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Effects of Gene-Environment Interactions on the Evolution of Social Behaviours

Vonica J. Flear, *The University of Western Ontario*

Supervisor: Lachance, Marc-André, *The University of Western Ontario*

Co-Supervisor: Hobson, Keith A., *The University of Western Ontario*

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Abstract

Inclusive fitness models in sociobiology emphasize the importance of relatedness, *R*, and synergy, *S* when exploring the evolution of social behaviours. Very few models explicitly consider 'role', or environmental stimuli, influencing the expression of behaviours, and none consider genetic-environment interactions where genotype predisposes individuals to certain roles. I propose a third key variable for inclusive fitness models, *Q*, which describes the overlooked potential bias in the genetic composition of individuals exposed to an environmental stimulus – here referred to as 'role'. I describe a model built from Price's formula which can be presented in a 'Hamilton's Rule' format. I consider classic social behaviour models using this format, and find that the inclusion of gene-environment interactions dramatically changes the results. This, in conjunction with the increasing evidence supporting gene-environment interactions in eusocial caste determination, suggests that current inclusive fitness models may be missing key details about the evolution of social behaviours.

Keywords

Inclusive fitness, kin selection, Price equation, social evolution, modelling, Hamilton's Rule, conditional expression, conditional strategies, Hawk/Dove/Bourgeois game, altruism, eusociality, gene-environment interactions.

Summary for Lay Audience

To understand the evolution of social behaviours, evolutionary theorists must consider the direct fitness effects of a behaviour – how the behaviour influences the reproductive success of those enacting it, or 'actors' – and the indirect fitness effects of the behaviour – how the behaviour influences the reproductive success of individuals interacting with the actors. The combination of these two fitness components, called inclusive fitness, can be described in a simple mathematical form: $RB - C > 0$. When the relatedness, *R*, between the actor and recipient of the behaviour, is multiplied by the effect, *B*, that the actor's behaviour has on the recipient, minus the effect of the actor's behaviour on itself, *C*, is greater than zero, the behaviour is favoured by selection. This mathematical expression, commonly referred to as Hamilton's Rule, helps to make clear how behaviours such as altruism can evolve. Hamilton's Rule does not explicitly consider behaviours that are expressed conditionally depending on environmental factors however, nor does it consider the interaction of environmental factors and the genes involved in a behavior's expression. For my thesis, I develop a mathematical model that expands upon Hamilton's Rule to include conditionally expressed behaviours and the possibility for an interaction between the genotype of actors and the environmental stimuli to which they are exposed. Using my model, I explore a classic social behaviour example and find that when conditional expression and gene-environment interactions are considered, the outcome of this example change dramatically. Current sociobiological models that do not include these factors may not be getting the full picture of how social behaviours can evolve.

Co-Authorship Statement

The work involved in this project was conducted by Vonica Flear under the tutorship of Geoff Wild. The project was initiated by Graham Thompson. The publications to arise from this work will be coauthored by Graham Thompson and Geoff Wild.

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List of Variables and Symbols

$\propto$: proportional to

β_{FI}: partial regression of a focal individual's fitness on its own genotype, which can also be written as $\beta_{W,Z|z}$

β_{Part}: partial regression of a focal individual's fitness on the genotype of a social partner, which can also be written as $\beta_{W,Z|z'}$

β_{WZ} : regression coefficient of fitness and genotype, used in a modified form of the Price equation. Defined as β_{WZ} = Cov(W,Z)/Var(Z)

Δp: change in allele or gene frequency

$\Delta_s p$: change in allele or gene frequency due to selection alone

ΔZ_i: average difference in genotype between *i* and *i's* offspring

A_0: label for wild-type allele(s)

A_1: label for mutant allele(s)/focal allele(s)

B: bourgeois strategy

B: regression coefficient used in Hamilton's Rule to describe the fitness effect a focal individual's behaviour causes to its social partner, colloquially called "benefit"

C: regression coefficient used in Hamilton's rule to describe the fitness effect a focal individual's behaviour causes to itself. Alternatively: symbol used to represent "cost" in Maynard Smith's hawk/dove/bourgeois game

Cov: covariance

D: dove strategy

$D_{Obszz'}$: observed residual fitness values, calculated as the difference between individual fitness and expected average fitness, $W_{FI} - \mathrm{E}[W]$. The subtext label *zz'* refers to the pair type of interest, for example D_{Obs01} refers to the residual fitness of an A_0 focal individual with an A_1 partner

$D_{Fitzz'}$: residual fitness values fitted to regression coefficients β_{FI} and β_{Part}, calculated as $\beta_{FI}(z - E[Z]) + \beta_{Part}(z' - E[Z])$

E[_]: expected value of _, calculated as the weighted average, or mean, of the variable of interest. Examples: E[W] = expected fitness, E[Z] = expected genotypic value, or genotype

F0: parent generation

F1: daughter generation

F_0: frequency of pairings which consist of two A_0 carriers

F_1: frequency of pairings which consist of one A_0 carrier and one A_1 carrier, i.e. frequency of heterogeneous pairings

F_2: frequency of pairings which consist of two A_1 carriers

$f_{zz'}$: frequency of pairings in which P1 possesses genotype *z* and P2 possesses genotype *z'*, for example f_{10} indicates P1 is an A_1 carrier, and P2 is an A_0 carrier

H: hawk strategy

HDB: hawk/dove/bourgeois

h: frequency at which mixed wild type strategists adopt strategy H

i: identifier for one of several individuals. For example, in a dataset of 5 individuals, for the 5th individual $i = 5$.

MSE: mean squared error

N: placeholder for a yet undefined number of individuals in a population

P1: label for role 1

P2: label for role 2

p: frequency of the focal allele in the parent generation of a population

p': frequency of the focal allele in the daughter generation of a population

Q: asymmetry quotient. Defined as the proportion of heterogeneous pairs in which the A_1 carrier is in the P2 role, calculated as $f_{01}/(f_{10} + f_{01})$ or f_{01}/F_1

R: coefficient of relationship between an actor and recipient of an action/behaviour. Defined various ways (see Chapter 1) but for the purposes of this thesis, *R* is defined as $R = Cov(z, z')/var(Z)$

S: synergistic effect, described as the proportion of A_1 carriers with an A_1 social partner, calculated as F_2/p

V: value of a resource, used in Maynard Smith's hawk/dove/bourgeois game

$v1_{zz'}$: fitness outcome to P1 after an interaction with P2, given P1 has genotype *z* and P2 has genotype *z'*

$v2_{zz'}$: fitness outcome to P2 after an interaction with P1, given P1 has genotype *z* and P2 has genotype *z'*

Var: variance

W_i: fitness of individual *i*

WT: wild type

z: focal individual's genotype, or genotypic value, at a focal locus

z': genotype, or genotypic value, of focal individual's social partner at a focal locus

Z_i: genotypic value of individual *i*

Z_i': average

1 Introduction

Natural selection does not act solely on the individual. Discussed *ad nauseum* by evolutionary theorists for decades, the concept of indirect selection was touched upon even in Charles Darwin's *The Origin of Species* (1859). While the majority of Darwin's arguments supporting natural selection focus on examples of direct fitness effects – i.e., how an individual's traits may affect its own reproductive success – he also touched upon the concept of selection acting on traits in one individual that affect the reproductive success of others, what are now known as indirect fitness effects. In his monumental book, Darwin discusses potential real-life examples of behaviours that may be difficult to explain with his theory of natural selection. One such example, which he referred to as "one special difficulty," involved the evolution of sterile castes in eusocial insects.

Eusocial insects such as ants, bees, wasps, and termites are differentiated into reproductive castes, such as queens and drones, and nonreproductive castes, such as workers. Nonreproductive castes in eusocial colonies – as the name suggests – do not produce their own offspring, and instead invest energy into helping reproducing relatives in ways that increase their (the relatives') reproductive output. If natural selection only acted on traits that improve an individual's direct fitness, sterile castes would be considered an anomaly that went against Darwin's theory. Darwin understood this potential problem however, and resolved it to some degree by considering other ways in which selection may act, writing:

> *"This difficulty, though appearing insuperable, is lessened, or, as I believe, disappears, when it is remembered that selection may be applied to the family, as well as to the individual, and may thus gain the desired end."*

In this way, ahead of his time, Darwin speculated upon the concept of indirect selection.

1.1 Indirect selection theories

Following *The Origin of Species*, the prevailing understanding of how traits detrimental to an organism's direct fitness (for example, sterility in eusocial insects) might evolve became the topic of much investigation (Boomsma 2016). One highly controversial book exploring this topic, written by Wynne-Edwards (1962), strongly argued in support of group selection theory, which he defined as selection acting on traits which contribute to "the welfare and survival of the group… and when necessary subordinating the interests of the individual." The history of literature on group selection is somewhat conflicting, with some works describing Wynne-Edwards as the first to coin the term (e.g., Williams 1966, p. 96) despite its use much earlier in the literature (e.g., Fisher 1930), and others describing the concept as being widely accepted up until 1966 (e.g., Wilson 1983). It has been argued (Borello 2005) that Darwin also used group selection – though he did not use that terminology – as a solution for some of the difficulties he faced when applying natural selection to, for example, sterile worker castes in eusocial insects. The confusion here is likely due to the inconsistency in defining group selection, and a lack of historical reflection on the subject by some authors. David Sloan Wilson (1983), in discussing the mathematical approach to group selection, called the subject "polyphyletic," as models of group selection seem to have stemmed from multiple authors (e.g., Wright 1945, Wynne-

Edwards 1962, Maynard Smith 1964), with some neglecting to cite others' previous works.

In the meantime, other methods for exploring indirect selection were developing. Fisher (1930), for example, followed Darwin in considering the possibility of natural selection favoring traits detrimental to an individual if the individual's relatives benefitted as a result. Haldane, often cited for his alleged comments about jumping into a river to save two brothers or eight cousins, also wrote of relatedness as a potential means for behaviours which are "socially valuable but individually disadvantageous" to evolve (1932, p. 207):

> *"For a character of this type can only spread through the population if the genes determining it are borne by a group of related individuals whose chances of leaving offspring are increased by the presence of these genes in an individual member of the group whose own private viability they lower."*

Sewall Wright, in his time, seems to have come the closest to developing an effective mathematical representation of indirect selection involving relatives, but failed to put the pieces together effectively. In 1922, Wright developed a coefficient of relationship, *r*, which he used to measure the level of relatedness between two focal individuals. Later, in a 1945 book review, Wright described a group selection model that illustrated how a "character of value to the population, but disadvantageous at any given moment to the individuals" may evolve, depending on a minimum benefit to the population caused by the trait, and a maximum disadvantage experienced by the individuals possessing the trait. He did not, however, include his coefficient of relatedness in this model. While it is

impossible to know why these researchers – known by some as being the founders of population genetics – were unable to develop an effective mathematical model of the problem they so clearly could describe verbally, Alan Grafen had some insight on the matter (Dugatkin 2007). It is possible that, because the economic-based approach to population genetics had not become popularized during the times of Fisher, Haldane and Wright, they did not have the foundational perspective required to make the appropriate connection in their work. The individual who did have this economic perspective was William Donald Hamilton.

1.2 Inclusive fitness theory and Hamilton's rule

Hamilton was an undergraduate student at Cambridge University when he discovered Fisher's *The Genetical Theory of Natural Selection* – a book which absorbed Hamilton to the point where his coursework suffered. Captivated by Fisher's work, Hamilton was lucky enough to meet the professor himself, who happened to be teaching genetics at Cambridge University at that time. Despite getting along well with Fisher, Hamilton struggled to enjoy his undergraduate degree due to the lack of support for evolutionary theory from some of his professors (Grafen 2004). Possibly as a result of this, for his graduate degree Hamilton initially enrolled in an MSc in human demography at the London School of Economics. As his work shifted towards his interests in genetics and mathematics however, Hamilton transferred into a joint supervision at both the London School of Economics and University College London (Dugatkin 2007). During his graduate education, Hamilton remained interested in evolution and genetics and was greatly influenced by Fisher's 'Fundamental Theorem of Natural Selection' (1930) which states:

> *"The rate of increase in fitness of any organism at any time is equal to its genetic variance in fitness at that time."*

Hamilton sought to develop a mathematical model that was as generally applicable as Fisher's theorem while remaining useful even for scenarios involving social behaviour, in which individuals behave in such a way that influences the fitness of others (of the same species). In his studies, Hamilton recognized the connection between relatedness and social behaviours referenced by previous researchers (see previous section for examples) (Hamilton 1964 II). Making use of Wright's coefficient of relationship, Hamilton worked to create a model which considered the fitness effects of social behaviours and the relatedness between the social interactants involved. One difficulty he faced was the implication of Fisher's fundamental theorem that the number of offspring an individual produced was dependent on its own genotype, not the genotype of other individuals (Grafen 2004).

To fully understand the complication Hamilton faced, consider nonreproductive castes in eusocial insect colonies as an example. Nonreproductive workers in these colonies have evolved such that they themselves do not reproduce, but instead convey fitness benefits to reproducing queens. If we focus on the fitness benefits conveyed to the queen when modeling the evolution of the workers' behaviour, we must take into consideration not only the queen's genotype, but the genotype of the workers, because their behaviour is (presumably) caused by their genotype. In doing so, we imply that the number of offspring produced by the queen is dependent on both her genotype and that of the worker, but do not consider this interaction from the perspective of the workers. The question thus remains: why would workers give up their own fitness to help the queen?

