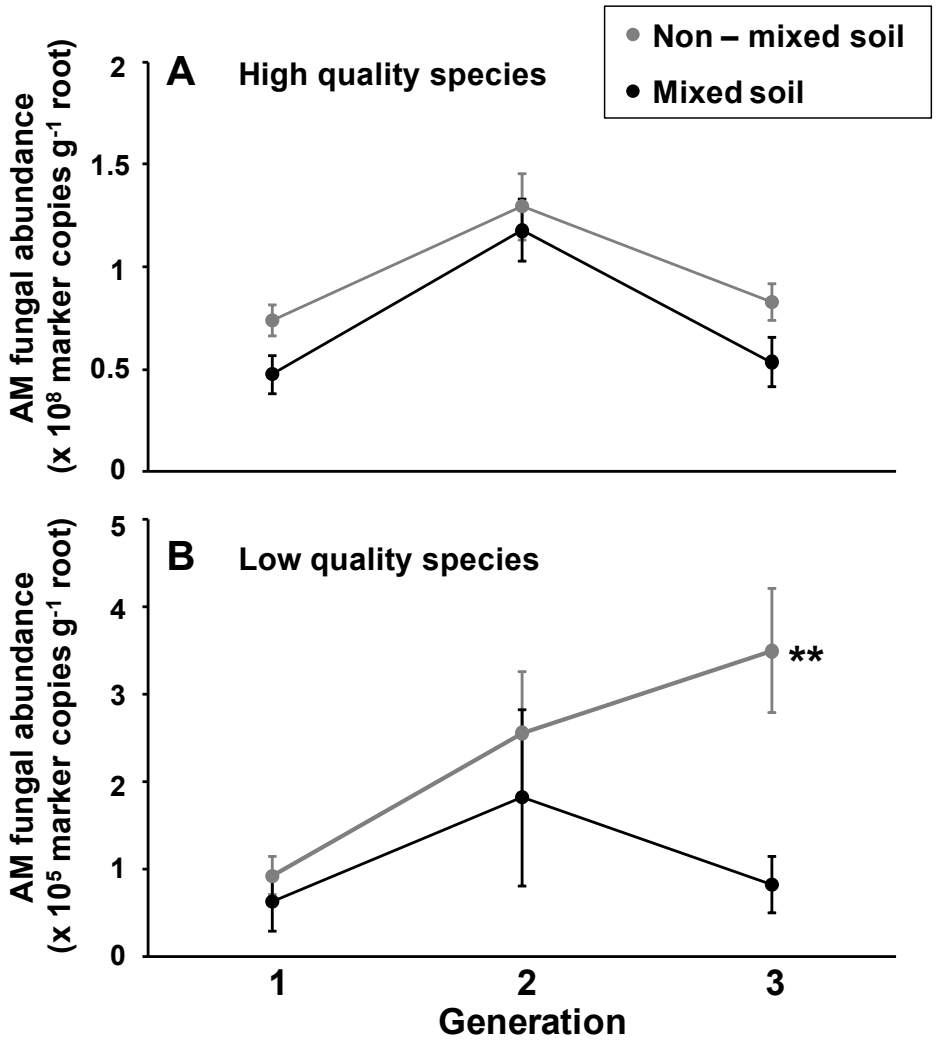


Figure 7.3. Effect of soil treatments on abundance of the fungal species, (*A*) high-quality *Glomus intraradices* and (*B*) low-quality *Glomus custos* in the "combined" treatment for each. In generation three, abundance of the low-quality species after mixing was significantly reduced compared to the non - mixed control. Error bars represent 1 standard error. Significant differences between mixed versus non - mixed are indicated with asterisks (** = $P \leq .01$). Note that mixing occurred twice (generation 1 to 2 and generation 2 to 3).

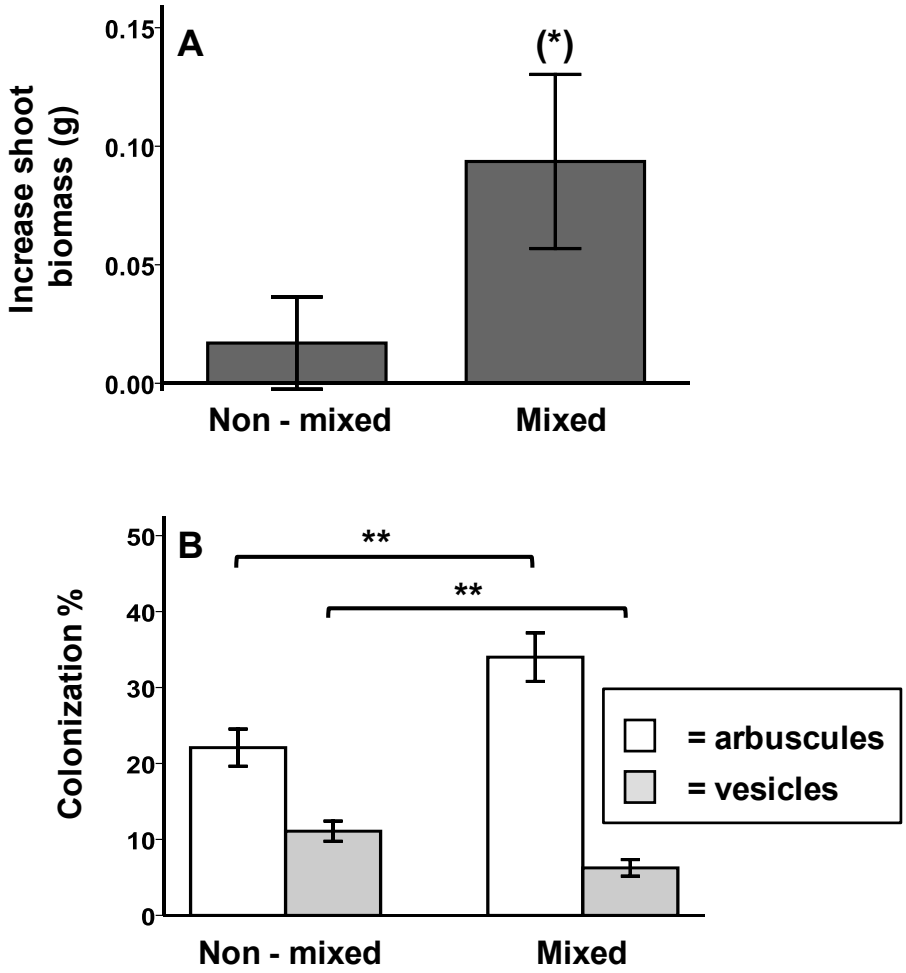


Figure 7.4 Effect of soil mixing on plant biomass and mycorrhizal colonization in "combined" treatment. Host plant increase in biomass from first to third generation when both AMF species were present (*A*) was marginally significantly larger (i.e. (*), $P = .079$) when soil was mixed. This effect was associated (*B*) with a significant increase of arbuscular and decrease of vesicular colonization. Asterisks indicate significant difference (** = $P \leq 0.01$). Hyphal colonization did not differ between treatments (not shown). Bars indicate mean ± 1 standard error.

7.5 Discussion

Spatial structure is frequently assumed to favour cooperation within mutualistic interactions (Frank 1994; Doebeli and Knowlton 1998; Bever and Simms 2000; Hoeksema and Kummel 2003; Foster and Wenseleers 2006; Gardner et al. 2007; Lion and van Baalen 2008; Platt and Bever 2009; Hodge et al. 2010). Other models (e.g. (Sherratt and Roberts 2002) have found that cooperation can be maintained even in

the absence of spatial structure depending on investment-response rules. Our model predicts that spatial structuring can indeed have contrasting effects on selection for cooperation, depending on the scale (fine or coarse) at which hosts enforce cooperation (fig. 7.1). We tested these predictions using the arbuscular mycorrhizal symbiosis as a model system. We found that when we mixed the soil (reduced spatial structuring) in the combined treatment, this decreased the success of the low-quality species (fig. 7.3*B*). In contrast, mixing soil had no significant effect on the success of the high-quality species (fig. 7.3*A*).

There is a large theoretical literature examining how spatial structure would influence the evolution of cooperation. These include models for cooperation both between (see above) and within (reviewed by Lehmann and Rousset 2010; Nowak et al. 2010) species. Within species, population structuring keeps relatives together, and hence can favour cooperation through kin selection (Hamilton 1964), as long as this is not negated by increased competition between relatives (West et al. 2002b; Lehmann and Rousset 2010). Between species, population structure can generate spatial correlations in the tendency to cooperate, which can favour cooperation (Frank 1994). In all cases, a general feature is that population structuring usually favours cooperation, because of its tendency to create genetic correlations either within or between species (for an exception, see Hauert and Doebeli 2004). We gain the opposite prediction, because in our model, population structure influences the evolution of cooperation in a very different way. Specifically, it alters the extent to which one partner is able to preferentially reward more cooperative partners.

We used soil mixing as a mechanism to decrease the extent to which fungi populations are structured (i.e. to produce a lower F_{st}). Our model predicts that if plants can enforce cooperation at a fine-scale then lower spatial structuring will be relatively detrimental to the success of low-quality species (fig. 7.1). This effect arises

because it will lead to patches containing both high- and low-quality mutualists, which facilitates the extent to which plants can differentially allocate resources to high-quality mutualists. Our empirical results are consistent with this model prediction (fig. 7.3*B*), although there may be other explanations (see discussion below). We also found that soil mixing led to a small increase in benefit for the host itself (fig. 7.4*A*). This benefit likely arose because the host was associating predominately with the high-quality species.

Our work suggests that if hosts are able to enforce cooperation on a fine scale then high spatial structuring of the symbiont community may not be critical for stabilizing the partnership, at least in the model-system tested here. Recent work has highlighted potential mechanisms plants may employ to enforce cooperation at the fine-scale assumed in our model (e.g. level of arbuscule). For example, stunted arbuscule morphology has been described when arbuscular-specific plant phosphate transporters were knocked out (Javot et al. 2007), suggesting that when fungal symbionts fail to deliver P, plant hosts may reduce C allocation locally (Parniske 2008) or even digest individual arbuscles (Kobae and Hata 2010). Other work has pointed to the role of lysophosphatidylcholine (LPc), a compound that may help hosts to sense P concentrations, potentially allowing hosts to evaluate the amount of P delivered via the mycorrhizal pathway (Bucher et al. 2009). Now more work is needed to establish how plants physically control C transport across individual interfaces.

Are there alternative explanations for how soil mixing could favour the spread of the high-quality symbiont? Other differences between the species, such as ability to tolerate disturbance, could explain why the high-quality symbiont responds better to mixing. However, we can tentatively reject this hypothesis because the low-quality species appears to have greater inherent tolerance of mixing. In the single species

treatments, mixing reduced the abundance of the high-quality species (decrease of ~50%, $P = .023$), whereas mixing had no significant effect (even a small mean increase of ~60%, $P = .083$) on the low-quality species.