To tackle this question, Hamilton developed the concept of inclusive fitness, which considered the fitness effects of an individual's behaviour towards relatives as indirectly affecting its own fitness (Hamilton 1964 I). Thus in our eusocial insect example, we would consider the fitness benefit conveyed from a worker to a queen as indirectly affecting the worker's own fitness. This concept can be formally written as an inequality, now commonly known as 'Hamilton's Rule', which weighs the fitness effect that a focal individual (FI) has on another individual by the degree to which they are related, and compares this to the fitness effect that the FI has on itself. This can be written as $RB > C$, where *R* is approximate to Wright's coefficient of relationship, *RB* is the indirect fitness effect experienced by the FI (calculated by multiplying *R* by the fitness effect the FI has on another individual, *B*), and *C* is the fitness effect that the FI causes to itself (alternatively written as $RB - C > 0$, depending on the signs of each coefficient). When Hamilton's Rule is fulfilled, the FI is said to be working to maximize its inclusive fitness – a measure of all direct and indirect fitness effects to the FI caused by its behaviour. See sections 2.1 and 2.2 for a full derivation of Hamilton's Rule.

In the years following his original 1964 publications, Hamilton worked to improve upon the minor weaknesses in his inclusive fitness formulas. With help from George R. Price, a population geneticist who has significantly contributed to selection mathematics, Hamilton re-derived his measure of relatedness *R* to encompass a wider variety of circumstances, such as inbred populations or spiteful behaviours (Grafen 2004). This new derivation of *R* has been the source of some confusion in previous literature (Gardner et al. 2011), as well as discussion of alternative measures of relatedness (Michod and Anderson 1979). For clarity, I will briefly review here both Wright's (1922) coefficient

of relationship used as an estimator in Hamilton's original (1964) models, and Hamilton's re-derived 'coefficient of relatedness' described in his 1970 and 1972 works.

Wright's coefficient of relationship is calculated using inbreeding coefficients – a measure of the likelihood that an individual bears two copies of the focal allele as opposed to just one (i.e. it is autozygous for the focal allele) (Hamilton 1972). Using these inbreeding coefficients, Wright was able to trace back pedigrees to calculate the likelihood that two focal individuals share alleles at a given locus that are 'identical by descent' (IBD), or descended without mutation from the same ancestral allele in a common ancestor (Hill 1996; Michod and Anderson 1979). Wright's relationship coefficient is therefore limited to positive values between 0 and 1. This seems reasonable; intuitively, we would not expect to find a "negative" relatedness value between individuals. For inbred populations, however, this type of measurement will be less useful, as two random individuals in the population may be more likely to share identical alleles at a given locus than two focal individuals. To avoid this problem, Hamilton re-derived his inclusive fitness formula to include a coefficient of relatedness which incorporates Wright's coefficient weighted against the likelihood that any two random individuals in the population will share genes IBD (Hamilton 1970). Many authors have since attempted to add to or modify this relatedness formula, with varying limitations or assumptions required for each. An overview of the various derivations of *R* was published in 1980 by Michod and Hamilton, who showed that these alternative formulas were effectively equivalent. The most effective and widely applicable form of *R* supported in Michod and Hamilton's review paper was originally described by Orlove (1975) and Orlove and Wood (1978). The formula is described in Michod and Hamilton's

(1980) publication as the covariance between a FI's genotype at a given locus, *z*, and the genotype of its social partner, *z'*, divided by the variance of the focal allele in the population *Z*, or $R = Cov(z, z')/var(Z)$. This can be interpreted as the extent to which the genotype of any given FI in the population matches that of its partner, divided by the extent to which the genotypes of any two random individuals in the population match. This regression form of *R* has fewer limitations than Wright's coefficient of relationship. If, for example, the FI and partner are less related than two random individuals in the population, the resulting *R* would be negative. With this less restrictive measurement of relatedness, we can now model behaviours like spite, which tend to involve negative *R* values (Gardner and West 2004). I will be using this formula of *R* for my methods.

1.3 Controversy of inclusive fitness theory

Hamilton's inclusive fitness theory has often been cited as 'revolutionizing' the study of social behaviours (e.g., Michod 1982), and its utility has been supported in numerous studies (Bourke 2014). Despite this, controversy has surrounded Hamilton's work for decades. Perhaps the most famous source of disagreement is the so-called 'haplodiploidy hypothesis'. In his 1964 papers, Hamilton discussed the prevalence of high relatedness values between haplodiploid eusocial insect workers: full-sibling workers in these colonies often show *R* values of 0.75 at any given locus (as opposed to 0.5 in full sibling diploids). According to Hamilton (in 1964, at least), this high relatedness might help explain why eusociality is so prevalent in haplodiploid hymenopterans. At the time, however, Hamilton failed to recognize the resulting lower relatedness between sisters and brothers in haplodiploids (0.25). Several critics of inclusive fitness theory leapt at this oversight, treating it as key evidence that Hamilton's rule was incorrect (for example, see

Wilson 2005, Nowak et al. 2010). Hamilton's haplodiploidy idea is not required for inclusive fitness to work however, despite the insistence otherwise by some (Foster et al. 2006, Gardner et al. 2011).

Another source of confusion regarding the utility of Hamilton's rule involves the perceived requirement of additive fitness effects. When the fitness effects of a social interaction are additive, the change in fitness to the FI after an interaction with its partner is equal to the sum of the fitness effects of each individual's behaviour. For example, if the FI's behaviour increases its own fitness by X, and the partner's behaviour increases the FI's fitness by Y, the additive fitness effect of a social interaction between the FI and its partner would be $X + Y$. If there is a synergistic effect, however, and the combined behaviours of the FI and its partner result in a fitness effect greater than (or alternatively, less than) $X + Y$, then the fitness effects of their behaviours would be considered nonadditive. Several authors (e.g., Queller 1992, Fletcher and Doebeli 2006, Nowak et al. 2010) have argued that there is an implicit assumption of additive fitness effects in Hamilton's rule, with many regarding B and C to be additive fitness parameters that can be freely and independently varied in any given model. This has led some (e.g., Queller 1992) to believe that a third fitness parameter, sometimes labeled D, must be added to Hamilton's rule to represent nonadditive or synergistic effects not included in B and C. In reality, B and C are regression coefficients derived from a least-squares regression analysis, the formulas for which encapsulate both additive and nonadditive fitness effects (see full derivation in sections 2.1 and 2.2) (Gardner et al. 2011). As explained in Gardner et al. (2011), alternative inclusive fitness models that partition the nonadditive and additive fitness effects, such as those described by Queller (1992), are still useful for

understanding how different components of inclusive fitness influence selection. The point here is that Hamilton's rule is still valid with nonadditive effects, which are already encapsulated by Hamilton's original coefficients *B* and *C*. In fact, different types of fitness effects can be partitioned out of these coefficients directly, the methods for which I will describe in Section 2.2.

1.4 Applications

Despite ample criticism, Hamilton's inclusive fitness theory was eventually widely supported by evolutionary geneticists and is considered to have significantly contributed to the Modern Synthesis and the genetical theory of natural selection. Prominent supporters of inclusive fitness, such as George C. Williams (who was among the first to support the theory publicly), hail Hamilton's rule for its wide applicability and utility for understanding the evolution of adaptation and social behaviours (Bourke 2011, Boomsma 2016). As discussed above, Hamilton's rule can be applied not only to cooperative and altruistic behaviours, but also selfish and spiteful ones, depending on the values of *R*, *B* and *C*. This formula is not limited to the study of theory either – Hamilton's rule can be applied to real-world data to investigate the mechanisms behind the evolution of a wide variety of behaviours. Loeb (2002), for example, used Hamilton's rule to study the evolution of egg dumping in the lace bug *Gargaphia solani*, and found the fitness effects of this behaviour support it being altruistic (as opposed to selfish or cooperative) in nature. These practical applications of Hamilton's rule help to answer specific questions about social behaviour, often with a focus on specific organisms. Theoretical applications, by contrast, look to explore the overarching mechanisms behind the evolution of such behaviours – the why and how as it applies to all life on earth. Theory

provides the foundation for practical research to build from – Hamilton's inclusive fitness theory, for example, has illuminated the importance of indirect fitness effects in the evolution of some social behaviours.

Since Hamilton's original 1964 papers, some evolutionary theorists have worked to further investigate the evolution of social behaviours through inclusive fitness models. It is now well understood that inclusive fitness plays an important role in behaviours such as altruism and spite, but how does that manifest in life? Questions of dispersal by individuals from their natal sites, the conditional expression of genes for social behaviours, gene penetrance, intra-genomic conflicts, and many more have been investigated and are still being explored via inclusive fitness models. Charlesworth (1978), for example, compared models of the evolution of altruistic behaviour by means of parental manipulation (in which reproductive mothers influence their offspring to behave altruistically) and kin selection, where individuals behaved altruistically towards their siblings with a specified probability. The results of these models revealed several interesting implications, such as how the *B* and *C* values required for altruism to evolve are influenced by the probability of expressing altruism. This makes sense for the model because, as Charlesworth himself (1978) put it:

> *"The higher the probability of expression of the gene for altruistic behaviour, the higher the probability that the sibs being helped do not carry the gene, or that if they do, they themselves express it and suffer the concomitant loss in fitness."*

Charlesworth calls these models "crude" because – as many models do – they require broad assumptions that leave out important aspects of the modeled organism's life

history. Despite this, his and many theoretical works allow for a better understanding of specific aspects of behavioural evolution. While any one study cannot give us a full picture of how, for example, altruistic behaviour evolves, each investigation can illuminate pieces of the puzzle which, together, paint a larger picture.

Another example of research 'illuminating a piece of the puzzle' regarding the evolution of altruism is Parker's (1989) research on role-based conditional expression. In nature, there are many circumstances in which animals may fall into different roles; birth order (or age), territory establishment (owner or invader), size (assuming correlation of nutrition with size), and social status are all examples of potential asymmetric roles between interacting individuals which generally do not involve differing genotypes. This is important for the study of altruism because in many cases, an individual will behave altruistically or not depending on an environmental stimulus such as asymmetric roles. In honeybees (*Apis mellifera*), for example, larvae differentially develop into queens or workers depending on the diet they are fed by nurse bees (Mao et al. 2015). The conditional expression of social behaviours like altruism is thus an important aspect to study, and while Charlesworth (1978) used a probability of expression to this end, Parker (1989) explored this concept by dividing his model population into roles X and Y. Several examples are explored in the paper, but in essence, individuals that possess a specific allele or genotype for a given behaviour are modeled such that they only express the behaviour associated with their genotype when in a specific role. For example, in the sibling competition models, carriers of Parker's hypothetical focal allele will take more resources than their siblings when in role X, but not when in role Y.

1.5 Developing Inclusive Fitness Theory

In this thesis I aim to expand upon the current understanding of conditionally expressed social behaviours by developing a general inclusive fitness model based on the methods of Gardner et al. (2011). In the model building process, I describe how Hamilton's Rule can be derived from a general evolutionary model, the Price equation, and introduce the variables *B* and *C* in Hamilton's Rule as partial regression coefficients of fitness and genotype (see sections 2.1 and 2.2). I then begin the development of my model by describing a basic structure for the scenarios I will be modelling, identifying the parameters involved, and assigning appropriate variables for each component – collectively called my *Model Foundation* (Section 2.3). Using the variables and formulae compiled in my model foundation, I find mathematical expressions for the partial regression coefficients *B* and *C* in Hamilton's Rule (see Section 2.4). I then apply these expressions to Hamilton's rule (as derived from the Price equation) and describe the sociobiological significance of each component of the resulting formula, with particular emphasis on the coefficient of relatedness, *R*, as described by Michod and Hamilton (1980), and a coefficient of synergetic pairings, *S* (see Section 2.5).

After assembling a base model structure, I explore several themes of conditionally expressed social behaviours touched upon by previous researchers such as Parker (1989) and Charlesworth (1978), and consider effects that were not included in previous inclusive fitness models. From Parker's work I adopt a similar concept of 'roles' for my model, such that individuals occupy one of two different roles in an interaction. In nature, these roles could represent any asymmetry between the two interactants, such as habitat

defense vs invasion, food availability, age, birth order, etc., which may influence the conditional expression of a focal social behaviour.

Current inclusive fitness papers modelling conditionally-expressed social behaviours often do so in such a way where the 'condition' – the stimulus influencing a behaviour's expression – is allocated at random, or with a given probability, without consideration of the genotype of the focal individual. In other words, these models do not allow for gene-environment interactions that bias an individual's likelihood of exposure to an environmental stimulus. For example, in Parker's (1989) role-dependent expression model, he explicitly states "To obey Hamilton's rule, roles must be assigned randomly with respect to the genotype of the pair of relatives, and a gene must be expressed in one role only; i.e. its expression must be conditional upon role." Given that Hamilton's inclusive fitness theory was somewhat inspired by the behaviours of social insects (Grafen 2004), it is understandable why Parker and others may have made this assumption. Until relatively recently, it was assumed that eusocial insect castes, such as workers and queens, were determined entirely by environmental factors such as diet, or the presence of specific pheromones (Schwander et al. 2010). Intuitively, this seems to make sense; if castes were genetically determined, queens would be unable to produce non-reproductive castes like workers, and these non-reproductive castes would rapidly go extinct. With the allowance of *conditional* gene action (or conditional behaviour expression), we may once more consider the prospect of genes influencing the determination of caste – i.e., gene-environment interaction.

To include the possibility of a gene-environment interaction in my model, I introduce a third key variable in addition to the coefficient of relatedness R and the synergistic

pairings coefficient *S*, called the 'asymmetry quotient,' *Q*. The asymmetry quotient accounts for genetic bias in role by measuring the proportion of individuals carrying a focal allele that are found disproportionately in one role over another. To use Parker's roles X and Y (discussed in section 1.4) as an example, *Q* in Parker's model could be calculated by taking the number of individuals with the focal allele that are in role X in a heterogeneous pairing divided by the total number of individuals with the focal allele in a heterogeneous pairing. This calculation can be done with either role as the point of focus (i.e., it does not matter whether role X or Y is used in the formula, so long as the role used is consistent throughout the model).