A second way in which soil mixing could favour one symbiont over the other would be through changes in the competitive dynamics between the two species not related to host C-rewards. For instance, variation in life-history traits could mean that one species proliferates more, or earlier than the other, depending on our experimental conditions. However, by choosing closely-related species, we reduced the variation associated with diverse life-history strategies typically found across genera (Denison and Kiers 2011). Future work should aim to extend these types of multi-generational tests to more fungal species pairs so broader conclusions can be drawn.

One important assumption of our work was that the control treatment, without soil mixing, showed a higher level of spatial structuring than the mixed treatment. Spatial structuring of AM fungal communities can arise from differential soil colonization patterns of strains (Smith et al. 2000; Oehl et al. 2005) or clustered sporulation (Jansa et al. 2002). In line with previous recommendations (e.g. Bever et al. 2009), we examined fungal competition under relatively realistic spatial conditions, i.e. the structuring that originates during one or two generations of a successful symbiotic interaction (non-mixed) compared to a reduction of that structuring (mixed). We assume that even our non-mixed soil treatment was exposed to some mixing from root forging and other processes. No soil in nature would ever be free from mixing, especially at the fine-level of the arbuscule. Manipulative tests of spatial structure should be extended to the field where natural processes such as non random host-AMF associations (Öpik et al. 2009), burrowing animals (Mangan and Adler 2002) and fungal aggregation in response to plant signaling cues (Yoneyama et al. 2008) can both increase and decrease spatial structure of symbiont communities.

Is there evidence for strong spatial structuring of symbiont communities in nature? While studies have demonstrated spatial structuring at the field or plot level (Mummey and Rillig 2008), the few studies that have examined spatial distribution of AM fungal species within individual plant roots, find fungal strains to be relatively well-mixed (Alkan et al. 2006). This could be because relatively large root fragments (± 5 cm) were examined (Jansa et al. 2003) or small fragments were pooled (Wolfe et al. 2007). The smallest root fragments studied thus far to our knowledge (~1 cm), did not reveal strong structuring (van Tuinen et al. 1998; Janouskova et al. 2009). A quantitative approach (e.g. Alkan et al. 2006) in which strain abundance (rather than presence-absence) is measured in small root fragments, will be the most successful at determining the degree of spatial structuring of AM fungal communities.

We found that the low-quality mutualist was not completely replaced by the high-quality mutualist. If the experiment had been continued for more generations, the abundance of the low-quality mutualist would have likely decreased further. However, our results suggest that the high-quality mutualist does not completely replace the other. One explanation is that host plants are physically unable to exclude colonization of a particular AM fungal species. Another explanation is that some fungi may provide benefits not measured (e.g. defense against pathogens; Herre et al. 2007), or only provide benefits under different conditions, and thus even 'putative' low-quality mutualists are retained in low abundances (Smith et al. 2009; Palmer et al. 2010).

Can our model be extended to other systems? It would be interesting to test our model predictions in the legume-rhizobia mutualism, where fine-scale rewarding (at the level of the individual nodule) is known to operate (Kiers et al. 2006; Simms et al. 2006; Oono et al. 2011). Like in the fungal system, we would predict that soil mixing has the potential to be positive (or at least not negative) on the success of the high-

quality strain by exposing the host to a greater diversity of potential partners. The only complication is that 'mixed nodules' (containing more than one rhizobial strain) are known to occur with some frequency in the field (e.g. 12-32%; Moawad and Schmidt 1987). There is currently no evidence that hosts can differentially control resource distribution to strains within a single nodule (Denison and Kiers 2011). So if soil mixing also increased the frequency of 'mixed nodules', this could undercut any positive benefit for the host plant (Kiers et al. 2002; Friesen and Mathias 2010); Till and no-till agricultural systems could be useful for field-level experimental tests of our predictions, especially in cases where high-quality fungal and rhizobial inoculum have been introduced. We predict that tillage could have a positive effect in promoting cooperation in the mycorrhizal symbiosis, but this effect is likely overridden by life history differences among fungi in respect to their ability to handle extreme disturbances (Verbruggen and Kiers 2010).

More generally, whether spatial structure will stimulate or decrease mutualistic cooperation depends on the biology of the interaction. If a mutualism depends upon partners cooperating to produce a beneficial common good, without a mechanism to reward more cooperative partners (or punish less cooperative partners), then increased spatial structure favors cooperation, because it increases relatedness amongst symbionts leading to a shared interest between symbiont and host (Frank 1994; Bever and Simms 2000; West et al. 2002a; Foster and Wenseleers 2006). This is analogous to how spatial structuring favours cooperation within species (e.g.: Griffin et al. 2004; Diggle et al. 2007; Cornwallis et al. 2009; Lehmann and Rousset 2010). In other symbiotic interactions, like the mycorrhizal symbiosis, mixing may increase the potential suite of partners available to host plants. This could increase host plant benefit as long as plants can discriminate among partners at a fine-scale. Research is now needed to (i) identify potential physiological mechanisms that would allow

plants to control allocation processes, and (ii) test these dynamics in the field to determine the actual effect of symbiont mixing under natural conditions.

7.6 Appendix

7.6.1 Quantitative real-time PCR (qPCR) markers design, validation, optimization, and Calibration

Design of qPCR markers (primers with hydrolysis probes) for specific detection and quantification of *Glomus intraradices* and *Glomus custos* (isolates obtained from Mycovitro S.L. Biotechnología ecológica, Granada, Spain) targeting the mitochondrial large subunit RNA gene.

DNA was extracted from AMF spores produced monoxenically with carrot root organ cultures, or from the colonized carrot roots from the same cultures, using the DNeasy Plant Mini kit (Qiagen, Hombrechtikon, Switzerland), following manufacturer recommendations. The final volume of the DNA preparations from spores was 20 µl (instead of 100 µl recommended by the manufacturer) to maximize DNA concentrations before PCR. The roots samples were eluted with 100 µl of the elution buffer. Monoxenic fungal cultures were provided by A. Bago, H. Bucking and M. Hart.

DNA was subjected to PCR amplification of the mitochondrial large ribosomal subunit (mtLSU) RNA gene with following primer pair combinations, according to Börstler *et al* (2008): RNL11- RNL17, RNL1-RNL14, or RNL1-RNL15. The PCR was carried out using Taq PCR Core kit with CoralLoad reaction buffer (Qiagen), using 25 µl PCR reaction volume, 1µM concentration of each primer, and 38 cycles (denaturation at 95°C for 10s, annealing at 50°C for 90s and amplification at 72°C for

90s). Amplified DNA fragments were cloned into a blue-script vector (pGEM-T Easy vector system; Promega, Dübendorf, Switzerland) and sequenced at Microsynth (Balgach, Switzerland). The sequences were individually edited and the clones re-sequenced if quality of the reads proved insufficient. The identity of the sequences was revealed by BLAST search (http://blast.ncbi.nlm.nih.gov/Blast.cgi) to sort out contaminant sequences (e.g., bacteria, unspecific amplifications of other genome regions etc.). Several partial sequences of the mitochondrial large subunit of the different AMF species were obtained, which were deposited in the GenBank under the accession numbers HQ706096-HQ706103.

These sequences were aligned with other mtLSU sequences available in the GenBank (e.g., *Glomus intraradices*, *Glomus proliferum*, *Glomus clarum*) and at least two combinations per AMF species of species-discriminating qPCR primers with associated hydrolysis probes were designed using the AlleleID version 6 software (Premier Biosoft International, Palo Alto, California, USA). Care was taken to target mtLSU regions coding for the ribosomal RNA, i.e. avoid putative introns described recently by Thiery *et al* (2010). Specificity of the primers and fluorescent probes was confirmed with a BLAST search and the oligonucleotides (primers and dually labeled hydrolysis probes, labeled with fluorescein at 5`-end and BHQ-1 quencher at 3`- end) were then synthesized in Microsynth. The primers were purified by preparative HPLC and the probes by preparative polyacrylamide gel electrophoresis before lyophilization. Both primers and probes were diluted with PCR-grade water to achieve 25 µM concentrations, aliquoted (20 µl each) and frozen at -20°C.

Marker	Target	Sequences 5`→3` (forward primer, reverse primer, hydrolysis probe)	Nr cycles	Denaturation (°C / s)	Annealing (°C / s)	Amplification (°C / s)
intra mt5	*Glomus intraradices*	TTTTAGCGATAGCGTAACAGC, TACATCTAGGACAGGGTTTCG ***FAM***-AAACTGCCACTCCCTCCAT ATCCAA-***BHQ1***	65	95 / 10	60 / 10	72 / 1
cust	*Glomus custos*	TCTAACCCCAGAAATGTATAG, AAGGACTGCCTTGTGTTC ***FAM***-ATACAATAATGGGCAATC AGACATATCGT-***BHQ1***	65	95 / 10	62 / 15	72 /1
CMV	*Cassava Mosaic Virus*	CGAACCTGGACTGTTATGATG, AATAAACAATCCCCTGTATT TCAC *FAM*-CACCAGGCACCAACA ACGACCATT-*BHQ1*	45	95/10	50/30	72/1

FAM – flourseceín; ***BHQ1*** – flourescece quencher

Table 7.1: qPCR markers for specific quantification of *Glomus intraradices* and *Glomus custos* by means of measuring gene copies of the mitochondrial large ribosomal subunit of the respective AMF species. Also represented are the markers for partial Cassava Mosaic Virus DNA sequence (Genbank accession number: AJ427910; inserted in vector pUC19) quantification, used as a control in the experiment.

7.6.1 Marker selection, optimization of cycling conditions, cross-reactivity testing

First, the markers were tested for their specificity under low stringency cycling conditions (denaturation at 95°C for 10 s, annealing at 52°C for 30 s, and amplification at 72°C for 5 s). In this assay, DNA extracts from *Medicago truncatula* roots colonized by the different AMF (3 individual samples per each AMF species) were used as templates. Second, markers showing greatest specificity towards their target species (either no cross-amplification with other species or the greatest difference in Cq value between target and non-target species) were selected for further optimization (Table 7.1). Stringency of cycling conditions was then stepwise increased for each of the markers so as to achieve a complete avoidance of

amplification with non-target samples (see Table 7.1 for details of the optimized cycling conditions). The markers were confirmed to amplify their target AMF species and to avoid non-target AMF species as well as the plant DNA (tested on spores and colonized roots of *Medicago truncatula* and *Daucus carota* roots). All qPCR assays were carried out in 9 µl reaction format, using the LightCycler® 2.0 instrument, LightCycler TaqMan chemistry (LightCycler® TaqMan® Master, cat. Nr 04735536001) and 20µl-Lightcycler® glass capillaries (cat. Nr 04929292001). Final concentration of the primers was 0.5 µM each and the final concentration of the hydrolysis probe was 0.11 µM. 2.25 µl DNA template (i.e, sample) was included in each reaction.