With a base model structure with roles assembled, I apply my model to two example social evolution scenarios for illustrative purposes. The first scenario, named the 'Hawk/Dove/Bourgeois Game' (or HDB for short), is based on Maynard Smith's game theory model of the same name, and describes the evolutionary dynamics between three different behaviour strategies, Hawk, Dove, and Bourgeois, which are adopted by individuals when either defending a territory from, or invading the territory of, another individual. Inclusive fitness models are often used to investigate social behaviours like cooperation, altruism, and occasionally spite, but they are less frequently used for selfish behaviours like territory defense. In applying my model to a modified form (see section 3.1) of Maynard Smith's HDB game, I hope to show that inclusive fitness models can provide insight into how factors like relatedness, synergy, and gene-environment interactions can influence the evolution of what seems to be purely selfish behaviour. The second scenario I apply my model to is of my own design, and explores the evolution of a conditionally expressed altruistic behaviour that is prone to some degree of 'error' in its

conditional expression. In this scenario, ‘altruism’ is defined as a behaviour that reduces the fitness of the FI behaving altruistically and increases the fitness of a social partner that shares the same focal allele. I integrate a modified form of Charlesworth’s (1978) ‘penetrance’ value here, where instead of a probability of expression as Charlesworth used, I apply a frequency of error, *e* (see section 3.2), such that individuals make errors in their expression of altruism with frequency *e*. These errors are meant to represent any roadblocks in a FI’s lifetime that prevent it from expressing altruism towards relatives. For example, one type of error would be misidentifying an unrelated social partner as a relative, and behaving altruistically towards that non-relative.

With the two example applications of my model described, I explain the results in detail, and discuss the implications of my model in the context of current evolutionary theory.

2 Model Description

In the following sections I derive the two main formulae upon which my model is built: the Price equation and Hamilton's rule. I begin with a description of the basic variables involved in the Price equation, and how to build Price's formula using these components. I then show how Hamilton's rule can be derived from a simple rearrangement of the Price equation using regression coefficients of fitness and genotype. Following this, I describe the various parameters involved in my own application of the Price equation, called my model foundation, and how to find expressions for the aforementioned regression coefficients using these parameters. Once my base formula is created, I explain how the model can be simplified by rearranging it to include the variables *R* and *S*, which allow for more intuitive interpretation of results. This more simplified formula is explained in sociobiological terms, and I introduce the new component I wish to add to the formula, *Q*, to describe fitness effects not clearly represented in the base form of the model.

2.1 The Price equation

To construct my inclusive fitness model, I begin with a formula developed by Price (1970) that has been used by various researchers – including Hamilton himself (Hamilton 1970) – to describe the allele frequency change, Δp, resulting from natural selection and other evolutionary forces (e.g., mutation). To derive Price's formula, consider first a simplified scenario involving a population of N haploid, asexual organisms. Each individual i (where $i = 1, 2, \ldots N$) in the F0 (parent) generation has a genotypic value Z_i, where $Z_i = 1$ indicates that individual i possesses a focal allele, and $Z_i = 0$ indicates that i does not possess this allele. Fitness W_i for each individual in this model is expressed as

the number of offspring produced, thus $W_i = 2$ indicates individual *i* contributes two offspring to the F1 (daughter) generation. The average genotypic value of *i*'s offspring can be found with $Z_i + \Delta Z_i$, where ΔZ_i is the average difference in genotypic value between *i* and *i*'s offspring. This difference in genotypic value in a diploid, sexual population could be the result of individual *i*'s offspring inheriting a different allele from the other parent, but in this haploid, asexual scenario differences in genotypic value between parent and offspring may be caused by mutation. Table 2.1 illustrates how these data may be laid out for analysis purposes.

Table 2.1. Example dataset of hypothetical population described for model building, where *i* denotes a number label for each individual from 1 to *N*. Data in each row correspond to the individual *i* in the far-left column. Z_i is the genotypic value of individual *i*, such that a Z_i of 1 indicates that individual *i* possesses the focal allele, and a Z_i of 0 indicates that it does not. W_i is the number offspring produced by individual *i*. Z_i' is the average genotypic value of *i*'s offspring, calculated by summing Z_i and ΔZ_i, which is the average difference in genotypic value between *i* and *i*'s offspring.

i	Z_i	Wi	$Z_i' = Z_i + \Delta Z_i$
1	1	2	1 – 0.5
2	0	5	0 + 0
3	1	3	1 + 0
⋮	⋮	⋮	⋮
N	1	2	1 + 0

To find the change in allele frequency Δp, or $p' - p$, we first calculate p, which is equivalent to the expected value of Z, or $E[Z] = \sum \frac{1}{N} Z_i$.[1] Similarly, allele frequency for the F1 generation, p', can be calculated by multiplying fitness W_i, or the number of offspring produced by i, with the average genotype of i's offspring, Z_i', summed across all is and divided by the number of individuals in the F1 generation, N'. We can easily find the size of the F1 generation by summing all W_i values, thus $p' = \sum W_i(Z_i')/\sum W_i$, which can be rewritten (by multiplying the numerator and denominator by $\frac{1}{N'}$) as $p' = \frac{E[WZ]+E[W\Delta Z]}{E[W]}$. Calculating Δp can now be done by taking $p' - p$ to find

$$\Delta p = \frac{E[WZ] + E[W\Delta Z]}{E[W]} - E[Z] \quad \textbf{(1)}$$

which can be rewritten as[2]

$$\Delta p = \frac{Cov(W,Z)}{E[W]} + \frac{E[W\Delta Z]}{E[W]} \quad \textbf{(2)}$$

---

[1] The expected value of a random variable, X, is the sum of all the values X may take on weighted by the probability that X will take on that value. In many cases, this can be considered the same as the weighted average of X. For this thesis, I will use the terms "expected value" and "average" interchangeably to describe parts of my models. In the current example, each individual i contributes equally to the population as reproduction only occurs once per organism, thus $E[Z] = \sum \frac{1}{N} Z_i$.

[2] Note: The covariance of two random variables, in this case W and Z, is defined as the expected product of the individual values minus expected values of each variable, or $Cov(W,Z) = E[(W_i - E[W])(Z_i - E[Z])]$. This formula can be simplified to $E[WZ] - E[W]E[Z]$ (see full derivation in Appendix). Thus, Equation 1 has been simplified as follows:

$$\Delta p = \frac{E[WZ] + E[W\Delta Z]}{E[W]} - E[Z] = \frac{E[WZ] - E[W]E[Z] + E[W\Delta Z]}{E[W]} = \frac{Cov(W,Z) + E[W\Delta Z]}{E[W]}$$

This final simplified version is the Price equation. The first term in Equation 2 describes the partial change in allele frequency due to selection, $\Delta_s p = \frac{Cov(W,Z)}{E[W]}$, as a covariance between individual fitness, *W* and genotypic value, *Z*, for all individuals in the population divided by the average population fitness. The second term describes the partial change in allele frequency due to other, non-selective effects such as mutation, drive, or even cultural transmission (Price 1970; Gardner 2008). Interpreting the above expression is relatively straightforward: assuming that the effects of transmission bias (differences in genotypic and/or phenotypic value between parents and offspring) are small (though this may not always be the case), and because average fitness cannot be negative, we may simply focus on the sign of the covariance expression. A positive covariance between *W* and *Z* indicates an increase in frequency of the focal allele due to selective action, while a negative covariance indicates a decrease due to selection against the focal allele. The covariance in Price's formula is valuable in its intuitively appealing results: if carrying a focal allele is positively correlated with fitness, one would naturally expect the frequency of this allele to increase.

2.2 Regression

An alternative form of the Price equation, which may appeal more to quantitative geneticists, involves multiplying the variance of the focal allele by a regression coefficient of fitness and genotype, such that

$$\Delta p = \frac{\beta_{WZ} Var(Z)}{E[W]} + \frac{E[W\Delta Z]}{E[W]} \quad \textbf{(3)}$$

where β_{WZ} = Cov(W,Z)/Var(Z) (Gardner et al. 2011). This format allows us to consider the partial change in allele frequency due to selection, $\Delta_s p$, as a selection gradient multiplied by the genetic variance of the population, weighted by the population average fitness. To develop my own model predictions in an inclusive fitness context, I will employ an alternative approach to analyzing this regression form of the Price equation with my model data. Because I am focusing specifically on changes in allele frequency due to selective effects in my models, I will from this point be referring solely to the $\Delta_s p$ formula, and omit the second term of Price's original equation.

Describing the inclusive fitness effects of a hypothetical allele which influences social behaviour (henceforth called a 'social allele' for short) involves not only considering how a focal individual's (FI) behaviour will affect its own fitness, but also how any social partners' (other individuals interacting, either indirectly or directly, with the FI) behaviours will indirectly affect the FI's fitness. The coefficient in Equation 3 can be partitioned into two for this purpose, such that one coefficient represents the partial regression of the FI's fitness on its own genotype ($\beta_{W,Z|z} = \beta_{FI}$) and the other represents the partial regression of the FI's fitness on the genotype of a social partner ($\beta_{W,Z|z'} = \beta_{Part}$), which gives

$$\Delta_s p = \frac{\beta_{FI} var(z) + \beta_{Part} Cov(z, z')}{E[W]} \quad \textbf{(4)}$$

Each of the coefficients is multiplied by its respective genotype covariance: β_{FI} with the covariance of its own genotype, i.e. the variance of the focal allele (Cov(z,z)=var(Z)), and β_{Part} with the covariance of the FI's genotype on its social partner's genotype. With the

understanding that the average genetic relatedness between social partners, *R*, is defined as $R = Cov(z, z')/var(Z)$ (Michod and Hamilton 1980), we can rearrange Equation 4 to show

$$\Delta_s p = \frac{(\beta_{FI} + \beta_{Part} R) var(Z)}{E[W]} \quad \textbf{(5)}$$

(Gardner et al. 2011). This formula is, in essence, Hamilton's rule: so long as $\beta_{Part} R > \beta_{FI}$, $\Delta_s p$ will be positive and the focal allele will increase in frequency over one generation. If one were to consider this formula in the context of altruistic behaviours, β_{Part} would be Hamilton's benefit term, *B*, and β_{FI} would be cost, *C* (Gardner, 2008). The interpretation of β_{FI} and β_{Part} here is straightforward when considering only additive fitness effects from interactions, meaning that we assume the resulting fitness to the FI after an interaction is equal to the sum of the fitness effects of the FI's behaviour and its social partner's behaviour. In this case, β_{FI} would simply measure the average difference in residual (individual minus average) fitness to the FI when it possesses the focal allele versus when it does not. The term β_{Part} would then measure the average change in residual fitness to the FI when its *partner* possesses the focal allele versus when it does not. If, however, the total fitness effect of an interaction is not equal to the sum of the individual fitness effects of the FI and partner's behaviours (i.e. it is nonadditive), the above formula remains valid (Gardner et al. 2011; Queller 1992) but the interpretation of each coefficient becomes more complicated, because (in a departure from Hamilton's original formulation) coefficients depend directly on allele frequency. If, for example, two carriers of the focal allele interact more effectively than

heterogeneous (carrier with non-carrier) pairings, the total fitness effect on the FI may be greater than simply the sum of the individual fitness effects.

To find the mathematical expressions for β_{FI} and β_{Part} which encompass both the additive and nonadditive fitness effects of the social behaviours I wish to model, I will first describe an evolutionary "game" outlining the rules that will act as the foundation of my model.

2.3 Model Foundation

Consider a finite population of haploid, asexual organisms, each of which, at one point in its lifetime, engages in a pairwise interaction with another individual in the population. The behaviour of an individual during this interaction is controlled by a single locus where genotype $Z = 0$ indicates possession of the wild-type allele (A_0), and $Z = 1$ indicates the mutant allele (A_1). Interactions of each type occur at frequency $F_{z+z'}$. Therefore, interactions between two carriers of the mutant allele A_1 occur with frequency F_2, interactions between two carriers of the wild-type allele A_0 occur with frequency F_0, and interactions between one A_1 carrier and one A_0 carrier occur with frequency F_1. Each individual *i* may experience a fitness cost or benefit dependent on its own behaviour and that of its partner.

The frequency of allele A_1, $E[Z]$, in the F0 generation can be calculated by summing the frequency values of all pairings involving A_1 carriers weighted by the frequency of A_1 interactants in each pairing, giving $E[Z] = F_2 + \frac{1}{2}F_1$. Average fitness $E[W]$ in the F0 population can be similarly calculated by taking the fitness outcome to the FI in an interaction weighted by the frequency of that pair summed across all interactions.

2.4 Mean Squared Error

The expressions for β_{FI} and β_{Part} in Equation 5 can now be obtained using the formulae outlined in the previous section. Note that there are four possible residual fitness (W_i – E[W]) outcomes that can be calculated using the input values in section 2.3, one for each possible interaction type that a focal individual can have (interactions between A_1 and A_0 carriers account for two of these, one for each type of FI in this pair). These fitness outcomes are not separated into those resulting from the FI's behaviour or that of its partner, and thus may be less informative in this form. To partition residual fitness outcomes into the coefficients β_{FI} and β_{Part}, I fit these coefficients such that they fulfill the same function in the model as each of the observed residual fitness values. I accomplish this by finding the mean squared error (MSE), which calculates the average squared difference between a set of estimated values and the observed values being estimated. The fitted residual fitness values used to calculate the MSE are expressed using the β_{FI} and β_{Part} coefficients in the following formula:

$$W_{FI} - E[W] = \beta_{FI}(z - E[Z]) + \beta_{Part}(z' - E[Z]). \quad \textbf{(6)}$$

All fitted and observed residual fitness values are laid out in Table 2.2, labelled D_{Obs} and D_{Fit} respectively (uppercase D is used to remind us that we are comparing the difference between observed, D_{Obs}, and fitted, D_{Fit}, residual fitness values).

Table 2.2. Expressions used to develop the MSE formula. Fitness and genotype values from each row correspond to a specific focal individual in each pair. For example, D_{Obs10} is the residual fitness to all A_1 carriers in heterogeneous (A_1 with A_0) pairings.