Marker system	**Detection limit (Cq)**	**Threshold mtLSU gene copy concentration (copies μl^{-1})**
intra mt5	37.62	199
cust	35.6	10

Table 7.2: Detection limits and minimal detectable target gene concentrations of the two qPCR assays

7.6.3 qPCR calibration, detection limits

Two to four individual plasmid preparations per AMF species were used for calibration of the qPCR markers. These plasmids were carrying fragments of mtLSU of the respective AMF species with 100% sequence match to the region amplified by the markers (confirmed by sequencing). Plasmids were isolated from overnight cultures of the transformed *E.coli* JM109 (Promega), grown on LB medium supplemented with Ampicillin (100 µg ml-1), using the Miniprep procedure (Sambrook *et al*. 1989). The plasmids were linearized using EcoRI+ (Fermentas, Le Mont-sur-Lausanne, Switzerland) digestion, incubating the samples at 37°C for 2h and then at 65°C for 20 min. The concentration of the DNA was then measured by PicoGreen fluorescence assay (Invitrogen, cat. Nr P7589), using LighCycler 2.0 at

45°C and measuring the emission at 530 nm. The concentration of plasmid copies per unit of sample volume was calculated according to Jansa *et al* (2008), by knowing the concentration of DNA in each sample, length of insert (176 bp for *G. intraradices* and 438 bp for *G. custos*) , length of vector (3015 bp), and assuming molecular weight per nucleotide double-stranded DNA to be 660 Da. Plasmid preparations were serially diluted (5fold and 10fold) to achieve a range of plasmid concentration from few billions through less than 1 per microliter. These were used for establishment of:

1. Calibration curves for conversion of the qPCR detection cycle (Cq) to gene copy concentrations

2. Assessment of detection limits of the qPCR markers.

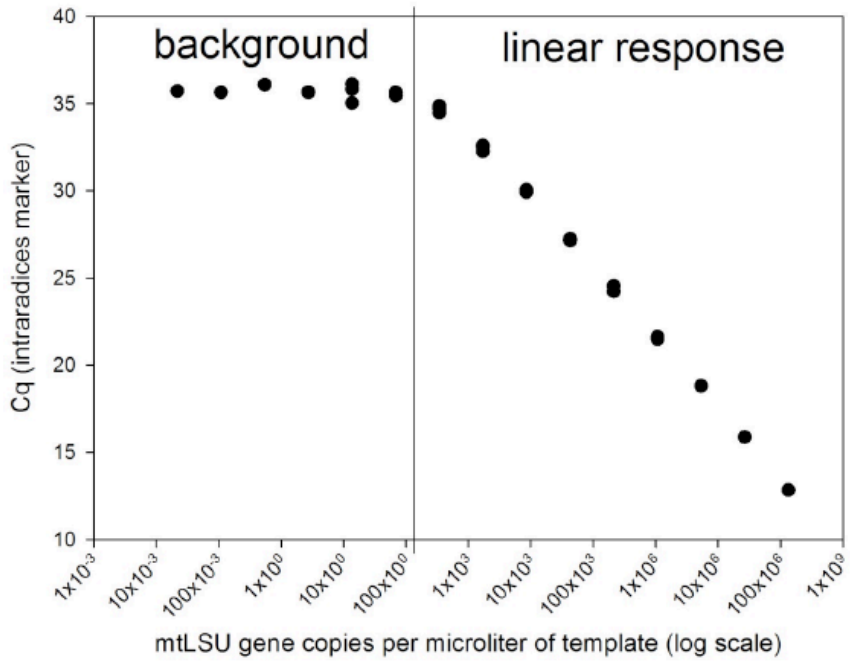


Figure 7.4 Typical response of qPCR assay to DNA template dilution. Linear response region contains values used for calibration of the qPCR assay and the background region has been used for assessment of detection limit of the qPCR assay.

Namely, the Cq is typically negatively and linearly correlated to the log-transformed template concentrations (Fig. 7.4, linear response region), until the detection limit of

the assay is reached and the Cq becomes independent of the further dilution (Fig. 7.4, background region), or there is no response at all.

Linear response region of each of the calibration assay has been used to derive equations allowing conversion of Cq values to mtLSU gene copies per unit volume of the template (Fig. 7.5)

Detection limits were derived from the background region of the qPCR response curve as follows:

$$DL = AV_{Cq(back)} - 3 \times SD\left(AV_{Cq(back)}\right),$$

Where DL stays for detection limit of the assay (Cq value), $AV_{Cq(back)}$ stays for mean of the Cq values in the background region and SD($AV_{Cq(back)}$) stays for standard deviation of this mean. The detection limits of the three marker systems and corresponding threshold mtLSU concentrations are given in Table 7.2.

These assays were then used for assessment of mtLSU gene copy concentrations in DNA samples taking into account any dilutions of the template during the sample processing.

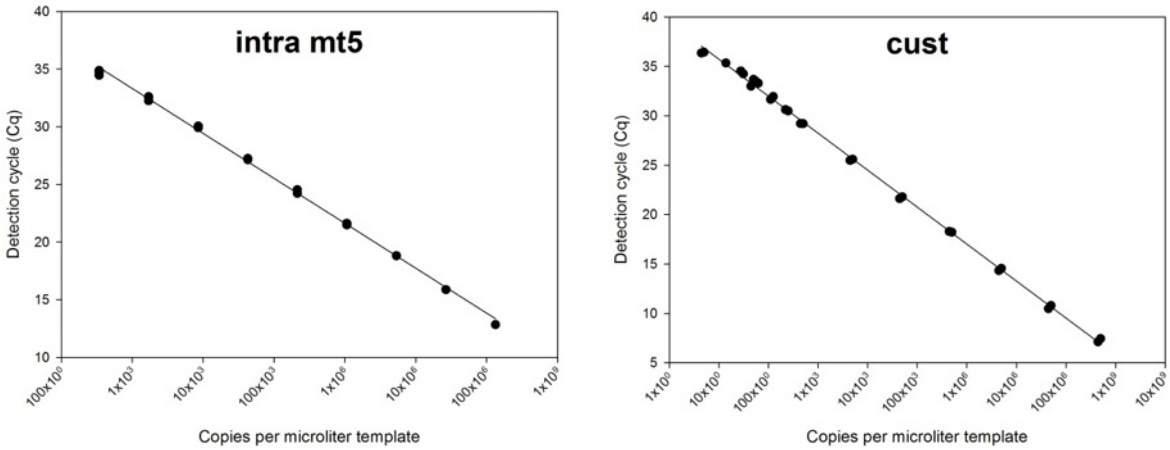


Figure 7.5 Calibration curves for the qPCR assays targeting mtLSU of *Glomus intraradices* and *G. custos*. Equations for conversion of the qPCR signal to mtLSU copy concentrations in template are given for each assay. CP stands for number of target gene copies per microliter template. (*Glomus intradadices*: R^2 = 0.998, $CP = 10^{((47.33655877 - Cq)/4.22866042)}$; *Glomus custos*: R^2 = 0.999, $CP = 10^{((39.38265200 - Cq)/3.74460986)}$.

Chapter 8. Discussion.

Each thesis chapter contained its own detailed discussion. The aim of this chapter is to summarise the main conclusions, highlight avenues for future research and consider some of the general issues my work raises about social evolution research, specifically when applied to the evolution of cooperation in humans.

8.1 Chapter summaries

In Chapter 2, I examined whether knowing ones dispersal status could affect the evolution of social behaviours. This is because, in general, non-dispersers are more highly related to their neighbours whilst dispersers are less related. I extended Taylor's infinite island model (Taylor 1992a), to compare the case when individuals cannot adjust their behaviour depending on their dispersal status, with the case when they can. I found that helping was more readily favoured among non-dispersers and harming more readily favoured among dispersers. I also showed that harming by dispersers evolved more readily than does helping by non-dispersers. This is because zero relatedness (on average) is guaranteed by one's own dispersal, irrespective of the population level of dispersal, whereas the relatedness achieved by not dispersing is nonzero only insofar as one's social partners are also non-dispersers. In the future, I would like to build upon this work to examine the coevolution of dispersal and social behaviours in more detail, in particular the coevolution of dispersal and harming.

In chapter 3, I extended earlier work by Frank (1995, 1996b, 2003, 2009) to examine the evolution of cooperation by policing. The original aim was to investigate how

different population structures alter selection for enforcement mechanisms within groups. However, the focus became more general when I examined Frank's model and found qualitatively different results were obtained by relaxing some potentially unrealistic implicit assumptions. One of these implicit assumptions was that the individual cost of investment into policing fell as selfishness increased, and it was this which generated the model's result that policing favours high levels of selfishness. By relaxing this assumption, I found that policing was favoured only at intermediate levels of relatedness and that when it evolved it led to a reduction in selfishness. I also relaxed the assumption that policing perfectly recovers the group resources that are lost due to selfishness and found this modification meant full policing was disfavoured. I considered the impact of demography on the coevolution of policing and cooperation and showed that policing is maintained more readily than it invades (Figure 3.4). A future goal is to model the evolution of inequality by extending Frank's (1996b) model of policing when resources vary, to consider the coevolution of selfishness and mechanisms that could enforce an inequitable distribution of resources in a group.

In chapter 4, I examined how competition between groups affected selection for social behaviours. Conflict, or warfare between groups is seen as a potentially important mechanism favouring the evolution of altruism in humans (Choi and Bowles 2007; Bowles 2006; 2009), however the evolutionary logic underpinning this idea was not clear as I identified several inconsistencies in their model assumptions. Using this previous work as a starting point, I investigated what forms of sociality are favoured when groups compete. I showed that in contrast with previous results, group competition could favour the evolution of any social behaviour within a group so long as it improves the group's ability to compete. In the future, I would like to investigate the evolution of group competition as a means to maintain within-group

cooperation. This builds upon Hamilton's (1996) idea that if relatives cannot avoid bunching in groups, then the group itself must expand at the expense of others because to be effective, altruism must put offspring in competition with non-altruists, not bunch them into wasteful competition with their own kind.

Chapter 5 extends the model developed in chapter 3 to investigate the idea of reproductive levelling. Reproductive levelling is the term used to refer to policing mechanisms in the human literature and has been suggested as an important factor in the emergence of egalitarian societies (Bowles 2006; Boehm 1997, 2001). Previous models of reproductive levelling examined how culturally inherited traits (e.g. norms for sharing resources) could aid the evolution of genetic cooperation (Bowles 2006). However this work assumed a culturally inherited norm for reproductive levelling was already present in the population at no cost; it did not model how it might evolve in the first place. I considered the coevolution of a genetically inherited trait for competitiveness and a culturally inherited trait for reproductive levelling and demonstrated that costly, culturally-inherited traits for reproductive levelling could be favoured in such a way that they select for genetically-inherited self-restraint. I showed that reproductive levelling is most strongly favoured when group members are genetically unrelated but share the same culture. Therefore, the gene-culture coevolution of reduced competitiveness and reproductive levelling may explain why the simplest societies tend to be both cooperative and egalitarian. However, this result comes with an important caveat: it is unclear if cultures evolve in the way this model assumes. More work is needed to model cultural change realistically and for this reason, one of my research priorities is to use the formal Darwinism approach (Grafen 1999, 2007) to investigate whether or when the dynamics of cultural transmission can to lead to a maximisation principle.