FI + partner	Frequency of pairing	$D_{Obszz'} = W_{FI} - E[W]$	$D_{Fitzz'} = \beta_{FI}(z - E[Z]) + \beta_{Part}(z' - E[Z])$
$A_1 + A_1$	F_2	D_{Obs11}	$\beta_{FI}(1 - E[Z]) + \beta_{Part}(1 - E[Z])$
$A_1 + A_0$	$\frac{F_1}{2}$	D_{Obs10}	$\beta_{FI}(1 - E[Z]) + \beta_{Part}(0 - E[Z])$
$A_0 + A_1$	$\frac{F_1}{2}$	D_{Obs01}	$\beta_{FI}(0 - E[Z]) + \beta_{Part}(1 - E[Z])$
$A_0 + A_0$	F_0	D_{Obs00}	$\beta_{FI}(0 - E[Z]) + \beta_{Part}(0 - E[Z])$

Using the $F_{z+z'}$ values to weigh each partial squared difference, I derive the MSE using the formula[3]

$$MSE = \sum F_{z+z'}(D_{Obs} - D_{Fit})^2$$

$$= F_2(D_{Obs11} - D_{Fit11})^2 + \frac{F_1}{2}(D_{Obs10} - D_{Fit10})^2 + \frac{F_1}{2}(D_{Obs10} - D_{Fit10})^2$$

$$+ F_0(D_{Obs00} - D_{Fit00})^2 \quad \textbf{(7)}$$

where $D_{Obs\,zz'}$ and $D_{Fit\,zz'}$ represent the observed and fitted residual fitnesses respectively of a focal individual with genotype *z* whose partner possesses genotype *z'*.

[3] Note that F_1 here is divided in two, because half of the interactants in heterogeneous pairs are A_1 carriers, and half are A_0 carriers.

Using the mathematics program *Maple* (2020) to carry out the calculations, I determine the expressions for β_{FI} and β_{Part} which minimize Equation 7 (the mean squared error between the observed residual fitnesses and those predicted by Equation 6) (see equations 8a and 8b in appendix). While the expressions resulting from my MSE analysis can be substituted directly into Equation 5 for calculation purposes, they are difficult to interpret in this form. In the following section, I will illustrate simplifications that can be used to interpret the outcomes of selection studied with this type of social behaviour model.

2.5 Sociobiological Interpretation

To make the interpretation of a somewhat unwieldy, large equation more intuitive, we may assign simplifying variables to represent some of the frequency-dependent components of a formula. With these modifications, the interacting dynamics of the model can be more easily described using sociobiological language, as opposed to purely frequency-dependent terms. For my model, I focused on isolating two key variables frequently used in inclusive fitness analyses: relatedness, *R*, which I have previously defined in Section 2.2 as $R = Cov(z, z')/var(Z)$ (see Equation 5), and synergy, *S*. Fitness effects may be considered synergistic if the combined effect of two interacting individuals is greater than (or conversely, less than) the sum of the effects caused independently by each interactant. In this model, I focus on the synergistic effects resulting from the interaction of two A_1 individuals. The proportion of individuals with the focal allele, A_1, that interact with an A_1-carrying social partner can be written as F_2/p. Assigning the variable *S* to this fraction (i.e., $S = F_2/p$) provides a means of weighting synergistic fitness effects by the frequency at which A_1 carriers are in a

homogeneous pairing. Using the synergy coefficient *S* in combination with Hamilton's coefficient of relationship *R*, expressions (8a) and (8b) may be rearranged as follows:

$$\beta_{FI} = \frac{S(D_{Obs11} - D_{Obs10} - D_{Obs01} + D_{Obs00}) + (D_{Obs10} - D_{Obs00})(R + 1)}{R + 1}$$

(9a)

and

$$\beta_{Part} = \frac{S(D_{Obs11} - D_{Obs10} - D_{Obs01} + D_{Obs00}) + (D_{Obs01} - D_{Obs00})(R + 1)}{R + 1}$$

(9b)

Focusing on the first portion of the formula in Equation 5 again, and replacing the beta coefficients there with the above expressions, I find[4]

$$\Delta_s p \propto R(D_{Obs01} - D_{Obs00}) + D_{Obs10} - D_{Obs00} + S\big((D_{Obs11} - D_{Obs10}) - (D_{Obs01} - D_{Obs00})\big)$$

(10)

Unlike equations 8a and 8b, the above formula can be more easily broken into the specific behavioural phenomena at play. The first component, $R(D_{Obs01} - D_{Obs00})$, can be broken into the two factors, *R* and $(D_{Obs01} - D_{Obs00})$, the latter of which can be interpreted as the difference in additive direct fitness effects to individuals when their partner is switched from an A_0 to A_1 carrier. In other words, this measures how an

[4] The symbol ∝ used in Equation 10 indicates "proportional to," meaning the change in *p* is proportional to the right-hand side of the equation.

individual's fitness is affected by a partner exhibiting wild type behaviour (A_0) compared to mutant behaviour (A_1). To understand how these effects influence the mutant's own fitness, I multiply by *R*, a measure of the extent to which interactants are more related to each other than two random individuals in the population. In doing so, our perspective is then redirected to the A_1 carrier itself, and the resulting product measures the difference in additive *indirect* fitness effects to A_1 carriers in comparison to A_0 carriers. It is helpful here to consider a hypothetical scenario where individuals are somehow able to 'will' their alleles to change – in which case the first component would measure the change in additive indirect fitness effects to individuals that 'switch' their social allele from A_0 to A_1.

The second component in Equation 10, $D_{Obs10} - D_{Obs00}$, is the difference in additive direct fitness effects between A_0 and A_1 carriers. Following the above hypothetical scenario, this could be thought of as the change in additive direct fitness effects to individuals that 'switch' their social allele from A_0 to A_1.

The third component in Equation 10, $S\big((D_{Obs11} - D_{Obs10}) - (D_{Obs01} - D_{Obs00})\big)$ encapsulates the nonadditive fitness effects resulting from pairings between two A_1 carriers, i.e. the *synergistic* effects. This can be expanded slightly for explanatory purposes to $S((Dobs_{11} - Dobs_{00}) - (Dobs_{10} - Dobs_{00}) - (Dobs_{01} - Dobs_{00}))$. In this expanded form, we can see that the synergistic component first takes the difference in fitness effects to A_1 carriers in homogeneous pairings – that is, pairings consisting of individuals with the same social allele – and fitness effects to A_0 carriers in homogeneous pairings, or $(Dobs_{11} - Dobs_{00})$. From this expression, both the direct $(Dobs_{10} -$

$Dobs_{00}$) and indirect ($Dobs_{01} - Dobs_{00}$) additive effects are subtracted such that only the nonadditive effects remain. Considering again the hypothetical allele switching scenario, we may consider the synergistic component to be measured as the change in fitness effects to individuals when both they and their partner simultaneously 'switch' their social allele from A_0 to A_1, minus the additive effects described above. The result is then weighted by *S*, because these effects are only experienced by A_1 carriers in homogeneous pairings. When simplified, the synergistic component becomes $S\big((D_{Obs11} - D_{Obs10}) - (D_{Obs01} - D_{Obs00})\big)$, as shown above.

In its current form, Equation 10 is informative in its ability to partition the fitness effects of an allele for social behaviour into additive and nonadditive parts while also providing insight into the influence that both relatedness between interactants (*R*) and proportion of synergistic pairings (*S*) have on allele frequency. As is, the model contains factors that describe the dynamics between a FI's genotype and that of its social partner. What is missing, however, is a descriptor for the dynamics between a FI's genotype and its environment, i.e., a measure of gene-environment interaction effects.

To incorporate environmental effects in my model, I designate a role label to each individual in an interacting pair, labelled P1 and P2. 'Role' here will be used to represent a difference in environmental stimulus experienced by a FI in comparison to the stimulus experienced by the FI's social partner. In other words, roles P1 and P2 will be used to model an environmental asymmetry between the FI and its partner that causes them to differentially express their behaviour depending on the role they occupy. This asymmetry can be thought of as, for example, a difference in size (nourishment), territory defender

versus invader, birth order, or any other asymmetry that may occur that is not a direct result of genetic differences between individuals.

To quantify a measure of gene-environment interaction, I assign a new simplifying variable, *Q*, which describes the proportion of individuals carrying a focal allele – in this case, A_1 – that are disproportionately found in one role over another. This asymmetry quotient provides insight into the significance of gene-environment interaction effects on the evolution of a social behaviour.

Consider, for example, an asymmetric game in which individuals in the P1 role have an advantage over those in role P2, such as already owning (as opposed to seeking) a territory which positively influences fitness of the territory holder. If alleles A_1 and A_0 are associated with differing territory invasion and/or defense behaviours, then an asymmetry in the proportion of A_1 carriers in each role may have an influence on which allele outcompetes the other. In the following chapter I will go through a few example applications of my model, illustrating how different levels of gene-environment interaction (as measured by *Q*) can influence the resulting change in allele frequency $\Delta_s p$.

3 Examples

The final model built and described in Chapter 2 (Equation 10) is a generalized formula that can be applied to a wide variety of social behaviours, including behaviours that are not typically associated with inclusive fitness theory such as aggression or territory defense. The model does not require a kin selection effect to be applicable, but it can help reveal whether relatedness or synergy play a part in the selection for (or conversely, against) a social behaviour. The asymmetry quotient *Q* is not included in this base model form, because *Q* is directly involved in the fitness outcomes of each interaction. If an individual's behaviour – and therefore, its fitness – is influenced by which role it occupies, the average fitness outcome of each type of interaction (A_1 with A_1, A_1 with A_0, etc.) is dependent on the proportion of A_1 carriers disproportionately occupying each role, which is quantified by *Q*.

In the following sections I describe two examples of how my model can be applied to social behaviours, and how *Q* is applied to the fitness outcomes of these behaviours.

3.1 Hawk/Dove/Bourgeois Game

In Maynard Smith's (1982) Hawk-Dove-Bourgeois (HDB) game, an asymmetry is described in which the focal individual (FI) occupies one of two roles: owner or invader of a resource which increases an owner's fitness by amount *V*. Individuals in a population may employ one of three strategies:

> H or "Hawk": Defend resource (if owner) or fight to obtain resource (if invader) until either the individual is injured (in which case the individual would flee,

losing the resource) or the opponent retreats. Injury causes the individual to experience a fitness cost *C*.

D or "Dove": Retreat before injury (if opponent is *H*) or share the resource without escalation to injury (if opponent is *D*).

B or "Bourgeois": If already owner of the resource, act as H; if invader, act as D.

For this game, Maynard Smith assumes an infinite, randomly mixing population consisting mostly of individuals adopting behaviour strategies H or D, in which a rare mutant behaviour B is introduced to the population. The game was initially developed to help explain why same-species conflicts rarely result in life-threatening injury (Bacaër 2011), and therefore was not originally designed with an inclusive fitness context in mind. As a result, relatedness is not taken into consideration, and thus individuals do not interact with relatives more often than by chance (i.e., $R = 0$). Using the parameters described in the HDB game, Maynard Smith describes the requirements for each evolutionary stable strategy (ESS) – a behavioural strategy or phenotype which, when adopted by a population, cannot be supplanted by a competing mutant strategy. In his original HDB game, Maynard Smith finds that the values of V and C determine which strategy is the ESS; when $V > C$, H is the ESS, and when $C > V$, B is the ESS.

In this game, Maynard Smith fails to consider biases in strategies adopted by territory owners or invaders. Consider, for example, the fitness outcomes of interactions between H and B strategists if B individuals were always territory invaders (and therefore always acted like doves)? What if B strategists were always territory owners?

To apply the HDB game to my model and explore these possible scenarios, I must make some modifications to Maynard Smith's original design. Because my model is designed to compare only two competing alleles at a time (A_1 and A_0), I combine the two pure strategies H and D to create a mixed strategy associated with the allele A_0. Similarly to Maynard Smith, I will consider B to be the mutant strategy in my model. Territory owners and invaders will be considered separate roles, where owners are labelled P1, and invaders labelled P2.

If H is considered to be a strategy employed with frequency h by carriers of the wild-type allele A_0, D is considered to be a strategy employed with frequency $(1 - h)$ by A_0 carriers, and B is considered to be a strategy employed by all carriers of the mutant allele A_1, the fitness payoffs for interactions between A_0 and A_1 carriers can easily be applied to the W_i values as described in Section 2.3. Here, however, W_i values for each interaction are partitioned into the fitness outcome to P1 and the fitness outcome to P2, labelled $v1_{zz'}$ and $v2_{zz'}$, respectively, where z and z' represent genotypes of P1 and P2. Figure 3.1 illustrates how fitness outcomes $v1_{zz'}$ and $v2_{zz'}$ are applied to each possible interaction type between A_1 and A_0 carriers in each role. This guide can be used for scenarios other than the current HDB game by substituting the placeholder labels $v1_{zz'}$ and $v2_{zz'}$ with fitness values for any social behaviour one wishes to model.