In chapter 6, I developed a class-structured model using an inclusive fitness approach to consider how parental promiscuity affected selection on juveniles to stay and help their parents breed. Previous authors (Hughes *et al.* 2008; Boomsma 2009) have suggested that low promiscuity or monogamy may favour the evolution of cooperative breeding as the relatedness between parent and offspring and the relatedness between siblings will be similar, meaning that the fitness benefits of raising siblings is potentially the same as the fitness benefits of reproducing independently. By contrast, I found that promiscuity had very little effect on selection for helping. This was because of a trade-off: staying to help benefitted the parent by increasing their survival, but it led to increased competition with siblings for the breeding position. This competition meant that when there was monogamy (high relatedness), cooperative breeding was disfavoured as the best way to aid relatives was by dispersing to avoid competing with them. In nature, the cost of competition between siblings must be decoupled from the benefits of cooperative breeding, for example via delayed dispersal, as there is ample evidence to suggest that monogamy does favour the evolution of cooperation (Hughes *et al.* 2008; Boomsma *et al.* 2009; Cornwallis *et al.* 2010). This work illustrates the value of formalising verbal arguments as even straightforward ideas, like the monogamy hypothesis, can yield surprisingly complex results.

In chapter 7, I presented the results of some collaborative work I undertook with plant science researchers from the Netherlands. They wanted to know whether the soil-tilling practices used in industrial agriculture might affect the stability of the mutualism between plants and their arbuscular mycorrhizal (AM) fungal symbionts. I developed a simple model to consider how changing the soil's spatial structure (i.e. how mixed the soil is) interacts with the scale at which a plant has evolved to reward its fungal partners. The model showed that the effect of tilling on plant fitness

depended on the scale at which the plant rewarded its mutualistic partners.

This work led to an idea for some future work concerning mutualisms where a single host has a very large number of symbionts, such as the squid-bioluminescent bacteria mutualism. In these types of mutualism, if an empiricist wishes to estimate the inclusive fitness of the symbionts with the host, they must take great care as the relevant relatedness they need to measure will depend upon the scale at which the hosts meters out rewards or sanctions (Foster and Wenseleers 2006). At the extremes, if the host rewards or sanctions at the level of an individual symbiont cell, relatedness $r=1$ whereas if they reward or sanction at the symbiont population level then, in the limit, relatedness $r \rightarrow 0$). As high symbiont relatedness limits selection for free-riding, this suggests that mutualisms will be more stable if hosts possess adaptations that allow them to use targeted rewards or sanctions.

8.2 General themes

8.2.1 Implicit assumptions matter

Three of my chapters resulted from relaxing potentially unrealistic implicit assumptions in existing models. As many people who read theory papers are not mathematically inclined, they must trust that the theoretician's written description reflects their maths accurately. If a model's results rely upon unexplained assumptions, it is bound to cause problems. First, if the maths and written arguments are inconsistent, it obscures why cooperation occurs in the model and second, it makes testing the model's predictions extremely difficult. If empirical researchers try to test such as model, they are likely to measure unsuitable parameters and may draw incorrect conclusions from their observations (for a related discussion from an

experimental perspective, see Appendix section 2). Implicit assumptions have led to widespread confusion in the human evolution literature where there are many instances of verbal arguments not describing properly how a model works (reviewed by West *et al.* 2011). For example Lehmann *et al.* (2007c) evaluated the implicit assumptions in two models that claimed to examine the coevolution of cooperation and punishment (Bowles and Gintis 2004; Gintis 2000). Contrary to the papers' verbal arguments, Lehmann *et al.* (2007c) showed that the cooperative trait in these models merely acted as a tag for who carried the punishment trait; cooperation in itself did not generate the models' results. Thus these models actually examined the evolution of spiteful greenbeards, which is a biologically unrealistic explanation for the evolution of cooperation in humans. I plan to continue to highlight the problem of implicit assumptions in models that examine the evolution of cooperation in humans. I also plan to establish collaborations with empiricists who study human cooperation so as to formalize their research questions using simple, testable models, which may be presented alongside their quantitative results (akin to the project which resulted in chapter 7).

8.2.2 It is often easier to be nasty than nice

My work illustrates that punishment and enforcement mechanisms often evolve more readily than unenforced cooperation. In chapter 2, I found that harming by dispersers evolves more readily than helping by non-dispersers, as it is easier to guarantee the low relatedness needed for harming than it is the high relatedness needed for helping. Unenforced cooperation evolves when relatedness is high, but as I illustrated in chapter 3, even with high relatedness there is significant scope for selfishness and the presence of enforcement mechanisms such as policing can still yield additional benefits. Furthermore, as Bourke (2011 and see my review in

Appendix section 1) emphasizes, once present, cooperation is not guaranteed to persist; if selfish individuals are not checked, they can make groups collapse or even cause species to go extinct (Rankin and López-Sepulcre 2005; Rankin *et al*. 2007).