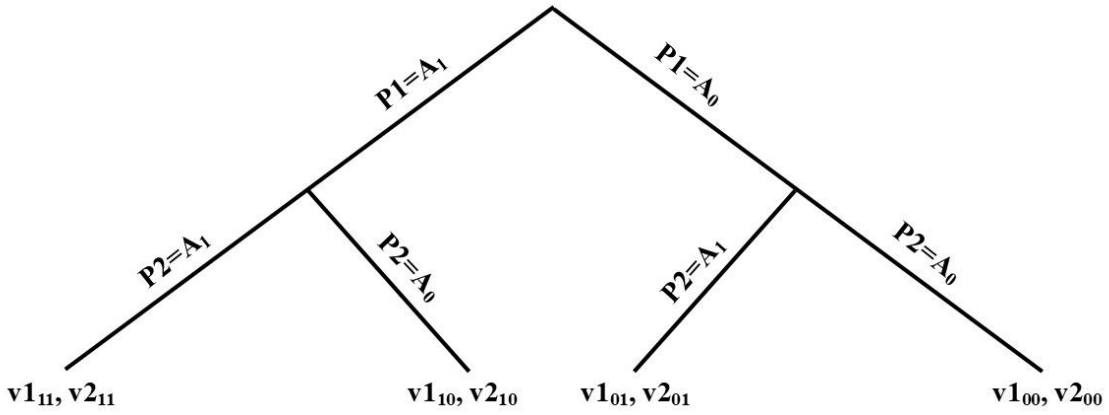

Figure 3.1. A decision tree illustrating the fitness outcomes, labelled v1zz' and v2zz', to individuals in roles P1 and P2, respectively, following pairwise interactions between individuals carrying one of two alleles: the mutant allele A_1 or the wild-type allele A_0. Fitness outcome labels *z* and *z'* indicate the genotypes of individuals in roles P1 and P2, respectively, where 1 indicates A_1 and 0 indicates A_0.

Thus, $v1_{10}$ represents, for example, the fitness outcome (measured as the number of offspring directly produced in the next generation) for P1 in an A_1:A_0 interaction, and $v2_{10}$ is the outcome for P2 in that same interaction. As there are now four distinct interaction types when role is taken into account (A_1:A_1, A_1:A_0, A_0:A_1, and A_0:A_0), interaction frequencies for each type will now be labelled $f_{zz'}$.

The resulting fitness outcomes from Maynard Smith's HDB game to resource owners, P1, and resource invaders, P2, upon enacting either strategy H or *D*, is illustrated in Figure 3.2 below. Strategy B is not labelled here explicitly, as individuals employing this strategy will ultimately act as either H or *D*, depending on which role it occupies.

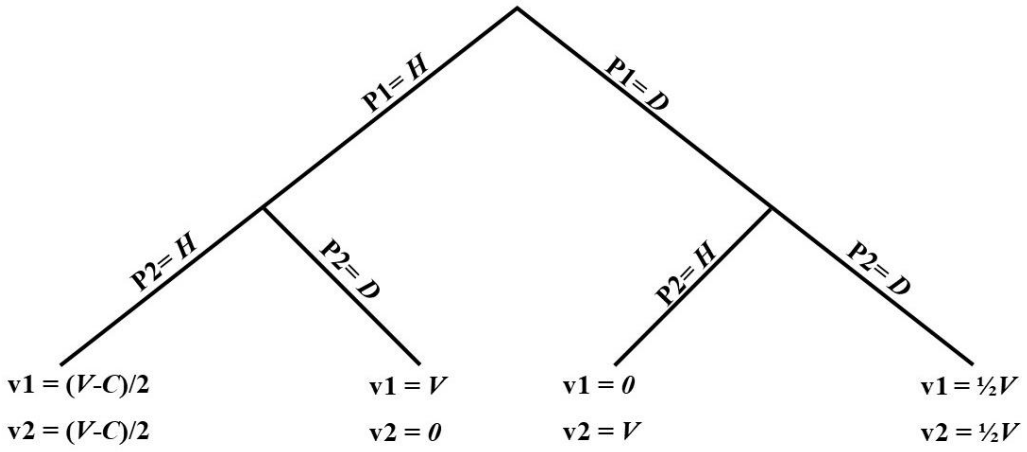

Figure 3.2. A decision tree illustrating the fitness outcomes – *V* indicating an increase in fitness, *C* indicating a fitness decrease – to individuals in roles P1 and P2, respectively, following pairwise interactions between individuals employing one of two strategies: H or *D*. Genotype labels *z* and *z'* for each fitness outcome are not specified here.

To vary the level of bias in the genotypic composition of each role (i.e., to vary the level of gene-environment interaction), I designate a mathematical expression for the asymmetry quotient, *Q*. In this model, the focal (or mutant) allele is A_1, and roles are assigned labels P1 and P2. As described in section 1.5, *Q* is found by taking the number of individuals with the focal allele that are in a focal role in a heterogeneous (A_1:A_0) pairing, divided by the total number of individuals with the focal allele in a heterogeneous pairing. For this example, P2 will be the focal role, and therefore $Q = f_{01}/(f_{01} + f_{10})$.

Table 3.1 below outlines the average fitness outcomes, $v1_{zz'}$ and $v2_{zz'}$ to interactants in roles P1 and P2 respectively, for all possible allele and strategy combinations. Note that *Q* is integrated into these fitness outcomes, because the average fitness outcome to, for

example, A_1 carriers in heterogeneous pairings, is dependent on how frequently A_1 carriers are found in each role.

Table 3.1. Average fitness outcomes – *V* indicating an increase in fitness, *C* indicating a fitness decrease – to individuals carrying either allele A_0 or A_1 and occupying either role P1 or P2 following pairwise interactions of each possible pair type. For example, the average fitness outcome to P2 in an A_0:A_1 pairing, or $v2_{01}$, is (1-*h*)*V*/2. Because A_0 carriers employ H with frequency *h* and D with frequency (1 – *h*), the average fitness outcome to P2 (which, as a B invader, is employing D) is found by taking the weighted sum of the outcome when A_0 enacts H and when it enacts D.

Pair type = *zz'*	Fitness to P1 = v1	Fitness to P2 = v2
11	V	0
10	$(1-Q)\left(\frac{h(V-C)}{2}+(1-h)V\right)$	$(1-Q)\left(\frac{h(V-C)}{2}\right)$
01	$Q\left(hV+\frac{(1-h)V}{2}\right)$	$Q\left(\frac{(1-h)V}{2}\right)$
00	$\frac{V-h^2C}{2}$	$\frac{V-h^2C}{2}$

Substituting fitness outcomes $v1_{zz'}$ and $v2_{zz'}$ from Table 3.1 into the $D_{Obszz'}$ expressions in Equation 10 produces

$$\Delta_s p \propto R\left[\frac{Q(hV+V)}{2}+\frac{(1-Q)\big(h(V-C)\big)}{2}-\frac{(V-h^2C)}{2}\right]+\left[\frac{Q(1-h)V}{2}+(1-Q)\left(V-\frac{h(V+C)}{2}\right)-\frac{(V-h^2C)}{2}\right]$$
$$+S\left[\frac{V}{2}-\left(\frac{Q(1-h)V}{2}+(1-Q)\left(V-\frac{h(V+C)}{2}\right)\right)-\left(\frac{Q(hV+V)}{2}+\frac{(1-Q)\big(h(V-C)\big)}{2}-\frac{(V-h^2C)}{2}\right)\right] \quad \textbf{(11a)}$$

which can be simplified to Equation 11b below:

$$\Delta_s p \propto \frac{R(Q+h-1)(Ch+V)}{2} + \frac{(Q+h-1)(Ch-V)}{2} + S\left(Ch\left((1-Q)-\frac{h}{2}\right)\right)$$

(11b)

Notice here that both equations 11a and 11b can be broken into the three main partitions as seen in Equation 10: additive indirect ($R(D_{Obs01} - D_{Obs00})$) additive direct ($D_{Obs10} - D_{Obs00}$) and nonadditive fitness effects ($S\big((D_{Obs11} - D_{Obs10}) - (D_{Obs01} - D_{Obs00})\big)$. I will describe equation 11a first, because its respective parts are more easily connected back to Equation 10. To describe the principal results of this formula, I will use the simplified form, Equation 11b.

Beginning with the simplest of the three components in (11a), the additive direct effect ($D_{Obs10} - D_{Obs00}$), we can understand the meaning behind the formula by considering a hypothetical scenario similar to that discussed in Section 2.4, in which a wild type (WT) focal individual is making a unilateral "decision" to switch from the WT strategy of H with probability *h* and D with probability (1 – *h*) (or the H/D strategy for short), to the mutant behaviour B. While obviously in nature organisms cannot 'will' their alleles to mutate or change suddenly, adaptation of game theory helps us understand the equation more intuitively. This allows us to compare the fitness outcomes experienced by A_1 carriers resulting from the mutant behaviour with those experienced by A_0 carriers. In following this hypothetical scenario, we can consider *Q* to be the probability of being in the P2 role when an individual is making a *unilateral decision*. Therefore the FI in the additive direct effect portion of Equation 11a is P2 (resource invader) with probability *Q* and P1 (resource owner) with probability (1 – *Q*). Following Table 3.1, the fitness payoff

received by the FI here is[5] $Q(1-h)V/2 + (1-Q)(h(V-C)/2 + (1-h)V)$. Had the FI not changed behavioural strategies, it would have received a fitness payoff of $h^2(V-C))/2 + h(1-h)V + (1-h)^2\,V/2$, which simplifies to $(V-h^2C)/2$. To calculate the change in fitness to the FI caused by the switch in behavioural strategies, or $(D_{Obs10} - D_{Obs00})$, we simply subtract the 'final' fitness effect from the 'initial', as shown in the second component of Equation 11a. The result is then the additive direct fitness effect to B strategists caused by their mutant behaviour.

Moving on to the indirect additive effect $R(D_{Obs01} - D_{Obs00})$, we now imagine a scenario where the FI's *partner* is switching strategies from H/D to B. This time, Q measures the probability that the P2 role is held by the partner as opposed to the FI, because the partner here is making the unilateral decision. The fitness payoff received by the FI due to its partner's change in behaviour therefore is $Q(hV + (1-h)V/2) + (1-Q)h(V-C)/2$. Had the partner not changed behaviours, the FI's fitness payoff would have been the same as in the additive direct scenario above when the FI did not switch to B and was paired with another H/D, or D_{Obs00}. The change in fitness to the FI due to its partner's switch in strategies is again calculated by subtracting the 'final' (D_{Obs01}) minus 'initial' (D_{Obs00}). Here however, the result is multiplied by *R*, thereby shifting our perspective such that the individual switching strategies is now considered the FI, and thus weighing the fitness effects which this FI (who switched from H/D to B strategies) by the relatedness between the FI and its social partner. The resulting product can then be

[5] Some minor simplification was applied to this and the following formulae to reduce the size of Equation 11b.

considered the additive indirect fitness effect to B strategists caused by their mutant behaviour.

Finally proceeding to the nonadditive component, $(S\big((D_{Obs11} - D_{Obs10}) - (D_{Obs01} - D_{Obs00})\big)$, we see the exact same simplified form as that described in Equation 10. With the last three fitness effect terms (D_{Obs10}, D_{Obs01}, and D_{Obs00}) already described above, the foremost fitness effect, D_{Obs11}, is simply the effect experienced by B strategist individuals in homogeneous pairings. Notice that a *Q* factor is not included here – as both individuals in this type of pairing implement the same strategy, B, there can be no bias in roles (i.e. half the B strategists in this type of pair are P1, and the other half are P2). There is also no *h* frequency involved, as there are no H/D strategists in this pairing, and thus the D_{Obs11} term here is simply *V*/2. The entire synergy component, when assembled, measures all nonadditive (synergistic) fitness effects resulting from homogeneous pairings between B strategists.

What does Equation 11b tell us? To answer this question, I first set up a baseline analysis to test whether the results obtained with this model match those of Maynard Smith's (1982) original model. Several assumptions are made in Maynard Smith's basic Hawk-Dove- Bourgeois game that can be used to enter values for variables in my own formula and recover Maynard Smith's original results. Mainly, Maynard Smith assumes an infinite, random mixing population, with a rare introduced mutant strategy (B) and individuals that do not interact with relatives more often than by chance. For my model, these assumptions translate to my *R* and *S* values being set to zero. In doing so, the first and third component of Equation 11b reduce to zero, leaving only the additive direct

effect $(Q + h - 1)(Ch - V)/2$. In his model, Maynard Smith found that when $V > C$, strategy H is the only 'evolutionarily stable strategy,' or ESS, a strategy in which, when adopted by the entire population, cannot be invaded by other mutant strategies. In other words, when $V > C$ in Maynard Smith's HDB model, strategy H cannot be supplanted once fixed in the population. In contrast, when $C > V$, strategy B is the only ESS in Maynard Smith's model. These same results can be found in the expression $(Q + h - 1)(Ch - V)/2$ by setting $h = 1$, meaning that all wild type A_0 carriers enact strategy H. When this is done, I find that $\Delta_s p \propto Q(C - V)/2$. When $V > C$, the change in A_1 allele frequency $\Delta_s p$ becomes negative, meaning that strategy H cannot be outcompeted by B once it (H) is sufficiently high frequency in the population, and the reverse is true when $C > V$ – a rare B can invade a population of individuals playing only H. When h is set to zero, $\Delta_s p$ is always positive, assuming that strategy (i.e. genotype) is independent of role. This assumption was also made by Maynard Smith, and can be adopted here by setting Q equal to one-half, meaning that B strategists are equally likely to be territory owners or invaders.

Maynard Smith considered H and D to be pure strategies in his basic HDB model, meaning that he considered H and D strategists to be separate entities, as opposed to the mixed strategy H/D used in my model. As a result, when considering intermediate values of h (between 0 and 1) I find some interesting results. This is more easily shown if the additive direct component of Equation 11b is rearranged to $(h - (1 - Q))(Ch - V)/2$. When $V > C$, $\Delta_s p$ is only negative (meaning that A_1 allele frequency is decreasing) if wild type individuals enact strategy H more frequently than mutants are territory owners ($h > (1 - Q)$). This makes sense because the role occupied by a B strategist dictates its

behaviour. If mutants are behaving as H more frequently than wild-type (WT) individuals, they have more opportunities to reap the full value of a territory *V* from social partners enacting strategy D. Also, as a higher frequency of enacting strategy H involves a lower frequency of enacting D, when mutants behave as H more often than wild type individuals (in other words, WT individuals behave as D more than mutants), a partner acting as D is more likely to be WT than mutant.

When $C > V$, the sign of $\Delta_s p$ becomes not only dependent on the relationship between h and $(1 - Q)$, but also the ratio of V/C. To understand how each of these variables interact to influence the sign of $\Delta_s p$, I set the expression $(h - (1 - Q))(Ch - V)/2$ equal to zero, and solve for h, which gives the two expressions stated above: $(1 - Q)$ and V/C. These two expressions are linear functions of h which intersect, and between these two functions is where $\Delta_s p$ becomes negative. For visualization purposes, these functions can be plotted in three dimensions using the command *plot3d* in Maple by setting values for V and C. Figures 3.3 and 3.4 illustrate two such examples, with V/C ratios of 1/2 and 3/4, respectively.

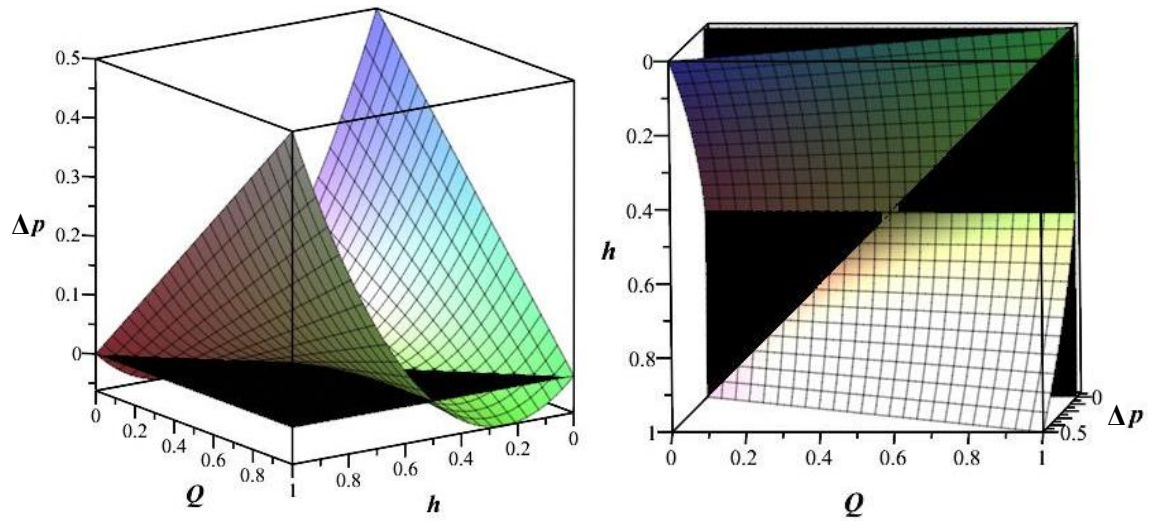

Figure 3.3. Two perspectives of a 3D plot of $\Delta_s p$, Q, and h, (in colour) when $R = 0$, $S = 0$, $V = 1$ and $C = 2$. A secondary plane (black) at $\Delta_s p = 0$ has been added for illustrative purposes.

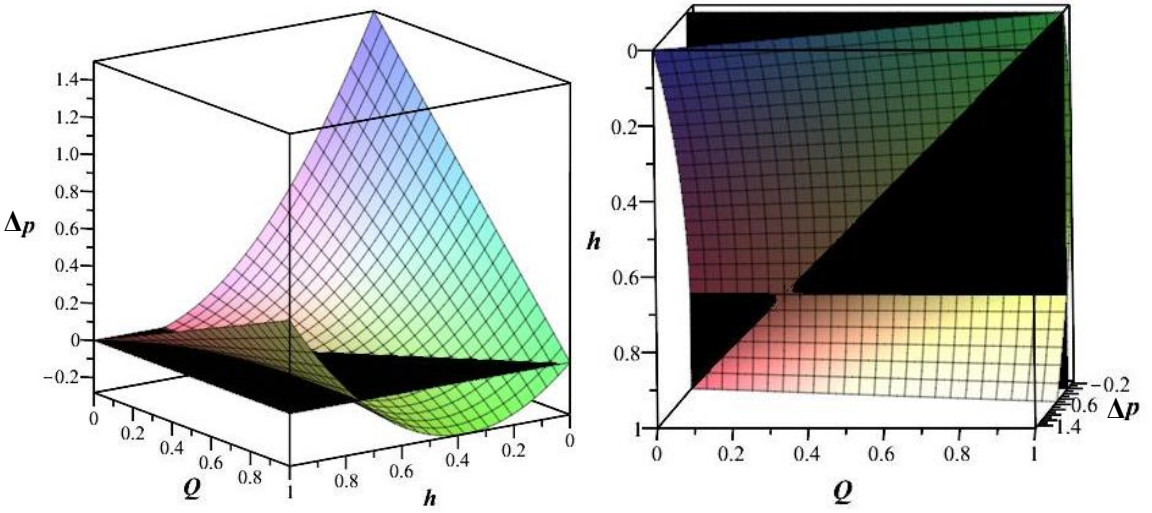

Figure 3.4. Two perspectives of a 3D plot of $\Delta_s p$, Q, and h, (in colour) when $R = 0$, $S = 0$, $V = 3$ and $C = 4$. A secondary plane (black) at $\Delta_s p = 0$ has been added for illustrative purposes.

In the secondary (right-hand side) perspectives of each example plot, the functions $h = (1 - Q)$ and $h = V/C$ are clearly visible as borders through which $\Delta_s p$ crosses as it dips below zero. Based on the expressions found above and the example figures shown, when $C > V$, $\Delta_s p$ is only positive when h is either greater than both V/C *and* $(1 - Q)$ or when h is less than both V/C *and* $(1 - Q)$. If h is only greater or less than one of these functions and not the other, $\Delta_s p$ becomes negative.

One final interesting scenario involving the additive direct component is when $h = 0$ and $Q = 1$, meaning that B strategists are always playing D, as are mixed strategists (individuals choosing H with frequency h, and D with frequency $(1 - h)$). When this occurs, $\Delta_s p = 0$, because selection cannot differentiate between mutants and WTs, as both are behaving as D in all interactions. The same goes for when $h = 1$ and $Q = 0$, in which case both WTs and mutants are behaving as H in all interactions. Based on these and the results described above, it is already clear that role is important for modelling natural selection, without yet even considering these dynamics in connection to relatedness or synergy.

With the baseline of Maynard Smith's HDB game established, I can now relax the assumptions made earlier, starting with the assumption of a rare mutant allele. As I allow the mutant allele A_1 to appear more frequently in the population (increasing allele frequency p), the likelihood of homogeneous A_1 pairs increases, and therefore so does S. When S is greater than zero, the nonadditive component of Equation 11b, $S(Ch(1 - Q - h/2))$, influences $\Delta_s p$ accordingly. To understand if synergy contributes positively or negatively to the change in A_1 allele frequency, we may look again at the relationship

between h and $(1 - Q)$. Whereas in the additive direct component, the requirements for $\Delta_s p$ to be positive or negative depended on whether $(1 - Q)$ exceeded h or vice-versa, the sign of the synergy component seems to more heavily favor a positive $\Delta_s p$. For the component to contribute positively to $\Delta_s p$, $(1 - Q)$ must exceed $h/2$, meaning that wild-type individuals must behave as H twice as often as the frequency at which mutants own territories $(1 - Q)$ in order for A_0 to outcompete A_1. These results are interesting, because they imply that synergy can significantly influence the selection of certain social behaviours, and models which assume only rare alleles with no homogeneous interactions may produce different results if those assumptions are relaxed, particularly if role-based expression is also considered.

Finally moving on to relaxing the assumption of random mixing, meaning that R is no longer zero (reminder that R may be either positive *or* negative). The entire additive indirect component, $R(Q + h - 1)(Ch + V)/2$, contributes to $\Delta_s p$ relative to the size and sign of R. This component may be rearranged to $R(h - (1 - Q))(Ch + V)/2$ for easier interpretation, and in doing so it looks similar to the additive direct component described previously ($(Q + h - 1)(Ch - V)/2$), differing only in the sign of V and the factor of R. With C and V both positive, whether the additive indirect component contributes positively or negatively to $\Delta_s p$ – assuming, for now, that R is positive – becomes entirely dependent on R and the ratio of h and $(1 - Q)$. This time, however, $(1 - Q)$ exceeding h does not contribute positively to $\Delta_s p$, but negatively, and the opposite is true for h exceeding $(1 - Q)$. Even more interesting is when we consider negative values of R, which reverses this dynamic back such that values of $(1 - Q)$ exceeding h cause the additive indirect component to contribute positively to $\Delta_s p$. To understand the

interactions between these variables, I adopt the same methods used for the additive direct component. Using arbitrary values for *C* and *V* (in this case, I used *C* = 2 and *V* = 1), I set the expression $R(h-(1-Q))(Ch+V)/2 = 0$ and solve for *h*, which gives the expressions (1 – *Q*) and $-(R-1)/2(R+1)$. As before, these two expressions are linear functions of *h* which intersect, and between them is where $\Delta_s p$ dips below zero. Figures 3.5 and 3.6 visualize two examples of these functions in 3D plots with values for *Q* set to 1/2 and 1/4, respectively.

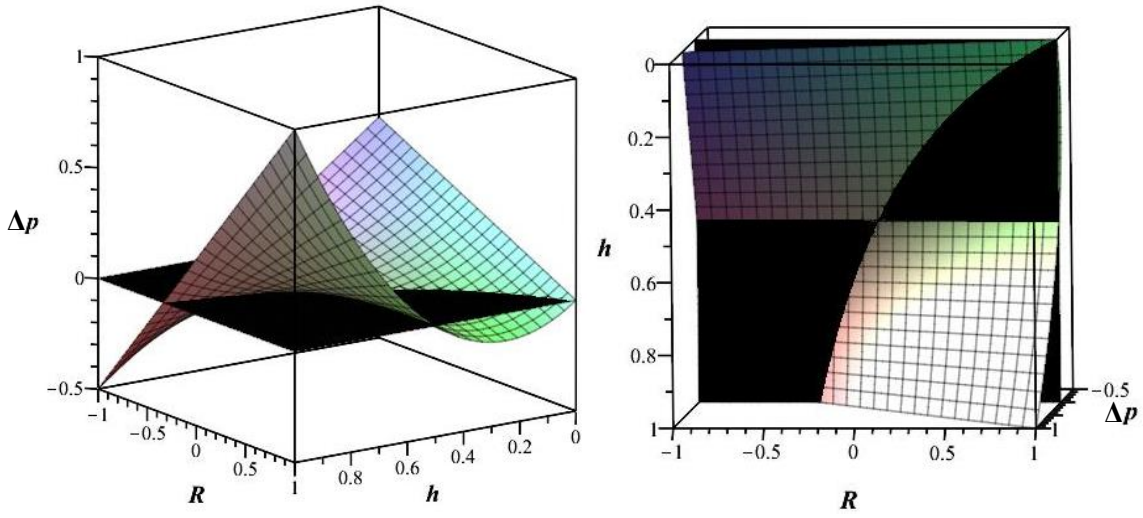

Figure 3.5. Two perspectives of a 3D plot of $\Delta_s p$, *R*, and *h*, (in colour) when *Q* = 1/2, *S* = 0, *V* = 1 and *C* = 2. A secondary plane (black) at $\Delta_s p$ = 0 has been added for illustrative purposes.

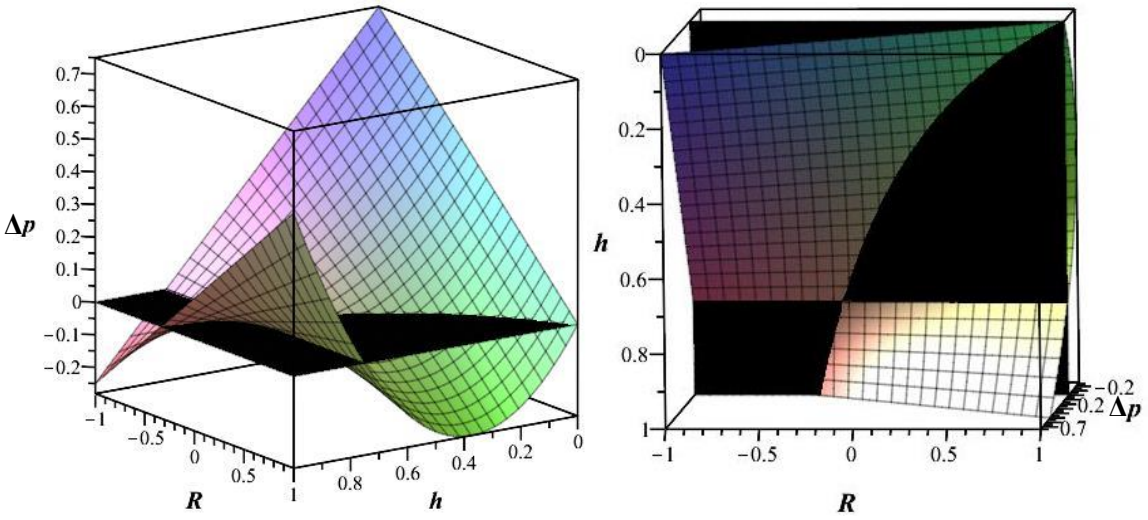

Figure 3.6. Two perspectives of a 3D plot of $\Delta_s p$, *R*, and *h*, (in colour) when *Q* = 1/4, *S* = 0, *V* = 1 and *C* = 2. A secondary plane (black) at $\Delta_s p$ = 0 has been added for illustrative purposes.

The secondary (right-hand side) perspectives of each example plot show the functions $h = (1 - Q)$ and $h = -(R - 1)/2(R + 1)$ as borders through which $\Delta_s p$ crosses as it dips below zero. Based on the expressions found for *h* in the additive indirect component and the example figures shown, when *C* and *V* are positive values, $\Delta_s p$ is only positive when *h* is less than both $(1 - Q)$ and $-(R - 1)/2(R + 1)$ or when *h* is greater than both $(1 - Q)$ and $-(R - 1)/2(R + 1)$. As with the additive direct component, if *h* is only greater (or conversely, less than) one of these two functions, $\Delta_s p$ will be negative.