My work also highlights why it is important to distinguish between evolutionary classifications for cooperation and the proximate details of how fitness effects are achieved. For example, in chapter 3, I showed that policing is altruistic in an evolutionary sense, however the proximate details of how groups enforce the distribution of resources need not pleasant. Similarly, in chapter 4, whilst social behaviours associated with group competition are always altruistic or mutually beneficial, I showed they could be anti-social and reduce the partial fitness of fellow group mates. For this reason, care is needed when engaging with research on human cooperation where the aim is typically to explain more proximate definitions of altruism, such as the altruistic act of helping another when there is no prospect of a reward. In Chapter 1 and in West *et al* (2011, reprinted in Appendix section 3), I explain that as we cannot know precisely what selective pressures favoured the behaviours we see today, it is impossible to say whether the adaptations which underpin specific behaviours, such as blood donation, voting, or recycling, which are described as altruistic, were favoured due to direct or indirect benefits.

8.2.3 Understanding how group and population structure affects the evolution of cooperation in humans is an important area for future research

My work highlights that details of a population's structure, such as dispersal patterns (chapter 2), group size and dispersal rates (chapter 3), the intensity of competition between groups (chapter 4) and the degree of mixing (chapter 7) are important determinants of whether or when social behaviours evolve. The importance of population structure for the evolution of cooperation is well known (Hamilton 1964,

Taylor 1992a) and there is an extensive body of work in the biological literature which examines how different facets of a population's structure affects the evolution of cooperation, such as: overlapping generations (Taylor and Irwin 2000; Irwin and Taylor 2001; Grafen 2007; Lehmann *et al*. 2007a; Taylor, *et al*. 2007), trans-generational social behaviours (Lehmann 2007), grouped (or 'budded') dispersal (chapter 1; Gardner and West 2006; Lehmann *et al*. 2006), sex-biased dispersal (Johnstone and Cant 2008) competition between groups (chapter 4; Hamilton 1996; Bowles 2006; 2009; Lehmann and Feldman 2008b) and changes in group size (Kokko *et al*. 2011). Unfortunately, most of this research is unknown to researchers concerned with the evolution of cooperation in humans. Therefore, an important task for the future is to take this body of work and make it relevant to human cooperation researchers by developing specific models reflecting human demography that generate testable predictions.

8.2.4 The evolutionary importance of culture is unclear.

It is frequently assumed that cultural evolution is able to explain cooperation in cases where genetic selection cannot (Richerson and Boyd 2004; Henrich 2005, 2008). Whilst humans do appear to be unique in the extent to which social learning allows them to adapt to local conditions, it is unclear what role culture plays in the evolution of cooperation. First, contrary to verbal claims (Henrich and Boyd 1998; Henrich and Gil-White 2001; Henrich and McElreath 2003), whether or not cultural evolution promotes cooperation depends upon the exact mechanism of cultural transmission (Feldman *et al*. 1985; Lehmann *et al*. 2007c, 2008; Lehmann and Feldman 2008). For example, prestige-biased transmission, where individuals copy a single successful individual in their group, may encourage the spread of harming behaviours if, in the short term, defectors appear more successful (Lehmann *et al*. 2008). Second, as

discussed in chapter 5, it is unclear whether culture itself possesses the inheritance properties necessary for natural selection to operate and lead to any maximising dynamics. It may be more accurate to view culture as a phenotypic tool where change is primarily guided by clever minds, which themselves are the product of natural selection.

One reason why cultural evolution receives so much attention appears to stem from misconceptions about the scope of genetic natural selection to favour cooperation (see chapter 1 and Appendix section 3 for more discussion). It is widely assumed that evolutionary explanations for genetically inherited cooperation are limited to kin-selection and reciprocity. This means the role of genetic selection in the evolution of behaviours that stabilize cooperation in large groups or between non-kin is often neglected. Furthermore, the idea that culturally inherited institutions can enhance or encourage cooperative behaviours is receiving ever-more attention from think tanks and politicians (Bowles 2008; Willetts 2010). There is a real danger that in the near future, the results of evolutionary models that rely on untested or unrealistic assumptions will be used to inform public policy.

8.3 Conclusion

To conclude, the rapidly expanding field of human cooperation research offers great opportunities for synergistic collaborations between biologists, psychologists, economists, anthropologists, archaeologists and social scientists. One particularly challenging, yet rewarding, feature of human cooperation research is that because it spans so many disciplines, successful research is only possible when people from different traditions themselves learn how to cooperate. In order to facilitate future collaboration and the progression of the field's research, there are several major

outstanding tasks. These include addressing the misconceptions about evolutionary theory before they become accepted wisdom, determining whether or when cultures evolve and how culture influences levels of cooperation and considering whether it is the insights from these diverse disciplines may be drawn together in a way that is appropriate for use by policy makers.

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Appendix

This Appendix contains the other publications resulting from my DPhil:

Section 1. El Mouden, C. (2011) Life: social to its core. *Evolution and Human Behaviour* 33(1): 79-80.

This is a book review I wrote of Bourke, A. F. G. (2011). *Principles of Social Evolution.* OUP.

Section 2: Harrison, F. H. and El Mouden, C. (2011). 'Money for nothing, cooperation for free: exploring the effects of working for endowments on behaviour in standard economic games', PLoS ONE 6(11): e27623.

This is an experimental paper examining whether individuals behave differently in economic games if they must first earn their endowment by performing dull or difficult tasks.

Section 3: West, S.A., El Mouden, C. and Gardner, A. (2011). 'Sixteen common misconceptions about the evolution of cooperation in humans', *Evolution and Human Behavior* 32(4): 231-262.

This paper reviews recent work on the evolution of cooperation in humans and highlights common misunderstandings regarding how natural selection operates and the conditions under which cooperation can be favoured.