These results, in conjunction with the results from the synergy component, indicate that there is a potential cost to B strategists disproportionately favoring other B strategists as their partners as opposed to H/D mixed strategists. However, when the frequency of B strategists is high in the population and homogeneous A_1 pairs occur by virtue of this high allele frequency, there is a potential nonadditive benefit to these pairings. In terms of

my model, this means that when *R* is low but *S* is high (which can occur when there is random or disassortative pairing but high A_1 frequency) the requirements for mutants to outcompete WT individuals are less restrictive than for WT individuals to outcompete mutants.

Clearly, relatedness, synergy, and role-based expression each significantly influence the selection of social behaviours, even when the behaviour is non-cooperative like conflict over territory ownership.

3.2 Altruism

Price's equation has classically been used as a means of exploring different ways to model altruistic behaviour (Grafen 2006; Queller 1992; Frank 1995), and due to the complications of inclusive fitness, the utility of my heterogeneous pair composition variable *Q* may be best illustrated with this example.

Previous literature has emphasized the importance of altruistic behaviour being expressed conditionally (e.g., Charlesworth 1978; Parker 1989). Queller (2000) has particularly emphasized this in the context of sterile castes in eusocial insect species, where altruists forgo any personal fitness in order to convey a fitness benefit (in the form of brood care, for example) to a reproducing relative. Recognition systems, which often coevolve with altruistic behaviour (Axelrod et al. 2004), allow individuals to discriminate between kin and non-kin, and can be vital in the evolution of conditionally expressed altruistic behaviours with significant fitness costs.

To explore the importance of kin recognition systems with my model, I consider the same haploid, asexual population of organisms as described in the *Model Foundation* section,

with individuals in the population again carrying one of two alleles, the wild-type allele A_0 or the mutant A_1. As in the previous example, individuals in the population occupy asymmetric roles, P1 and P2, and exhibit pairwise interactions resulting in individual fitness outcomes $v1_{zz'}$ and $v2_{zz'}$. Each interactant in a pair may employ one of two strategies:

> *A* or altruist: invests energy in helping partners, reducing its own reproduction as a result, incurring fitness cost *c* on the FI but conferring fitness benefit *b* to its social partner.
>
> *E* or egoist: reproduces as usual.

In this example, carriers of the wild-type allele A_0 always employ strategy *E*, whereas carriers of the mutant allele A_1 employ one of either strategy depending on their role (P1 or P2) and their social partner's genotype. I will assume, for now, that individuals in this population possess perfect kin recognition systems. In other words, A_1 carriers recognize their social partner's genotype with perfect accuracy. In this model, individuals in the P1 role will be considered to establish a social environment for individuals in the P2 role to react to. In nature, this would be analogous to, for example, P1 locating a habitat first and P2 reacting by either moving on to a new habitat area (egoist) or staying to help P1 care for its offspring, forgoing its own reproduction as a result (altruist). By virtue of the above allele descriptions, A_0 carriers will not vary in their strategy choice, regardless of role. By contrast, individuals carrying A_1 will only employ strategy *A* when in the P2 role and – with the assumption of perfect kin recognition – their social partner is *also* an A_1

carrier. The fitness outcomes of each type of interaction according to the above description are laid out in Table 3.2.

Table 3.2. Average fitness outcomes to individuals carrying either allele A_0 or A_1, where A_1 carriers employ strategy *A* conditionally on their own role and the genotype of their social partner. In this example, individuals exhibit perfect kin recognition.

Pair type = *zz'*	Fitness to P1 = v1	Fitness to P2 = v2
11	*b*	*-c*
10	0	0
01	0	0
00	0	0

Substituting these values into the $D_{Obszz'}$ expressions in Equation 10 produces β_{FI} and β_{Part} coefficients that are identical:

$$\Delta_s p \propto \frac{RS(b-c)}{2R+2} + \frac{S(b-c)}{2R+2} \propto \frac{S(b-c)}{2} \quad \textbf{(12)}$$

where $\beta_{FI} = \beta_{Part} = \frac{S(b-c)}{2R+2}$. This result is expected: when A_1 carriers express strategy *A* exclusively towards other A_1 carriers, and never towards A_0 carriers, fitness effects condense to be solely dependent on the size of *b* and *c*, and the likelihood that interacting pairs share the same allele (measured via *R* and *S*).

To vary the accuracy of recognition between A_1 carriers in this example, I modify the fitness outcomes in Table 3.2 such that with frequency *e*, A_1 reactors (i.e., those in P2 role) will make an error in the recognition of their social partner and behave accordingly. When such an error occurs in an A_1 – A_1 pairing for example, P2 would erroneously

recognize its partner as an A_0 carrier, and employ strategy *E* instead of *A*. Table 3.3 outlines these modified fitness outcomes.

Table 3.3. Average fitness outcomes to individuals carrying either allele A_0 or A_1. With frequency (1 – *e*), A_1 carriers in the P2 role employ strategy *A* conditionally on the genotype of their social partner; with frequency *e*, A_1 carriers make an error in recognizing their partner, and behave according to said error. In this example, A_1 carriers in the P1 role are unable to make an error, as they are not reacting to the genotype of their partner, and instead setting the phenotypic environment for P2 individuals to react to.

Pair type = *zz'*	Fitness to P1 = v1	Fitness to P2 = v2
11	$(1-e)b$	$(1-e)(-c)$
10	0	0
01	eb	$e(-c)$
00	0	0

When these outcomes are substituted into the $D_{Obszz'}$ expressions in Equation 10, we find[6]

$$\Delta_s p \propto RQbe - Q(ce) + S\left[\frac{(1-e)(b-c)}{2} - Qe(b-c)\right]. \quad \textbf{(13)}$$

As with the Hawk/Dove/Bourgeois example, the three components of Equation 13 correspond to the additive indirect, additive direct, and nonadditive fitness effect components described in Section 2.5 for Equation 10. Applying the same hypothetical

[6] Equation 13 simplifies immediately to the formula shown here due to several factors of zero being removed. The fully expanded form of this equation is

$$\Delta_s p \propto R[Qbe + (1-Q)0 - 0] + [Q(-ce) + (1-Q)0 - 0] + S\left[\left(\frac{(1-e)(b-c)}{2} - Q(-ce) + (1-Q)0\right) - (Qbe + (1-Q)0 - 0)\right].$$

scenario as before, we may think of each component based on the idea of interactants 'switching' their alleles. For the additive direct component, $(D_{Obs10} - D_{Obs00})$, a wild type (WT) focal individual is making a unilateral 'decision' to switch from its original allele A_0 to the mutant allele A_1. In doing so, this individual moves from experiencing zero fitness effects due to its own behaviour (a reminder that this does not mean the FI experiences zero fitness, simply that its base fitness has not been influenced), to potentially experiencing an additive fitness effect due to its behaviour, depending on its role and the likelihood of error, *e*. When in the P1 role with probability (1 – *Q*), the mutant is establishing the social environment (arriving in a new territory first, for example) and thus is not in a position to 'react' to an already established social environment and potentially make an error in doing so. In contrast, when the mutant is in the P2 role with probability *Q*, it is in a position to react to the social environment established by its social partner, P1. If the FI recognizes its social partner to be a WT individual and behaves accordingly, it will adopt strategy *E*. If, however, an 'error' occurs, the FI will instead adopt strategy *A*, thereby experiencing a fitness cost – *c*. This 'error' can manifest in a number of ways, including problems with the FI's ability to recognize kin, epigenetic effects involving penetrance of the allele involved, or even cultural effects. The entire additive effect component, when taking the 'final' (D_{Obs10}) minus 'initial' (D_{Obs00}) additive effects due to the FI switching from the WT to mutant allele, produces $-Qce$.

For the indirect additive effect $R(D_{Obs01} - D_{Obs00})$, we may imagine the FI's *partner* switching from A_0 to A_1, thereby switching from both individuals experiencing zero fitness effects to the mutant potentially conferring a fitness benefit *b* to the FI depending

on its role and on the frequency of error *e*. Again, the mutant is only in a position to make an error when in the P2 role, which occurs with probability *Q*. When in the P2 role, the mutant will recognize its social partner's genotype with probability (1 – *e*) and adopt strategy E accordingly. When an error occurs with probability *e*, the mutant will instead adopt strategy A and confer a fitness benefit *b* to the WT individual. When this effect is multiplied by *R*, we reorient the perspective such that the individual switching from WT to mutant is now the FI. The indirect effect to this FI caused by its switch in alleles is measured as the fitness effect that the FI causes to its partner weighted by the extent to which the interactants are related, or *Qbe*.

Moving on to the nonadditive component, $S\big((D_{Obs11} - D_{Obs10}) - (D_{Obs01} - D_{Obs00})\big)$, the last three fitness terms have again already been described above ($D_{Obs10} = -Qce, D_{Obs01} = Qbe, D_{Obs00} = 0$) leaving only D_{Obs11} to describe. This effect encompasses the outcome to a mutant FI in a homogeneous pairing. When the FI is in the P2 role, it may recognize the genotype of its mutant partner and adopt strategy A accordingly with probability (1 – *e*), conferring a fitness benefit *b* to its partner and experiencing a fitness cost *c* due to its behaviour. When an error occurs with probability *e*, the FI in role P2 does not confer this benefit to its partner and does not experience a cost from its behaviour. Because the FI in a homogeneous A_1 pairing has an equal likelihood of being in either role P1 or P2, the effects to each individual in such an interaction are simply added together and divided by two to find $D_{Obs11} = (1 - e)(b - c)/2$.

Using Equation 12 as a baseline, Equation 13 can be analyzed in a manner similar to the analysis of the HDB game in the previous section (3.1). Without errors, the sign of $\Delta_s p$ in the altruism model depends only on three variables: *S*, *b* and *c*. This indicates that the degree to which interactants are more related to each other than what is expected by random chance, or *R*, does not influence the frequency of A_1 when altruism is expressed conditionally on role and the partner's genotype, without any errors. This makes sense because if mutants only confer benefits exclusively towards other mutants, and only when the other mutant (the partner) is behaving as an egoist, then the proportion of mutants in homogeneous pairs is the only factor influencing the size of $\Delta_s p$. Whether the rate of mutant homogeneous pairs is greater than or less than what is expected by chance doesn't matter: assuming $b > c$, so long as *S* is greater than zero, $\Delta_s p$ will be positive. When *S* is zero, obviously $\Delta_s p$ is also zero, as A_1 carriers are never paired with other carriers and thus the fitness effects of this allele are no different than those of A_0.

Relaxing the assumption of no errors, the variables *Q* and *R* reappear in the model. If there is again a simplifying assumption of a rare mutant allele and random mixing, the frequency of homogeneous A_1 pairings approaches zero (and therefore $R = S = 0$) leaving only the additive direct cost $-Qce$. As the mutant allele becomes more frequent, *S* will begin to rise accordingly. *R*, however, may not necessarily rise with an increase in A_1 allele frequency: depending on whether individuals pair randomly, assortatively ('like with like') or disassortatively, *R* may be zero, positive, or negative. For now, I assume random pairing (therefore $R = 0$) in order to focus on the synergistic effect, $S\left[\frac{(1-e)(b-c)}{2} - Qe(b-c)\right]$, with error rates above zero now considered. This component is fairly straightforward, with a form similar to that shown in Equation 12. With errors

incorporated, the expression $S(b-c)/2$ is now partitioned into the fitness effects resulting from an interaction without errors, which occurs with frequency (1 – e), minus those same fitness effects multiplied by the rate of error, e. In essence, this component is measuring the difference between the average fitness effects experienced by an individual in a homogeneous A_1 pairing and the average fitness effects experienced by an individual in a heterogeneous pairing. The importance of kin recognition, conditional expression, and role in my model is clear in this nonadditive component, as whether it contributes positively or negatively to $\Delta_s p$ depends on the rate of non-error, (1 – e), in proportion to the rate of error, e, and the probability of a mutant being in the P2 role, Q. So long as the rate of non-error is more than double the likelihood of an error occurring multiplied by the likelihood that mutants are in the P2 role (because errors are only possible when P2 is a mutant), or $(1-e) > 2eQ$, $\Delta_s p$ will be positive. This relationship between e and Q is illustrated in Figure 3.7: as Q increases, the maximum level of e allowable for $\Delta_s p$ to be positive decreases. At $Q = 0.5$ (meaning mutants are in the P2 role 50% of the time), the rate of errors must be less than 0.5 for A_1 carriers to spread in the population.

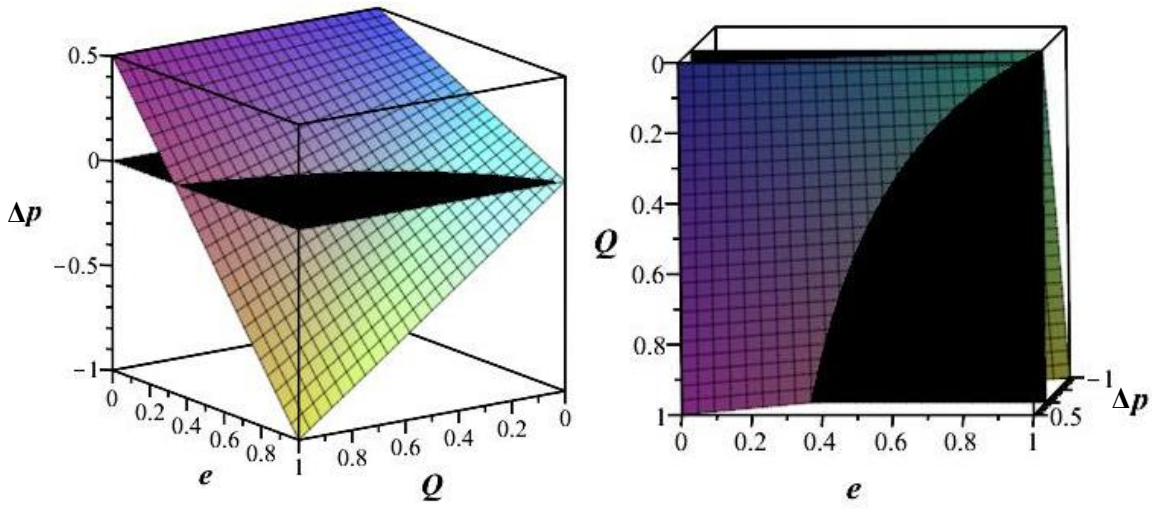

Figure 3.7. Two perspectives of a 3D plot of the nonadditive component of $\Delta_s p$ ($S[(1-e)(b-c)/2 - Qe(b-c)]$) in relation to Q and e (in colour), with S and (b – c) held constant (b = 2, c = 1). A secondary plane (black) at $\Delta_s p$ = 0 has been added for illustrative purposes.

Finally relaxing the assumption of random pairing, the additive indirect component *RQbe* begins to come into play. This component's contribution to $\Delta_s p$ is relatively straightforward: when *R* is positive, the expression *RQbe* contributes positively to $\Delta_s p$ proportionately to *Qe*, because *Q* and *e* must be above or equal to zero, and when *R* is negative the expression contributes negatively to $\Delta_s p$. It seems perplexing, at first, that the additive indirect component includes both *Q* and *e* in its expression; *Q* implies a unilateral 'switch' to mutant, and *e* implies that an error is made in the strategy chosen (*A* or *E*). It is important to remember, however, that indirect additive effects are measured based on heterogeneous pairings, to avoid including any nonadditive effects from a homogeneous interaction.

4 Discussion

My original goal for this thesis was to explore the evolution of conditionally expressed social behaviours using mathematical modelling techniques. More specifically, I aimed to study how selection acts on genes for social behaviours that are conditionally expressed based on an environmental trigger, particularly when carriers of said genes disproportionately experience the trigger more or less often than non-carriers. To answer this question, I developed a mathematical model following the methods of Gardner et al. (2011) and those described therein. Beginning with the covariance form of Price's formula, which describes a population's change in allele frequency over a single generation, I developed my model in an inclusive fitness framework such that individuals occupy one of two roles (the conditional 'trigger') when interacting with a social partner. I then used the mathematics software *Maple* (2020) to streamline the process of rearranging my model into a form that can be more easily interpreted verbally, with particular emphasis on the inclusive fitness variables R and S, as well as my newly described variable, Q. This new variable, Q, was added to describe biases in whether carriers of a focal allele are disproportionately found in one role over another. To illustrate the utility of this new variable, I applied my model to two social behaviour scenarios. The first is a modified form of Maynard Smith's Hawk/Dove/Bourgeois game in which the pure strategies 'Hawk' and 'Dove' are combined to create a mixed wild-type strategy, such that individuals behave as 'Hawk' or 'Dove' depending on a set frequency h, and the 'Bourgeois' strategy is considered mutant. The second model, of my own design, features an altruism scenario, where mutant strategists behave as altruists conditionally depending on what role they occupy *and* the genotype of their social

partner (i.e., whether they are kin or non-kin), with wild-type individuals acting as non-altruists. To explore possibilities in which errors may occur in an individual's ability to recognize kin, or its ability to express the behaviour, among other errors, I introduced an additional variable *e*, representing the frequency of such errors. I interpreted the components of the resulting formulae using *Maple* (2020) to create three-dimensional figures describing some of the results of these two scenarios.

In the following sections, I will provide an overview of the results described for both models and their significance in the greater context of sociobiology and evolutionary genetics. I will also describe the limitations of this study and how it may be developed further to increase our understanding of inclusive fitness and evolutionary theory.

4.1 Results overview

In both the Hawk/Dove/Bourgeois and altruism models, the extent to which mutants occupied one role more than the other dramatically influenced the resulting change in mutant allele frequency. A clear visual representation of this influence can be seen by comparing figures 3.5 and 3.6 from the HDB model, where the value of *Q*, the frequency of mutants that are territory invaders, can even affect whether relatedness between interactants positively or negatively contributes to the survival of the mutant allele in the next generation. In the altruism model, lower values of *Q* (particularly when $Q < 0.5$) allow for greater flexibility in the frequency of errors, *e*, in conditionally expressed altruism. These results imply that models which do not include roles, or asymmetries in stimuli that trigger the conditional expression of social behaviours, may yield significantly different results if these details were included. Indeed, when we consider the HDB model without relatedness or synergy effects, as it was originally described by

Maynard Smith, *Q* still holds influence over the change in allele frequency regardless of whether mutants are more frequently paired with H or D strategists (see figures 3.3 and 3.4).

4.2 The significance of role and the variable *Q*

When discussing inclusive fitness, particularly in the context of altruistic behaviours, there is a generally accepted assumption of – as described by Parker (1989) "conditionality of gene action". In other words, genes associated with a behaviour, such as altruism in eusocial insects, cannot be unconditionally expressed such that all carriers always behave altruistically. If this were the case, members of sterile castes in eusocial colonies would not have a reproducing relative to receive their help and indirectly pass their genes on to the next generation, effectively leading to the colony's extinction. While the assumption of conditional expression is important, I have found very few inclusive fitness papers that explicitly model mechanisms for this conditional action of genes for social behaviour. Parker's (1989) method of doing so, which I have partially adopted for my model, involves creating an asymmetry between two interacting individuals such that one occupies role P1 and the other P2 (labeled 'X' and 'Y' in Parker's model). These roles, as explained in Section 1.4 of this thesis, may represent any asymmetry between the two interacting individuals, such as birth order/age, territory establishment (owner/invader), size, and social status.

One clear example of such an asymmetry in nature, where phenotypic expression is influenced by which 'role' an organism occupies, can be observed in the European honeybee *Apis mellifera*. Females in this eusocial species have two main castes, queens and workers, which are reared from larvae by nurse workers in the hive (males, or drones,

are also reared in this way). The specific diet fed to each female larva, as opposed to a larva's genotype, is the main contributor to caste differentiation (Evans and Wheeler 1999). The molecular mechanisms behind this process are still being studied, but thus far it is clear that both the type of food given to larvae (with queens-to-be fed primarily a substance called 'royal jelly', and workers fed a mixture of royal jelly and nectar) and the amount that each larva is fed play major parts in whether an individual will develop into a queen or worker bee (Slater et al. 2020). If these differential diets are thought of as binary environmental stimuli that influence the phenotypic expression of a specific gene (or group of genes) in European honeybees, it is easy to see how 'role' (in this case, if a larva is fed a queen diet vs a worker diet) might be an important aspect to the evolution of conditional phenotypes.

Examples of asymmetric roles in nature aren't limited to diet alone, of course. Parker (1989) described other mechanisms such as age, relative physical strength, and social dominance/subordination. In his models, Parker also explored potential genetic mechanisms involved: for example, it may be that one locus determines an individual's behaviour when in role X, and another locus determines behaviour when the individual occupies role Y. An aspect of asymmetric roles which Parker fails to consider, and which is yet to be explored in modelling literature, is the potential for bias in the genotypes found in each role. If, for example, there was a rare allele in honeybees which increases the chances of carriers being chosen as new queens by nurse bees, how might that affect the evolution of these eusocial behaviours?

Interestingly enough, evidence has already been found of genetic predispositions to castes in some eusocial species. Volny and Gordon (2002), for example, describe differences in

genotype at a particular microsatellite locus between reproductive and non-reproductive members of the red harvester ant *Pogonomyrmex barbatus*. This is possible because, according to Volny and Gordon, non-reproductives inherit heterozygous genotypes and reproductives inherit homozygous genotypes at the focal locus, which allows both alleles found in workers to be carried by homozygous queens and drones. Genetic predispositions influencing caste determination have been observed in other eusocial insect species as well, such as members of the stingless bee genus *Melipona* (Hartfelder et al. 2006). Despite this, the potential effect of genotype on caste differentiation seems generally to be overlooked in the literature. Schwander et al. (2010) discussed this in further depth, arguing that there is an assumption commonly made that evidence for environmental influences on caste differentiation suggests that the environment is the main or only factor involved. In fact, according to Schwander et al, there is growing evidence that caste differentiation is caused by a combination of both environmental and genetic factors in several eusocial species.

To integrate concepts of genetic inheritance and developmental biology (environmental influence) into a modern evolutionary synthesis is a huge undertaking. Weitekamp et al. (2017) discuss some of the complexities of considering environment-gene interactions in empirical studies, such as a significantly higher number of variables that must be controlled for. Mary Jane West-Eberhard has been a strong advocate for the integration of environmental effects into evolutionary theory. In much of her work (1987, 1989, 2005), she argues that current research puts too little emphasis on the contribution of development and the environment to the evolutionary change of organisms, resulting in a gap in our understanding of the evolution of complex traits such as eusocial behaviours.

The use of mathematical modelling techniques – such as those used in this thesis – may help to bridge this gap, and possibly provide valuable insight in situations where it is impossible to control all variables necessary for informative results.

4.3 Limitations and future development

When I first began the development of my model, I decided to use the Price formula for a number of reasons, including its common usage in the inclusive fitness theory literature and the ease with which the model can be converted into Hamilton's Rule. I was also aware of the limitations of Price's equation, which can be summarized as follows.

Most likely the greatest criticism of Price's formula is its lack of dynamic sufficiency; in other words, the model cannot be iterated over and over to explore the evolution of a trait though several generations. The source of this limitation stems from the use of pair frequencies, such as F_2 (the frequency of $A_1 - A_1$ pairings). Because these frequencies are assumed to be unknown (so that it's possible to explore different pair frequency effects), we do not know the composition of different pairs in the daughter generation, and thus cannot calculate the fitness effects of their interactions. If needed, dynamic sufficiency can be forced into the model by creating a calculation 'rule' for social pair distributions. For example, if a rule is created such that the frequency of each pair type is always proportional to the population allele frequencies (thus $F_2 = p^2$), both the HDB and altruism models can be made to be dynamically sufficient. The results from such a modification, however, provide little novel information, and thus I have not provided them in this thesis.

Another limitation is the exponential increase in complexity when attempting to model multiple alleles and/or more than two interactants with the Price equation. To model more than two alternative alleles for example, or to model diploid organisms, fitness effects must be created for each possible combination of alleles (assuming nonadditive effects – less so if the effects are additive). Since role is emphasized here as well, we would also need to consider how the fitness effects of different allele combinations may vary depending on which carriers are in which roles. To model more than two interactants, new model parameters would need to be designed to consider how many interactants exist in each role per interaction, and how the various allele combinations may influence the fitness effects resulting from these interactions. While these added details can absolutely be explored using the concepts introduced in my thesis, I aimed to make clear the importance of conditional expression with my model, and so avoided adding too many details for simplicity's sake.

4.4 Conclusion

Inclusive fitness models in sociobiology commonly emphasize the importance of relatedness, *R*, and synergy, *S*. In this thesis I propose a third key variable for inclusive fitness models, *Q*, which describes the commonly overlooked potential bias in the genetic composition of individuals exposed to an environmental stimulus – here referred to as 'role'. I describe an inclusive fitness model built from Price's formula which can be converted into Hamilton's rule, and provide results from two example applications of the model – one in which I consider the classic Hawk/Dove/Bourgeois game originally described by Maynard Smith, and another where I consider fitness outcomes of altruistic behaviour prone to errors in kin recognition. Both the Hawk-Dove-Bourgeois and

altruism applications of my model show dramatically different results when *Q* is considered (i.e., when biases in allele frequency for each role are allowed) versus when the models only focus on *R* and *S*. Based on these results, it is possible that inclusive fitness models which do not consider biases in role composition may be missing key details in their results. Bearing in mind the current increase in evidence supporting gene-environment interactions involved in eusocial insect behaviours, the integration of details which take these interactions into account – such as the variable *Q* – in inclusive fitness models may be an important step to furthering our understanding of social evolution.

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Appendix A: Supplementary Equations

$$\beta_{FI} = \frac{2p^2(D_{Obs10} - D_{Obs00}) + p(F_2(D_{Obs11} - D_{Obs10} - D_{Obs01} + D_{Obs00}) - D_{Obs10} + D_{Obs00}) - F_2(D_{Obs11} - D_{Obs01})}{2p^2 - p - F_2}$$

(8a)

$$\beta_{Part} = \frac{2p^2(D_{Obs01} - D_{Obs00}) + p(F_2(D_{Obs11} - D_{Obs10} - D_{Obs01} + D_{Obs00}) - D_{Obs01} + D_{Obs00}) - F_2(D_{Obs11} - D_{Obs10})}{2p^2 - p - F_2}$$

(8b)

Curriculum Vitae

Name:	Vonica Flear
Post-secondary Education and Degrees:	Cape Breton University Sydney, Nova Scotia, Canada 2012-2017 B.Sc. The University of Western Ontario London, Ontario, Canada 2018 – present M.Sc.
Related Work Experience	Teaching Assistant The University of Western Ontario 2018 – 2020 Paleontological Lab Technician and Illustrator Cape Breton University Oct – Dec 2017 (work term) Fisheries Technician LFA27 Management Board May – Sept 2017 (seasonal) Paleontological Lab Technician and Illustrator Cape Breton University May – Jul 2017 (work term) Curatorial Assistant Cape Breton University 2016 – 2017 Fisheries Technician Fishermen Scientists Research Society (FSRS) May – Aug 2016 (seasonal) Paleontological Lab Technician and Illustrator Cape Breton University Apr – Jul 2015 (work term)

Publications:

Modesto, Sean P., **Flear, Vonica J.**, Dilney, Melissa M., Reisz, Robert R. 2016. ”A large moradisaurine tooth plate from the Lower Permian of Texas and its biostratigraphic implications”, Journal of Vertebrate Paleontology (36).